International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
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ARTICLE TITLE	VITAMIN D DEFICIENCY AND RISK OF OVERUSE MUSCULOSKELETAL INJURIES IN RECREATIONAL RUNNERS
DOI	https://doi.org/10.31435/ijitss.1(49).2026.4858
RECEIVED	29 January 2026
ACCEPTED	15 March 2026
PUBLISHED	30 March 2026
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VITAMIN D DEFICIENCY AND RISK OF OVERUSE MUSCULOSKELETAL INJURIES IN RECREATIONAL RUNNERS

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ABSTRACT

Recreational distance running provides substantial cardiovascular and metabolic benefits but is associated with a high burden of overuse musculoskeletal injuries, particularly bone stress injuries and medial tibial stress syndrome. Low vitamin D status is common in athletes, including endurance runners living at higher latitudes, and may adversely affect bone, muscle and tendon adaptation to repetitive loading.

This narrative review synthesises current evidence on vitamin D metabolism, its roles in the musculoskeletal system and the relationship between serum 25-hydroxyvitamin D [25(OH)D] and overuse injuries in recreational and sub-elite runners. Electronic databases were searched for observational, interventional and review studies addressing vitamin D status, bone stress injuries, soft-tissue overuse conditions and determinants of 25(OH)D in endurance athletes, with particular emphasis on running cohorts and military populations exposed to similar mechanical loads.

Observational and interventional data in military recruits and athletes show that low 25(OH)D status is consistently associated with a higher risk of bone stress injuries, whereas evidence for muscle strains, tendinopathies and non-specific musculoskeletal pain remains limited and heterogeneous. Studies in endurance athletes indicate that vitamin D insufficiency is frequent, especially during winter, at higher latitudes and in those training predominantly indoors or outside peak sunlight hours. However, few prospective studies have specifically examined recreational runners, and virtually no trials have tested whether correcting vitamin D insufficiency reduces overuse injury incidence beyond standard load management and nutritional strategies.

Available evidence supports viewing vitamin D as one modifiable systemic factor within multifactorial overuse injury prevention in runners, rather than an isolated causal agent. Maintaining serum 25(OH)D at least above 50 nmol/L, and possibly around 75–100 nmol/L in runners at elevated risk of bone stress injuries, appears reasonable, particularly in those with limited sun exposure or features of relative energy deficiency. Well-designed prospective cohorts and randomized trials in recreational runners are needed to clarify causal effects and refine screening and supplementation recommendations.

KEYWORDS

Vitamin D Deficiency, Overuse Musculoskeletal Injuries, Bone Stress Injuries, Recreational Distance Runners, Serum 25-Hydroxyvitamin D

CITATION

Nel Geworkian, Paulina Malon, Aleksandra Trojańska, Anita Pieńkowska, Karol Śliwa, Natalia Hariasz, Martyna Pietz, Anita Szymańska. (2026) Vitamin D Deficiency and Risk of Overuse Musculoskeletal Injuries in Recreational Runners. *International Journal of Innovative Technologies in Social Science*. 1(49). doi: 10.31435/ijitss.1(49).2026.4858

1.Introduction

Running is one of the most widely practised forms of physical activity worldwide and is associated with substantial cardiovascular, metabolic, and psychological benefits (Lopes et al., 2012; Kakouris et al., 2021). With the continuous growth of the running population, the burden of overuse injuries has become increasingly recognised (Lopes et al., 2012; Kakouris et al., 2021). The literature reports wide variation in the incidence of musculoskeletal injuries among runners, largely due to differences in population characteristics, study design, and injury definitions (Lopes et al., 2012; van der Worp et al., 2015; Hespanhol et al., 2016). Running-related injuries also impose a considerable health and economic burden on recreational runners (Hespanhol et al., 2016).

The musculoskeletal complaints most frequently reported by runners include stress fractures, medial tibial stress syndrome (MTSS), patellofemoral pain syndrome, and Achilles tendinopathy, among others (Lopes et al., 2012; van der Worp et al., 2015). These conditions typically develop when repetitive mechanical loading exceeds the capacity of bone, muscle, or tendon to recover and adapt (Hreljac, 2004; Tenforde et al., 2016). Training errors, biomechanical factors, and footwear choices are commonly discussed contributors to such injuries (Bahr & Holme, 2003; Hreljac, 2004). The relationship between training load and injury risk is complex, with both excessive and insufficient load potentially contributing to overuse injuries (Gabbett, 2016). In recent years, however, growing attention has been directed toward the possible role of nutritional and biochemical factors, which may also influence the load tolerance of musculoskeletal tissues (Tenforde et al., 2016; Mountjoy et al., 2018).

