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# The Role of Vitamin D in Muscle Damage and Recovery Following Exercise: A Narrative Review

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## Abstract

**Background:** Exercise-induced muscle damage (EIMD) is characterized by transient strength loss, muscle soreness, inflammation, and increases in circulating muscle-derived markers. Vitamin D may influence skeletal muscle recovery through effects on vitamin D receptor signaling, calcium handling, mitochondrial function, oxidative stress, inflammatory pathways, and satellite-cell activity.

**Aim:** This narrative review aimed to evaluate the relationship between vitamin D status or supplementation and recovery from EIMD, with particular emphasis on biochemical markers, inflammatory responses, delayed-onset muscle soreness, and restoration of muscle function.

**Materials and methods:** A structured literature search was conducted in PubMed/MEDLINE, Scopus, and Web of Science up to 14 July 2026. Human observational and intervention studies involving healthy adults, physically active individuals, and athletes were prioritized. Preclinical studies were included only to explain potential mechanisms. The evidence was synthesized narratively according to biochemical, inflammatory, subjective, and functional outcomes.

**Results:** Higher baseline 25-hydroxyvitamin D concentrations and vitamin $D_3$ supplementation were associated with improved recovery of muscle strength in several small studies. However, findings concerning creatine kinase, lactate dehydrogenase, myoglobin, inflammatory mediators, and muscle soreness were inconsistent. Effects appeared to vary according to baseline vitamin D status, vitamin D form, dose, supplementation duration, exercise protocol, training status, season, and timing of assessment.

**Conclusions:** Vitamin D may support selected aspects of post-exercise recovery, particularly restoration of muscle strength, but current evidence does not justify its use as a universal recovery-enhancing supplement. Further well-designed randomized trials are required.

**Keywords:** *vitamin D, exercise-induced muscle damage, muscle recovery, inflammation, delayed-onset muscle soreness, skeletal muscle*

## 1. Introduction

Exercise-induced muscle damage (EIMD) commonly develops after unaccustomed or strenuous exercise, particularly when a large number of eccentric muscle contractions are performed. It is typically characterized by a transient reduction in muscle strength, increased muscle soreness, impaired range of motion, and elevated circulating muscle-derived proteins [1]. The initial mechanical disturbance of muscle fibers triggers an inflammatory response

that, when appropriately regulated, contributes to muscle repair, regeneration, and remodeling [2].

Vitamin D may contribute to the regulation of skeletal muscle function and regeneration. Its potential effects involve vitamin D receptor signaling, calcium handling, mitochondrial function, oxidative stress regulation, inflammatory pathways, and satellite-cell activity [3, 4]. This relationship may be particularly relevant to physically active populations because vitamin D insufficiency is relatively common among elite athletes, particularly during periods of limited sunlight exposure [5].

Several studies have investigated whether vitamin D status or supplementation influences recovery after strenuous exercise. In one intervention study, vitamin D supplementation improved the early recovery of peak isometric force and attenuated increases in selected circulating enzymes, particularly alanine and aspartate aminotransferases, but did not reduce muscle soreness [6]. Nevertheless, the overall evidence remains inconsistent. Bello et al. concluded that the available data on circulating muscle damage markers were insufficient to confirm a significant effect of supplementation on post-exercise recovery [7]. In contrast, Rojano-Ortega and Berral-de la Rosa suggested that regular supplementation may attenuate selected indicators of muscle damage and inflammation, although its effects on muscle function, soreness, and oxidative stress require further confirmation. These differences may be related to baseline 25-hydroxyvitamin D concentrations, supplementation protocols, exercise models, participant characteristics, and the timing and selection of outcome measurements [8].

**Research Objective**

The objective of this narrative review is to summarize and critically evaluate current evidence concerning the relationship between vitamin D status, vitamin D supplementation, and recovery from exercise-induced muscle damage. Particular attention is given to inflammatory responses, biochemical markers of muscle damage, delayed-onset muscle soreness, and the restoration of muscle strength and function.

**Research Problems**

The principal research problems addressed in this review are:

1. Does baseline vitamin D status influence the severity of exercise-induced muscle damage and the subsequent recovery process?
2. Can vitamin D supplementation attenuate biochemical and inflammatory responses following muscle-damaging exercise?
3. Does vitamin D supplementation reduce delayed-onset muscle soreness or accelerate the restoration of muscle strength and function?
4. Are the effects of supplementation influenced by baseline 25-hydroxyvitamin D concentrations, supplementation protocols, exercise type, or participant characteristics?

