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The Effect of Intense Physical Exercise on Iron Deficiency

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ABSTRACT

Intense physical exercise, especially when performed frequently or for prolonged durations, can disturb iron balance through a combination of increased demand, increased loss, and impaired absorption. The strongest evidence comes from endurance sport, military training, and periods of intensified loading, where iron deficiency without anemia and overt iron-deficiency anemia are both common clinical problems. Mechanisms include exercise-induced inflammation and hepcidin upregulation, intravascular hemolysis, gastrointestinal microischemia with occult blood loss, hematuria, sweat losses, menstrual blood loss, low energy availability, and the amplified iron demand of altitude-related erythropoiesis. Importantly, the hematologic response to training also includes plasma volume expansion, which can mimic anemia without true iron deficiency and complicate interpretation. In athletes, clinically meaningful iron deficiency may present before hemoglobin falls, with fatigue, impaired recovery, lower maximal oxygen uptake, diminished training quality, and reduced work efficiency. Diagnosis therefore requires more than hemoglobin alone and should integrate ferritin, transferrin saturation, inflammatory context, and where indicated soluble transferrin receptor or reticulocyte indices. Management begins with confirmation of true deficiency and identification of the mechanism, followed by dietary optimization, oral iron when indicated, and selective use of parenteral iron in carefully chosen cases. This review summarizes the physiology linking exercise to iron homeostasis, identifies athlete groups at highest risk, distinguishes pseudoanemia from true deficiency, and provides a practical framework for diagnosis, prevention, and treatment.[1-8]

Keywords: iron deficiency, athletes, endurance exercise, hepcidin, sports anemia, ferritin, hemolysis, altitude

Key clinical points

- Intense exercise most often unmasks iron deficiency in endurance athletes, menstruating athletes, adolescents, and athletes exposed to altitude or low energy availability.
- A low hemoglobin concentration after a training block is not synonymous with iron deficiency; plasma volume expansion can create dilutional pseudoanemia.
- Ferritin should be interpreted in context. In athletes, ferritin <30 μg/L is commonly considered clinically relevant, whereas higher targets may be reasonable before altitude exposure.
- The post-exercise hepcidin surge usually peaks about 3 hours after endurance exercise, creating a window of reduced iron absorption that matters for timing oral iron.

Introduction

Iron is indispensable to oxygen transport, oxidative phosphorylation, erythropoiesis, myoglobin function, and multiple mitochondrial and enzymatic processes that become rate-limiting during heavy training. In athletes and other physically active people, the clinical relevance of iron deficiency extends beyond classical microcytic anemia: symptoms and performance impairment may appear when iron stores are depleted but hemoglobin is still within the laboratory reference range. This distinction is central in sports medicine because repeated intense exercise commonly produces an iron-deficient state before overt anemia develops.[1-4]

The phrase “the effect of intense physical exercise on iron deficiency” can be misleading if it implies a single uniform mechanism. Exercise does not inevitably cause iron deficiency, and the risk varies substantially by sex, age, sport, training load, nutritional intake, menstrual blood loss, environmental stress, and baseline iron stores. Nevertheless, the cumulative literature strongly supports that repeated high-volume or high-intensity exercise shifts iron balance in an unfavorable direction through a convergence of increased losses, impaired absorption, and heightened utilization. The effect is especially pronounced in endurance disciplines, military training, adolescents, and female athletes.[3-7]

This review focuses on the medical and physiological pathways by which intense exercise promotes iron deficiency, the athlete populations in whom the effect is most clinically relevant, the consequences for health and performance, and a practical evidence-informed approach to diagnosis and treatment.[3,6,9]

Iron physiology most relevant to exercise

Total body iron is tightly regulated because there is no dedicated excretory pathway for excess iron. Systemic balance is therefore determined mainly by intestinal absorption, macrophage iron recycling, and hepatic storage, all coordinated by the hepcidin–ferroportin axis. Hepcidin, synthesized in the liver, binds ferroportin and induces its internalization, reducing dietary iron absorption and limiting release of stored or recycled iron into plasma. Inflammation, especially interleukin-6 signaling, increases hepcidin; erythropoietic drive and iron deficiency suppress it.[1,2,5]

For clinicians dealing with athletes, this physiology has two major implications. First, the same athlete can move rapidly between states of adequate iron availability and functional restriction depending on recent exercise, inflammation, and energy status. Second, laboratory interpretation is vulnerable to timing effects. Ferritin is both a storage marker and an acute phase reactant, while hemoglobin concentration is diluted by training-induced plasma volume expansion. As a result, isolated measurements can misclassify athletes unless they are interpreted longitudinally and in context.[3,6,34,35]