Among the various nutritional and biochemical factors implicated in musculoskeletal health, vitamin D has gained particular attention due to its recognised role in bone and muscle function (Holick, 2007; Cannell et al., 2009; Girgis et al., 2013). Vitamin D plays an essential role in calcium–phosphate homeostasis, bone mineralisation, neuromuscular function, and modulation of inflammatory pathways (Holick, 2007; Holick et al., 2011). Serum 25-hydroxyvitamin D [25(OH)D] is widely recognised as the most reliable indicator of vitamin D status (Holick et al., 2011). Vitamin D deficiency or inadequacy remains common among athletes, including outdoor endurance athletes, due to seasonal variation in ultraviolet exposure, training schedules, skin pigmentation, and dietary factors (Farrokhyar et al., 2015; Dahlquist et al., 2015). Systematic reviews indicate that a substantial proportion of athletes present with low 25(OH)D levels across different sports and latitudes (Farrokhyar et al., 2015).

Growing evidence suggests that inadequate vitamin D levels may increase the risk of musculoskeletal injuries, particularly bone stress injuries (Tenforde et al., 2016; Millward et al., 2020). Prospective and observational studies have demonstrated associations between low 25(OH)D levels and a higher incidence of stress fractures in physically active populations (Ruohola et al., 2006; Lappe et al., 2008). Furthermore, both clinical and experimental data support a role of vitamin D and calcium in the prevention and management of stress fractures (Lappe et al., 2008; Millward et al., 2020). Additionally, emerging work has identified potential links between vitamin D deficiency and tendon pathology, impaired muscle recovery, and altered muscle and tendon structure, which may predispose athletes to overuse injuries (Yoon et al., 2021; Tarantino et al., 2024). The biological plausibility of these associations is supported by the presence of vitamin D receptors in osteoblasts, myocytes, and tenocytes, and by experimental evidence indicating that vitamin D influences bone microarchitecture, muscle metabolism, collagen synthesis, and inflammatory signalling (Girgis et al., 2013; Cannell et al., 2009).

Despite the expanding body of literature exploring vitamin D and musculoskeletal health, there is a lack of reviews specifically addressing this relationship in recreational runners, a population with distinct biomechanical, training, and exposure characteristics compared with elite athletes (Farrokhyar et al., 2015; Dahlquist et al., 2015). Most studies include heterogeneous athletic cohorts, limiting the ability to draw conclusions applicable to runners alone or to distinguish between elite and recreational populations (Tenforde et al., 2016; Millward et al., 2020).

This narrative review aims to synthesise and critically evaluate current evidence regarding vitamin D status and the risk of overuse injuries in recreational runners. Specifically, it summarises key aspects of vitamin D and musculoskeletal function, examines evidence from military and athletic cohorts with a focus on runners, discusses practical implications for screening and prevention, and highlights knowledge gaps and priorities for future research. In this context, overuse musculoskeletal injuries in recreational runners represent a multifactorial problem in which repetitive mechanical loading interacts with systemic factors, including nutritional and endocrine status such as vitamin D availability (Tenforde et al., 2016; Mountjoy et al., 2018; Bouillon et al., 2020).

The present article is a narrative review. Relevant studies were identified by searching PubMed and Scopus using combinations of the terms “vitamin D”, “25-hydroxyvitamin D”, “stress fracture”, “overuse injury”, “runner” and “endurance athlete”. Priority was given to human observational and interventional studies, systematic reviews and meta-analyses published in English, with particular emphasis on recreational and sub-elite endurance runners and military cohorts exposed to similar lower-limb loading.

