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The role of curcumin in the management of pain and inflammation across clinical conditions. A narrative review

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ABSTRACT

Background: Chronic pain frequently coexists with low-grade inflammation, and long-term use of NSAIDs and opioids is limited by adverse effects. Curcumin, a primary bioactive polyphenol derived from turmeric *(Curcuma longa)*, modulates inflammatory and oxidative pathways.

Aim: To synthesize current clinical evidence on the effects of curcumin on pain, functional

outcomes, and inflammatory markers across selected conditions.
Material and Methods: This narrative review was based on PubMed searches combining terms related to curcumin/turmeric with pain, inflammation, and specific diseases. Open Access randomized controlled trials, systematic reviews, and meta-analyses were prioritized; mechanistic studies were included to contextualize clinical findings. No formal risk-of-bias assessment was performed.
Results: The most consistent evidence concerns osteoarthritis and rheumatoid arthritis (RA), where pain reduction and improvements in functional and inflammatory parameters have been reported. In exercise-induced muscle damage (EIMD), curcumin more consistently reduces delayed-onset muscle soreness (DOMS) and creatine kinase (CK) than systemic cytokine responses. In metabolic disorders, effects on inflammatory markers (e.g., CRP) and insulin resistance appear modest. In non-alcoholic fatty liver disease (NAFLD), benefits do not clearly exceed those of lifestyle modification. Variability likely reflects differences in dose, formulation, bioavailability and duration.
Conclusions: Curcumin appears safe and may serve as an adjunct in the management of inflammatory pain. Well-designed long-term trials are needed to define optimal formulations and target populations.
Key words: curcumin; pain; inflammation; osteoarthritis; exercise-induced muscle damage; metabolic disorders

1. INTRODUCTION

Pain is defined as an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage. The updated definition by the International Association for the Study of Pain (IASP) emphasizes the multidimensional nature of pain, encompassing both biological and psychosocial components [1, 2].

Chronic pain represents a significant public health concern. A substantial proportion of the adult population is estimated to experience persistent pain, which is associated with reduced quality of life, impaired physical functioning, and increased healthcare utilization. Epidemiological reviews indicate that chronic pain is among the leading causes of medical consultations and work disability [3]. Growing evidence suggests that inflammatory processes play a key role in the pathophysiology of many forms of chronic pain. In inflammatory pain and pain associated with chronic diseases, the involvement of pro-inflammatory cytokines, such as interleukin-1β

(IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α), as well as the activation of signaling pathways related to the inflammatory response, has been described. These mechanisms may contribute to peripheral and central sensitization and the persistence of pain symptoms [4]. Furthermore, chronic low-grade inflammation is increasingly recognized as a common underlying mechanism in numerous chronic diseases, including metabolic, degenerative, and autoimmune disorders. Sustained immune activation and the production of inflammatory mediators may contribute to disease development and progression [5].

The management of pain and inflammation relies primarily on pharmacotherapy, including nonsteroidal anti-inflammatory drugs (NSAIDs), glucocorticoids, and opioids [6]. Although NSAIDs are well established in pain reduction, their use is associated with cardiovascular and gastrointestinal complications [7, 8]. Long-term opioid therapy carries substantial risks, including endocrine disturbances (such as hypogonadism), immunological, cardiovascular, respiratory, gastrointestinal, and neurological adverse effects, as well as an increased risk of fractures, infections, sleep-disordered breathing, overdose, and mortality [9]. These limitations have stimulated interest in adjunctive therapies with a more favorable safety profile.

In this context, increasing attention has been directed toward nutraceuticals and plant-derived compounds with potential anti-inflammatory and antioxidant properties [10]. One of the most extensively studied compounds in this group is curcumin, the principal polyphenol derived from the rhizome of *Curcuma longa*. The literature describes its pleiotropic biological activity, including modulation of inflammatory mediators and signaling pathways involved in the inflammatory response [11].

Therefore, a comprehensive synthesis of current evidence regarding the role of curcumin in pain relief and modulation of inflammatory processes across different clinical conditions appears warranted.

2. RESEARCH MATERIALS AND METHODS

This narrative review aims to synthesize current evidence on the role of curcumin in pain relief and the modulation of inflammatory processes across various clinical conditions. The analysis focused on clinically relevant endpoints, including pain intensity, functional outcomes, inflammatory markers, tissue damage parameters, and supplementation safety.