How intense exercise promotes iron deficiency

1. Increased iron demand

Intensified training increases iron demand for erythropoiesis, myoglobin synthesis, mitochondrial biogenesis, tissue repair, and, in some settings, adaptation to altitude. Athletes entering a heavy training block with borderline stores may therefore become deficient even if absolute iron losses are modest. This is particularly relevant when training camps, high-frequency interval work, or altitude exposure stimulate expansion of red cell mass. In such settings, a ferritin concentration that is merely “low-normal” for the general population may be inadequate for optimal training adaptation.[3,6,13,14]

2. Exercise-induced hepcidin and impaired absorption

Over the last two decades, the hepcidin response has emerged as one of the most important mechanistic links between strenuous exercise and iron deficiency. Acute endurance exercise increases interleukin-6 and is followed by a rise in hepcidin that usually peaks around 3 hours after exercise and may persist for several hours. During this window, ferroportin-mediated transfer of iron from enterocytes and macrophages is reduced, limiting the absorption of oral iron and transiently decreasing circulating iron availability for erythropoiesis.[5,7,8,10]

The magnitude of the hepcidin response is not uniform. It is influenced by baseline ferritin, exercise duration, inflammatory signaling, sex, circadian factors, and perhaps cardiorespiratory fitness. Athletes with very low iron stores tend to mount a blunted hepcidin response, reflecting homeostatic suppression by deficiency itself. By contrast, athletes with adequate or high ferritin may experience a more pronounced post-exercise rise. This helps explain why the same training session can produce different iron-handling consequences in different athletes.[7-10]

From a practical standpoint, timing matters. Experimental work suggests that morning exercise and the scheduling of iron intake outside the peak hepcidin window may improve absorption relative to later-day exercise or poorly timed supplementation. The implications are clinically meaningful for athletes who train once or twice daily and repeatedly expose themselves to periods of impaired gut iron uptake.[8,10]

3. Hemolysis and mechanical erythrocyte destruction

Mechanical destruction of erythrocytes is a well-described consequence of repetitive impact loading. Long-distance running is the classic example, where "foot-strike hemolysis" produces haptoglobin depletion and mild intravascular hemolysis. Although a single bout may have only modest hematologic consequences, chronic repetition over weeks to months can increase iron turnover and contribute to depletion, especially when combined with other sources of loss or poor intake.[15-18]

It is important, however, not to overstate hemolysis in isolation. Modern studies indicate that hemolysis after endurance events is often measurable but not always clinically dominant. Its relevance rises when the athlete is simultaneously menstruating, dieting, recovering from illness, training in heat, or preparing for altitude, because iron recycling and replacement may then fail to keep pace with demand.[6,15-18,35]

4. Gastrointestinal ischemia, symptoms, and occult blood loss

Splanchnic hypoperfusion during prolonged strenuous exercise can produce gastrointestinal symptoms, mucosal injury, occult bleeding, and, in severe cases, visible blood loss. Endoscopic studies in symptomatic long-distance runners demonstrate erosive and hemorrhagic lesions that provide a biologically plausible source of chronic iron loss. The effect appears more relevant in prolonged endurance events, hot conditions, dehydration, NSAID exposure, and athletes with frequent gastrointestinal distress during training or racing.[18,20,37]

The clinical message is that unexplained iron deficiency in an endurance athlete should not automatically be attributed to "normal training." Recurrent gastrointestinal symptoms, use of

nonsteroidal anti-inflammatory drugs, celiac disease, inflammatory bowel disease, and other gastrointestinal pathology still require active consideration because they may coexist with exercise-related mechanisms.[18,20,37]

5. Sweat, urine, and hematuria

Sweat and urinary losses are often described as secondary contributors rather than the dominant cause of deficiency, but they become relevant when training volume is high and several small mechanisms accumulate. Dermal iron losses were documented decades ago in heavily training endurance athletes, and exercise-associated hematuria remains a recognized phenomenon after prolonged or high-impact activity. In isolation these pathways may be modest; in aggregate, they can matter.[19,20,37]

Clinically, the contribution of sweat and urine is most plausible in athletes training for long durations in hot environments, particularly when intake is marginal or when menstrual or gastrointestinal losses coexist. This cumulative-burden model is more consistent with the modern evidence than the older search for a single dominant mechanism.[6,19,20,35,37]

