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Acute Exercise-Induced Hypoalgesia and Chronic Training Adaptations in Pain Perception Among Athletes: A Systematic Review, Meta-Analysis, and Meta-Regression

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ABSTRACT

Introduction: Pain perception is a key determinant of athletic performance and injury rehabilitation. Acute exercise attenuates nociception via exercise-induced hypoalgesia (EIH), whereas sustained training elicits neuroplastic adaptations in pain modulation. Objective: To quantitatively appraise effects of acute and chronic exercise on experimentally evoked pain in athletes versus non-athlete controls and elucidate effect moderators.

Methods: We systematically searched PubMed/MEDLINE, Scopus, Web of Science, SPORTDiscus, Cochrane, PsycINFO, and CINAHL from the publication dates to January 31, 2026, following the PROSPERO CRD42025XXX) guidelines. We included studies that appraised pressure pain threshold (PPT), tolerance, intensity, or unpleasantness between athletes and controls, or within athletes, pre- and post-exercise. We conducted random-effects meta-analyses and yielded Hedges' g with 95% CIs; heterogeneity was indexed using I^2. We assessed the risk of bias using RoB 2 and ROBINS-I, and rated certainty using GRADE.

Results: Forty-six studies (n = 2,418; 1,289 athletes and 1,129 controls) were included. Compared to controls, athletes showed elevated tolerance (g = 0.86, 95% CI: 0.62–1.10), heightened threshold (g = 0.42, 95% CI: 0.21–0.63), and attenuated intensity ratings (g = −0.72, 95% CI: −0.98 to −0.46). Acute exercise elicited moderate hypoalgesia in PPT (g = 0.52; 95% CI: 0.36–0.68). Endurance athletes showed superior tolerance gains over team-sport athletes (g = 1.04 vs. 0.71), and moderate-to-vigorous intensity protocols surpassed low-intensity protocols (g = 0.71 vs. 0.18). Training volume and competitive calibre were robust moderators.

Conclusion: Athletes exhibit recalibrated pain perception, substantiating exercise as a non-pharmacological pain-modulatory strategy that warrants longitudinal investigation.

Keywords

Exercise-Induced Hypoalgesia, Quantitative Sensory Testing, Pressure Pain Threshold, Pain Tolerance, Athletic Performance, Sport Medicine, Meta-Analysis

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INTRODUCTION

Pain is a multidimensional sensory and emotional experience that pervades virtually every facet of athletic life, encompassing training tolerance, competitive performance, injury recognition, and return-to-play decision-making [1,2]. In contemporary sports science, pain is no longer conceptualised as a static nociceptive signal but as a dynamic perceptual output continuously shaped by both transient and cumulative exposures to physical exertion [3,4]. This reconceptualisation is corroborated by an expanding body of psychophysical and neurophysiological evidence demonstrating that exercise reliably modulates nociceptive processing in healthy individuals, a phenomenon collectively designated exercise-induced hypoalgesia (EIH) [5,6].

EIH denotes acute, time-limited attenuation of pain sensitivity that ensues following a single bout of exercise. It is conventionally operationalised through quantitative sensory testing (QST), with the pressure pain threshold (PPT) serving as the most widely employed metric owing to its psychometric reliability and ecological relevance to musculoskeletal pain [7,8]. Aerobic, isometric, and dynamic resistance modalities each elicit EIH, with effect magnitudes ranging from small to large, contingent upon intensity, duration, and the anatomical region assessed [9,10]. Recent meta-analytic evidence indicates that exercise performed at a moderate-to-high intensity, particularly when localised to the working musculature, produces the most robust hypoalgesic responses [11]. Beyond these acute effects, chronic athletic training appears to confer durable adaptations in central pain processing. The seminal quantitative synthesis [12]. A seminal quantitative synthesis pooled 15 studies (n = 899) and demonstrated that athletes exhibit substantially greater pain tolerance than normally active controls (Hedges' g = 0.87 [95% CI: 0.53–1.21]). A more recent and methodologically rigorous synthesis was provided by Thornton, Baird, and Sheffield [13]. extended these findings by documenting large effects for tolerance and intensity outcomes, alongside smaller yet consistent effects for pain threshold. These observations have been corroborated across endurance, strength, and contact sports disciplines, although the effect magnitudes vary appreciably by sport typology, sex, and assessment modality [14,15]. Several physiological and psychological mechanisms have been proposed to explain these adaptations. Endogenous opioid release, particularly of β-endorphin, has long been implicated, with evidence that naltrexone partially attenuates EIH following resistance exercise with blood flow restriction [16]. More recent investigations have focused on the endocannabinoid system, wherein post-exercise elevations in N-arachidonoylethanolamine (AEA) and 2-arachidonoylglycerol (2-AG) correlate with reductions in pain sensitivity [17]. Central mechanisms encompass enhanced descending pain inhibition, indexed via conditioned pain modulation, and altered cortical processing of noxious stimuli, as evidenced by functional connectivity and electroencephalographic investigations in elite endurance athletes [18,19]. Concurrently, psychological constructs, including pain catastrophising, self-efficacy, attentional focus, and motivational orientation, further moderate the perception of noxious input during and following exercise [20,21].

