



QUALITY IN SPORT

eISSN 2450-3118 · Open Access · Peer-reviewed

apcz.umk.pl/QS Nicolaus Copernicus University in Toruń



WOJTANOWICZ, Julia, SKALNIAK, Kinga, and BOCHNAK, Mikołaj. Iron Deficiency in Athletes – Current State of Knowledge. *Quality in Sport*. 2026;77;76037. <https://doi.org/10.12775/QS.2026.77.76037>

ARTICLE TIMELINE

Received: 12.09.2026. Revised: 13.09.2026 Accepted: 14.09.2026. Published: 28.09.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych). © The Authors 2026.

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Iron Deficiency in Athletes — Current State of Knowledge

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Abstract

Introduction and Purpose. Iron deficiency is common among athletes and may develop before anemia becomes apparent. Its clinical significance is particularly difficult to determine when iron stores are depleted but hemoglobin remains within the normal range. This review summarizes current evidence on iron deficiency in athletes, with particular attention to iron deficiency without anemia (IDNA), its relationship with athletic performance, and practical aspects of diagnosis and management.

Materials and Methods. A structured literature search of PubMed/MEDLINE was performed and supplemented by Google Scholar and reference screening. Publications from January 2015 to August 2026 were considered, with older studies included when relevant to fundamental concepts of iron metabolism, diagnosis, or treatment.

Results. Iron deficiency in athletes reflects the interaction of increased requirements, inadequate intake, impaired availability, and iron losses, with female and younger athletes among the groups at increased risk. Ferritin remains the principal marker of iron stores, but its interpretation is limited by inflammation and the lack of a universal athlete-specific threshold. The functional consequences of IDNA are heterogeneous. More pronounced depletion appears more likely to be associated with impaired endurance performance, whereas improvements in ferritin after supplementation do not consistently translate into improvements in maximal aerobic capacity or other performance outcomes.

Conclusions. Iron status in athletes should be interpreted in the context of laboratory findings, symptoms, risk factors, and training demands. Management should focus on correction of documented deficiency and identification of its underlying cause rather than supplementation aimed solely at increasing iron stores or enhancing performance.

Keywords: iron deficiency, athletes, iron deficiency without anemia, ferritin, hepcidin, athletic performance, iron supplementation

1. Introduction

Iron is essential for oxygen transport, cellular energy metabolism, and skeletal muscle function, making adequate iron availability particularly relevant to physically active individuals. Although the adverse effects of iron-deficiency anemia on exercise capacity are well established, depletion of iron stores may develop before hemoglobin falls below the reference range. This earlier stage can be more difficult to recognize, and its functional significance in athletes remains less clearly defined [1–3].

The development of iron deficiency in athletes is typically multifactorial. Increased requirements may coexist with insufficient dietary intake and exercise-associated iron losses. Exercise can also modify iron availability through changes in hepcidin, the principal regulator of systemic iron homeostasis. These mechanisms may become particularly relevant when intensive training occurs in athletes with marginal iron stores or insufficient nutritional intake [2,3].

Iron deficiency without anemia (IDNA) is of particular interest in sports medicine because reduced iron stores may be present despite normal hemoglobin concentrations. While iron-deficiency anemia clearly compromises exercise capacity, the impact of IDNA on athletic performance is less consistent. Some athletes appear to benefit from correction of pronounced iron depletion, whereas biochemical improvement after supplementation does not invariably translate into measurable performance gains. This uncertainty complicates decisions regarding screening and treatment [1,3–5].

Important questions therefore remain regarding how iron deficiency should be defined in athletes, which individuals are most likely to experience functional consequences, and when intervention is warranted. This review summarizes current evidence on the epidemiology, mechanisms, performance consequences, diagnosis, prevention, and management of iron deficiency in athletes, with particular emphasis on IDNA and its relevance to athletic performance [1–3].

2. Materials and Methods

A structured literature search was conducted in PubMed/MEDLINE and supplemented by Google Scholar and reference lists of relevant publications. The search covered articles published from January 2015 to August 2026, with the final search performed in August 2026.

Earlier publications were included when they provided fundamental evidence relevant to iron metabolism, diagnostic concepts, or sports medicine practice.

Search terms combined concepts related to iron deficiency, exercise, athletic performance, and iron supplementation, with the search focused on peer-reviewed English-language publications.

Systematic reviews, meta-analyses, consensus statements, randomized and non-randomized intervention studies, and relevant observational studies addressing iron status in physically active populations were considered. Studies were selected when they provided evidence on epidemiology, risk factors, exercise-related iron metabolism, diagnosis, performance consequences, prevention, or treatment. Publications focusing exclusively on conditions substantially affecting iron metabolism independently of physical activity were generally excluded.

As this study was designed as a narrative review, no formal meta-analysis was performed. The available evidence was synthesized with particular attention to differences in definitions of iron deficiency, study populations, and clinically relevant outcomes.

3. Epidemiology and Risk Factors

Iron deficiency is frequently reported in athletic populations, although prevalence varies considerably according to the population studied and the diagnostic criteria applied. Sex, age, training characteristics, sport discipline, and nutritional status all contribute to this variability. Interpretation is further complicated by the use of different serum ferritin thresholds, which substantially affects the proportion of athletes classified as iron deficient [1–3].

A recent systematic review and meta-analysis including 17,519 collegiate athletes from 23 countries and 62 sports illustrates this problem. Using serum ferritin <20 µg/L, approximately 23% of athletes met criteria for absolute iron deficiency, whereas 53.9% were classified as having hypoferritinemia when a threshold of <50 µg/L was applied. Although these categories are not clinically equivalent, the findings demonstrate both the frequency of reduced iron stores in athletes and the importance of the definition used to identify them [6].