**Review Hypotheses**

1. Adequate vitamin D status may be associated with faster functional recovery following muscle-damaging exercise.
2. Vitamin D supplementation may attenuate selected inflammatory and biochemical indicators of muscle damage, whereas its effects on muscle soreness may be less consistent.
3. The potential benefits of vitamin D supplementation may be greater in individuals with low baseline 25-hydroxyvitamin D concentrations than in individuals with sufficient vitamin D status.
4. Differences in supplementation dose, intervention duration, exercise protocol, participant characteristics, and outcome assessment contribute to the heterogeneity of the available evidence.

## 2. Research Materials and Methods

This study was conducted as a narrative review supported by a structured literature search. The review evaluated the relationship between vitamin D status or supplementation and exercise-induced muscle damage, inflammatory responses, delayed-onset muscle soreness, and functional recovery.

### 2.1. Participants

No participants were directly recruited. The review primarily included studies involving healthy adults, physically active individuals, recreationally trained participants, and athletes. Human observational and intervention studies were prioritized. Animal and in vitro studies were included only when they provided relevant information on the biological mechanisms potentially linking vitamin D with skeletal muscle repair and regeneration.

Studies involving patients with severe neuromuscular disorders, postoperative rehabilitation, or chronic diseases unrelated to exercise-induced muscle damage were excluded from the main clinical synthesis.

### 2.2. Procedure / Search Protocol / Measures

The literature search was conducted in PubMed/MEDLINE, Scopus, and Web of Science. The final search was completed on 14 July 2026. No strict lower publication-date limit was applied; however, emphasis was placed on studies published from 2010 onward. Earlier foundational studies were included when they provided essential information on the mechanisms, symptoms, assessment, or time course of exercise-induced muscle damage.

The search combined terms related to vitamin D, muscle damage, and post-exercise recovery. The following core strategy was used in PubMed and adapted to the syntax of Scopus and Web of Science:

("vitamin D" OR cholecalciferol OR ergocalciferol OR "vitamin D3" OR "vitamin D2" OR "25-hydroxyvitamin D" OR "25(OH)D") AND ("exercise-induced muscle damage" OR EIMD OR "eccentric exercise" OR "muscle damage" OR "delayed-onset muscle soreness" OR DOMS) AND (recovery OR inflammation OR "creatine kinase" OR myoglobin OR strength OR torque OR soreness OR regeneration)

The reference lists of relevant original studies, systematic reviews, and meta-analyses were also screened manually to identify additional publications. Duplicate publications identified across databases were excluded during study selection. Titles and abstracts were screened first, followed by full-text assessment of potentially eligible publications.

Studies were included when they:

1. evaluated vitamin D status or vitamin D supplementation;
2. assessed outcomes related to exercise-induced muscle damage or post-exercise recovery;
3. reported biochemical, inflammatory, subjective, or functional outcomes;
4. were available as full-text publications in English.

Studies were excluded when they assessed vitamin D only in relation to general physical performance without measuring muscle damage or recovery, were available only as abstracts, or provided insufficient methodological or outcome data.

The principal outcomes included serum 25(OH)D concentrations, supplementation dose and duration, creatine kinase (CK) and lactate dehydrogenase (LDH) activity, myoglobin and troponin concentrations, inflammatory mediators, perceived muscle soreness, range of motion, muscle strength, peak torque, and time required for functional recovery.

### 2.3. Data Collection and Analysis

Data were extracted on study design, sample characteristics, training status, baseline 25(OH)D concentration, vitamin D form, supplementation dose and duration, exercise protocol, follow-up period, measured outcomes, principal findings, and reported limitations.

The findings were synthesized narratively and grouped into four main outcome categories: biochemical markers of muscle damage, inflammatory responses, muscle soreness, and functional recovery. Differences between studies were interpreted in relation to baseline vitamin D status, supplementation strategy, vitamin D form, exercise model, participant characteristics, and timing of post-exercise measurements.

Randomized controlled trials and other human intervention studies were prioritized when evaluating clinical effects. Systematic reviews and meta-analyses were used to contextualize the overall consistency of the evidence, whereas observational and preclinical studies were used mainly to describe associations and biological plausibility.

#### 2.3.1. Statistical Software

No dedicated statistical software was used because no meta-analysis, pooled effect calculation, or reanalysis of individual participant data was performed.