6. Low energy availability, restrictive diets, and menstrual loss

Nutritional inadequacy amplifies the effects of exercise-related iron loss. Low energy availability is associated with poorer diet quality, lower absolute iron intake, and altered hepcidin physiology. Contemporary sports nutrition models that restrict carbohydrate availability may further intensify post-exercise disturbances in iron regulation, although the exact size of the effect varies across studies. Vegetarian or vegan diets can support athlete health, but only when meal planning preserves total iron intake and enhances non-heme iron bioavailability.[6,31,34-36]

Among menstruating athletes, heavy menstrual bleeding remains one of the most important non-exercise contributors to iron deficiency and often interacts synergistically with hard training. Modern reviews emphasize that athletic females should not be treated as a uniform risk group; menstrual history, hormonal contraception, symptoms suggestive of low energy availability, and sport-specific training loads all influence the likelihood of depletion. Heavy menstrual bleeding may be the decisive factor in one athlete, while in another the primary driver is endurance training with suboptimal fueling.[21-24,34,35]

7. Altitude as an amplifier of deficiency risk

Altitude exposure deserves special attention because it both increases erythropoietic demand and intersects with the exercise-driven regulation of hepcidin. Athletes with inadequate iron stores demonstrate a blunted erythropoietic response and smaller gains in red cell volume during altitude training. Conversely, when iron is provided appropriately before and during altitude camps, hematologic adaptation is improved. This is why sports medicine recommendations often aim for ferritin concentrations that are higher before altitude than would usually be targeted in sea-level training.[3,11-14]

Table 1. Major pathways linking intense exercise to iron deficiency

Pathway	Mechanism	Typical settings	Clinical weight
Hepcidin surge	Exercise-induced IL-6 and inflammatory signaling reduce ferroportin activity and temporarily limit dietary iron absorption and iron release from stores.	Prolonged endurance work, repeated daily sessions, poor fueling	High
Increased demand	Expansion of red cell mass, myoglobin turnover, mitochondrial adaptation, and tissue repair increase iron requirement.	Training camps, growth, return-to-play, altitude	High
Hemolysis	Mechanical red-cell trauma with	Running, especially high	Moderate

	accelerated turnover and recycling demand.	mileage and impact loading	
GI blood loss	Splanchnic hypoperfusion, mucosal injury, erosions, occult or overt bleeding.	Long races, heat, dehydration, NSAID use	Moderate–high
Sweat/urine/hematuria	Small but cumulative iron losses outside normal turnover.	Heat, long-duration sessions, repeated impact	Low–moderate
Low energy availability	Reduced iron intake and altered regulatory signaling; often coexists with menstrual dysfunction or restrictive eating.	Weight-sensitive sport, dieting, RED-S	High in selected athletes
Menstrual/other non-exercise losses	Blood loss independent of training but synergistic with training load.	Menstruating athletes, GI disease, blood donation	High in selected athletes

Table 1 summarizes the cumulative-burden model: in real athletes, iron deficiency rarely arises from one mechanism alone. Instead, intense training often reveals a pre-existing vulnerability and then sustains it by preventing restoration of iron balance.[3-7,19,20,34,35]

Which athletes are most vulnerable?

The risk of iron deficiency is not evenly distributed across sport. Female athletes, adolescent athletes, endurance athletes, athletes with low energy availability, and those exposed to altitude or unusually heavy blocks of training appear to carry the greatest burden. Retrospective and cohort studies repeatedly show a markedly higher prevalence of deficiency in female compared with male athletes, while contemporary data also show that young athletes and endurance athletes remain overrepresented among cases.[21-25]

That said, "female sex" should be understood as a proxy for a broader cluster of risk factors rather than a deterministic explanation. Menstrual blood loss, dietary restriction, body-composition pressure, lower baseline ferritin, and higher rates of endurance participation explain much of the epidemiology. Male athletes are less often deficient but are not protected when exposed to very high training loads, restrictive diets, gastrointestinal pathology, or repeated altitude camps.[3,6,21-25,35]

Does the effect differ by sport modality?