Female athletes are consistently at greater risk than males, with menstrual blood loss representing an important additional source of iron loss. Heavy menstrual bleeding may further increase susceptibility, particularly when combined with high training demands or inadequate

dietary intake. Nevertheless, iron deficiency also occurs in male athletes and should not be overlooked when compatible symptoms or other risk factors are present [2,7,8].

Adolescence and early adulthood may represent additional periods of vulnerability because the nutritional demands of growth can coincide with increasing training loads. This may be particularly relevant in adolescent female athletes, in whom growth, training demands, and menstrual iron losses occur simultaneously [1,2].

Although endurance athletes have traditionally received the greatest attention, low iron stores are not confined to endurance disciplines. Athletes participating in team, aesthetic, and weight-sensitive sports may also be affected. Training volume and intensity should therefore be considered alongside sport discipline and individual risk factors rather than assuming that iron deficiency is primarily a problem of endurance exercise [2,6].

Nutritional factors are also important. Insufficient energy intake, restrictive eating practices, intentional weight reduction, and limited intake of bioavailable iron may increase the risk of depletion. Vegetarian and vegan athletes may require particular attention to dietary planning, although plant-based diets should not themselves be considered evidence of deficiency. Low energy availability may further increase risk by limiting overall nutrient intake and is particularly relevant in athletes exposed to high training volumes or weight-related pressures [2,9].

Iron deficiency in athletes is therefore best understood as the result of interacting rather than isolated risk factors. Female sex, younger age, substantial menstrual blood loss, high training demands, previous iron deficiency, and inadequate or restrictive dietary intake can identify athletes in whom closer assessment of iron status may be warranted [1,2,7,9].

4. Iron Metabolism and the Role of Hepcidin

Systemic iron homeostasis depends primarily on the regulation of intestinal absorption and recycling because the body has no active mechanism for controlled iron excretion. Dietary iron is absorbed mainly in the proximal small intestine, while a substantial proportion of circulating iron is continuously recovered from senescent erythrocytes. Once released into plasma through ferroportin, iron binds to transferrin and becomes available for erythropoiesis and other iron-dependent processes [10].

Hepcidin, a peptide hormone produced mainly by the liver, is the principal regulator of systemic iron availability. It binds to ferroportin and promotes its internalization and degradation, reducing both intestinal iron absorption and the release of recycled iron from macrophages. Hepcidin production increases in response to iron loading and inflammation, whereas iron depletion, hypoxia, and increased erythropoietic demand tend to suppress its activity [10].

The hepcidin response to exercise is particularly relevant in athletes. Physical exercise can induce a transient inflammatory response, including an increase in interleukin-6 (IL-6), which contributes to hepatic hepcidin production. Post-exercise increases in hepcidin have been reported following both endurance and resistance exercise, although the magnitude of the response varies with baseline iron status, exercise characteristics, inflammatory response, and time of day [2,11,12].

Hepcidin concentrations typically rise during the hours following exercise, with peak values commonly observed approximately 3–6 hours after a training session. During this period, intestinal iron absorption and release of recycled iron may be reduced. Repeated exposure to such periods of reduced availability could contribute to an unfavorable iron balance when combined with insufficient dietary intake, increased requirements, or already depleted stores. Exercise-induced hepcidin elevation should therefore be considered a contributing mechanism rather than an independent cause of iron deficiency [2,11,12].

Hepcidin also follows a diurnal rhythm, with generally lower concentrations earlier in the day. Together with the post-exercise response, this has prompted interest in whether the timing of iron intake can influence absorption in athletes. Some experimental evidence suggests greater iron availability when intake occurs earlier in the day or before the post-exercise hepcidin peak, but current evidence does not support rigid timing recommendations. In practice, adequate total iron intake and correction of established deficiency remain more important than precise nutrient timing [2,13].

5. Mechanisms of Iron Deficiency in Athletes

Iron deficiency in athletes usually results from the interaction of several mechanisms rather than a single isolated cause. Increased physiological requirements may coexist with insufficient dietary intake, menstrual blood loss, exercise-associated losses, and transient reductions in iron availability. Their relative contribution varies between athletes according to sex, age, training load, nutritional status, environmental exposure, and baseline iron stores. Exercise should

therefore be regarded as a potential contributor to negative iron balance rather than a universal cause of deficiency [1,2].

5.1. Increased Iron Requirements

Regular training may increase iron requirements through adaptations involving erythropoiesis, skeletal muscle metabolism, and mitochondrial function. This becomes particularly relevant during rapid growth, substantial increases in training volume, or exposure to hypoxic conditions such as altitude training. Expansion of hemoglobin mass requires adequate iron availability, and athletes entering periods of increased physiological demand with marginal stores may be particularly susceptible to progressive depletion. The effect of training therefore depends partly on baseline iron status and the ability of dietary intake to compensate for increased requirements [1,2].

5.2. Exercise-Induced Hemolysis

Exercise-induced hemolysis is one of the characteristic mechanisms proposed to contribute to altered iron balance in athletes. Repetitive mechanical stress, particularly during running, can damage circulating erythrocytes and increase their turnover. The term *foot-strike hemolysis* describes erythrocyte destruction associated with repeated impact of the feet against the ground, although mechanical trauma is unlikely to be the only mechanism involved. Oxidative stress, increased body temperature, osmotic changes, and compression of erythrocytes within the microcirculation may also contribute during strenuous exercise [14,15].