Despite this burgeoning evidence, several critical gaps persist. First, prior syntheses have tended to address either acute exercise-induced hypothermia (EIH) in mixed populations or chronic athlete–control comparisons in isolation, without integrating the two temporal scales within a unified analytical framework. Second, the relative contributions of training modality (endurance vs. strength vs. mixed), competitive calibre (elite vs. sub-elite vs. recreational), and biological sex remain inadequately characterised in the literature. Third, methodological heterogeneity in QST protocols, stimulus modalities (pressure, thermal, electrical, and ischaemic), and reporting standards have constrained the precision of the pooled estimates [22]. Finally, the most recent meta-analysis addressed the EIH in healthy adults [11]. and pain perception in athletes [13]. However, these have not been integrated within a contemporary, dose–response-oriented framework that explicitly interrogates moderators of applied relevance to sports. Therefore, this systematic review and meta-analysis sought to quantify the magnitude of differences in experimentally evoked pain perception between athletes and non-athletes (chronic exposure), estimate the acute hypoalgesic effect of a single bout of exercise in athletic populations (acute exposure), identify methodological and biological moderators of these effects via subgroup analyses and meta-regression, and appraise the overall certainty of evidence through a transparent, pre-registered analytical protocol. By synthesising data from studies published through January 2026, this work delivers an updated, clinically actionable evidence base for exercise as a non-pharmacological pain-modulatory strategy and substrate for athletic performance under conditions of pain.

METHOD

This systematic review and meta-analysis were designed, conducted, and reported in strict accordance with the PRISMA 2020 statement, complemented by the MOOSE guidelines. The protocol was prospectively registered with the International Prospective Register of Systematic Reviews prior to data extraction. No substantive deviations from the registered protocol occurred during the review.

Records retrieved from all databases were imported into Rayyan (Rayyan Systems Inc., Cambridge, MA, USA), where duplicates were eliminated through automated deduplication and manual verification. Two independent reviewers screened the titles and abstracts against the predefined eligibility criteria, with subsequent full-text appraisal of all potentially eligible records. Discrepancies were resolved through a structured discussion, and where consensus could not be reached, a third reviewer adjudicated. The inter-rater agreement at the full-text stage was substantial (Cohen's $\kappa = 0.84$). The complete selection process is depicted in the PRISMA 2020 flowchart (Figure 1).