2. Role of vitamin D in the musculoskeletal system

2.1. Vitamin D metabolism

Vitamin D is obtained from cutaneous synthesis under ultraviolet B radiation and from dietary sources or supplements as vitamin D_2 (ergocalciferol) and D_3 (cholecalciferol) (Holick, 2007; Lips, 2006). After entering the circulation, it is transported to the liver, where 25-hydroxylase enzymes, mainly CYP2R1, convert it to 25-hydroxyvitamin D [25(OH)D], the major circulating form and primary marker of vitamin D status (Holick et al., 2011; Bikle, 2014). Subsequently, 25(OH)D is converted in the kidney by 1α-hydroxylase (CYP27B1) to the biologically active hormone 1,25-dihydroxyvitamin D [1,25$(OH)_2$D], whose production is tightly regulated by parathyroid hormone, fibroblast growth factor-23, and serum calcium and phosphate (Holick, 2007; Bouillon et al., 2019). Both 25(OH)D and 1,25$(OH)_2$D are catabolised by 24-hydroxylase (CYP24A1), limiting excessive vitamin D signalling in target tissues (Bikle, 2014). Serum 25(OH)D is widely accepted as the most reliable indicator of vitamin D status because it reflects cutaneous synthesis and intake and has a relatively long half-life compared with vitamin D and 1,25$(OH)_2$D (Holick et al., 2011; Holick, 2007).

Clinical and public health guidelines commonly define deficiency as 25(OH)D below approximately 25–30 nmol/L, insufficiency as 30–50 nmol/L, and sufficiency as above 50 nmol/L, although thresholds vary slightly between organisations (Holick et al., 2011; Bouillon et al., 2019). Concentrations below 50 nmol/L are consistently associated with adverse skeletal outcomes, including reduced bone mineral density and increased fracture risk, whereas more severe deficiency (for example <25 nmol/L) is linked with osteomalacia and pronounced musculoskeletal symptoms (Bouillon et al., 2019; Wranicz and Szostak-Węgierek, 2014).

2.2. Mechanisms of action

The active metabolite $1,25(OH)_2D$ binds to the intracellular vitamin D receptor (VDR), a nuclear transcription factor expressed in many tissues, including osteoblasts, myocytes and tenocytes (Bikle, 2014; Girgis et al., 2013). The $1,25(OH)_2D$–VDR complex regulates transcription of genes involved in calcium and phosphate transport, matrix protein synthesis, and cell proliferation and differentiation, exerting pleiotropic effects on bone, muscle and tendon (Bikle, 2014; Dougherty et al., 2016).

One of the key classical actions of vitamin D is regulation of calcium and phosphate homeostasis through increased intestinal absorption, renal tubular reabsorption and coordination with parathyroid hormone, maintaining mineral availability for bone and muscle function (Holick, 2007; Bouillon et al., 2019). By enhancing mineral supply and directly stimulating osteoblast activity, vitamin D supports bone formation and mineralisation, whereas deficiency predisposes to increased bone turnover, bone loss and impaired microarchitecture (Bouillon et al., 2019; Wranicz and Szostak-Węgierek, 2014).

In skeletal muscle, vitamin D influences fibre size, mitochondrial function and protein synthesis through VDR-mediated genomic pathways and rapid non-genomic signalling (Girgis et al., 2013). Low 25(OH)D levels have been associated with reduced muscle mass, decreased neuromuscular strength and poorer physical performance, suggesting that deficiency and insufficiency may compromise strength, power and recovery after exercise or injury (Bischoff-Ferrari et al., 2004; Beaudart et al., 2014).

Vitamin D also exerts immunomodulatory and anti-inflammatory effects, modulating cytokine production and immune responses, which may be relevant for tissue healing and adaptation to mechanical load (Wranicz and Szostak-Węgierek, 2014; Dougherty et al., 2016). Experimental and clinical data indicate that vitamin D can regulate collagen synthesis, extracellular matrix remodelling and mineralisation at the tendon–bone interface, and VDR expression in tenocytes supports a potential role in tendon health and enthesis repair (Dougherty et al., 2016; Tarantino et al., 2024).

Overall, adequate vitamin D status appears important for optimal bone mineral density, muscular strength and recovery, and possibly tendon integrity, whereas suboptimal 25(OH)D levels may impair these adaptations and increase vulnerability to overuse injury (Bouillon et al., 2019; Girgis et al., 2013; Wranicz and Szostak-Węgierek, 2014).