Biochemical changes consistent with hemolysis have been observed following prolonged running, including reductions in haptoglobin and changes in markers of erythrocyte turnover. However, clinically meaningful reductions in hemoglobin are not consistently observed. Exercise-induced hemolysis is therefore more appropriately considered a contributor to increased erythrocyte turnover and iron requirements than an independent cause of iron-deficiency anemia [15].

5.3. Exercise-Associated Blood and Iron Losses

Prolonged or high-intensity exercise may be accompanied by small gastrointestinal and urinary blood losses. Gastrointestinal bleeding has been associated with transient intestinal hypoperfusion, mechanical stress, and exercise-related changes in mucosal integrity. Exercise-

associated hematuria, particularly after prolonged running, may result from mechanical trauma and transient renal or bladder-related changes. These events are usually self-limited, but repeated episodes may contribute to negative iron balance when other risk factors are present [2].

Iron is also lost in sweat. The amount appears to vary considerably between individuals and is difficult to quantify accurately, making its contribution to chronic iron depletion uncertain. Sweat-related losses are unlikely to be a dominant mechanism in most athletes but may become relevant during prolonged training, high sweat rates, or exercise in hot environments [2].

Persistent gastrointestinal bleeding, recurrent hematuria, or iron deficiency that does not respond as expected to nutritional intervention should not automatically be attributed to exercise. Such findings warrant assessment for non-sport-related causes of blood or iron loss [2,10].

5.4. Menstrual Blood Loss

Menstrual blood loss is an important contributor to iron depletion in female athletes. The magnitude of iron loss varies considerably between individuals, with heavy menstrual bleeding substantially increasing the risk of depleted stores. Its effect may be amplified when menstrual losses coexist with intensive training, inadequate energy intake, or insufficient dietary iron [7,8].

Menstrual history should therefore form part of the assessment of female athletes with low or progressively declining iron stores. Hormonal contraceptive use may modify bleeding patterns and consequently iron loss, although the effect depends on the contraceptive method and individual response. Assessment should therefore focus primarily on the actual pattern and magnitude of menstrual bleeding [7].

5.5. Dietary Intake and Iron Bioavailability

Insufficient dietary intake is one of the most modifiable contributors to iron deficiency in athletes. Low total energy intake, intentional weight reduction, restrictive eating practices, and limited consumption of iron-rich foods may prevent dietary supply from matching physiological requirements [2,9].

Iron bioavailability also influences the amount available for absorption. Heme iron is generally more readily absorbed than non-heme iron, while absorption of non-heme iron is affected by meal composition. This is particularly relevant for athletes consuming predominantly plant-based diets, although vegetarian or vegan dietary patterns can provide adequate iron when appropriately planned [2].

Dietary intake should be considered in relation to physiological demand rather than in isolation. An intake sufficient under normal conditions may become inadequate during periods of rapid growth, intensive training, altitude exposure, or increased blood loss [1,2].

5.6. Exercise, Hepcidin, and Iron Availability

As discussed above, exercise-induced increases in hepcidin can transiently reduce intestinal iron absorption and the release of recycled iron. Repeated periods of reduced availability may contribute to negative iron balance when combined with insufficient intake, increased requirements, or ongoing losses. Hepcidin should therefore be considered one component of the multifactorial development of iron deficiency rather than an independent explanation for its occurrence in athletes [2,11,12].

Overall, the mechanisms underlying iron deficiency in athletes are closely interconnected. Increased requirements, inadequate intake, reduced availability, and iron losses may each have a relatively modest effect in isolation but become clinically relevant when several occur simultaneously. Identifying the predominant contributors in an individual athlete is therefore important not only for correcting depleted stores but also for reducing the risk of recurrence [1,2].

6. Iron Deficiency Without Anemia

Iron deficiency does not necessarily coexist with anemia. In athletes, reduced iron stores may be present while hemoglobin remains within the reference range, a condition commonly referred to as iron deficiency without anemia (IDNA). This distinction is clinically relevant because normal hemoglobin may create a false impression of adequate iron status, particularly when assessment is limited to a complete blood count [1–3].

The potential significance of IDNA reflects the functions of iron beyond hemoglobin synthesis. Iron participates in mitochondrial energy production, oxidative metabolism, myoglobin function, and multiple enzymatic processes. Depletion may therefore affect physiological

function before erythropoiesis is sufficiently impaired to cause anemia. Fatigue, reduced training tolerance, and impaired performance have been reported in athletes with IDNA, although these symptoms are nonspecific and may also reflect inadequate recovery, low energy availability, illness, sleep disturbance, or excessive training load [2,3].

A major difficulty is the lack of a consistent definition of IDNA in sports medicine. Serum ferritin is commonly used to identify depleted iron stores, but thresholds vary substantially between studies. Ferritin <30 µg/L is frequently used in otherwise healthy adult athletes, while concentrations <15–20 µg/L indicate more pronounced depletion. Higher thresholds have been proposed in selected circumstances, particularly before altitude training. These differences complicate comparison between studies and argue against interpreting a single ferritin value as a universal indicator of functional impairment [1,2].

The functional significance of IDNA remains uncertain. A low ferritin concentration does not necessarily indicate impaired athletic performance, and improvement in iron stores after supplementation does not consistently translate into functional benefit. Available evidence suggests that athletes with more pronounced iron depletion may be more likely to experience functional impairment and respond to treatment, although differences in diagnostic thresholds, study populations, and performance outcomes limit firm conclusions [4,5,16].