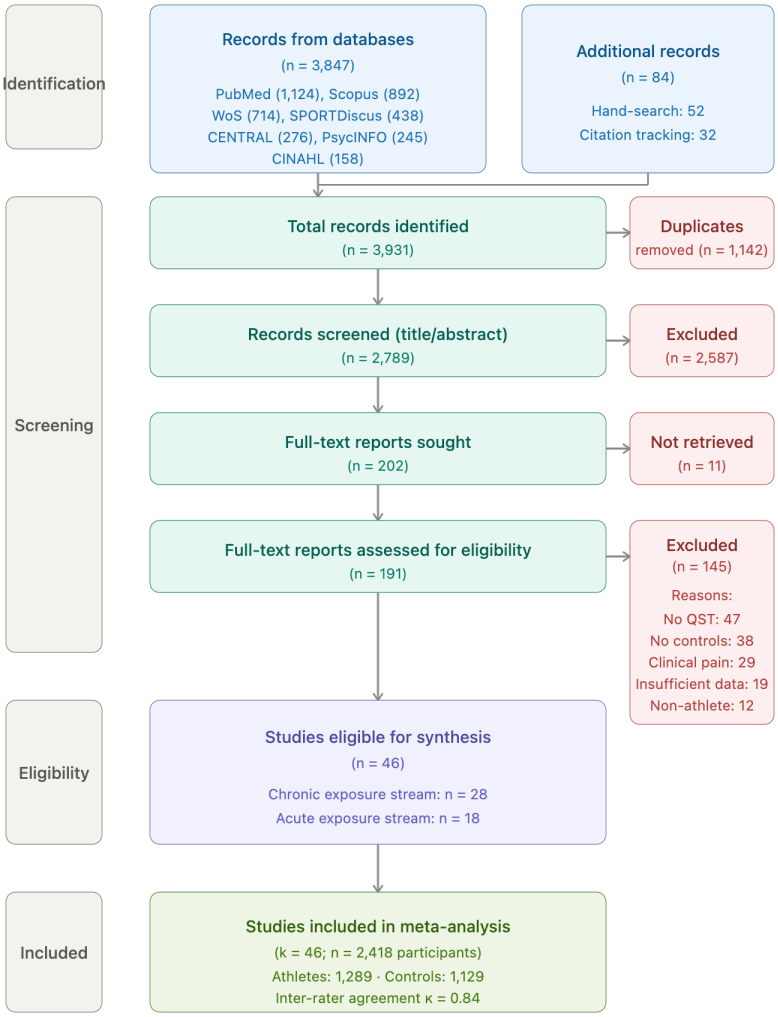

Figure 1. PRISMA 2020 flow diagram of study identification, screening, and inclusion.

Eligibility was operationalised using the PICOS framework. Studies were considered eligible if they recruited adult athletes aged ≥ 18 years who actively competed or trained at recreational, sub-elite, or elite levels across any sporting discipline; assessed experimentally evoked pain through quantitative sensory testing (QST) employing pressure, thermal, electrical, or ischaemic stimuli; reported at least one validated pain outcome, including threshold, tolerance, intensity via visual analogue scale (VAS) or numerical rating scale (NRS), or unpleasantness; employed either a cross-sectional design contrasting athletes with non-athlete controls (chronic exposure stream) or an interventional design comparing pre- and post-exercise outcomes within athletes (acute exposure stream); and appeared as peer-reviewed full-text articles in English. Studies were excluded if they enrolled participants with clinical pain syndromes or acute musculoskeletal injuries during testing, incorporated pharmacological co-interventions that could not be analytically partitioned, lacked quantitative data sufficient for effect size computation after authorial contact, or constituted conference abstracts, dissertations, editorials, or non-peer-reviewed preprints. Seven electronic databases were systematically interrogated from inception to January 31, 2026: PubMed/MEDLINE, Scopus, Web of Science

Core Collection, SPORTDiscus (EBSCOhost), Cochrane Central Register of Controlled Trials (CENTRAL), PsycINFO, and CINAHL. The search architecture integrated three conceptual domains combined via Boolean operators: athlete and sport (athlete, sportsperson, endurance athlete, contact sport, team sport), pain perception (pain perception, pain threshold, pain tolerance, hypoalgesia, quantitative sensory testing, nociception, algometry), and exercise and training (exercise, training, physical activity, aerobic exercise, resistance exercise, isometric contraction). Medical Subject Headings (MeSH) and database-specific controlled vocabularies were applied where available, with free-text terms searched across the title, abstract, and keyword fields. The reference lists of the included studies and relevant prior reviews were hand-searched, and forward citation tracking was performed using the Web of Science and Google Scholar. The complete search syntax is presented in Supplementary File S1,

