Narrative review

Vitamin D Supplementation and Chronic Musculoskeletal Pain Among Obese Patients: A Narrative Review

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Abstract

Chronic musculoskeletal pain (CMP) is a complex clinical condition that is particularly prevalent among individuals with obesity. In addition to increased mechanical overload, its pathophysiology involves chronic low-grade systemic inflammation, adipose tissue dysfunction, metabolic abnormalities, and altered pain processing. Obesity is also associated with a higher risk of vitamin D deficiency.

The aim of this narrative review was to summarize current evidence on the relationship between vitamin D deficiency, vitamin D supplementation and chronic musculoskeletal pain in overweight and obese individuals. A comprehensive literature search was conducted using the PubMed/MEDLINE and Web of Science (WoS) databases. Original research articles, systematic reviews, meta-analyses, and relevant narrative review articles were included.

Current evidence suggests that vitamin D supplementation may reduce pain intensity, particularly in individuals with documented hypovitaminosis D, whereas findings in the general population remain inconsistent. Although only a limited number of studies have specifically investigated overweight and obese individuals, available randomized controlled trials suggest potential benefits of vitamin D supplementation on pain-related outcomes, especially when combined with physical activity. The underlying mechanisms are likely multifactorial and may involve vitamin D immunomodulatory and anti-inflammatory properties, improvements in musculoskeletal function and modulation of nociceptive pathways; however, these mechanisms remain incompletely understood. Current evidence does not support routine vitamin D supplementation as a sole treatment for chronic musculoskeletal pain. Nevertheless, identifying and correcting hypovitaminosis D may be a part of a multimodal treatment strategy for overweight and obese individuals.

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Introduction

Chronic musculoskeletal pain (CMP) can be localized or widespread and can affect muscles, ligaments, tendons and bones. CMP is associated with reduced physical activity, impaired sleep quality, and mood disturbances, all of which adversely affect quality of life. It can also reduce work capacity and increase healthcare costs. Although its exact pathophysiology has not been fully elucidated, CMP is thought to arise from sensitization of the central and peripheral nervous systems, accompanied by systemic inflammation and dysfunction of the descending pain modulatory network (1, 2). Analgesics remain an important component of chronic pain management; however, they are ineffective in approximately 20% of patients (3). The mechanisms underlying this limited efficacy have not yet been fully clarified, but are believed to reflect the complexity and neuroplasticity of chronic pain (3). Furthermore, opioid analgesics are frequently prescribed for CMP despite their well-known limitations, including tolerance and the risk of dependence. Consequently, considerable efforts have focused on identifying more effective therapeutic strategies for managing CMP.

Obesity is associated with an increased risk of several chronic pain conditions, including chronic widespread pain, fibromyalgia syndrome, neck pain, low back pain, shoulder pain, sciatica and chronic daily headache (1, 4–9). These conditions occur significantly more often in obese individuals than in those with normal body weight, and obesity and chronic pain frequently coexist as comorbid conditions (10, 11).

Because CMP management remains challenging and treatment responses vary considerably among patients, ongoing research continues to explore novel therapeutic approaches. Growing evidence suggests that patients with chronic pain frequently exhibit vitamin D deficiency and that vitamin D may play an important role in pain modulation and musculoskeletal function, in addition to its well-established skeletal effects (12, 13). Epidemiological studies indicate that the prevalence of hypovitaminosis D exceeds 80% among individuals older than 65 years, a population that is also at particularly high risk of developing CMP (14).

Researchers have extensively investigated the relationship between vitamin D and chronic musculoskeletal pain. Several studies have reported that correcting vitamin D deficiency is associated with pain reduction in selected patients with musculoskeletal disorders. As mentioned above, obesity is closely associated with CMP and is characterized by a higher prevalence of hypovitaminosis D. Emerging evidence also suggests that treating vitamin D deficiency may reduce pain intensity in individuals with musculoskeletal pain. In light of these observations, this narrative review summarizes current evidence regarding the association between vitamin D deficiency, vitamin D supplementation and chronic musculoskeletal pain in individuals with obesity.