#### 2.3.2. Statistical Methods

No original statistical analyses were conducted. Effect estimates, measures of variability, confidence intervals, and statistical significance were considered when reported by the original studies. Results were compared descriptively according to the direction and consistency of the observed effects. No formal risk-of-bias assessment tool was applied because the study was designed as a narrative rather than a systematic review.

Formal analyses of heterogeneity, publication bias, or pooled treatment effects were not performed. Methodological differences were considered qualitatively when interpreting inconsistencies between studies.

## 3. Results - Literature Findings

### 3.1. Exercise-Induced Muscle Damage

Exercise-induced muscle damage (EIMD) commonly develops after unaccustomed or strenuous physical activity, particularly exercise involving repeated eccentric muscle contractions. During eccentric contractions, active muscle fibers are lengthened under tension, and intense eccentric exercise may disrupt myofibrillar organization, including sarcomere and Z-line architecture [9].

The initial mechanical disturbance is followed by a complex biological response involving local inflammation, immune-cell infiltration, and activation of tissue-repair mechanisms [2]. Leukocyte populations, including CD16+ and CD68+ cells, accumulate in exercised muscle during the hours and days following severe eccentric exercise [10]. Although prolonged or dysregulated inflammation may contribute to secondary tissue stress, a coordinated inflammatory response supports the removal of damaged cellular material and the initiation of muscle repair and remodeling [2]. Changes in inflammatory mediators do not always correspond directly with the severity of perceived muscle soreness or alterations in biochemical markers, illustrating the complexity of the post-exercise response [11].

Typical manifestations of EIMD include a temporary reduction in muscle strength, stiffness, swelling, restricted range of motion, and delayed-onset muscle soreness (DOMS). These symptoms follow different time courses. Loss of muscle function may occur immediately after exercise, whereas muscle soreness commonly develops several hours later and reaches its greatest intensity approximately 24–72 hours after the damaging activity [12]. Importantly, DOMS should not be regarded as a direct measure of structural muscle damage. Its severity correlates only weakly with changes in muscle strength, swelling, range of motion, and circulating creatine kinase activity [13].

Biochemical indicators commonly used to evaluate exercise-induced muscle damage include circulating creatine kinase and myoglobin, although these markers provide only indirect information about the extent of muscle injury [11, 14]. Their concentrations may increase following disruption of muscle-cell membrane integrity, but the magnitude and time course of

these responses vary considerably between individuals. In particular, plasma creatine kinase activity does not always correspond to the severity of histologically assessed muscle damage [14]. Therefore, biochemical markers should not be interpreted in isolation. A more reliable assessment of EIMD requires the combined evaluation of circulating markers, perceived muscle soreness, changes in range of motion, and functional outcomes such as loss and recovery of muscle strength.

The recovery process involves the restoration of muscle function and remodeling of affected tissue. Satellite-cell activation represents one component of this response and may be particularly pronounced following eccentric contractions [15]. The magnitude of EIMD is also influenced by previous exposure to similar exercise. A prior bout of eccentric activity may reduce strength loss, soreness, and biochemical responses during a subsequent session, a phenomenon known as the repeated-bout effect [16]. These factors should be considered when interpreting studies evaluating nutritional strategies, including vitamin D supplementation, for post-exercise muscle recovery.

### 3.2. Potential Mechanisms of Vitamin D

Vitamin D may influence skeletal muscle function and regeneration through genomic and rapid non-genomic mechanisms. The biologically active metabolite, 1,25-dihydroxyvitamin $D_3$ [$1,25(OH)_2D_3$], binds to the vitamin D receptor (VDR), which regulates the transcription of genes involved in myogenesis, cellular metabolism, and protein synthesis. Vitamin D-related signaling may also involve MAPK and PI3K/Akt/mTOR pathways, although the physiological significance of these mechanisms in adult human skeletal muscle remains incompletely understood [17, 18].

Rapid vitamin D signaling may also affect intracellular calcium transport and the activity of calcium-dependent signaling proteins, potentially influencing muscle contraction and intracellular signaling [19]. However, direct evidence that these mechanisms attenuate exercise-induced muscle damage in humans remains limited.

Another proposed mechanism involves mitochondrial function. In cultured human skeletal muscle cells, $1,25(OH)_2D_3$ increased mitochondrial oxygen consumption and modified mitochondrial dynamics, suggesting a potential role in cellular energy metabolism [20]. Preclinical evidence also indicates that vitamin D may influence reactive oxygen species

production and antioxidant capacity. These effects may be relevant to energy-dependent repair processes and oxidative stress regulation during recovery from muscle damage [3].

