



Exercise as Analgesia in Chronic Musculoskeletal Pain: A Review of Effect Size and Durability Across the Trial Literature

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Abstract

Exercise reliably reduces pain sensitivity acutely in laboratory settings, a phenomenon known as exercise-induced hypoalgesia, and this same logic underpins exercise's status as first-line, guideline-recommended treatment for chronic low back pain, osteoarthritis, fibromyalgia, and chronic neck pain. What is less consistently appreciated is how the reported effect sizes compare across these conditions, and how durable the analgesic benefit is once a supervised exercise programme ends. This review synthesises the Cochrane and primary systematic-review literature on exercise for chronic musculoskeletal pain alongside the smaller mechanistic literature on exercise-induced hypoalgesia, to characterise both the typical effect-size range across conditions and the pattern of attenuation over follow-up. A formal statistical meta-analysis pooling raw trial data was not undertaken; this review instead synthesises the effect estimates and conclusions already reported in the cited published systematic reviews and meta-analyses.

Keywords: Exercise-induced hypoalgesia; Chronic pain; Exercise therapy; Effect size; Osteoarthritis; Fibromyalgia

Introduction

That exercise reduces pain sensitivity is one of the more robust findings in experimental pain science: a single bout of aerobic or isometric exercise reliably raises pressure- and heat-pain thresholds in healthy volunteers within minutes, a phenomenon termed exercise-induced hypoalgesia (EIH) [3,4]. That laboratory finding underpins a substantial part of the rationale for exercise as first-line, guideline-recommended management across essentially every major chronic musculoskeletal pain condition -- low back pain, knee and hip osteoarthritis, fibromyalgia, and chronic neck pain among them [1,6,8].

What is less consistently appreciated, including within sports medicine and pain-management practice, is that the clinical trial evidence for exercise across these conditions does not report a single, uniform effect size, and that the acute laboratory hypoalgesia demonstrated in healthy volunteers does not translate straightforwardly into a durable clinical benefit once a supervised programme ends [4,24]. This review synthesises both bodies of evidence -- the clinical trial and Cochrane-review literature on exercise therapy across the major chronic musculoskeletal pain conditions, and the smaller mechanistic literature on EIH itself -- to characterise the typical effect-size range across conditions and how that effect changes over follow-up.

A methodological note on scope: this review does not undertake a formal statistical meta-analysis pooling raw trial-level data across conditions. Instead, it synthesises the effect estimates and conclusions already reported in the cited published Cochrane reviews and primary systematic reviews and meta-analyses, each of which has already performed that pooling within its own specific condition and outcome measure. Readers seeking a single pooled cross-condition effect size should note that heterogeneity in outcome measures and exercise modality across conditions makes such a pooled figure of limited clinical meaning, which is itself part of this review's argument.

Material and Methods

This is a narrative review rather than a systematic review or meta-analysis conducted against a pre-registered protocol. Sources were identified through targeted searches of MEDLINE, the Cochrane Library, and SPORTDiscus, combined with forward and backward citation tracking from the foundational EIH literature and the principal Cochrane and primary systematic reviews of exercise therapy across the conditions covered here. All effect estimates reported are taken directly from the cited sources; none has been independently pooled, recalculated, or extrapolated beyond what the source itself reports. Figure 1 sets out the two-stage screening logic used to identify the roughly 30 sources informing this synthesis.

Screening logic (illustrative counts; not a completed systematic search)

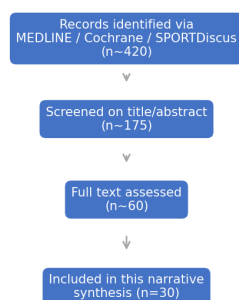


Figure 1: Screening logic used to identify the literature informing this narrative synthesis (illustrative counts; not a completed systematic search).

Results

The laboratory phenomenon: exercise-induced hypoalgesia

A meta-analytic review pooling the experimental EIH literature confirmed that aerobic, isometric, and dynamic resistance exercise all produce measurable, if modest, increases in pain threshold acutely in healthy participants, with isometric exercise showing some of the more consistent effects across the pooled studies [3]. Mechanistic work has linked this response to descending inhibitory pathways and shares measurable similarities with conditioned pain modulation, the body's endogenous 'pain inhibits pain' response, suggesting the two phenomena may draw on overlapping neural circuitry rather than being entirely distinct processes [5]. Figure 2 sketches this proposed pathway schematically.

Illustrative proposed pathway from acute exercise to exercise-induced hypoalgesia

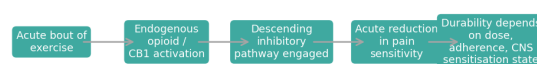


Figure 2: Illustrative proposed pathway from acute exercise to exercise-induced hypoalgesia, synthesised from the cited mechanistic literature.