IDNA should therefore be regarded as a clinically relevant but heterogeneous state rather than a laboratory diagnosis with a uniform effect on performance. Normal hemoglobin does not exclude meaningful iron depletion, but neither nonspecific symptoms nor a borderline ferritin concentration establishes iron deficiency as the cause of impaired performance. Assessment should integrate iron status with symptoms, risk factors, and training context, while treatment should aim to restore adequate physiological iron availability rather than increase iron stores solely in pursuit of an ergogenic effect [1–5].

7. Impact on Health and Athletic Performance

The impact of iron deficiency on athletic performance depends largely on its severity. Iron-deficiency anemia reduces oxygen-carrying capacity and consequently impairs exercise capacity, whereas the functional consequences of depleted iron stores without anemia are less predictable. Some athletes with IDNA demonstrate measurable impairment, while others remain largely unaffected [1,3,4,16].

7.1. Aerobic Capacity and Endurance Performance

Endurance performance has been studied most extensively. In iron-deficiency anemia, reduced oxygen delivery provides a direct mechanism for impaired aerobic capacity and exercise tolerance. In IDNA, oxygen-carrying capacity is preserved, and potential effects are more likely to involve reduced iron availability for mitochondrial and other iron-dependent metabolic processes [2,3,16].

A recent systematic review of 23 studies involving 669 high-level female athletes across 16 sports reported an approximately 3–4% reduction in endurance performance associated with iron deficiency. Improvements were observed in several studies following correction of deficiency, although responses varied considerably. These findings are relevant in competitive sport, where relatively small performance differences may be meaningful, but should be interpreted in light of small study populations and heterogeneous definitions of iron deficiency and performance outcomes [4].

Evidence regarding maximal aerobic capacity is similarly inconsistent. Earlier studies reported improvements in $\text{VO}_{2\text{max}}$ or $\text{VO}_{2\text{peak}}$ following iron treatment in some non-anemic iron-deficient athletes, particularly those with more pronounced depletion. In contrast, a 2024 meta-analysis of randomized trials demonstrated a clear increase in ferritin following oral iron supplementation but only a small, statistically non-significant pooled effect on $\text{VO}_{2\text{max}}$. The biochemical response was greatest among athletes with the lowest baseline iron stores, suggesting that baseline deficiency may influence the response to treatment [5,16].

Maximal oxygen uptake does not capture all aspects of endurance performance. Exercise economy, tolerance of submaximal workloads, and the ability to sustain a given intensity may change without a substantial alteration in $\text{VO}_{2\text{max}}$. Reliance on a single laboratory endpoint may therefore underestimate functionally relevant changes in some athletes [4].

7.2. Fatigue, Exercise Tolerance and Training Adaptation

Fatigue and reduced exercise tolerance are frequently associated with iron deficiency but are nonspecific in athletes. High training loads, inadequate recovery, sleep disturbance, low energy availability, illness, and psychological stress may produce similar symptoms. Iron status should therefore be considered as one component of the assessment rather than assumed to explain otherwise unexplained fatigue [2,3].

More pronounced iron depletion may nevertheless compromise an athlete's ability to tolerate or complete planned training. Although this could influence longer-term adaptation, direct evidence linking isolated IDNA with impaired recovery or training adaptation remains limited. Iron supplementation should therefore not be regarded as a general recovery strategy in athletes without documented deficiency [2,4,5].

7.3. Strength, Power and Other Performance Outcomes

Evidence regarding strength, power, and anaerobic performance is substantially more limited than for endurance outcomes. Available studies show inconsistent effects of iron deficiency and supplementation, while endurance athletes remain disproportionately represented in the literature. Current evidence is therefore insufficient to establish a consistent relationship between low iron stores and strength or power performance [4].

Overall, the relationship between iron status and athletic performance appears to be graded rather than binary. Iron-deficiency anemia has a well-established adverse effect on exercise capacity, whereas the consequences of IDNA vary according to the degree of depletion and individual context. Athletes with markedly depleted stores appear more likely to experience functional impairment, but correction of ferritin should not itself be interpreted as evidence of an ergogenic effect [4,5,16]. The relationship between progressive iron depletion and its variable functional consequences is summarized in Figure 1.

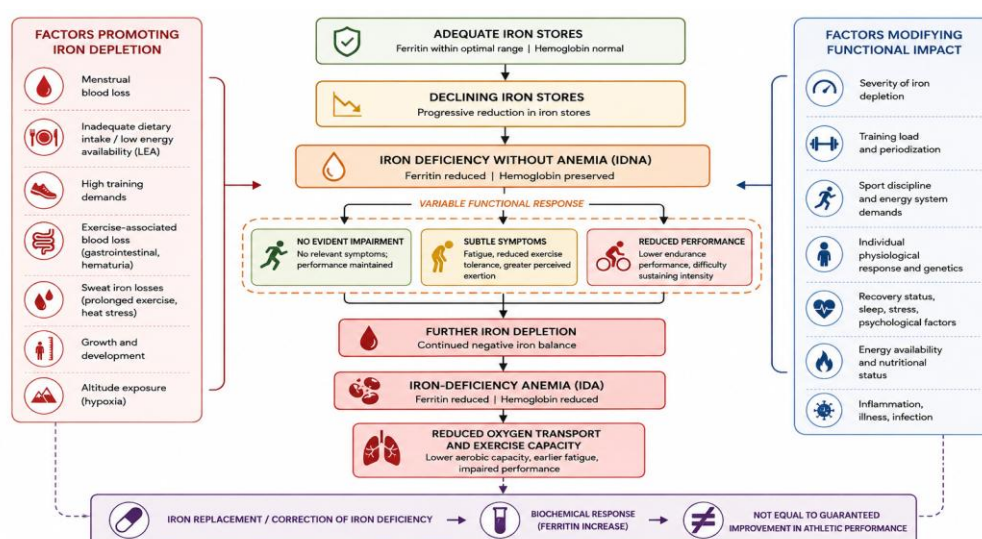


Figure 1. From low iron stores to impaired athletic performance: a proposed conceptual framework.