Vitamin D may also interact with inflammatory pathways. In human skeletal muscle, intramuscular VDR protein content has been associated with IL-6 gene and protein expression, suggesting a possible relationship between VDR signaling and local inflammatory processes [21]. However, this observational relationship does not demonstrate that vitamin D directly suppresses post-exercise inflammation. Moreover, an appropriately regulated inflammatory response is involved in the removal of damaged cellular material and subsequent muscle repair and remodeling [2].

Evidence from in vivo animal studies suggests that vitamin D status may influence satellite-cell proliferation and differentiation during muscle growth and regeneration [4]. These effects appear to depend on the physiological context and the administered dose, as vitamin D deficiency may impair satellite-cell differentiation, whereas high doses may impair muscle regeneration in some animal models. In human skeletal muscle precursor cells, $1,25(OH)_2D_3$ altered regulators of myogenesis and the FOXO3 and Notch pathways but also inhibited myoblast proliferation and differentiation under the examined experimental conditions [22].

Overall, vitamin D has several biologically plausible roles in muscle recovery, including the regulation of cellular signaling, calcium handling, mitochondrial function, inflammatory pathways, and satellite-cell activity. However, much of the mechanistic evidence is derived from cell and animal models, and its direct relevance to recovery from exercise-induced muscle damage in athletes remains uncertain.

### 3.3. Effects on Muscle Damage and Recovery

Current evidence regarding the effects of vitamin D on exercise-induced muscle damage remains inconsistent. Observational data suggest that baseline vitamin D status may be associated with functional recovery. Higher pre-exercise serum 25-hydroxyvitamin D concentrations predicted a smaller deficit in peak isometric force immediately and 48–72 h after a muscle-damaging exercise protocol [23]. However, this observational association does not establish a causal effect of vitamin D.

Intervention studies provide some evidence of a possible effect of vitamin $D_3$ on functional recovery. Supplementation with 4000 IU daily for 35 days improved the early restoration of

peak isometric force during the first 24 h after intense exercise but did not significantly reduce perceived muscle soreness [6]. Similarly, six weeks of vitamin $D_3$ supplementation in men with low baseline 25(OH)D concentrations improved the recovery of peak muscle torque 48 h and 7 days after eccentric exercise [24]. These findings suggest a possible influence on functional recovery, although the extent to which baseline vitamin D status modifies this response remains uncertain.

Results concerning circulating markers of muscle damage are less consistent. In endurance-trained runners, vitamin $D_3$ supplementation was associated with lower post-exercise troponin and myoglobin concentrations, lower CK activity after 24 h, and a reduced TNF-α response, whereas no significant effect on LDH was observed [25]. Pilch et al. found higher LDH activity in the suboptimal-status control group than in the remaining groups, whereas CK did not differ between groups and changes in myoglobin could not be attributed unequivocally to supplementation [26]. Other studies reported no clear effects on indices of muscle damage or inflammatory responses despite an improvement in vitamin D status [27, 28].

The form of vitamin D may also influence the observed response. In one study, vitamin $D_2$ supplementation increased circulating 25(OH)$D_2$ but reduced 25(OH)$D_3$ concentrations and was associated with greater post-exercise increases in creatine kinase and myoglobin [29]. These findings indicate that results obtained with ergocalciferol should not be assumed to apply directly to cholecalciferol.

Overall, findings concerning functional recovery appear somewhat more favorable than those concerning circulating biomarkers, inflammatory responses, and delayed-onset muscle soreness, although the number of relevant trials remains limited. A meta-analysis found no significant overall effect on creatine kinase, lactate dehydrogenase, or myoglobin, whereas a later systematic review suggested that regular supplementation may attenuate selected indicators of muscle damage and inflammation [7, 8]. Differences in baseline vitamin D status, supplementation dose and form, exercise protocol, training status, and timing of outcome assessment are likely to contribute to the heterogeneity of the available findings.

### 3.4. Factors Modifying the Effects

The effects of vitamin D on exercise-induced muscle damage and recovery may depend on several methodological and biological factors. Baseline vitamin D status is likely to be particularly important. Higher pre-exercise 25-hydroxyvitamin D concentrations have been

associated with a smaller post-exercise strength deficit, while some intervention studies have reported improved functional recovery in participants with initially low vitamin D status [23, 24]. However, findings concerning biochemical markers remain inconsistent, and a low baseline concentration does not necessarily predict a clear response to supplementation [26].