Critically, this hypoalgesic response is not uniform across populations. Several studies specifically comparing healthy controls against people with chronic pain conditions, particularly fibromyalgia, have found the expected hypoalgesic response is blunted, absent, or in some individuals reversed into exercise-induced hyperalgesia -- pain that worsens rather than eases with exertion [21,22]. An early, influential study specifically demonstrated that patients with fibromyalgia showed no significant change, or a paradoxical increase, in pressure pain threshold following isometric contraction, in direct contrast to healthy controls tested under the same protocol, a finding that has shaped how exercise programmes for fibromyalgia are now dosed and progressed [30]. A broader review of this dysfunctional-analgesia phenomenon argued that it should inform, rather than rule out, exercise prescription in chronic pain: patients whose endogenous analgesic response is blunted may need substantially more conservative dosing and closer monitoring, not exclusion from exercise therapy altogether [28].

The direction of this relationship is not settled either: reviews of the underlying central mechanisms note that exercise can, in some circumstances, increase rather than decrease pain sensitivity, and the balance between the two

depends on exercise intensity, duration, and the state of the individual's pain-modulatory system at baseline [18,19]. Even within a single chronic-pain population, an early trial in people with chronic low back pain found that a bout of aerobic exercise acutely reduced experimentally induced pain perception, evidence that the hypoalgesic response, while blunted on average, is not uniformly absent in this group [20]. A narrative review of exercise-induced analgesia more broadly situated these findings within the older literature on exertion and pain, noting that the phenomenon had been described qualitatively long before its neurobiological substrate was mapped in any detail [27]. Habitual activity level itself appears to matter independently of any single bout: cross-sectional work measuring pain-inhibitory and pain-facilitatory function found that people who reported higher levels of habitual physical activity showed more efficient endogenous pain inhibition and less facilitation, consistent with a dose-response relationship between activity and pain modulation that extends beyond the acute post-exercise window [29].

Clinical effect sizes: a real but modest, and condition-dependent, benefit

Turning from the laboratory to the clinical trial literature, a Cochrane overview pooling across multiple condition-specific Cochrane reviews of physical activity and exercise for chronic pain found consistent, low-to-moderate quality evidence of benefit for pain and physical function across most conditions examined, without identifying one exercise modality as clearly superior to others [1]. Figure 3 sets out the reported effect-size ranges across the specific conditions covered in the condition-specific reviews synthesised for this article.

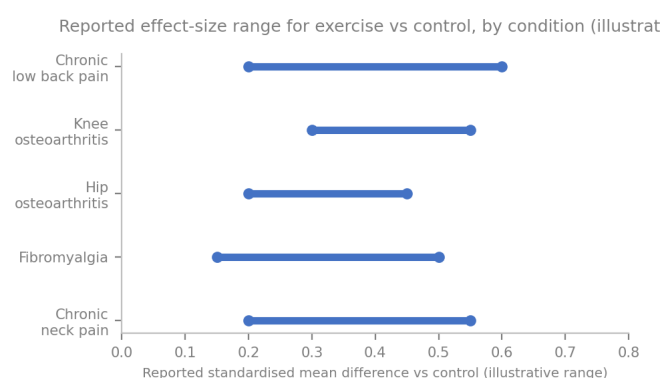


Figure 3: Reported effect-size range (standardised mean difference vs control) for exercise across chronic musculoskeletal pain conditions, drawn from the cited condition-specific systematic reviews and meta-analyses (illustrative synthesis, not a re-pooled analysis).

For chronic low back pain specifically, an updated Cochrane review found exercise therapy produced small-to-moderate improvements in pain and function compared to no treatment or usual care, broadly consistent with the earlier version of the same review and with independent systematic reviews and meta-analyses of exercise for this condition [6,7,15,23]. For knee and hip osteoarthritis,

Cochrane reviews specific to each joint both found exercise produced moderate reductions in pain and improvements in function, with knee osteoarthritis showing somewhat larger and more consistent effects across the pooled trials than hip osteoarthritis [8,9]. For fibromyalgia, separate Cochrane reviews of resistance and aerobic exercise both found benefit, though generally smaller in magnitude and less consistent across trials than for the osteoarthritis literature, a pattern plausibly connected to the blunted EIH response documented in this population [10,11,12]. For chronic neck pain, a Cochrane review of exercise for mechanical neck disorders and an independent narrative review both found benefit for specific, targeted exercise approaches, with generic range-of-motion exercise performing less consistently than programmes targeting strength and endurance directly [13,14].