The literature search was conducted using the PubMed database and included articles published up to February 2026. Search terms combined keywords related to the intervention (curcumin, turmeric), clinical outcomes (pain, inflammation, CRP, IL-6, TNF-α), and selected disease entities (including osteoarthritis, rheumatoid arthritis, exercise-induced muscle damage, metabolic syndrome, and NAFLD). Preference was given to Open Access publications and the most recent studies, particularly randomized controlled trials (RCTs), systematic reviews, and meta-analyses.

The selection process was qualitative and focused on studies of the highest clinical relevance. Within each thematic area, publications were chosen to reflect the clinical application of curcumin in the respective condition. Mechanistic studies were included only when they supported the interpretation of clinical findings.

No formal risk-of-bias assessment was performed, and no procedures consistent with PRISMA guidelines were applied, in accordance with the narrative nature of the review. The findings were synthesized descriptively and organized according to the main clinical domains addressed in the study.

3. RESEARCH RESULTS

3.1. Curcumin in osteoarthritis and degenerative joint disease

Knee osteoarthritis (KOA) is described as a chronic degenerative joint disease characterized by progressive cartilage degradation, subchondral bone remodeling, and synovial inflammation, leading to pain, stiffness, and functional impairment. An analysis of systematic reviews and meta-analyses emphasized that KOA is not solely a mechanical process but also involves an inflammatory component contributing to clinical symptoms and structural progression [12]. Inflammatory mediators such as interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), and interleukin-6 (IL-6), as well as activation of signaling pathways including NF-κB and MAPK, promote increased expression of extracellular matrix–degrading enzymes, such as matrix metalloproteinases (MMPs), and induction of COX-2, thereby contributing to cartilage degradation [13].

A systematic review published in *BMJ Open Sport & Exercise Medicine* highlighted the relevance of inflammatory mechanisms in KOA pathophysiology and identified them as potential therapeutic targets, including plant-derived supplements. Within this context, curcumin has been described as a compound with anti-inflammatory properties, providing a rationale for its clinical evaluation in patients with KOA [14].

In a randomized, double-blind, placebo-controlled trial, the effects of a standardized curcumin extract (Curcugen®) on KOA symptoms were assessed over 8 weeks. The primary outcome was the pain subscale of the KOOS (Knee Injury and Osteoarthritis Outcome Score), along with a numeric pain rating scale (0–10). Additional assessments included the Japanese Orthopaedic Association (JOA) score and functional tests (timed up-and-go test, 6-minute walk test, 30-second chair stand test, and 40-meter fast-paced walk test). Statistically significant improvements were observed in the KOOS pain subscale and numeric pain scores in the curcumin group compared with placebo. Improvements were also reported in the total JOA score and selected functional measures (timed up-and-go test and 6-minute walk test). Supplementation was well tolerated, with no serious adverse events reported and a comparable incidence of adverse events between groups [15].

A randomized study comparing curcumin with diclofenac in patients with KOA evaluated analgesic efficacy over 28 days. Both groups demonstrated pain reduction; however, gastrointestinal adverse effects were more frequently reported in the diclofenac group. The authors noted that the short duration of the intervention limited conclusions regarding long-term efficacy and safety [16].

Similar findings were reported in a study comparing *Curcuma domestica* extract (containing curcuminoids) with ibuprofen in KOA treatment. Over a 4-week intervention, comparable improvements in pain and function were observed using standardized clinical instruments, particularly the WOMAC index (pain, stiffness, and physical function) and the 6-minute walk test as primary endpoints. The authors concluded that the turmeric extract demonstrated efficacy comparable to ibuprofen in short-term observation [17].

A systematic review of randomized trials concluded that supplementation with turmeric or curcumin preparations may be associated with pain reduction and functional improvement in patients with KOA compared with placebo. However, substantial heterogeneity in formulations, dosages, and intervention duration was noted, limiting direct comparisons and the formulation of definitive clinical recommendations [14].