The functional consequences of iron depletion are not uniform across its progression. During iron deficiency without anemia (IDNA), hemoglobin remains preserved, while the effect on athletic performance may vary according to the severity of depletion and individual physiological, nutritional, and training-related factors. Progression to iron-deficiency anemia (IDA) results in reduced oxygen-carrying capacity and a more predictable impairment of exercise performance. Correction of iron stores produces a biochemical response but does not necessarily result in measurable performance improvement. *Source: authors' own elaboration based on the literature reviewed.*

8. Diagnosis and Screening

Assessment of iron status in athletes should not rely on a single laboratory parameter. Hemoglobin identifies anemia but may remain normal despite depleted iron stores, while ferritin can detect earlier depletion but is influenced by inflammation and the threshold used for interpretation. Laboratory findings should therefore be considered together with symptoms, dietary and menstrual history, training characteristics, and potential sources of iron loss [1,2,10].

8.1. Laboratory Assessment of Iron Status

A complete blood count and serum ferritin provide a practical basis for initial assessment. Hemoglobin distinguishes non-anemic iron deficiency from iron-deficiency anemia, while mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) may decrease as deficiency progresses. Normal erythrocyte indices, however, do not exclude depleted iron stores [1,10].

Ferritin remains the most widely used marker of storage iron. Transferrin saturation (TSAT) provides complementary information about circulating iron availability, while C-reactive protein (CRP) can assist interpretation when inflammation is suspected. Serum iron alone has limited diagnostic value because of substantial biological and diurnal variation and should not be used in isolation to diagnose iron deficiency [1,10,17].

Additional markers, including soluble transferrin receptor (sTfR) and reticulocyte hemoglobin content, may be useful when conventional results are inconclusive. Their routine use is limited by availability and variable reference ranges, and they are generally unnecessary when the clinical and laboratory picture is otherwise clear [1,10].

8.2. Interpreting Ferritin in Athletes

No universally accepted ferritin threshold defines iron deficiency across all athletic populations. Concentrations below approximately 30 µg/L are frequently used to indicate reduced stores in otherwise healthy adult athletes, while values below 15–20 µg/L generally reflect more pronounced depletion. These thresholds should be regarded as practical reference points rather than absolute biological boundaries [1,2].

The proportion of athletes classified as iron deficient varies substantially according to the ferritin threshold applied, emphasizing that different cut-offs should not be considered clinically equivalent. In particular, higher thresholds used to describe low or suboptimal iron stores should not automatically be interpreted as evidence of clinically significant deficiency [6].

Higher iron stores may be relevant before altitude exposure because increased erythropoietic demand requires adequate iron availability. This represents a specific physiological situation and should not be used to justify higher ferritin targets for all athletes [1,2].

Ferritin is also an acute-phase protein and may increase during inflammation, potentially masking depleted stores. Recent illness, inflammatory status, and training conditions should therefore be considered when interpreting results, with CRP providing additional context when appropriate [10,17].

8.3. Who Should Be Screened?

Evidence is insufficient to support a single screening schedule for all athletes. Assessment is most relevant in individuals with recognized risk factors, including previous iron deficiency, heavy menstrual bleeding, restrictive dietary practices, low energy availability, high training demands, or unexplained fatigue and deterioration in exercise tolerance. Female and younger athletes are frequently represented among higher-risk groups, but sex or sport discipline alone should not determine whether testing is performed [1,2,7,9].

Testing may also be appropriate before periods of increased iron demand, particularly altitude training, and follow-up is reasonable after treatment or when the underlying cause of deficiency persists. The interval between assessments should be individualized according to previous results, symptoms, and changes in training or nutritional status [1,2].

8.4. Timing and Longitudinal Interpretation

Exercise, hydration, recent illness, and inflammatory responses can influence hematological and iron-related measurements. When iron status is monitored over time, blood samples should therefore be obtained under reasonably comparable conditions, particularly when relatively small changes in ferritin are being interpreted [1,2].

Longitudinal trends may be more informative than an isolated borderline result. A progressive decline in ferritin, particularly in an athlete with relevant risk factors or symptoms, may warrant further assessment before anemia develops. Conversely, minor fluctuations around a selected threshold should not automatically prompt supplementation [1,2].

Overall, hemoglobin and ferritin form the practical core of assessment, with TSAT, CRP, and selected additional biomarkers providing context when required. The objective is to identify clinically meaningful iron depletion and its underlying cause rather than to treat an isolated laboratory value [1,2,10].