3. Evidence from military and athletic cohorts

3.1. Bone stress injuries

Observational studies in athletic and military populations consistently report that low serum 25(OH)D concentrations are associated with a higher risk of lower-limb bone stress injuries (Ruohola et al., 2006; Lappe et al., 2008; Ribeiro et al., 2020). In young Finnish male recruits, baseline 25(OH)D below the cohort median independently predicted a greater incidence of stress fractures during basic training, suggesting that vitamin D status influences skeletal resilience under high repetitive loading (Ruohola et al., 2006). Similar relationships have been observed in other military cohorts and in athletes exposed to high cumulative lower-limb loading, such as distance runners, track and field athletes and players of team sports involving repeated sprinting and jumping (Lappe et al., 2008; Ribbans et al., 2021; Ip et al., 2022). Some studies indicate that athletes with recurrent stress fractures more often present chronically low 25(OH)D levels, raising the possibility that inadequate vitamin D status contributes to persistent vulnerability to overuse bone pathology (Ribeiro et al., 2020; Ribbans et al., 2021).

Prospective cohort studies further suggest that low 25(OH)D status may precede and predict the occurrence of stress fractures in physically active populations (Ruohola et al., 2006; Armstrong et al., 2020). In military and other high-load training environments, lower pre-training 25(OH)D levels correlate with higher rates of lower-limb stress fractures during basic training and intensive conditioning (Armstrong et al., 2020). Randomised interventional trials indicate that correcting low vitamin D status, particularly when combined with adequate calcium intake, can reduce stress fracture risk in high-risk cohorts; in female Navy recruits, daily calcium and vitamin D supplementation during basic training significantly reduced the incidence of stress

fractures compared with placebo, supporting a causal contribution of suboptimal mineral and vitamin D status to these injuries (Lappe et al., 2008). These results from military recruits should be interpreted cautiously when applied to recreational runners because of important differences in training intensity, age, nutritional habits, and medical monitoring (Ruohola et al., 2006; Lappe et al., 2008; Armstrong et al., 2020). More recent prospective studies and supplementation programmes in military and athletic cohorts generally show similar trends, although several studies are underpowered and not all report statistically significant effects (Armstrong et al., 2020; Girgis et al., 2013). Overall, the available evidence supports maintaining adequate vitamin D and calcium as a plausible, low-risk component of multifactorial strategies to prevent stress fractures under high-repetitive mechanical loading (Ruohola et al., 2006; Lappe et al., 2008; Armstrong et al., 2020).

3.2. Soft-tissue and non-bony overuse injuries

Compared with bone stress injuries, evidence linking vitamin D status to soft-tissue overuse conditions such as tendinopathy and muscle strain is more limited and heterogeneous (Girgis et al., 2013; Sikora-Klak et al., 2018). Some observational studies in athletic and military populations report higher rates of non-specific muscle pain, time-loss musculoskeletal complaints or skeletal muscle injuries in individuals with low 25(OH)D, suggesting that inadequate vitamin D status might compromise muscle resilience and recovery under repetitive loading (Rebolledo et al., 2018; Sikora-Klak et al., 2018). Experimental and clinical data indicate that vitamin D can influence muscle fibre size, neuromuscular function and mitochondrial efficiency, and that deficiency is associated with reduced strength and greater susceptibility to fatigue, which could indirectly predispose to overuse muscle injuries (Girgis et al., 2013; Wyon et al., 2012). Supplementation trials in athletes and physically active individuals generally show that correcting low 25(OH)D can improve selected strength and power measures and may attenuate post-exercise increases in muscle damage biomarkers, although performance effects are inconsistent and most studies do not assess clinically defined overuse injuries (Jastrzębska et al., 2018; Wiciński et al., 2019).

Evidence directly linking vitamin D status to tendon pathology is sparse and mainly mechanistic: vitamin D receptor expression in musculoskeletal tissues and experimental modulation of collagen turnover provide a plausible basis for effects on tendon and enthesis health, but clinical data are scarce and often group soft-tissue injuries with other musculoskeletal outcomes (Girgis et al., 2013; Sikora-Klak et al., 2018). Data on vitamin D and non-specific musculoskeletal pain are similarly mixed, with some trials in deficient adults showing small benefits of supplementation and others finding no meaningful effect versus placebo. Overall, current evidence supports only cautious, hypothesis-generating inferences about an independent role of 25(OH)D in soft-tissue overuse injuries, in contrast to the clearer and more consistent associations observed for stress fractures (Girgis et al., 2013; Sikora-Klak et al., 2018).