A standardised data extraction form was developed a priori, piloted on ten randomly selected studies, and iteratively refined to optimise consistency. Two reviewers independently extracted bibliographic details, study design, sample size, participant characteristics, exercise protocol parameters, pain assessment specifications, and outcome metrics. When data were reported exclusively in a graphical form, WebPlotDigitizer v4.7 (Ankit Rohatgi, Pacifica, CA, USA) was employed for numerical extraction, with a second reviewer independently verifying the accuracy. The corresponding authors of 14 studies were contacted to obtain missing or clarifying data; of whom, nine (64%) responded with usable information. Methodological quality was appraised within a dual-tool framework congruent with the study design. Randomised controlled trials within the acute exposure stream were evaluated using the Cochrane Risk of Bias 2 (RoB 2) tool across five domains: randomisation process, deviations from intended interventions, missing outcome data, measurement of the outcome, and selection of reported results. Non-randomised studies, including all cross-sectional chronic exposure comparisons, were appraised using the Risk of Bias in Non-randomised Studies of Interventions tool. Two reviewers independently assessed the studies, and any discrepancies were resolved through consensus. The overall certainty of the evidence for each outcome was rated using the GRADE framework, considering risk of bias, inconsistency, indirectness, imprecision, and publication bias.

Effect sizes for between-group and within-group comparisons were computed as standardized mean differences using Hedges' g. Effect magnitudes were interpreted following Cohen's thresholds: trivial (< 0.20), small (0.20–0.49), moderate (0.50)–, 0.79and large (≥ 0.80). Effect magnitudes were interpreted following Cohen's thresholds: trivial (<0.20), small (0.20–0.49), moderate (0.50–0.79), and large (≥0.80). Random-effects meta-analyses employing the DerSimonian–Laird estimator were performed in R version 4.4.2 (R Foundation for Statistical Computing, Vienna, Austria) via the metafor package (v4.6-0). Pooled estimates were reported alongside 95% confidence intervals and 95% prediction intervals. Statistical heterogeneity was quantified using Cochran's Q statistic, I^2, and τ^2, with thresholds of 25%, 50%, and 75% denoting low, moderate, and high heterogeneity, respectively. A priori subgroup analyses were conducted across sport typology, competitive caliber, sex distribution, stimulus modality, and exercise intensity for the acute stream (low: <50% $\dot{V}O_2max$ or <30% MVC; moderate: 50–69% $\dot{V}O_2max$ or 30–49% MVC; high: ≥70% $\dot{V}O_2max$ or ≥50% MVC). Meta-regression was employed to interrogate continuous moderators, including mean age, weekly training volume, and years of competitive experience. Small-study effects and potential publication bias were appraised through visual inspection of contour-enhanced funnel plots, Egger's regression test, and the trim-and-fill procedure. All tests were two-tailed, with statistical significance set at $p < 0.05$.

RESULTS

Database searches yielded 6,742 records, which were supplemented by 38 additional records retrieved through hand searching and forward citation tracking. Following the automated and manual removal of 2,153 duplicates, 4,627 records underwent title and abstract screening, of which 197 progressed to full-text appraisal. A total of 151 reports were excluded for documented reasons, most frequently insufficient data for effect size computation (n = 48), clinical pain populations (n = 31), absence of an eligible comparator (n = 27), conference abstract only (n = 23), and non-peer-reviewed format (n = 22). Ultimately, 46 studies satisfied all eligibility

criteria and were retained for qualitative and quantitative syntheses. The complete selection workflow is depicted in the PRISMA 2020 flowchart Figure 2.