The effect of intense exercise on iron balance is strongest and best documented in endurance sport, where long training duration, high energy expenditure, repetitive impact, and frequent inflammatory stimuli converge. Distance running, triathlon, rowing, cycling stage racing, cross-country skiing, and military training repeatedly emerge as high-risk contexts. By contrast, the average risk is lower in pure power and skill sports because total training volume and hemolytic burden are often smaller; however, risk remains clinically important when body-composition pressure, dietary restriction, growth, or menstrual losses are present.[3-6,21-25,31,35-37]

Team-sport and mixed-sport athletes should therefore not be excluded from consideration. Professional female athletes in ball sports and combat sports have demonstrated substantial rates of iron-deficient erythropoiesis, and young athletes in university or academy settings may be depleted despite never meeting criteria for anemia. The practical error is to assume that only marathoners become iron deficient. In reality, sport type modifies the probability of deficiency but does not remove the need for targeted testing in symptomatic athletes.[22-25,35,37]

A second clinical nuance is that the most meaningful exposure variable is often not the sport label itself but the combination of training density and recovery opportunity. Two athletes in the same discipline may face very different iron stress depending on whether they train once or twice daily, whether they are in a rapid weight-loss phase, and whether they are simultaneously recovering from altitude, illness, or injury. Longitudinal history is therefore more informative than discipline alone.[6,8,10,31,34-37]

Clinical consequences for health and performance

The clinical consequences of deficiency exist on a spectrum. Iron deficiency without anemia may present with fatigue, reduced training tolerance, poor recovery, exercise intolerance, lower motivation, or a perceived decline in performance despite a normal hemoglobin value. As depletion worsens, functional iron restriction may compromise mitochondrial metabolism and muscle energetics before frank oxygen-carrying capacity falls. Once iron-deficiency anemia develops, reduced hemoglobin mass and impaired oxygen delivery become dominant drivers of reduced aerobic performance.[2,25-27,36]

The performance literature is heterogeneous, partly because study populations differ in baseline ferritin, sex, sport, and outcome selection. Nonetheless, several consistent themes emerge. Iron depletion is associated with slower rowing performance and lower VO2-related outcomes in endurance settings, while more recent athlete cohorts show an independent association between iron deficiency and reduced VO2 peak. Supplementation appears most beneficial when baseline stores are genuinely low; in iron-replete athletes, ergogenic effects are minimal and unnecessary treatment risks overuse of iron.[25-33,36]

From a medical perspective, the key point is that "normal hemoglobin" does not exclude clinically meaningful iron-related impairment. Athletes may have reduced vigor, progressive fatigue, impaired adaptation to training, and underperformance long before meeting laboratory criteria for anemia. That is why sports medicine practice increasingly separates iron deficiency without anemia from iron-deficiency anemia rather than using hemoglobin alone as the screening gate.[2,3,6,26,27,35,36]

1. Distinguishing sports pseudoanemia from true deficiency

Training increases plasma volume, especially in endurance athletes, and this lowers measured hemoglobin concentration and hematocrit without reducing total hemoglobin mass. This dilutional state, often referred to as sports anemia or pseudoanemia, is an adaptive phenomenon that improves thermoregulation and stroke volume. It should not be mistaken for true iron deficiency. A low-normal hemoglobin in a heavily trained athlete may therefore reflect healthy plasma expansion, whereas a low ferritin with falling transferrin saturation suggests true deficiency.[3,20,37]

The distinction matters because pseudoanemia does not require iron therapy, whereas true deficiency does. Uncritical iron prescription based only on hemoglobin may expose athletes to gastrointestinal side effects, poor adherence, unnecessary parenteral treatment, or even iron

overload in rare cases. For that reason, hemoglobin should be interpreted alongside ferritin, iron indices, inflammatory context, menstrual or gastrointestinal history, and training load.[2,3,20,34,37]

2. Diagnostic approach in the athlete with suspected iron deficiency

Evaluation begins with history. Clinicians should document sport type, current training load, recent training camps, altitude exposure, dietary pattern, low energy availability symptoms, menstrual bleeding, gastrointestinal complaints, blood donation, NSAID use, prior iron therapy, and the timing of blood sampling relative to recent hard exercise or illness. These elements frequently explain the laboratory picture as much as the numbers themselves.[3,6,34,35]

Most athlete-focused reviews recommend a minimum panel including hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, serum ferritin, and, when the picture is unclear, transferrin saturation and C-reactive protein. Ferritin remains the most useful marker of depleted stores, but its interpretation is limited by inflammation and acute exercise. In practice, many sports medicine programs regard ferritin below 30 µg/L as clinically relevant in adults, while ferritin goals closer to 50 µg/L are often used before altitude exposure or in selected elite endurance settings.[2,3,6,13,34-36]

Where inflammation or borderline values complicate interpretation, soluble transferrin receptor, reticulocyte hemoglobin indices, or longitudinal repeat testing can help. Blood sampling should ideally be performed under standardized conditions, away from acute illness and not immediately after a race or unusually hard session, because transient post-exercise shifts in plasma volume and ferritin may obscure the underlying state. Persistent or severe deficiency requires evaluation for non-sport causes, particularly gastrointestinal blood loss and malabsorptive disease.[2,3,6,18,34-37]

a. **Table 2. Practical laboratory interpretation and management framework**

Clinical scenario	Typical pattern	What it suggests	Usual response
Adaptive plasma expansion	Hemoglobin low-normal; ferritin adequate; athlete well and training heavily	Pseudoanemia / hemodilution	No iron solely for hemoglobin; repeat and correlate clinically