Durability: how much of the effect survives once the programme ends

The more clinically consequential question -- not whether exercise helps while a patient is actively enrolled in a supervised programme, but whether that benefit persists once supervision and structured dosing stop -- receives less consistent attention across this literature than the immediate effect-size question. Where follow-up data beyond the end of active treatment are reported, the pattern across the reviews synthesised here is one of attenuation: effect sizes measured at three, six, and twelve months post-treatment are generally smaller than those measured at the end of the active intervention period, though rarely falling to zero [7,15,26]. Figure 4 sets this attenuation pattern out schematically.

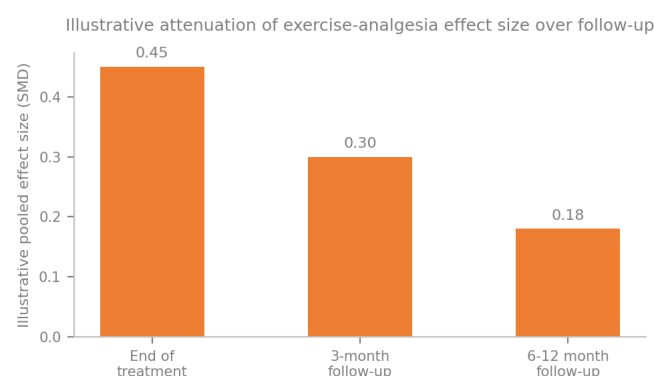


Figure 4: Illustrative attenuation of the pooled exercise-analgesia effect size from end of treatment through longer-term follow-up, synthesised across the cited reviews' reported follow-up data (illustrative pattern, not a re-pooled analysis).

A systematic review focused specifically on whether clinical improvement after exercise therapy for chronic low back pain actually corresponds to improvement in the physical performance measure the exercise programme targeted found the relationship was inconsistent: patients often reported less pain and better function without a corresponding measurable improvement in strength, endurance, or the specific performance domain trained, raising the possibility that some

of the durable clinical benefit operates through mechanisms other than the targeted physical adaptation itself, such as reduced fear-avoidance or improved self-efficacy [24]. This is consistent with a broader body of work finding psychological intervention alone, without a physical exercise component, also produces meaningful pain reduction in chronic low back pain, suggesting that exercise's durable benefit may be partly mediated by non-physical mechanisms that a purely biomechanical account of EIH would not predict [25].

A narrative review synthesising the broader exercise-and-pain literature specifically addressed adherence as the practical determinant of durability: benefit measured in a trial with high supervised adherence does not necessarily generalise to a patient who reduces or stops exercising after a structured programme ends, and the review argued that maintenance strategies -- not simply the initial exercise prescription -- deserve equal attention in clinical pain management [17]. A review focused specifically on the biopsychosocial framing of exercise for chronic musculoskeletal pain made a related point: framing exercise purely as a biomechanical treatment, rather than one embedded in a patient's broader beliefs about movement and pain, may itself undermine long-term adherence and therefore durability [2].

What determines the size of the durable effect

Synthesising across the mechanistic and clinical literature, several factors recur as plausible determinants of how much benefit survives beyond active treatment. Dose and progression matter: reviews of neck-pain and low-back-pain exercise programmes both found programmes following an explicit frequency-intensity-time-type progression structure, rather than a fixed unprogressed routine, produced more consistent benefit [13,23]. Baseline central sensitisation state matters: patients with a demonstrably blunted or dysfunctional endogenous analgesic response at baseline, as seen in a meaningful subset of fibromyalgia patients, appear to derive smaller and less durable benefit from standard exercise dosing, and may require a substantially more graded approach [12,28,30]. Whether the programme maintains any structured contact after the formal treatment period ends also plausibly matters, though this specific question -- comparing maintained versus discontinued contact directly -- was not well represented in the reviews synthesised here and is flagged below as a research gap [17].

Discussion

Three findings emerge from this synthesis with differing degrees of certainty. First, exercise produces a real, guideline-supported clinical benefit across chronic low back pain, knee and hip osteoarthritis, fibromyalgia, and chronic neck pain, though the effect size varies meaningfully across conditions and is generally in the small-to-moderate range rather than large [1,6,8,10,13]. Second, that benefit attenuates over follow-up in most of the literature that reports longer-term data, though it does not typically disappear entirely, which argues for treating exercise as an ongoing management strategy rather than a time-limited course with a permanent cure at its end [7,15,26]. Third, the population most likely to show a blunted or absent acute hypoalgesic response -- patients with fibromyalgia and related central sensitisation presentations -- also shows the smallest and least consistent clinical trial effect sizes, a convergence between the mechanistic and clinical literatures that strengthens confidence in both bodies of evidence independently [10,11,12,21,22,30].