Table 1. Laboratory markers used in the assessment of iron status in athletes

Marker	Clinical information	Interpretation in athletes	Main limitations
Hemoglobin (Hb)	Presence and severity of anemia	Reduced in IDA; may remain normal in IDNA	Changes relatively late during iron depletion; influenced by plasma volume
Serum ferritin	Body iron stores	Values <30 µg/L are commonly used to indicate reduced stores in adult athletes; lower concentrations indicate more pronounced depletion	Acute-phase reactant; interpretation may be affected by inflammation and recent strenuous exercise
Transferrin saturation (TSAT)	Circulating iron availability	Complements ferritin and Hb when assessing iron availability	Biological variation; limited value when interpreted alone

Serum iron	Circulating iron at the time of sampling	Limited role as an isolated diagnostic marker	Marked biological and diurnal variation; influenced by recent dietary intake
C-reactive protein (CRP)	Inflammatory status	Helps identify inflammation that may complicate ferritin interpretation	Does not directly assess iron stores
Soluble transferrin receptor (sTfR)	Cellular iron demand	May support assessment when ferritin is difficult to interpret	Limited availability and assay-dependent reference ranges
Reticulocyte hemoglobin content	Iron availability for recent erythropoiesis	May provide additional information on iron-restricted erythropoiesis	Availability, terminology, and reference ranges vary between analyzers

Abbreviations: Hb – hemoglobin; IDA – iron-deficiency anemia; IDNA – iron deficiency without anemia; TSAT – transferrin saturation; CRP – C-reactive protein; sTfR – soluble transferrin receptor.

9. Prevention and Treatment

Management of iron deficiency in athletes should address both depleted iron stores and the factors responsible for their development. Supplementation may provide only temporary correction if heavy menstrual bleeding, inadequate energy or iron intake, gastrointestinal losses, or increasing training demands remain unrecognized. Treatment should therefore be guided by the severity of deficiency, symptoms, likely cause, and response to previous interventions [1,2,18].

9.1. Nutritional Strategies and Prevention

Dietary assessment is an appropriate starting point, particularly in athletes with mild or recurrent depletion. Evaluation should consider total iron intake, energy availability, dietary

restrictions, and iron bioavailability. Heme iron is generally absorbed more efficiently than non-heme iron, while ascorbic acid can enhance non-heme iron absorption. Vegetarian and vegan diets can provide adequate iron but may require more deliberate planning when physiological requirements are high [2,18].

Nutritional intervention should also address the broader cause of deficiency. Increasing dietary iron alone may be insufficient when low energy availability, heavy menstrual bleeding, or another source of ongoing loss is the predominant factor [7,9,18].

The timing of iron intake has been investigated because hepcidin increases following exercise and varies throughout the day. Earlier intake or administration before the post-exercise hepcidin peak may favor absorption, but current evidence does not support rigid timing recommendations. Adequate total intake and correction of established deficiency remain more important than precise nutrient timing [11,13,18].

9.2. Oral Iron Supplementation

Oral iron is generally the preferred approach when dietary measures are insufficient or iron depletion is more pronounced. Ferrous preparations are commonly used, although studies in athletes vary considerably in dose, duration, and administration schedule. Available evidence consistently demonstrates improvement in iron stores, particularly among athletes with lower baseline ferritin concentrations [5,18].

Interest in lower-dose, once-daily, and alternate-day regimens has increased because oral iron itself can transiently increase hepcidin and reduce fractional absorption from subsequent doses. No single regimen has been established as optimal for all athletes, and treatment should balance efficacy with tolerability and adherence [18,19,20].

Gastrointestinal adverse effects, including nausea, abdominal discomfort, constipation, and diarrhea, may limit adherence. Adjustment of dose, formulation, or dosing frequency can therefore be appropriate. Treatment duration should be guided by clinical and laboratory response rather than continued indefinitely to achieve progressively higher ferritin concentrations [18,21].

9.3. When Is Intravenous Iron Appropriate?

Intravenous iron should be reserved for selected situations, including intolerance or inadequate response to oral treatment, impaired gastrointestinal absorption, severe deficiency, or circumstances in which rapid correction is clinically necessary. Rapid restoration of iron stores should not be interpreted as evidence of an additional performance benefit, particularly in non-anemic athletes with only mild depletion [18,22].

Although modern intravenous preparations are generally effective, potential adverse effects include infusion reactions, hypersensitivity, extravasation, and, with some formulations, hypophosphataemia. The indication for parenteral treatment should therefore remain clinical rather than performance driven [23].

Competitive athletes must also consider anti-doping regulations. Under current World Anti-Doping Agency (WADA) rules, intravenous infusions and/or injections exceeding a total of 100 mL within a 12-hour period are prohibited except when legitimately received during hospital treatment, surgical procedures, or clinical diagnostic investigations. This restriction applies to the intravenous method and volume even when the administered substance itself is not prohibited [24].

9.4. Monitoring and Prevention of Recurrence

Response to treatment should be assessed clinically and biochemically. Hemoglobin and ferritin provide the practical basis for follow-up, with additional markers used when necessary. The timing of reassessment should reflect the initial severity of deficiency, treatment route, symptoms, and likelihood of continued losses [1,2,18].

Recurrent depletion should prompt reassessment of the underlying cause rather than repeated empirical supplementation. Persistent menstrual blood loss, inadequate dietary intake, low energy availability, gastrointestinal disease, poor adherence, or other sources of blood loss should be considered when iron stores decline again after treatment [1,2,18].

The objective of management is restoration and maintenance of adequate iron status rather than maximization of ferritin concentrations. Supplementation is appropriate when deficiency is documented, but increasing iron stores in iron-replete athletes has no established performance benefit [4,5,18].