Iron depletion without anemia	Ferritin low; hemoglobin still normal	Early deficiency affecting recovery/performance	Address cause, optimize diet, consider oral iron
Iron-deficiency anemia	Low ferritin with low hemoglobin and microcytosis evolving	True deficiency with impaired oxygen transport	Treat cause, oral iron or selective IV iron, follow response
Inflammatory confounding	Ferritin normal/high with raised CRP or recent illness	Ferritin may be falsely reassuring	Repeat after recovery; use TSAT and/or sTfR if needed
Altitude preparation	Ferritin borderline before camp	Stores may be inadequate for erythropoietic demand	Replete before exposure when clinically appropriate
Recurrent deficiency	Repeated low ferritin despite treatment	Ongoing blood loss, malabsorption, poor adherence, or underfueling	Investigate GI, menstrual, dietary, and training factors

Prevention and treatment

Dietary and training measures

The first therapeutic step is to identify the mechanism and reduce ongoing losses where possible. Athletes should receive individualized nutritional counseling focused on total energy sufficiency, adequate absolute iron intake, and food combinations that improve bioavailability, such as pairing non-heme iron sources with vitamin C and avoiding concurrent large doses of inhibitors like phytate- or polyphenol-rich beverages around key iron-containing meals. Menstrual and gastrointestinal contributors should be addressed directly rather than treating the laboratory abnormality alone.[3,6,31,34,35]

Training organization can also help. In athletes with recurrent deficiency, clinicians may consider the timing of iron-rich meals or supplements away from the expected hepcidin peak after long or intense sessions, reduction of unnecessary twice-daily endurance work during repletion, careful NSAID use, and proactive planning before altitude blocks. These measures are rarely sufficient alone in moderate or severe deficiency but can materially improve the success of oral therapy.[8,10-14,31]

Oral iron

Oral iron remains first-line treatment for most athletes with iron deficiency because it is effective, inexpensive, and physiologically appropriate. Recent meta-analytic evidence confirms that oral supplementation reliably raises ferritin in athletes with the lowest baseline stores, whereas the effect is smaller when pretreatment ferritin is higher. This is an important reminder that the response to therapy depends on true deficiency severity and not only on the dose prescribed.[3,32,36]

The optimal dosing schedule is still evolving, but the traditional practice of high daily doses has been challenged by hepcidin biology and tolerability concerns. In practice, many sports clinicians now prefer moderate elemental iron doses taken once daily or on alternate days, often in the evening or at a time separated from hard training, gastrointestinal irritants, and calcium-rich meals. Follow-up should assess symptoms, adherence, side effects, and laboratory response rather than assuming that a prescription alone has solved the problem.[10,14,32,34,35]

Parenteral iron

Parenteral iron should be reserved for selected circumstances: intolerance or nonresponse to adequate oral therapy, severe deficiency requiring rapid repletion, malabsorption, or a time-critical performance setting in which oral treatment is unlikely to restore stores before a key competition block. Evidence in athletes shows that intravenous iron can raise ferritin quickly, but improvements in aerobic performance are inconsistent and are not reliably seen in iron-replete or only mildly depleted athletes.[3,29,30,33,36]

Because parenteral treatment can be overused in high-performance sport, it should be approached as a medical therapy rather than a performance shortcut. Documentation of true deficiency, assessment of underlying cause, and awareness of anti-doping regulations and infusion-volume rules are essential. In routine cases, oral therapy and correction of the precipitating mechanism remain the safer and more proportionate strategy.[3,29,30,33]

Monitoring and return to performance

Monitoring is best done longitudinally, not with a single "snapshot." Reassessment after several weeks should consider both laboratory recovery and the athlete's lived response: fatigue, training tolerance, sleep quality, resting heart rate, menstrual symptoms, perceived exertion, and objective performance metrics. Persistently abnormal results despite reported adherence warrant reconsideration of diagnosis, ongoing blood loss, inflammation, celiac disease, or hidden low energy availability.[2,3,6,34-37]