Methods

A comprehensive literature search was conducted using the PubMed/MEDLINE and Web of Science (WoS) databases to identify relevant studies published from database inception through July 2026. The search strategy included the following keywords and Medical Subject Headings (MeSH) and free-text terms with Boolean operators (AND/OR): ("overweight" OR "obesity") AND ("musculoskeletal pain" OR "chronic pain" OR "fibromyalgia") AND ("vitamin D" OR "vitamin D deficiency" OR "cholecalciferol" OR "calcitriol"). Original research articles, systematic reviews, meta-analyses, and relevant narrative review articles published in English were included, with a preference for the most recent literature. Exclusion criteria were studies involving pediatric populations, non-English publications and studies lacking relevance to pain mechanisms or supplementation outcomes. The authors independently evaluated titles, abstracts, and full texts and qualitatively synthesized relevant mechanistic and clinical data.

Association between Obesity, Vitamin D and Chronic Pain

Overweight and obese individuals have a higher prevalence of several chronic pain conditions, including musculoskeletal pain (90.3%), low back pain (46.8%) and knee pain (63.5%), compared with the general population (15, 16). Obesity is positively associated with both the incidence and severity of pain (1, 16, 17). In addition to experiencing pain more frequently, obese individuals use healthcare services more often because of painful musculoskeletal conditions and have higher healthcare utilisation related to musculoskeletal disorders (15, 18). The prevalence of obesity has increased substantially in Europe over recent decades (19). In Croatia, more than 65% of adults are classified as overweight or obese, placing the country among the European Union member states with the highest obesity rates (20).

Vitamin D is a fat-soluble secosteroid synthesized primarily in the skin following exposure to ultraviolet B (UV-B) radiation, with a smaller proportion obtained from dietary sources (13, 21). Vitamin D undergoes sequential hydroxylation in the liver to 25-hydroxyvitamin D [25(OH)D; calcidiol], the major circulating form and primary biomarker of vitamin D status, and subsequently in the kidneys to 1,25-dihydroxyvitamin D [1,25$(OH)_2$D; calcitriol], the biologically and hormonally active form (3, 22, 23). Vitamin D plays an essential role in calcium homeostasis and musculoskeletal health by facilitating calcium absorption and maintaining normal bone and muscle function (24).

Several mechanisms have been proposed to explain the high prevalence of hypovitaminosis D in overweight or obese individuals. One proposed mechanism is sequestration of vitamin D within adipose tissue, which may reduce its bioavailability in the circulation (25). Consequently, serum 25(OH)D concentrations inversely correlate with adipose tissue mass (26). Another important mechanism is volumetric dilution. Because individuals with obesity have a larger body and distribution volume, vitamin D is distributed into a larger volume, leading to lower measured serum concentrations despite similar total body vitamin D stores (27). In addition, lifestyle factors, including reduced physical activity and lower sunlight exposure, may also contribute to hypovitaminosis D in this population (28). Non-alcoholic fatty liver disease, which is common among obese individuals, has also been associated with lower 25(OH)D concentrations (29).

Mechanisms of Action of Vitamin D

Vitamin D exerts its biological effects primarily through its hormonally active form, 1,25-dihydroxyvitamin D [1,25$(OH)_2$D; calcitriol], acting via the vitamin D receptor (VDR), which is expressed in most tissues and cell types. Through VDR signaling, vitamin D contributes to myogenesis, cell proliferation and differentiation, protein synthesis and mitochondrial metabolism (13, 30, 31). Preclinical studies suggest that vitamin D deficiency may impair skeletal muscle structure and function, potentially contributing to CMP (30, 31). Furthermore, severe vitamin D deficiency can lead to osteomalacia and bone demineralisation, which may cause musculoskeletal pain (12). In patients with CMP, lower vitamin D status has been associated with greater pain intensity and impaired muscle function, although the available clinical evidence remains heterogeneous (14, 30, 31). At the same time, oxidative stress and mitochondrial dysfunction have been proposed as potential mechanisms underlying impaired muscle function (31).