Table 2. Management considerations in athletes with impaired iron status

Clinical context	Management considerations	Important points
Athlete at increased risk without confirmed deficiency	Assess dietary intake and modifiable risk factors; consider laboratory evaluation when clinically indicated	Routine iron supplementation solely for performance enhancement is not supported
Iron deficiency without anemia (IDNA)	Identify potential causes, optimize dietary iron intake, and consider oral supplementation according to the degree of depletion and clinical context	Functional consequences vary between athletes; biochemical correction does not guarantee performance improvement
Iron-deficiency anemia (IDA)	Investigate the underlying cause and initiate iron replacement; oral therapy is generally appropriate when tolerated	Recovery of Hb may precede complete restoration of iron stores
Poor tolerance of oral iron	Consider modification of dose, formulation, or dosing frequency	Less frequent or alternate-day strategies may improve tolerability in selected individuals; no single regimen is optimal for all athletes
Inadequate response to oral treatment	Reassess adherence, ongoing iron losses, diagnosis, and gastrointestinal absorption	Persistent deficiency should not lead automatically to repeated empirical supplementation
Recurrent iron deficiency	Re-evaluate menstrual, nutritional, gastrointestinal, and other potential sources of iron loss	Recurrence may indicate that the underlying cause has not been adequately addressed

Preparation for altitude training	Assess iron status before exposure and correct meaningful depletion when present	Increased erythropoietic demand makes adequate iron availability particularly important
Selected indication for intravenous iron	Consider when oral therapy is ineffective or poorly tolerated, absorption is impaired, or rapid correction is clinically required	Rapid restoration of iron stores does not necessarily improve performance; treatment should remain medically indicated

Discussion

The available evidence suggests that iron status in athletes is better understood as a continuum than as a binary distinction between deficiency and normality. This is particularly relevant in IDNA, where depleted iron stores coexist with preserved hemoglobin and the functional consequences vary considerably between individuals. Normal hemoglobin does not exclude clinically relevant iron depletion, but neither a low ferritin concentration nor nonspecific symptoms such as fatigue establish that iron deficiency is responsible for impaired performance [1–4,16].

A central difficulty is the interpretation of ferritin. The prevalence of iron deficiency changes substantially according to the threshold applied, and no single cut-off reliably predicts functional impairment across all athletes. Ferritin should therefore be interpreted in relation to hemoglobin, other indicators of iron availability, inflammatory status, symptoms, previous results, and the athlete's physiological and training context. This is particularly important for borderline values, where treatment decisions based solely on a numerical threshold may lead to unnecessary supplementation [1,2,6,17].

The distinction between biochemical and functional response is equally important. Iron supplementation generally improves depleted iron stores, but changes in performance are less consistent, particularly in non-anemic athletes. Available evidence suggests that athletes with more pronounced depletion are more likely to experience functional impairment and potentially benefit from correction, whereas increasing iron stores in athletes with adequate or only marginally reduced stores has no established ergogenic effect. The absence of improvement in

VO₂max should also be interpreted cautiously, as maximal aerobic capacity does not capture all aspects of endurance performance or training tolerance [4,5,16,18].

These uncertainties are compounded by important limitations in the literature. Definitions of iron deficiency, supplementation protocols, and performance outcomes vary substantially between studies, and many intervention trials include small samples. Female endurance athletes are disproportionately represented, while evidence in male athletes and participants in strength, power, and team sports remains limited. This restricts the generalizability of proposed thresholds and treatment strategies across sporting populations [4,5,16].

Future research should prioritize standardized definitions of iron deficiency and clinically meaningful outcomes that extend beyond changes in ferritin alone. Greater attention to baseline iron status, sex, sport discipline, training demands, and longitudinal changes may help identify which athletes are most likely to experience functional consequences from iron depletion and benefit from intervention. Until such evidence is available, management should focus on documented deficiency and its underlying cause rather than supplementation aimed solely at increasing iron stores or enhancing performance [4,5,18].

Conclusions

Iron deficiency is common among athletes and may develop before anemia becomes apparent. Normal hemoglobin therefore does not exclude depleted iron stores, particularly in athletes with relevant risk factors or unexplained changes in exercise tolerance.

The functional significance of iron deficiency without anemia remains less predictable. Athletes with more pronounced iron depletion appear more likely to experience performance impairment, while improvement in ferritin after supplementation does not necessarily translate into an ergogenic effect. Ferritin should therefore be interpreted alongside other laboratory findings and the individual clinical and training context rather than as an isolated treatment target.

Management should focus on correction of documented deficiency and identification of its underlying cause. Iron supplementation has no established performance benefit in iron-replete athletes, and treatment should aim to restore adequate physiological iron availability rather than maximize iron stores.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the author used ChatGPT (OpenAI) to assist with language refinement, improvement of clarity and readability, and manuscript structuring and formatting. After using this tool, the author critically reviewed and edited the content as needed and takes full responsibility for the final content of the manuscript.