3.2. Curcumin in rheumatoid arthritis and autoimmune inflammatory disorders

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disease characterized by synovial inflammation leading to cartilage and bone destruction and progressive loss of joint function. Histopathological findings include synovial hyperplasia (pannus formation), inflammatory cell infiltration, and production of inflammatory mediators that sustain disease activity [18, 19].

A central role in RA pathogenesis is attributed to the transcription factor NF-κB, which regulates the expression of TNF-α, IL-1β, IL-6, IL-8, and MCP-1, thereby promoting leukocyte recruitment and amplification of the inflammatory response [20, 21]. In addition, rheumatoid arthritis synovial fibroblasts (RASF) demonstrate increased expression of cyclooxygenase-2 (COX-2) and enhanced prostaglandin E2 (PGE2) production, contributing to inflammation and joint tissue destruction [22]. The JAK/STAT pathway, particularly STAT3, is also activated and maintains IL-6–dependent signaling and synovial cell proliferation [23]. MAPK kinases (p38, ERK, JNK) further regulate cytokine and matrix metalloproteinase production, accelerating tissue degradation [24]. Disturbances in apoptosis and persistent epigenetic alterations, including DNA methylation and histone modifications, contribute to stabilization of the pro-inflammatory synovial phenotype [25]. This complex signaling network constitutes the molecular basis of chronic inflammation and joint damage in RA.

Curcumin, the principal polyphenol of *Curcuma longa*, has demonstrated the ability in experimental models to inhibit activation of NF-κB and STAT3, thereby modulating the expression of TNF-α, IL-1β, IL-6, IL-8, and MCP-1—key mediators of chronic inflammation. It also reduces COX-2 activity and prostaglandin production. Additional effects include modulation of MAPK and JAK signaling pathways, regulation of apoptosis-related proteins, and influence on epigenetic mechanisms, including DNA methyltransferases and histone deacetylases [11, 26]. These pleiotropic actions suggest potential relevance as an adjunctive intervention in RA.

A meta-analysis of 10 randomized controlled trials (n = 539) demonstrated that curcumin supplementation was associated with significant improvements in both laboratory and clinical parameters. Reductions were observed in inflammatory markers, including erythrocyte sedimentation rate (ESR) and CRP, as well as in disease activity measured by DAS28. Significant decreases in tender joint count (TJC), swollen joint count (SJC), and pain intensity assessed using the VAS were also reported [27]. These findings indicate potential effects on both systemic inflammation and joint-specific disease activity.

A more recent 2026 meta-analysis of six placebo-controlled trials (n = 244) confirmed therapeutic effects of curcumin in RA. Improvements were reported in ACR20 response rates, DAS28, ESR, CRP, TJC, SJC, and rheumatoid factor (RF) levels. Subgroup analyses suggested that efficacy may depend on dosage and formulation, with doses above 250 mg/day and certain formulations (e.g., hydrogenated curcuminoids) demonstrating stronger effects. However, the quality of evidence for several outcomes was rated as low or very low due to small sample sizes, heterogeneity, and short follow-up periods [28]. Across analyzed studies, curcumin was generally well tolerated, with adverse events reported infrequently and typically mild [27, 28].

Current clinical evidence suggests that curcumin may represent a safe adjunct to standard RA therapy, potentially contributing to reductions in inflammatory activity and pain.

In a systematic review of 31 randomized controlled trials evaluating curcumin in autoimmune diseases, the most consistent clinical effects were observed in psoriasis and ulcerative colitis [29]. In psoriasis, adjunctive curcumin therapy increased the proportion of patients achieving PASI50, PASI75, and PASI90 responses according to the Psoriasis Area and Severity Index (PASI) compared with control groups. In ulcerative colitis, supplementation was associated with reductions in clinical disease activity indices, higher rates of clinical and endoscopic remission, and decreases in inflammatory markers such as ESR and CRP. No significant increase in adverse events was reported in these conditions. Nevertheless, the authors emphasized the need for further high-quality trials [29].

Positive signals have also been reported in isolated studies of Takayasu arteritis, ankylosing spondylitis, and Behçet’s disease, with improvements in disease activity indices and inflammatory parameters. However, due to the limited number of RCTs, no comprehensive meta-analyses were performed. In systemic lupus erythematosus, multiple sclerosis, and Crohn’s disease, available data remain inconsistent or insufficient to draw definitive conclusions regarding efficacy [29].