Beyond its effects on bone and skeletal muscle, vitamin D has been implicated in modulating nociceptive signaling. Vitamin D signaling and/or the vitamin D receptor expression have been demonstrated in several components of the pain pathway, including the skin (signal transduction), dorsal root ganglion (DRG) neurons (conduction), the spinal cord (transmission and modulation) and the brain (pain perception) (3). In experimental and cellular models, vitamin D has been shown to modulate nociceptive processing by downregulating nitric oxide synthase and cyclooxygenase-2 (COX-2) expression while upregulating 15-prostaglandin dehydrogenase (32). Furthermore, vitamin D

may modulate glial and astrocyte activation, potentially influencing processes involved in central sensitization (32). Vitamin D has recognized immunomodulatory and anti-inflammatory properties (3). Experimental and ex vivo studies suggest that vitamin D can modulate Toll-like receptor (TLR) signaling and inflammatory cytokine responses (33, 34). TLR signaling, including TLR2 and TLR4 pathways, has been implicated in neuroinflammation and neuropathic pain following nerve injury. Activation of these pathways can contribute to glial activation and pro-inflammatory cytokine release, processes that may contribute to central sensitization (35, 36). Although vitamin D influences pain through multiple biological pathways, its anti-inflammatory effects are considered among the potential mechanisms underlying its analgesic properties. Vitamin D can modulate adaptive immune responses by promoting the differentiation of Th2 and T regulatory (Treg) cells while suppressing pro-inflammatory Th1 and Th17 responses, which are involved in chronic inflammation and tissue injury (37, 38). In vitro studies also show that vitamin D can inhibit prostaglandin E_2 (PGE_2) synthesis in fibroblasts while promoting its degradation by upregulating 15-prostaglandin dehydrogenase (39). Vitamin D is also associated with reduced production of pro-inflammatory mediators, including TNF-α, IL-1, and PGE_2, and enhanced anti-inflammatory cytokine responses such as IL-10 (38). Inhibition of PGE_2 synthesis is particularly noteworthy because PGE_2 is an important mediator of inflammatory pain and its suppression provides a plausible mechanistic explanation for the analgesic effects of vitamin D (38, 39). The proposed mechanisms linking obesity, vitamin D deficiency, and chronic musculoskeletal pain are summarized in Figure 1. The potential effects of vitamin D supplementation are summarized in Figure 2.