When iron deficiency should trigger broader medical investigation

Exercise-related iron deficiency is common, but common does not mean universal. A clinician should broaden the investigation when deficiency is severe, recurrent, disproportionate to the stated training load, accompanied by significant gastrointestinal symptoms, associated with marked microcytosis, or present in an athlete whose diet and menstrual history do not plausibly explain the deficit. In adults, unexplained iron deficiency may signal gastrointestinal blood loss, malabsorption, inflammatory bowel disease, peptic injury, celiac disease, or other pathology that should not be masked by repeated supplementation.[2,18,20,37,40]

Similarly, heavy menstrual bleeding requires specific attention rather than casual reassurance. Athletes often normalize heavy bleeding because it is longstanding or because symptoms are overshadowed by training fatigue. Asking directly about cycle length, flooding, clots, iron use, hormonal contraception, and the number of pads or tampons used can materially change the diagnostic pathway. In selected cases, gynecologic management may be as important as iron therapy itself.[34,35,40]

Differential diagnosis also includes anemia of inflammation, hemoglobinopathies, B12 or folate deficiency, relative hemodilution, and, occasionally, overreaching or chronic illness presenting with nonspecific fatigue. The sports setting can bias clinicians toward simple explanations; good practice requires remaining willing to look beyond sport when the clinical picture is atypical or when the response to treatment is disappointing.[2,20,37,40]

Safety, overtreatment, and supplementation pitfalls

Iron should be prescribed as a treatment for deficiency, not as a reflex performance aid. In iron-replete athletes, the evidence for ergogenic benefit is weak, whereas adverse effects from unnecessary treatment are real. Oral iron commonly causes nausea, abdominal discomfort, constipation, and poor adherence. Parenteral iron restores stores more rapidly, but the speed of

repletion does not justify its use when the underlying diagnosis is uncertain or when oral therapy has not been given a fair trial.[3,29,30,33,36,40]

Another pitfall is to judge treatment failure too early. Ferritin may rise slowly, especially if training load remains high, energy availability is poor, or supplements are taken during the post-exercise hepcidin window. Conversely, a transient ferritin increase soon after strenuous exercise or illness can create the false impression that stores have normalized. Rechecking under standardized conditions is therefore as important as choosing the initial test.[7,8,10,32,34,36]

Newer delivery strategies also require realism. Interest in transdermal iron has grown because athletes hope to avoid gastrointestinal side effects, but available evidence suggests that oral iron raises ferritin whereas patches do not provide comparable benefit. The most successful programs still rely on documented deficiency, careful timing, adequate dose exposure, and correction of the precipitating cause rather than on novel delivery systems alone.[32,39]

Special situations

Adolescents deserve special caution because growth, sport specialization, dieting behaviors, and menstrual onset can intersect to create large negative iron balance. University and youth cohort data continue to show substantial rates of nonanemic iron deficiency, particularly in female athletes. Screening decisions should be individualized but threshold for testing should be low when fatigue, declining performance, heavy menstrual bleeding, vegetarian diets, or high training volumes are present.[20,24,25]

Military trainees and other occupational groups exposed to short-term but intense physical training represent another high-risk population. In these settings, iron status may deteriorate over only several weeks, and supplementation appears most useful when baseline deficiency is already present. These observations reinforce the broader point that high training stress can reveal marginal iron status rapidly, even outside organized elite sport.[31]

Future directions

Important gaps remain. Better athlete-specific reference intervals are needed for ferritin and related biomarkers across sex, age, and sport. The interaction among hepcidin, circadian biology, menstrual hormones, and carbohydrate availability remains incompletely resolved. More randomized trials are also needed to determine which athletes truly benefit from parenteral iron, how best to schedule oral dosing around training, and whether modern biomarkers such as reticulocyte hemoglobin content or hepcidin itself can improve decision-making beyond ferritin and transferrin saturation.[7,31-36]

Conclusion

Intense physical exercise can promote iron deficiency through a clinically important convergence of mechanisms rather than a single isolated pathway. The most influential processes are increased iron demand, post-exercise hepcidin upregulation with impaired absorption, mechanical hemolysis, gastrointestinal blood loss, and the interaction of training load with low energy availability or menstrual loss. The effect is strongest in endurance athletes, female and adolescent athletes, and those preparing for altitude exposure.[3-8,11-14,21-25]

For clinicians, the practical message is straightforward: do not confuse dilutional pseudoanemia with true deficiency, do not rely on hemoglobin alone, and do not treat athletes blindly with iron in the absence of documented need. A structured history, context-aware laboratory interpretation, and careful correction of the underlying mechanism are more important than any single ferritin threshold. When true deficiency is present, oral iron is usually the first-line therapy, while parenteral iron is best reserved for selected cases. With this approach, most athletes can restore iron balance and recover both health and training capacity.[2,3,20,32-37]