3.3. Curcumin in exercise-induced muscle pain and sports-related inflammation

Exercise-induced muscle damage (EIMD) represents a physiological response to intense or unaccustomed physical activity, particularly when eccentric contractions predominate.

Microtrauma to muscle fibers is associated with activation of the inflammatory response, immune cell infiltration, and increased levels of muscle damage markers such as creatine kinase (CK). Clinically, this process manifests as delayed-onset muscle soreness (DOMS), reduced range of motion (ROM), and transient decreases in maximal voluntary contraction (MVC) [30-32].

The biological rationale for curcumin use in post-exercise recovery is based on its ability to modulate oxidative stress and inflammatory pathways. Review articles describe mechanisms including reduction of NOX2 activity and reactive oxygen species (ROS) production, activation of the Nrf2 pathway, and inhibition of NF-κB signaling and pro-inflammatory cytokine expression [33]. These mechanisms support the hypothesis that curcumin may attenuate secondary inflammatory damage following exercise and potentially support recovery. DOMS and CK are the most frequently reported endpoints in studies investigating curcumin in EIMD, whereas cytokine responses (IL-6, TNF-α, CRP) appear more variable and dependent on exercise protocol and supplementation strategy [30]. It has been suggested that the effectiveness of curcumin may vary according to training status, exercise load, and the type of activity performed (eccentric vs. endurance), which complicates direct comparisons between studies [33].

In a randomized, double-blind study, curcumin supplementation was associated with reduced DOMS symptoms and a smaller increase in CK levels compared with placebo. Regarding IL-6, concentrations increased immediately post-exercise and at 48 hours, whereas a 20% decrease was observed at 24 hours relative to immediate post-exercise values. These findings do not confirm a consistent systemic anti-inflammatory effect [34].

More recent studies using enhanced-bioavailability formulations have also reported improvements in functional outcomes. Micellar and nanodispersed preparations (e.g., Theracurmin®) demonstrate improved absorption and may be particularly relevant during the acute 0–24-hour post-exercise window [33]. Supplementation with Theracurmin® has been associated with attenuation of CK elevation and partial recovery of muscle function, including faster restoration of MVC and ROM, particularly when administered after exercise [35]. In professional soccer players, curcumin supplementation was associated with reduced DOMS and smaller post-match declines in muscle performance [36].

A meta-analysis of 14 randomized controlled trials (n = 349) reported that curcumin supplementation significantly reduced markers of muscle damage and post-exercise soreness. Pooled analyses demonstrated significant reductions in CK and IL-6 levels, suggesting potential anti-inflammatory and protective effects against exercise-induced muscle damage, along with decreased subjective soreness. However, substantial heterogeneity was noted, reflecting differences in dosage (150 mg to 4 g/day), timing of administration (pre-exercise, post-exercise, or during recovery), duration of supplementation (single dose vs. ≥1 week), and training status of participants. Subgroup analyses indicated greater reductions in soreness at 96 hours post-exercise, while CK reduction appeared more pronounced in untrained individuals and in groups receiving doses below 0.5 g. Due to heterogeneity and limited data in certain subgroups, conclusions regarding optimal dosing and supplementation protocols should be interpreted cautiously [37].

The effects of curcumin on systemic inflammatory markers remain less consistent. Some trials reported reductions in pro-inflammatory cytokines such as IL-6 and TNF-α, suggesting a modulatory effect on post-exercise inflammation. In contrast, other studies did not demonstrate significant differences compared with placebo, with some findings limited to non-significant trends. Variability may be attributable to differences in dosage, formulation bioavailability, timing of supplementation (pre- vs. post-exercise), training status, and exercise protocols. Meta-analyses suggest a tendency toward IL-6 reduction; however, high heterogeneity limits definitive interpretation [30, 37]. Anti-inflammatory effects may be more apparent at the local muscle level than in systemic measurements, and variability may also reflect differences in formulation bioavailability [33].

In studies evaluating oxidative stress, reductions in lipid peroxidation markers (e.g., MDA) and improvements in total antioxidant capacity have been reported in certain athletic populations, although these effects were not consistently observed across all protocols [30, 33].