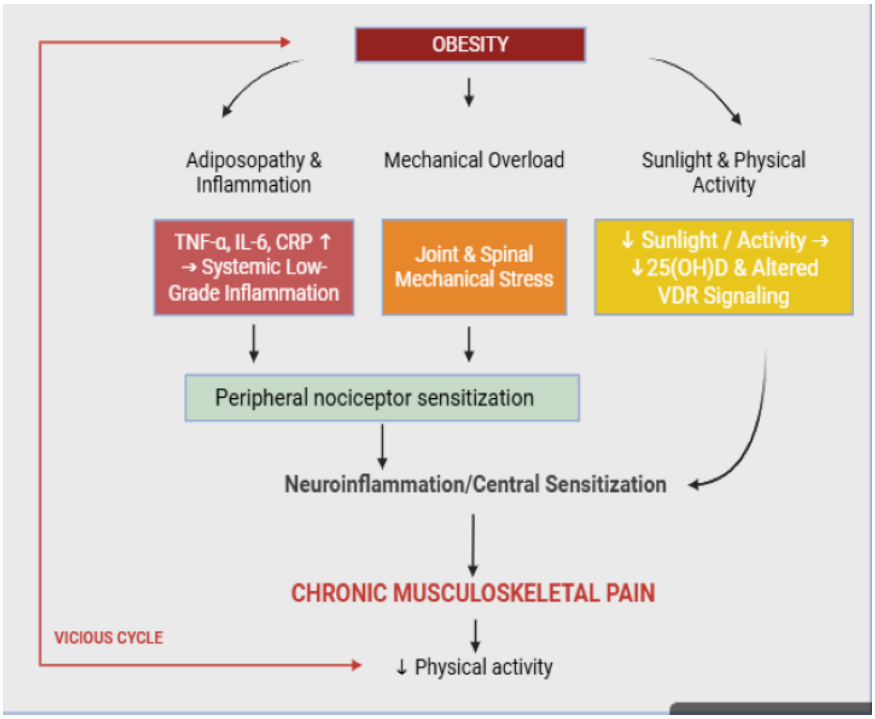


Figure 1. Proposed mechanisms linking obesity, vitamin D deficiency, and chronic musculoskeletal pain.

Obesity may contribute to chronic musculoskeletal pain through several interconnected pathways, including mechanical overload, adipose tissue dysfunction, chronic low-grade systemic inflammation, and reduced physical activity and sunlight exposure. Obesity-related alterations in vitamin D metabolism may lower circulating 25-hydroxyvitamin D [25(OH)D] concentrations and alter vitamin D receptor (VDR) signaling. Vitamin D deficiency may

further impair skeletal muscle function, cause mitochondrial dysfunction, and alter immune function, while modulating inflammatory and nociceptive pathways, potentially promoting peripheral and central sensitization.

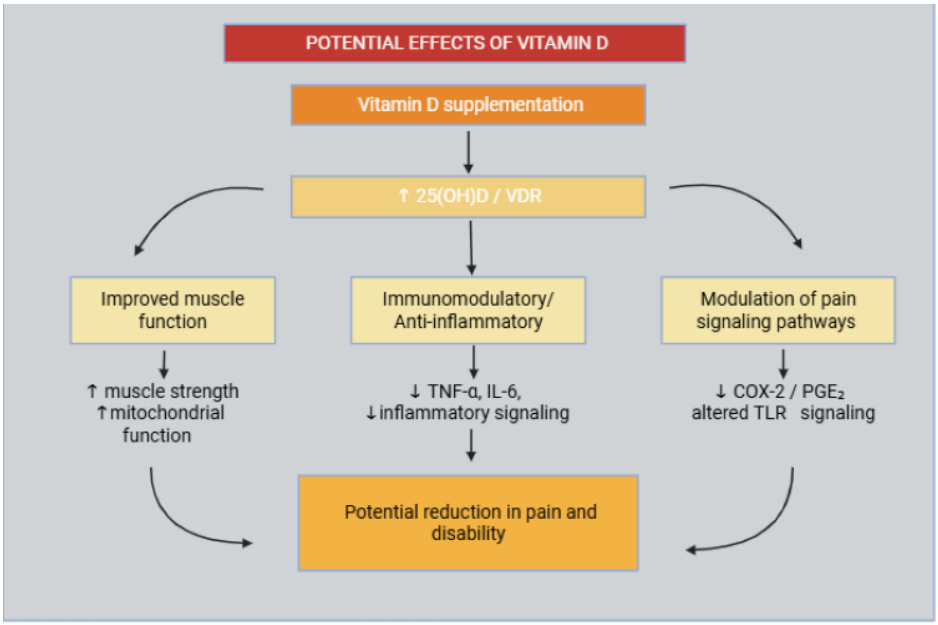


Figure 2. Potential effects of vitamin D supplementation.

Vitamin D supplementation may increase circulating 25(OH)D concentrations and activate vitamin D receptor (VDR)-dependent signaling. These effects may contribute to improved skeletal muscle function and mitochondrial activity, as well as immunomodulatory and anti-inflammatory responses. Vitamin D may also modulate inflammatory and nociceptive pathways by influencing cytokine production, Toll-like receptor (TLR) signaling, and prostaglandin pathways, including cyclooxygenase-2 (COX-2) and prostaglandin E2 (PGE_2). Collectively, these mechanisms may reduce peripheral nociceptor sensitization and neuroinflammation, thereby improving pain intensity and physical function.