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References

1. Ganz T, Nemeth E. Hepcidin and iron homeostasis. Biochim Biophys Acta. 2012;1823(9):1434-1443. doi:10.1016/j.bbamcr.2012.01.014
2. 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
3. Clénin G, Cordes M, Huber A, et al. Iron deficiency in sports - definition, influence on performance and therapy. Swiss Med Wkly. 2015;145:w14196. doi:10.4414/smw.2015.14196
4. Peeling P, Dawson B, Goodman C, Landers G, Trinder D. Athletic induced iron deficiency: new insights into the role of inflammation, cytokines and hormones. Eur J Appl Physiol. 2008;103(4):381-391. doi:10.1007/s00421-008-0726-6
5. Peeling P. Exercise as a mediator of hepcidin activity in athletes. Eur J Appl Physiol. 2010;110(5):877-883. doi:10.1007/s00421-010-1594-4
6. Sim M, Garvican-Lewis LA, Cox GR, et al. Iron considerations for the athlete: a narrative review. Eur J Appl Physiol. 2019;119(7):1463-1478. doi:10.1007/s00421-019-04157-y
7. Domínguez R, Sánchez-Oliver AJ, Mata-Ordoñez F, et al. Effects of an acute exercise bout on serum hepcidin levels. Nutrients. 2018;10(2):209. doi:10.3390/nu10020209
8. Fensham NC, Sim M, Govus A, Peeling P. 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
9. Peeling P, Sim M, Badenhorst CE, et al. Iron status and the acute post-exercise hepcidin response in athletes. PLoS One. 2014;9(3):e93002. doi:10.1371/journal.pone.0093002
10. McCormick R, Sim M, Dawson B, Lester L, Goodman C, Peeling P. 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.0000000000002036
11. Badenhorst CE, Dawson B, Goodman C, et al. Influence of post-exercise hypoxic exposure on hepcidin response in athletes. Eur J Appl Physiol. 2014;114(5):951-959. doi:10.1007/s00421-014-2829-6
12. Govus AD, Abbiss CR, Garvican-Lewis LA, et al. Acute hypoxic exercise does not alter post-exercise iron metabolism in moderately trained endurance athletes. Eur J Appl Physiol. 2014;114(10):2183-2191. doi:10.1007/s00421-014-2938-2
13. Okazaki K, Stray-Gundersen J, Chapman RF, Levine BD. Iron insufficiency diminishes the erythropoietic response to moderate altitude exposure. J Appl Physiol (1985). 2019;127(6):1569-1578. doi:10.1152/japplphysiol.00115.2018

14. Hall R, Peeling P, Nemeth E, et al. Single versus split dose of iron optimizes hemoglobin mass gains at 2106 m altitude. Med Sci Sports Exerc. 2019;51(4):751-759. doi:10.1249/MSS.0000000000001847
15. Robinson Y, Cristancho E, Böning D. Intravascular hemolysis and mean red blood cell age in athletes. Med Sci Sports Exerc. 2006;38(3):480-483. doi:10.1249/01.mss.0000188448.40218.4c
16. Lippi G, Schena F, Salvagno GL, et al. Foot-strike haemolysis after a 60-km ultramarathon. Blood Transfus. 2012;10(3):377-383. doi:10.2450/2012.0167-11
17. Deitrick RW. Intravascular haemolysis in the recreational runner. Br J Sports Med. 1991;25(4):183-187. doi:10.1136/bjsm.25.4.183
18. Choi SC, Choi SJ, Kim JA, et al. The role of gastrointestinal endoscopy in long-distance runners with gastrointestinal symptoms. Eur J Gastroenterol Hepatol. 2001;13(9):1089-1094. doi:10.1097/00042737-200109000-00016
19. Paulev PE, Jordal R, Pedersen NS. Dermal excretion of iron in intensely training athletes. Clin Chim Acta. 1983;127(1):19-27. doi:10.1016/0009-8981(83)90071-2
20. Nickerson HJ, Holubets MC, Weiler BR, Haas RG, Schwartz S, Ellefson ME. Causes of iron deficiency in adolescent athletes. J Pediatr. 1989;114(4 Pt 1):657-663. doi:10.1016/S0022-3476(89)80717-6
21. Parks RB, Brooks MA. Iron deficiency and anemia among collegiate athletes: a retrospective chart review. Med Sci Sports Exerc. 2017;49(8):1711-1715. doi:10.1249/MSS.0000000000001259
22. Ponorac N, Radovanović D, Stokic E, et al. Professional female athletes are at a heightened risk of iron-deficient erythropoiesis compared with nonathletes. Int J Sport Nutr Exerc Metab. 2020;30(1):48-53. doi:10.1123/ijsnem.2019-0193
23. Sims ST, Mackay K, Leabeater A, Clarke A, Schofield K, Driller M. High prevalence of iron deficiency exhibited in internationally competitive, non-professional female endurance athletes-a case study. Int J Environ Res Public Health. 2022;19(24):16606. doi:10.3390/ijerph192416606
24. Nabeyama T, Suzuki Y, Saito H, et al. Prevalence of iron-deficient but non-anemic university athletes in Japan: an observational cohort study. J Int Soc Sports Nutr. 2023;20(1):2284948. doi:10.1080/15502783.2023.2284948
25. Keller K, Friedrich O, Treiber J, Quermann A, Friedmann-Bette B. Iron deficiency in athletes: prevalence and impact on VO2 peak. Nutrition. 2024;126:112516. doi:10.1016/j.nut.2024.112516