References

1. Clénin G, Cordes M, Huber A, Schumacher YO, Noack P, Scales J, Kriemler S. Iron deficiency in sports – definition, influence on performance and therapy. *Swiss Med Wkly.* 2015;145:w14196. doi:10.4414/smw.2015.14196.
2. Sim M, Garvican-Lewis LA, Cox GR, Govus A, McKay AKA, Stellingwerff T, Peeling P. Iron considerations for the athlete: a narrative review. *Eur J Appl Physiol.* 2019;119(7):1463–1478. doi:10.1007/s00421-019-04157-y.
3. Solberg A, Reikvam H. Iron status and physical performance in athletes. *Life (Basel).* 2023;13(10):2007. doi:10.3390/life13102007.
4. Pengelly M, Pumpa K, Pyne DB, Etxebarria N. Iron deficiency, supplementation, and sports performance in female athletes: a systematic review. *J Sport Health Sci.* 2025;14:101009. doi:10.1016/j.jshs.2024.101009.
5. Šmid AN, Golja P, Hadžić V, Abazović E, Drole K, Paravlic AH. Effects of oral iron supplementation on blood iron status in athletes: a systematic review, meta-analysis and meta-regression of randomized controlled trials. *Sports Med.* 2024;54(5):1231–1247. doi:10.1007/s40279-024-01992-8.
6. Thompson C, Block D, Wang Z, Foster N, Hassett LC, Steien D, Galardy P, Al-Huniti A. The global prevalence of iron deficiency in collegiate athletes: a systematic review and meta-analysis. *Pediatr Blood Cancer.* 2025;72(2):e31415. doi:10.1002/pbc.31415.
7. Bruinvels G, Burden R, Brown N, Richards T, Pedlar C. The prevalence and impact of heavy menstrual bleeding (menorrhagia) in elite and non-elite athletes. *PLoS One.* 2016;11(2):e0149881. doi:10.1371/journal.pone.0149881.
8. Petkus DL, Murray-Kolb LE, De Souza MJ. The unexplored crossroads of the female athlete triad and iron deficiency: a narrative review. *Sports Med.* 2017;47(9):1721–1737. doi:10.1007/s40279-017-0706-2.

9. Mountjoy M, Ackerman KE, Bailey DM, et al. 2023 International Olympic Committee's (IOC) consensus statement on Relative Energy Deficiency in Sport (REDs). *Br J Sports Med.* 2023;57(17):1073–1097. doi:10.1136/bjsports-2023-106994.
10. Pasricha SR, Tye-Din J, Muckenthaler MU, Swinkels DW. Iron deficiency. *Lancet.* 2021;397(10270):233–248. doi:10.1016/S0140-6736(20)32594-0.
11. Fensham NC, Govus AD, Peeling P, Burke LM, McKay AKA. Factors influencing the hepcidin response to exercise: an individual participant data meta-analysis. *Sports Med.* 2023;53(10):1931–1949. doi:10.1007/s40279-023-01874-5.
12. Larsuphrom P, Latunde-Dada GO. Association of serum hepcidin levels with aerobic and resistance exercise: a systematic review. *Nutrients.* 2021;13(2):393. doi:10.3390/nu13020393.
13. McCormick R, Moretti D, McKay AKA, et al. The impact of morning versus afternoon exercise on iron absorption in athletes. *Med Sci Sports Exerc.* 2019;51(10):2147–2155. doi:10.1249/MSS.0000000000002026.
14. Telford RD, Sly GJ, Hahn AG, Cunningham RB, Bryant C, Smith JA. Footstrike is the major cause of hemolysis during running. *J Appl Physiol (1985).* 2003;94(1):38–42. doi:10.1152/japplphysiol.00631.2001.
15. Gartner A, Dombrowski N, Lowe N, Behzadpour V, Zackula R. Foot-strike hemolysis: a scoping review of long-distance runners. *Kans J Med.* 2024;17(5):119–124. doi:10.17161/kjm.vol17.22146.
16. Burden RJ, Morton K, Richards T, Whyte GP, Pedlar CR. Is iron treatment beneficial in iron-deficient but non-anaemic (IDNA) endurance athletes? A systematic review and meta-analysis. *Br J Sports Med.* 2015;49(21):1389–1397. doi:10.1136/bjsports-2014-093624.
17. World Health Organization. *WHO guideline on use of ferritin concentrations to assess iron status in individuals and populations.* Geneva: World Health Organization; 2020. ISBN 978-92-4-000012-4.
18. McCormick R, Sim M, Dawson B, Peeling P. Refining treatment strategies for iron deficient athletes. *Sports Med.* 2020;50(12):2111–2123. doi:10.1007/s40279-020-01360-2.
19. Moretti D, Goede JS, Zeder C, et al. Oral iron supplements increase hepcidin and decrease iron absorption from daily or twice-daily doses in iron-depleted young women. *Blood.* 2015;126(17):1981–1989. doi:10.1182/blood-2015-05-642223.

20. Stoffel NU, Cercamondi CI, Brittenham G, et al. Iron absorption from oral iron supplements given on consecutive versus alternate days and as single morning doses versus twice-daily split dosing in iron-depleted women: two open-label, randomised controlled trials. *Lancet Haematol.* 2017;4(11):e524–e533. doi:10.1016/S2352-3026(17)30182-5.
21. Tolkien Z, Stecher L, Mander AP, Pereira DIA, Powell JJ. Ferrous sulfate supplementation causes significant gastrointestinal side-effects in adults: a systematic review and meta-analysis. *PLoS One.* 2015;10(2):e0117383. doi:10.1371/journal.pone.0117383.
22. Garvican LA, Saunders PU, Cardoso T, et al. Intravenous iron supplementation in distance runners with low or suboptimal ferritin. *Med Sci Sports Exerc.* 2014;46(2):376–385. doi:10.1249/MSS.0b013e3182a53594.
23. Schaefer B, Tobiasch M, Viveiros A, et al. Hypophosphataemia after treatment of iron deficiency with intravenous ferric carboxymaltose or iron isomaltoside—a systematic review and meta-analysis. *Br J Clin Pharmacol.* 2021;87(5):2256–2273. doi:10.1111/bcp.14643.
24. World Anti-Doping Agency. *TUE Physician Guidelines: Intravenous Infusions and/or Injections.* Version 8.1. Montreal: World Anti-Doping Agency; 2025.