Characteristics of a Patient with Obesity

The pathophysiology of chronic musculoskeletal pain in overweight or obese individuals is multifactorial and possibly involves complex interactions among chronic low-grade inflammation, adiposity, mechanical overload, metabolic disturbances and altered nociceptive processing (1, 6, 40). Excess body weight imposes increased mechanical stress on weight-bearing joints and the spine, producing biomechanical alterations that may contribute to the development and persistence of musculoskeletal pain (1, 11). However, mechanical overload alone does not fully explain the increased prevalence of chronic pain among individuals with obesity. Adipose tissue functions as an active endocrine organ that secretes numerous pro-inflammatory mediators contributing to chronic low-grade systemic inflammation (11). Adiposopathy, characterized by dysfunctional expansion of adipose tissue, is associated with adipocyte hypertrophy, impaired tissue perfusion, local hypoxia, macrophage infiltration, and increased production of inflammatory biomarkers, including CRP, IL-6, TNF-α, and leptin (1, 40). These inflammatory mediators can sensitize peripheral nociceptors, lower their activation threshold, and promote persistent nociceptive

signaling (1, 36). Consequently, sustained inflammation may therefore contribute to peripheral sensitization and impair normal tissue repair (1, 36). Clinical studies have demonstrated elevated circulating TNF-α concentrations in individuals with obesity and chronic widespread pain, whereas increased CRP levels have been associated with low back pain in this population (11, 40). Chronic low back pain may also be associated with alterations in brain circuits involved in reward processing and food-related hedonic responses, including the ventral striatum and medial prefrontal regions, potentially contributing to the complex relationship between chronic pain and obesity (41). Conversely, weight reduction has been associated with improvements in pain intensity and physical function in obese individuals and chronic pain (11).

Taken together, chronic musculoskeletal pain in obesity cannot be attributed solely to increased mechanical overload. Rather, it appears to arise from a complex interaction among chronic systemic inflammation, adipose tissue dysfunction, altered nociceptive processing, biomechanical stress, and metabolic abnormalities, all of which may contribute to the initiation and maintenance of chronic pain.

Vitamin D Supplementation

Despite its well-established physiological functions, vitamin D deficiency remains a major global public health concern. Insufficient sunlight exposure associated with modern lifestyles is considered an important contributor to inadequate vitamin D status. At the same time, dietary sources alone may be inadequate to meet vitamin D requirements in many individuals (12). Epidemiological evidence suggests an association between lower serum 25(OH)D concentrations and chronic musculoskeletal pain, although the strength and consistency of this association vary across studies (14). Lower serum 25(OH)D concentrations have been associated with greater pain intensity in some adult populations with CMP (14).

Effect of Supplementation in the General Population

Several clinical studies, systematic reviews and meta-analyses suggest that vitamin D supplementation may provide greater benefit in individuals with low baseline 25(OH)D concentrations, although the magnitude of these effects varies across populations and studies (30, 38, 42–46). Some studies have reported that vitamin D supplementation is associated with reductions in chronic pain intensity, with greater effects reported among individuals with low baseline 25(OH)D concentrations (30, 38, 47–49). Other studies have reported improvements in pain intensity among individuals with chronic widespread pain and fibromyalgia following vitamin D supplementation, although substantial heterogeneity exists among studies (30, 47, 50). In addition, some studies have reported improvements in fibromyalgia-related functional status and quality of life following vitamin D supplementation (43, 47).

However, other studies have demonstrated little or no benefit of vitamin D supplementation in patients with chronic non-specific musculoskeletal pain (51–53) or chronic low back pain (44, 45, 54, 55). These inconsistent findings likely reflect substantial heterogeneity among studies, including differences in study design, patient populations, baseline vitamin D status, sunlight exposure, supplementation regimens, treatment duration, and the use of appropriate control groups.