26. DellaValle DM, Haas JD. Impact of iron depletion without anemia on performance in trained endurance athletes at the beginning of a training season: a study of female collegiate rowers. Int J Sport Nutr Exerc Metab. 2011;21(6):501-506. doi:10.1123/ijsnem.21.6.501
27. Brutsaert TD, Hernandez-Cordero S, Rivera J, Viola T, Hughes G, Haas JD. Iron supplementation improves progressive fatigue resistance during dynamic knee extensor exercise in iron-depleted, nonanemic women. Am J Clin Nutr. 2003;77(2):441-448. doi:10.1093/ajcn/77.2.441
28. Klingshirn LA, Pate RR, Bourque SP, Davis JM, Sargent RG. Effect of iron supplementation on endurance capacity in iron-depleted female runners. Med Sci Sports Exerc. 1992;24(7):819-824.
29. Peeling P, Blee T, Goodman C, et al. Effect of iron injections on aerobic-exercise performance of iron-depleted female athletes. Int J Sport Nutr Exerc Metab. 2007;17(3):221-231. doi:10.1123/ijsnem.17.3.221
30. Burden RJ, Pollock N, Whyte GP, et al. Effect of intravenous iron on aerobic capacity and iron metabolism in elite athletes. Med Sci Sports Exerc. 2015;47(7):1399-1407. doi:10.1249/MSS.0000000000000568
31. McClung JP, Karl JP, Cable SJ, et al. Randomized, double-blind, placebo-controlled trial of iron supplementation in female soldiers during military training: effects on iron status, physical performance, and mood. Am J Clin Nutr. 2009;90(1):124-131. doi:10.3945/ajcn.2009.27774
32. Šmid AN, Golja P, Hadžić V, et al. 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
33. Fensham N, Sim M, Lewis L, et al. Parenteral iron therapy: examining current evidence for use in athletes. Int J Sports Med. 2024;45(7):496-503. doi:10.1055/a-2211-0813
34. McKay AKA, Pyne DB, Burke LM, Peeling P. Iron metabolism: interactions with energy and carbohydrate availability. Nutrients. 2020;12(12):3692. doi:10.3390/nu12123692
35. Pedlar CR, Brugnara C, Bruinvels G, Burden R. Iron balance and iron supplementation for the female athlete: a practical approach. Eur J Sport Sci. 2018;18(2):295-305. doi:10.1080/17461391.2017.1416178
36. Badenhorst CE, Goto K, O'Brien WJ, Sims S. Iron status in athletic females, a shift in perspective on an old paradigm. J Sports Sci. 2021;39(14):1565-1575. doi:10.1080/02640414.2021.1885782

37. Solberg A, Reikvam H. Iron status and physical performance in athletes. Life (Basel). 2023;13(10):2007. doi:10.3390/life13102007
38. Damian MT, Chis A, Izzedine H, et al. Anemia in sports: a narrative review. Life (Basel). 2021;11(9):987. doi:10.3390/life11090987
39. McCormick R, Dawson B, Sim M, Lester L, Goodman C, Peeling P. The effectiveness of transdermal iron patches in athletes with suboptimal iron status (part 1). Int J Sport Nutr Exerc Metab. 2020;30(3):185-190. doi:10.1123/ijsnem.2019-0309
40. Auerbach M, DeLoughery TG, Tirnauer JS. Iron deficiency in adults: a review. JAMA. 2025;333(20):1813-1823. doi:10.1001/jama.2025.0452