Supplementation dose, duration, and route of administration may also influence outcomes. Daily vitamin $D_3$ supplementation for several weeks has been associated with favorable changes in muscle strength recovery and selected biochemical markers [6, 25]. In contrast, a single high-dose intramuscular injection improved vitamin D status but did not modify muscle-damage or inflammatory responses after resistance exercise [28]. These findings suggest that an improvement in vitamin D status does not necessarily translate into more favorable acute muscle-damage or inflammatory responses to exercise.

The form of vitamin D should also be considered. Vitamin $D_2$ supplementation increased circulating $25(OH)D_2$ but reduced $25(OH)D_3$ and was associated with greater post-exercise increases in creatine kinase and myoglobin in one study [29]. Therefore, findings obtained with ergocalciferol should not be directly extrapolated to cholecalciferol.

Differences in exercise protocols and participant characteristics further complicate comparisons between studies. Controlled eccentric exercise, resistance training, endurance-related protocols, and simulated team-sport activity may produce different patterns of muscle damage, inflammation, and functional impairment [6, 24, 25, 27, 28]. Training status may also modify the response, as previous exposure to eccentric exercise can attenuate muscle-damage responses during a subsequent bout, a phenomenon known as the repeated-bout effect [16]. Consequently, results obtained in untrained participants may not be directly applicable to trained athletes.

The timing of outcome assessment is another important factor. Muscle strength, soreness, creatine kinase, myoglobin, and inflammatory mediators may peak at different times after exercise. An effect observed during the first 24 h may therefore not persist at 48–72 h, while late biochemical responses may be missed in studies with short follow-up periods [6, 25].

Finally, season and sunlight exposure may influence both baseline and follow-up 25(OH)D concentrations. Considerable seasonal variation has been documented in athletes, particularly in indoor disciplines and at higher latitudes [30]. Season, sunlight exposure, training

environment, and concurrent supplement use should therefore be considered as potential confounding factors in supplementation studies.

Overall, differences in baseline vitamin D status, supplementation strategy, vitamin D form, exercise model, training status, season, and timing of measurements probably contribute substantially to the heterogeneity of the available evidence.

**Table 1. Factors potentially modifying the effects of vitamin D on exercise-induced muscle damage and recovery**

| Factor | Potential mechanism / explanation | Potential impact on recovery | Evidence consistency |
|---|---|---|---|
| **Baseline vitamin D status** | Vitamin D status may influence skeletal muscle function and the response to supplementation | Potentially greater benefit in individuals with low 25(OH)D concentrations | Mixed |
| **Vitamin $D_3$ supplementation** | May support muscle function through VDR signaling, mitochondrial activity, and other mechanisms | Possible improvement in strength recovery | Mixed |
| **Vitamin $D_2$ supplementation** | May produce different changes in vitamin D metabolites than vitamin $D_3$ | Uncertain; one study reported greater CK and myoglobin responses | Limited |
| **Dose and duration of supplementation** | Determine the magnitude and timing of changes in vitamin D status | May influence the magnitude and timing of recovery effects | Mixed |
| **Exercise type** | Different exercise protocols produce different degrees and patterns of EIMD | Important source of between-study variability | Consistent |
| **Training status / repeated-bout effect** | Previous eccentric exercise can attenuate subsequent muscle-damage responses | May reduce the apparent effect of supplementation | Consistent |

| | | | |
|---|---|---|---|
| **Timing of outcome assessment** | Strength, DOMS, CK, myoglobin, and inflammatory markers have different time courses | May determine whether an effect is detected | Consistent |
| **Season and sunlight exposure** | Influence baseline and follow-up 25(OH)D concentrations | Potential confounding of supplementation effects | Consistent |

## 4. Discussion

Current evidence suggests that vitamin D may influence selected aspects of recovery from exercise-induced muscle damage, but the overall findings remain inconsistent. Mechanistic studies indicate several biologically plausible pathways involving VDR signaling, mitochondrial function, calcium handling, oxidative stress, inflammatory regulation, and satellite-cell activity. However, much of this evidence originates from cell and animal models, and its relevance to post-exercise recovery in humans has not been fully established [3, 4, 17].

Human studies provide somewhat more favorable findings for functional recovery than for biochemical markers or muscle soreness. Higher baseline 25(OH)D concentrations and vitamin $D_3$ supplementation have been associated with improved restoration of muscle strength in several studies [6, 23, 24]. Nevertheless, these studies involved relatively small samples, and the results have not been consistently replicated. It also remains uncertain whether improved force production reflects accelerated structural repair or changes in other determinants of muscle function.