Vitamin D Supplementation in Patients with Obesity

The main clinical studies investigating vitamin D supplementation on musculoskeletal pain, including studies conducted specifically in overweight or obese, are summarized in Table 1. Evidence regarding vitamin D supplementation in overweight or obese individuals with CMP remains limited, with few randomized controlled trials directly addressing this population. One randomized trial evaluating vitamin D supplementation in obese individuals with low back pain showed no significant overall effect.

Table 1. Main clinical studies investigating vitamin D supplementation and musculoskeletal pain

Study	Study Design	Study Population	Vitamin D Dose & Regimen	Treatment Duration	Principal Findings
Brady et al., 2019	Randomized, placebo-controlled trial	65 overweight/obese adults (BMI ≥25 kg/m^2) with vitamin D deficiency (25(OH)D ≤50 nmol/L); 54 completed the study	100,000 IU oral loading dose of vitamin D_3, followed by 4,000 IU/day vs placebo	16 weeks	Vitamin D significantly increased serum 25(OH)D compared with placebo. Overall, back pain intensity and disability did not differ significantly between groups. However, in participants with severe vitamin D deficiency (25(OH)D <30 nmol/L), vitamin D supplementation significantly reduced back pain disability compared with placebo
Abdeen et al., 2021	Single-blind randomized controlled clinical trial	45 obese women, aged 30–40 years, with vitamin D deficiency and nonspecific myalgia; BMI 30.0–34.9 kg/m^2. Participants were randomized into 3 groups (15/group): vitamin D + aerobic exercise, vitamin D alone, or aerobic exercise alone.	50,000 IU/week vitamin D_3 in vitamin-D groups; vitamin D + aerobic exercise vs vitamin D alone vs exercise alone	12 weeks	Vitamin D + aerobic interval training produced the greatest improvement in pain intensity, serum vitamin D levels and quality of life compared with vitamin D or exercise alone. Functional capacity improved significantly with the combined intervention and exercise alone, but not with vitamin D alone. The authors concluded that combining vitamin D supplementation with aerobic exercise was more effective for managing myalgia in obese women
Gendelman et al., 2015	Randomized, double-blind, placebo-controlled trial	80 patients with musculoskeletal pain receiving analgesic treatment	4,000 IU/day vitamin D_3 vs placebo	12 weeks	Vitamin D supplementation resulted in a greater decline in VAS pain scores and reduced the need for rescue analgesics compared with placebo. TNF-α and PGE_2 also decreased in the vitamin D group.
Ghai et al., 2017	Open-label, single-arm clinical trial	68 patients with chronic low back pain ≥3 months, pain ≥50 on VAS, and 25(OH)D <30 ng/mL	60,000 IU/week vitamin D_3	8 weeks	Vitamin D supplementation was associated with improvement in pain intensity, functional disability and vitamin D status. The study did not include a placebo/control group
Goyal, Agrawal, 2021	Pre-post clinical intervention study	65 enrolled; 50 completed; adults aged 30–60 years with hypovitaminosis D and chronic nonspecific musculoskeletal pain	Vitamin D + calcium	3 months	Mean pain score decreased from 6.22 to 3.52, while serum 25(OH)D increased from 17.38 to 39.40 ng/mL. Physical activity increased and BMI and waist-to-hip ratio decreased
Ersoy et al., 2024	Prospective cross-sectional clinical study	180 women with fibromyalgia, aged 18–70 years; 77.8% had vitamin D levels requiring replacement	50,000 IU vitamin D_3	12 weeks	VAS pain scores decreased from 7.69 to 5.14 and FIQ scores from 67.46 to 53.65 after supplementation. The study was uncontrolled and therefore cannot establish a causal treatment effect

However, among participants with severe serum vitamin D deficiency (25(OH)D below 30 nmol/L), vitamin D supplementation was associated with a significantly greater reduction in back pain disability than placebo (56). Potential mechanisms may include vitamin D anti-inflammatory effects, modulation of nociceptive pathways, and effects on musculoskeletal and bone health. A second randomized controlled trial investigated the combined effects of vitamin D supplementation and aerobic exercise in obese women with myalgia. Participants receiving both interventions experienced significant reductions in pain intensity, improved quality of life, and enhanced functional capacity from baseline, as assessed with the VAS, among others (57). Furthermore, women receiving vitamin D supplementation achieved greater pain reduction and better pain-related outcomes than those doing aerobic exercise alone, although vitamin D supplementation alone did not improve functional capacity.