4. Vitamin D status and injuries in runners

The mechanistic pathways linking vitamin D to bone, muscle and tendon suggest several ways in which vitamin D status could influence overuse injury risk in recreational runners (Bikle, 2014; Girgis et al., 2013). Through its roles in calcium–phosphate homeostasis, bone remodelling, muscle function, and tendon–bone interface health, suboptimal vitamin D status may reduce tissue resilience and delay repair of microdamage under repetitive running loads, providing a biologically plausible rationale to consider vitamin D deficiency or insufficiency as a potentially modifiable systemic risk factor alongside mechanical and behavioural determinants (Holick et al., 2011; Bouillon et al., 2020; Dougherty et al., 2016).

4.1. Prevalence and determinants of low vitamin D in runners

Studies in endurance and recreational runners indicate that vitamin D insufficiency is common, with a substantial proportion of athletes presenting 25(OH)D concentrations below generally accepted sufficiency thresholds, particularly during winter and at higher latitudes (Farrokhyar et al., 2015; Dahlquist et al., 2015; Ip et al., 2022; Jastrzębska et al., 2018; Yoon et al., 2021). Cross-sectional work in distance runners and triathletes reports that many athletes have 25(OH)D levels in the range typically classified as insufficiency (around 30–50 nmol/L), despite regular outdoor training, suggesting that training time of day, clothing, skin pigmentation and sunscreen use can limit cutaneous synthesis (Farrokhyar et al., 2015; Ip et al., 2022; Dahlquist et al., 2015).

Seasonal variation is a consistent determinant of vitamin D status in runners, with markedly lower 25(OH)D concentrations observed in late winter compared with summer, and indoor or predominantly early-morning/evening training associated with lower levels than midday outdoor sessions (Farrokhyar et al., 2015; Dahlquist et al., 2015; Yoon et al., 2021). Additional factors linked to low 25(OH)D in endurance

athletes include higher latitude residence, darker skin phototype, low dietary vitamin D intake, and limited use of fortified foods, although findings for body composition are not entirely consistent across studies (Farrokhyar et al., 2015; Ip et al., 2022; Yoon et al., 2021). Overall, the available data suggest that a sizeable subset of recreational and sub-elite endurance runners may have suboptimal vitamin D status for musculoskeletal health, especially in winter and in those with limited sun exposure, highlighting the importance of context-specific assessment rather than assuming sufficiency based solely on outdoor training habits (Farrokhyar et al., 2015; Ip et al., 2022; Yoon et al., 2021).

4.2. Vitamin D and bone stress injuries in runners

Direct evidence specifically addressing the relationship between vitamin D status and overuse musculoskeletal injuries in recreational distance runners is limited, and most insights are extrapolated from broader athletic or mixed endurance cohorts (Moran et al., 2013; Knechtle et al., 2021). Existing work in endurance athletes suggests that baseline 25(OH)D concentrations below the cohort median, typically around 50–75 nmol/L, are associated with a higher prevalence of vitamin D insufficiency and markers of skeletal stress rather than a single uniform deficiency threshold for injury risk (Ip et al., 2022; Magee et al., 2013). Most available data on 25(OH)D and overuse injury risk derive from military cohorts, elite athletes, or mixed sporting populations, which differ from recreational runners in training load, supervision, and exposure to other risk factors (Ruohola et al., 2006; Ribbans et al., 2021). Athlete-focused reviews suggest that levels ≥75 nmol/L may be preferable for bone and musculoskeletal health in high-load endurance sports, even though conventional deficiency is often defined at <50 nmol/L, but the applicability of these findings to recreational runners with more variable training patterns, footwear choices, and injury histories remains only partially understood (Ip et al., 2022; Yoon et al., 2021; Moran et al., 2013; Kakouris et al., 2021).

Studies specifically focusing on runners have mainly described the prevalence and determinants of vitamin D insufficiency in endurance athletes rather than directly linking 25(OH)D levels to prospectively recorded overuse injuries (Yoon et al., 2021; Moran et al., 2013). Limited data suggest that runners with a history of stress fractures or recurrent bone stress injuries may more often present low or insufficient vitamin D compared with uninjured controls, but these analyses are typically retrospective, involve small samples, and lack comprehensive adjustment for confounders such as energy availability and menstrual function (Moran et al., 2013; Ribbans et al., 2021).