3.4. Curcumin in metabolic disorders associated with chronic low-grade inflammation

Chronic low-grade inflammation is recognized as a key mechanism linking obesity, metabolic syndrome, type 2 diabetes mellitus (T2DM), and non-alcoholic fatty liver disease (NAFLD/MAFLD). Unlike acute inflammation, this process is subclinical and persistent, characterized by sustained but moderate elevations of inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α) [38, 39].

Visceral adipose tissue functions as an active immunological and endocrine organ and plays a central role in the pathogenesis of metabolic disorders. In obesity, adipocyte hypertrophy, endoplasmic reticulum stress, and increased production of reactive oxygen species (ROS) lead to activation of pro-inflammatory signaling pathways, including NF-κB and JNK. These disturbances result in increased secretion of free fatty acids (FFAs) and pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6, accompanied by reduced levels of the anti-inflammatory adipokine adiponectin. Concurrently, recruitment of M1 macrophages amplifies both local and systemic inflammation [39]. Activation of the NLRP3 inflammasome further enhances inflammatory signaling through increased IL-1β production, contributing to the development of insulin resistance and T2DM [40, 41]. Persistent activation of NF-κB, JNK, and NLRP3 pathways impairs insulin signaling via abnormal phosphorylation of insulin receptor substrate (IRS) proteins and dysfunction of the insulin receptor, representing a key mechanism in T2DM pathogenesis. Additionally, oxidative stress and lipotoxicity induced by excess FFAs exacerbate inflammatory responses in adipocytes and hepatocytes, promoting NAFLD progression and worsening insulin resistance [39, 40, 42]. Thus, dysfunctional adipose tissue appears to be a central link between obesity, T2DM, and NAFLD through mechanisms involving chronic inflammation and impaired insulin signaling.

In the context of obesity-associated chronic inflammation, curcumin has been shown to inhibit NF-κB activation through suppression of IKK activity, stabilization of IκBα, and reduced nuclear translocation of the p65 subunit, resulting in decreased expression of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. It also attenuates MAPK pathway activation (ERK, JNK, p38), which is involved in inflammatory signaling and insulin resistance. Furthermore, curcumin inhibits NLRP3 inflammasome activation, reducing IL-1β production and limiting persistence of inflammation in adipose tissue. Activation of the Nrf2/ARE pathway represents another important mechanism, enhancing antioxidant enzyme expression and reducing oxidative stress [40]. Experimental studies have also suggested that curcumin may alleviate endoplasmic reticulum stress in adipocytes and improve insulin sensitivity [39]. These findings indicate a pleiotropic effect on inflammatory and metabolic pathways.

A review of meta-analyses of randomized controlled trials reported that curcumin supplementation in populations with metabolic disorders (including metabolic syndrome, T2DM, NAFLD, and obesity) was associated with significant reductions in CRP, IL-6, and TNF-α, as well as improvements in HOMA-IR in some studies. However, the magnitude of effect was generally modest, and heterogeneity related to dosage, formulation, and duration of intervention limited the strength of clinical recommendations [38].

In metabolic syndrome, curcumin supplementation has been associated with reductions in inflammatory markers and improvements in metabolic parameters, including fasting glucose and lipid profile, along with decreases in oxidative stress indices. Improvements in lipid profile were primarily related to increases in HDL-C, whereas triglyceride levels did not show consistent significant changes [43].

In a randomized placebo-controlled trial in patients with NAFLD, curcumin supplementation was associated with decreased NF-κB activity and reductions in liver enzymes (ALT, AST) and TNF-α compared with baseline values. However, similar improvements were observed in the placebo group undergoing lifestyle modification, and between-group differences did not reach statistical significance. These findings suggest that curcumin does not demonstrate clear superiority over lifestyle modification alone, and its clinical relevance requires further investigation [44].

Overall, heterogeneity across studies and variability in dosage and formulation limit definitive therapeutic recommendations. Current evidence supports considering curcumin as a potential adjunct in metabolic disorders associated with chronic low-grade inflammation, pending confirmation in well-designed, long-term clinical trials [38, 43].

4. DISCUSSION

The present analysis indicates that curcumin demonstrates anti-inflammatory and analgesic potential across various clinical conditions. However, the strength and consistency of the observed effects vary depending on the disease entity, studied population, and supplementation protocol.