Although the available randomized evidence is insufficient to support firm clinical recommendations, current findings suggest that patients with documented hypovitaminosis D may benefit from vitamin D supplementation. However, the available studies have relatively small sample sizes and heterogeneous supplementation protocols, and results should therefore be interpreted with caution. Although myalgia associated with vitamin D deficiency is not synonymous with CMP, these studies suggest that vitamin D supplementation may warrant consideration in overweight or obese individuals with documented hypovitaminosis D and chronic pain, particularly with severe vitamin D deficiency, although further studies are needed. In contrast, vitamin D supplementation has not consistently reduced low back pain in the general population (44, 54).

Although the potential analgesic effects of vitamin D in obesity are generally attributed to its anti-inflammatory properties, the available evidence remains inconclusive. Meta-analyses in otherwise healthy overweight or obese individuals have not consistently shown reductions in circulating CRP, IL-6, or TNF-α following vitamin D supplementation (58). In contrast, a study in individuals with musculoskeletal pain reported decreases in pain-related inflammatory mediators, including PGE_2 and TNF-α, following vitamin D supplementation (59). These findings may reflect differences in study populations, baseline vitamin D status, supplementation protocols, and treatment duration. Furthermore, evidence suggests that overweight or obese individuals may require longer supplementation periods to achieve serum vitamin D concentrations comparable to those observed in individuals with normal body weight (60). In one observational analysis, 25(OH)D levels had reached a steady state by approximately six months in normal-weight adults. Still, they continued to increase between six and twelve months in overweight and obese participants (60). Reductions in abdominal adiposity have also been associated with decreased systemic inflammatory markers in patients with chronic pain (1). Individuals with obesity are also more likely to have sedentary lifestyles and lower levels of physical activity. CMP in obese patients may further limit their ability to engage in regular physical activity. Notably, regular physical activity may reduce systemic inflammation, even without significant weight loss (1). Consequently, sustainable lifestyle interventions that combine regular physical activity with correction of hypovitaminosis D may present a reasonable approach to reduce CMP. Still, the contribution of vitamin D supplementation to pain reduction remains uncertain.

Overweight or obese individuals constitute a population in whom chronic low-grade inflammation, altered vitamin D metabolism, and increased susceptibility to CMP coexist. Consequently, this group may benefit from supplementation to correct vitamin D deficiency. However, it remains unclear whether vitamin D potential analgesic effects are driven primarily by anti-inflammatory mechanisms, improved muscle function, modulation of nociceptive pathways, or a combination of these mechanisms. The relative contribution of these pathways may vary according to the degree of obesity, the severity of vitamin D deficiency, and the presence of comorbidities.