4.3. Vitamin D and broader overuse musculoskeletal injuries

Beyond runners, studies in endurance and team-sport athletes have examined 25(OH)D and related markers in relation to both stress fractures and broader musculoskeletal time-loss injuries, but associations for non-bony injuries are less consistent and remain exploratory (Yoon et al., 2021; Ip et al., 2022). Relationships between low vitamin D status and lower-limb stress fractures appear most robust, whereas links with muscle strains, tendinopathies, or non-specific joint pain are more heterogeneous across cohorts, and there is a paucity of studies that concurrently assess training load, 25(OH)D status, bone mineral density, and detailed injury outcomes in recreational runners, limiting the ability to delineate the independent contribution of vitamin D to overuse injury risk (Ribeiro et al., 2020; Knechtle et al., 2021; Ip et al., 2022). Given the high prevalence of overuse injuries and the documented frequency of low vitamin D status in endurance athletes, targeted research in recreational distance runners is warranted, including well-designed prospective cohort studies with regular monitoring of 25(OH)D, training exposure, and clinically verified overuse injuries, and randomised controlled trials of vitamin D supplementation in deficient recreational runners (Kakouris et al., 2021; Moran et al., 2013; Knechtle et al., 2021; Ip et al., 2022).

5. Practical implications for recreational runners

For recreational and sub-elite endurance runners, vitamin D should be viewed as one modifiable systemic factor that interacts with mechanical loading, energy availability and other intrinsic and extrinsic risk factors for overuse musculoskeletal injuries, rather than as an isolated causal agent (Larson-Meyer and Willis, 2010; Owens et al., 2018). Evidence from athletic and military cohorts indicates that low 25(OH)D status is associated with a higher risk of bone stress injuries and may contribute to impaired muscle function and delayed recovery, which can in turn alter load distribution and increase tissue strain during running (Farrokhyar et al., 2015; Ruohola et al., 2006; Lappe et al., 2008; Ip et al., 2022; Moran et al., 2013). In this context, vitamin D optimisation is best considered as part of a broader injury-prevention strategy, alongside load management, adequate nutrition and correction of biomechanical factors (Tenforde et al., 2016; Mountjoy et al., 2018; Ip et al., 2022).

Given the high prevalence of vitamin D inadequacy in athletes at latitudes above approximately 40°N and in those training predominantly indoors or in the early morning and evening, it is not appropriate to assume sufficiency in recreational runners solely based on regular outdoor training (Farrokhyar et al., 2015; Ip et al., 2022; Yoon et al., 2021). Context-specific assessment of 25(OH)D can be considered particularly in runners with recurrent or unexplained bone stress injuries, persistent medial tibial stress syndrome or multiple overuse complaints over a short period (Tenforde et al., 2016; Millward et al., 2020; Knechtle et al., 2021). Assessment is also particularly relevant for those with known low bone mineral density or clinical features of relative energy deficiency in sport, such as menstrual disturbance, low body mass, and impaired recovery (Mountjoy et al., 2018; Ackerman et al., 2019). Testing may additionally be reasonable before periods of substantially increased training load in runners with minimal sun exposure, darker skin phototype, limited intake of vitamin D-rich or fortified foods, or a history of stress fractures (Holick, 2007; Farrokhyar et al., 2015; Ip et al., 2022; Yoon et al., 2021).

For musculoskeletal health in athletes, several expert groups and narrative reviews recommend maintaining 25(OH)D concentrations of at least 50 nmol/L as a minimum threshold for bone health (Holick et al., 2011; Bouillon et al., 2019). Several authors further propose that levels around 75–100 nmol/L may be a pragmatic target range for physically active individuals at risk of bone stress injuries, based on observational and interventional data linking these concentrations to lower fracture and stress-fracture incidence (Farrokhyar et al., 2015; Ip et al., 2022; Bischoff-Ferrari et al., 2005). Within this range, reductions in fracture and bone stress injury risk appear most plausible, whereas current evidence does not support additional benefit from substantially higher 25(OH)D concentrations and raises concerns about unnecessarily high-dose supplementation in the absence of documented deficiency (Ip et al., 2022; Ross et al., 2011). When low 25(OH)D is identified in a recreational runner, correction should be individualised based on baseline status, dietary intake and sun exposure, and combined with ensuring adequate calcium intake and overall energy availability, rather than implemented as a stand-alone intervention (Holick, 2007; Ogan and Pritchett, 2013; Ip et al., 2022).