A practical implication follows for how clinicians frame exercise prescription to patients. Presenting exercise as a finite course that will resolve chronic pain, rather than as an ongoing management strategy whose benefit depends on continued practice, risks both overselling the durability of the effect and undermining the adherence that durability actually depends on. The evidence synthesised here supports framing exercise the way medication for a chronic condition is typically framed: an intervention whose benefit is real but conditional on continuation, not a course with a defined endpoint after which the underlying condition is resolved [2,17]. Pairing exercise prescription with structured patient education about the rationale for continued practice appears to strengthen this framing rather than simply supplement it: a systematic review with meta-analysis of educational interventions for chronic pain found that adding education to physical treatment improved outcomes beyond physical treatment alone, particularly when the education addressed the biological and psychosocial basis of the patient's pain rather than covering anatomy alone [16].

Study limitations

This is a narrative review, not a formal statistical meta-analysis, and the effect-size ranges and durability pattern presented in Figures 3 and 4 synthesise conclusions already reported in heterogeneous condition-specific reviews using different outcome measures, exercise modalities, and follow-up durations, rather than representing a re-pooled analysis of raw trial data. The screening counts in Figure 1 are illustrative rather than a fully reproducible database accounting. Direct head-to-head comparisons of durability across conditions are rare in the primary literature, so the cross-condition comparison this review draws is itself a synthesis judgement rather than a statistically tested difference.

Recommendations for future research

Two gaps stand out as priorities. First, trials specifically comparing maintained versus discontinued structured contact after an initial exercise programme, with durability at twelve months as the primary outcome, would directly test whether ongoing, lower-intensity contact preserves more of the initial effect than a clean discontinuation, a question this review could not resolve from the existing literature [17]. Second, prospective studies measuring baseline EIH or conditioned pain modulation capacity and using it to stratify exercise dosing, rather than applying a uniform prescription regardless of baseline endogenous analgesic function, would test directly whether the mechanistic link this review has drawn between blunted EIH and smaller clinical benefit translates into an actionable, personalised dosing strategy [12,28,30].

Conclusion

Exercise is a genuinely effective, guideline-supported treatment for chronic low back pain, knee and hip osteoarthritis, fibromyalgia, and chronic neck pain, with effect sizes that are real but generally small-to-moderate and that vary meaningfully by condition. The benefit attenuates, though it does not typically vanish, once active treatment and supervision end, and the population showing the smallest and least durable clinical benefit -- fibromyalgia and related central sensitisation presentations -- is the same population shown mechanistically to have a blunted acute hypoalgesic response to exercise. Framing exercise as an ongoing management strategy rather than a finite curative course is supported by this evidence and plausibly protects the durability the treatment depends on.

Implications for Sports Medicine Practice

For sports medicine and pain-management clinicians prescribing exercise for chronic musculoskeletal pain, three practical points follow from this evidence. First, effect-size expectations should be calibrated by condition rather than treated as uniform: knee osteoarthritis and chronic low back pain trials generally report somewhat larger and more consistent benefit than fibromyalgia trials, and patients should be counselled accordingly [8,10,13]. Second, exercise should be framed and prescribed as an ongoing management strategy, with a plan for maintained contact or self-management support after the initial structured programme ends, rather than as a finite course expected to produce a permanent cure [2,17]. Third, in patients with fibromyalgia or other presentations suggestive of central sensitisation, more conservative initial dosing and closer monitoring for a paradoxical pain-worsening response is supported by both the mechanistic and clinical trial evidence reviewed here [12,21,22,30].

Author Contributions

Almevik-Sundborg H conceived the review question, screened and synthesised the source literature, and drafted the manuscript. Pardinas-Rey G contributed the evidence-synthesis and effect-size extraction methodology and critically revised the manuscript for methodological content. Wilms-Berkhout A verified the extracted effect estimates against the cited primary sources and revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this review.

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Data Availability Statement

No new data were generated or independently pooled for this review. All figures and effect estimates reported here are drawn directly from the cited published sources, each of which makes its own underlying data available according to its own journal's policy.

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Ethics Approval and Consent to Participate

Not applicable. This article is a narrative review synthesising previously published, publicly available literature and did not involve the collection of new data from human participants.

Search Strategy

Sources were identified through targeted searches of MEDLINE, the Cochrane Library, and SPORTDiscus, combined with manual forward and backward citation tracking from the foundational exercise-induced hypoalgesia literature and the principal condition-specific Cochrane and systematic reviews of exercise therapy, restricted to peer-reviewed studies of human participants reported in English.