Limitations of the Current Evidence

The available evidence is limited by the relatively small number of studies examining the relationship between CMP and vitamin D supplementation in overweight or obese individuals. Because only a limited number of literature databases were searched, we cannot rule out the possibility that not all relevant studies were identified and included in the review. Existing studies vary considerably in supplementation dose, treatment duration, baseline vitamin D status, study populations, and outcome measures, making direct comparisons difficult. Although vitamin D supplementation has shown inconsistent effects on inflammatory biomarkers, some studies have reported reductions in pain intensity, particularly in populations with documented vitamin D deficiency and when combined with other interventions such as regular physical activity and weight reduction. Consequently, the current evidence does not support vitamin D supplementation as a universal stand-alone treatment for CMP. Nevertheless, correcting vitamin D deficiency in obese individuals may represent a useful adjunctive therapeutic strategy that offers modest clinical benefit as part of a multimodal treatment approach. Further high-quality randomized controlled trials with larger cohorts of overweight or obese individuals are needed to clarify the mechanisms by which vitamin D influences chronic pain, determine the optimal supplementation regimens and treatment duration, and establish evidence-based recommendations regarding the intensity and frequency of physical activity. Such studies may facilitate the development of more effective, individualized therapeutic strategies for chronic musculoskeletal pain in overweight or obese. Alongside clinical outcomes, these investigations should include assessment of laboratory inflammatory biomarkers, immunohistochemical analyses, and evaluation of brain, adipose, and muscle tissues. In addition, research using cellular and animal models could provide valuable insights into the molecular and pathophysiological mechanisms linking vitamin D, obesity, and musculoskeletal pain.

Conclusion

Chronic musculoskeletal pain represents a major public health challenge, particularly among overweight or obese individuals. Obesity is also associated with an increased risk of vitamin D deficiency, suggesting a potential interplay between these conditions. Although relatively few studies have specifically examined vitamin D supplementation in overweight or obese individuals with chronic musculoskeletal pain, the available evidence remains heterogeneous. Some findings suggest that individuals with documented vitamin D deficiency may benefit from correcting hypovitaminosis D, particularly for selected pain-related outcomes; however, studies have not consistently demonstrated these effects.

Therefore, vitamin D supplementation should not be considered a universal analgesic treatment for CMP. Assessing and correcting documented vitamin D deficiency may be considered as part of a multimodal management strategy that includes physical activity, weight reduction, and other nonpharmacological and pharmacological interventions. However, the current evidence does not support routine vitamin D supplementation solely for analgesic purposes. Further well-designed randomized controlled trials in overweight and obese individuals with chronic musculoskeletal pain are needed to determine whether vitamin D supplementation provides clinically meaningful benefits.

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Vitamin D kao dodatak prehrani pretilih bolesnika i kronična mišićno-koštana bol: pregledni rad

Sažetak

Kronična mišićno-koštana bol čest je i složen klinički problem, osobito izražen u osoba s pretilošću. Osim povećanog mehaničkog opterećenja, njezinu nastanku pridonose kronična sistemska upala, disfunkcija masnog tkiva, metabolički poremećaji i promjene u središnjoj obradi boli. Pretilost je istodobno povezana s povećanim rizikom za razvoj manjka vitamina D.

Cilj ovog narativnog pregleda bio je prikazati dosadašnje spoznaje o povezanosti manjka vitamina D, njegove suplementacije i kronične mišićno-koštane boli u pretilih osoba. Provedena je pretraga literature u bazama PubMed/MEDLINE i Web of Science (WoS), pri čemu su uključene kliničke studije, sustavni pregledi, meta-analize i relevantni pregledni radovi.

Dosadašnji dokazi upućuju da bi suplementacija vitamina D mogla smanjiti intenzitet boli, osobito kod osoba s dokumentiranom hipovitaminozom D, dok su rezultati u općoj populaciji nedosljedni. U pretilih osoba broj dostupnih istraživanja još je malen, no postojeće randomizirane studije pokazuju povoljan učinak na intenzitet boli, osobito u kombinaciji s umjerenom tjelesnom aktivnošću. Mehanizam djelovanja vjerojatno uključuje imunomodulacijske i protuupalne učinke vitamina D, poboljšanje funkcije mišićno-koštanog sustava te modulaciju nociceptivnih putova, iako još nije u potpunosti razjašnjen. Trenutačni dokazi ne podupiru rutinsku primjenu vitamina D isključivo u svrhu liječenja kronične mišićno-koštane boli, ali opravdavaju prepoznavanje i korekciju hipovitaminoze D kao dijela multimodalnog liječenja pretilih osoba.

Ključne riječi: kronična bol; manjak vitamina D; mišićno-koštana bol; pretilost; vitamin D