The most consistent clinical evidence concerns knee osteoarthritis (KOA) and rheumatoid arthritis (RA). In KOA, reductions in pain and improvements in functional outcomes have been reported in randomized trials as well as systematic reviews [14-17]. In RA, meta-analyses indicate improvements in clinical parameters (e.g., DAS28, VAS, tender and swollen joint counts) along with reductions in inflammatory markers [27, 28]. Nevertheless, for several outcomes, the quality of evidence has been rated as low to moderate, and sample sizes are often limited, warranting cautious interpretation.

In the context of exercise-induced muscle damage (EIMD), the most consistently observed effects include reductions in delayed-onset muscle soreness (DOMS) and attenuation of creatine kinase (CK) elevation [34, 37]. Effects on systemic inflammatory markers (IL-6, TNF-α, CRP) appear less consistent and are influenced by exercise protocol, timing of supplementation, and formulation characteristics [30, 33]. Available data suggest that clinical outcomes, such as perceived soreness and functional recovery, may demonstrate greater consistency than changes in circulating biomarkers.

In metabolic disorders associated with chronic low-grade inflammation, curcumin may lead to modest reductions in inflammatory markers and improvements in insulin resistance [38, 43]. However, in NAFLD, improvements in metabolic parameters have been reported to be comparable to those achieved with lifestyle modification, without clear superiority of supplementation [44]. This suggests that, in this context, curcumin should be regarded as a potential adjunct rather than a primary therapeutic strategy.

A common feature across the analyzed conditions is the involvement of inflammatory pathways, including NF-κB activation, overproduction of pro-inflammatory cytokines, and oxidative stress [13, 20, 26, 40]. The biological plausibility of curcumin's effects—stemming from its pleiotropic modulation of these pathways—aligns with the observed clinical findings. However, concordance between molecular mechanisms and clinical outcomes is not always reflected in uniform results across trials, particularly in short-term interventions.

A major limitation in interpretation remains the heterogeneity of available studies, including variability in dosage (ranging from several hundred milligrams to several grams per day), duration of supplementation, formulation bioavailability, and characteristics of study populations [30, 37, 38]. Moreover, an umbrella review of curcumin meta-analyses indicates that although the overall direction of effect across many conditions appears favorable, the quality of evidence is frequently rated as moderate or low, and risk of bias is sometimes considerable [45]. In practice, this underscores the need to avoid overgeneralization and overinterpretation.

From a safety perspective, curcumin was generally well tolerated in the analyzed trials, and adverse events were typically mild [15, 27, 28]. Considering the known limitations of conventional anti-inflammatory and analgesic pharmacotherapy [7-9], this safety profile may support the role of curcumin as an adjunctive option, particularly in populations requiring long-term symptomatic management. Nevertheless, variability in product quality and the potential for drug interactions should be taken into account [10].

In summary, curcumin appears most promising as supportive therapy in inflammatory joint diseases and selected contexts of exercise-related pain. In metabolic disorders, its effects are generally modest and context-dependent. Future research should focus on standardization of dosage and formulations, longer follow-up periods, and the assessment of clinically meaningful endpoints.

5. CONCLUSIONS

Curcumin demonstrates potential anti-inflammatory and analgesic effects across various clinical conditions. However, the strength of evidence remains heterogeneous. The most consistent findings concern pain reduction and functional improvement in knee osteoarthritis, as well as improvements in clinical and inflammatory parameters in rheumatoid arthritis [14-17, 27, 28]. In exercise-induced muscle damage, supplementation may reduce muscle soreness and partially attenuate markers of muscle damage, whereas effects on systemic inflammatory markers remain less conclusive [34, 37]. In metabolic disorders associated with chronic low-grade inflammation, modest improvements in selected inflammatory and metabolic parameters have been observed. However, there is no clear evidence of superiority over standard lifestyle modification [38, 43, 44].

Current evidence supports considering curcumin as a potential adjunctive therapy rather than an alternative to standard treatment. Well-designed, long-term clinical trials are needed to determine optimal dosing strategies, formulations, and patient populations in which clinical benefits are most reproducible.

Disclosure:

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