From a practical clinical and coaching perspective, vitamin D optimisation should be integrated with careful progression of running volume and intensity, appropriate rest, strength and neuromuscular training, and attention to footwear and running surface, especially in runners with a history of tibial stress injuries, metatarsal stress fractures or Achilles and patellar tendinopathy (Tenforde et al., 2016; Kakouris et al., 2021). In such cases, maintaining 25(OH)D within the sufficient range can be considered a low-risk, potentially beneficial element of comprehensive care, particularly during winter and in those living at higher latitudes, but it cannot substitute for systematic monitoring and adjustment of mechanical loading or for addressing broader issues such as relative energy deficiency in sport and low bone mineral density (Farrokhyar et al., 2015; Lappe et al., 2008; Mountjoy et al., 2018; Ip et al., 2022).

6. Knowledge gaps and conclusions

Current evidence indicates that low 25(OH)D status is common among athletes and is consistently associated with a higher risk of bone stress injuries in military cohorts and selected high-risk sports, particularly when combined with high mechanical loads and other systemic risk factors (Farrokhyar et al., 2015; Ruohola et al., 2006; Lappe et al., 2008; Ip et al., 2022). Vitamin D deficiency and insufficiency have also been linked to impaired muscle function, altered recovery and potentially increased susceptibility to overuse injuries, although data for soft-tissue conditions such as tendinopathy and muscle strain remain limited and heterogeneous (Moran et al., 2013; Ip et al., 2022; Yoon et al., 2021; Ogan and Pritchett, 2013). Importantly, most studies include mixed athletic populations, with relatively few analyses focusing specifically on adult recreational runners, which restricts the direct transferability of findings to this group (Kakouris et al., 2021; Farrokhyar et al., 2015; Yoon et al., 2021).

Key knowledge gaps concern the lack of prospective cohort studies in recreational and sub-elite endurance runners that simultaneously assess vitamin D status, detailed training load and well-defined overuse injury outcomes over time (Tenforde et al., 2016; Millward et al., 2020; Knechtle et al., 2021). There is also a paucity of recognizing controlled trials testing whether correcting vitamin D insufficiency, with or without calcium, reduces the incidence of bone stress injuries or broader overuse musculoskeletal complaints in runners beyond the effects of standard load management and nutritional recognizing (Farrokhyar et al., 2015; Lappe et al., 2008; Ip et al., 2022; Williams et al., 2020). Furthermore, the relative contributions of total versus free and bioavailable 25(OH)D, as well as other vitamin D metabolites, to bone and tendon adaptation in endurance

athletes have not been fully clarified and warrant further mechanistic and clinical investigation (Bouillon et al., 2019; Ip et al., 2022; Książek et al., 2022).

Within these limitations, the available evidence supports a cautious, pragmatic approach for recreational runners and clinicians working with them (Farrokhyar et al., 2015; Ip et al., 2022; Yoon et al., 2021). Maintaining serum 25(OH)D at least above 50 nmol/L, and preferably within the range of approximately 75–100 nmol/L in runners at elevated risk of bone stress injuries, appears reasonable in light of observational data and interventional trials in related athletic and military populations (Holick et al., 2011; Farrokhyar et al., 2015; Ip et al., 2022; Bischoff-Ferrari et al., 2005). Vitamin D assessment and, where appropriate, supplementation should be ecognizing in runners with recurrent bone stress injuries, persistent medial tibial stress syndrome, low bone mineral density or features of relative energy deficiency, particularly during winter and in those with limited sun exposure (Mountjoy et al., 2018; Ruohola et al., 2006; Lappe et al., 2008; Ackerman et al., 2019). At the same time, vitamin D recognizing should be integrated into multifactorial prevention strategies that emphasise progressive training loads, adequate recovery, sufficient energy and calcium intake, and correction of biomechanical and footwear-related factors, recognizing that no single intervention is likely to prevent overuse injuries in isolation (Kakouris et al., 2021; Tenforde et al., 2016; Ip et al., 2022; Ogan and Pritchett, 2013).

The study has not received any external funding.
The authors declare that there are no conflicts of interest.

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