Evidence concerning CK, LDH, myoglobin, inflammatory mediators, and DOMS is more heterogeneous. Some studies reported favorable changes in selected biomarkers, whereas others found no meaningful effects despite increased serum 25(OH)D concentrations. Meta-analytic evidence has not confirmed a significant overall effect on commonly measured circulating markers, although a later systematic review suggested possible benefits for selected indicators of muscle damage and inflammation [7, 8]. Muscle soreness should not be considered the primary measure of effectiveness because DOMS does not directly reflect the magnitude of structural muscle damage [13].

Differences in baseline vitamin D status, supplementation dose, vitamin D form, exercise protocol, participant characteristics, and timing of measurements probably explain part of the inconsistency. Potential benefits may be more likely when correcting low vitamin D status,

but the available evidence does not establish a specific 25(OH)D threshold at which improved recovery can be expected. Results obtained with ergocalciferol, cholecalciferol, daily supplementation, and single high-dose administration should also not be considered directly comparable.

### 4.1. Practical Implications

Current evidence does not support vitamin D as a universal recovery-enhancing supplement. Correction of documented deficiency may be appropriate for general musculoskeletal health, but its specific effect on recovery from EIMD remains uncertain. Athletes training indoors, living at higher latitudes, or experiencing limited sunlight exposure may be at greater risk of low vitamin D status [5, 30].

Vitamin D supplementation should complement rather than replace established recovery strategies. Adequate energy and protein intake are important for post-exercise recovery and skeletal muscle adaptation [31, 32]. Sufficient sleep and appropriate progression and monitoring of training loads are also important components of recovery management [33, 34]. At present, no specific vitamin D dose or target 25(OH)D concentration can be recommended solely for reducing exercise-induced muscle damage.

### 4.2. Limitations

The available studies generally included small samples, predominantly young men, and used heterogeneous supplementation protocols and exercise models. Differences in training status, baseline 25(OH)D concentrations, follow-up periods, and outcome measures limit direct comparisons between studies. The restriction to full-text English-language publications may have introduced language and publication bias.

Biochemical markers, DOMS, inflammatory mediators, and muscle strength also follow different post-exercise time courses. Moreover, circulating biomarkers show considerable interindividual variability and do not necessarily correspond to structural muscle damage or functional impairment [1, 2, 14].

Season, sunlight exposure, dietary intake, concurrent supplementation, recent training load, and previous exposure to eccentric exercise were not consistently controlled. The repeated-bout effect may further reduce the magnitude of EIMD in trained individuals and influence the apparent effectiveness of supplementation [16].

### 4.3. Future Directions

Future randomized controlled trials should include larger and more diverse populations, particularly women, elite athletes, and participants with clearly defined vitamin D deficiency. Studies should stratify participants according to baseline 25(OH)D concentrations and report supplementation adherence and achieved serum concentrations.

Standardized vitamin $D_3$ protocols should be used to compare the correction of deficiency with supplementation in vitamin D-sufficient individuals. Serial assessments should cover at least 72 hours after muscle-damaging exercise and combine biochemical, inflammatory, subjective, and functional outcomes. Restoration of muscle strength, return to training, and performance during subsequent exercise should receive particular attention because these outcomes are more relevant to athletes than isolated changes in circulating biomarkers.

Overall, vitamin D may contribute to selected aspects of muscle recovery, but current evidence is insufficient to recommend it specifically as an ergogenic recovery aid. Maintaining an adequate vitamin D status remains reasonable for general health and athlete care.

## 5. Conclusions

Vitamin D may influence selected aspects of recovery from exercise-induced muscle damage, with the most promising findings concerning the restoration of muscle strength. However, its effects on biochemical markers, inflammatory responses, and delayed-onset muscle soreness remain inconsistent. Potential benefits may depend on baseline 25(OH)D concentration, vitamin D form, supplementation strategy, exercise protocol, and training status. At present, vitamin D should not be considered a universal recovery-enhancing supplement. Maintaining an adequate vitamin D status may support general musculoskeletal health, but further well-designed randomized trials are required to determine its specific role in post-exercise muscle recovery.

## DISCLOSURE

### Author's Contributions

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

In preparing this work, the authors used ChatGPT (OpenAI, San Francisco, CA, USA) for linguistic refinement, text organization, formatting support, and improvement of academic writing style. The authors critically reviewed, edited, and verified all generated content and accept full responsibility for the final content of the manuscript.

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