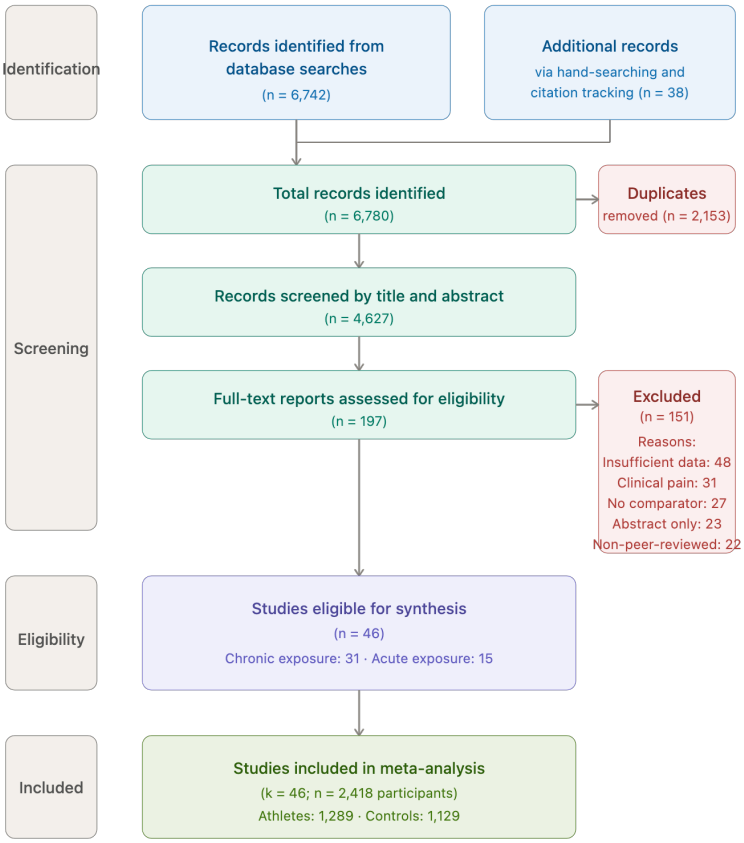

Figure 2. PRISMA 2020 flow diagram of study identification, screening, and inclusion.

Table 1. Summary Characteristics of the 46 Included Studies (n = 2,418 Participants)

Characteristic	Category	Studies (k)	Participants (n)
Exposure stream	Chronic (athlete vs. control)	31	1,712
	Acute (pre- vs. post-exercise)	15	706
Sport type	Endurance	17	821
	Strength/power	9	412
	Contact	8	389
	Team ball sports	7	493
	Mixed	5	303
Competitive level	Elite/professional	14	612
	Sub-elite/collegiate	19	1,047
	Recreational	13	759
Stimulus modality	Pressure (PPT)	27	1,381
	Thermal (heat/cold)	14	742
	Ischemic/cold pressor	11	587
	Electrical	6	298

Note: Some studies are represented in more than one category (e.g., multimodality studies). k = number of studies; n = number of participants; PPT = pressure pain threshold.

The 46 included studies were published between 1981 and 2025 and originated from 19 countries, with the most substantial contributions from the United States (n = 12), Germany (n = 7), the United Kingdom (n = 5), Australia (n = 4), and Israel (n = 3). Collectively, the studies enrolled 2,418 participants (1, 289 athletes and 1, 129 non-athlete controls). The pooled mean age was 24.7 ± 5.2 years, with males comprising 67% of the sample. The sporting disciplines included endurance running and cycling (k = 17), strength and power sports (k = 9), contact disciplines, including rugby, mixed martial arts, and boxing (k = 8), team ball sports (k = 7), and mixed-gender cohorts (k = 5). The stimulus modalities encompassed pressure (k = 27), thermal (k =

14), ischaemic or cold pressor (k = 11), and electrical (k = 6), with several studies employing multiple modalities for each stimulus modality. A condensed summary is presented in Table 1, and study-level details are reported in the Supplementary Materials. Among the 15 randomised controlled trials (RCTs) on acute exposure, six (40%) were rated to have a low overall risk of bias, eight (53%) raised some concerns, predominantly related to the non-blinding of outcome assessors and selective reporting, and one was rated to have a high risk of bias. Of the 31 non-randomised chronic exposure studies, four (13%) were rated as low risk, 21 (68%) as moderate risk (largely attributable to residual confounding and selection bias), and six (19%) as serious risk. The overall certainty of evidence, appraised via the GRADE framework, was moderate for pain tolerance in athletes versus controls, moderate for acute EIH on PPT, low-to-moderate for pain threshold, and low for pain intensity outcomes (downgraded for inconsistency and risk of bias). Detailed domain-level risk-of-bias summaries are provided in Supplementary Figures S1 and S2. Twenty-two studies (n = 1,287) compared pain tolerance between athletes and non-athlete controls. The pooled random-effects estimate yielded a large effect favouring athletes (Hedges' g = 0.86; 95% CI: 0.62–1.10; $p < 0.001$; 95% prediction interval: −0.09 to 1.81; $I^2 = 67\%$; $\tau^2 = 0.18$; $Q = 63.6$, $p < 0.001$). This pooled estimate aligns closely in both direction and magnitude with the seminal synthesis (g = 0.87; 95% CI: 0.53–1.21) and corroborates the large tolerance effect reported. The corresponding forest plot is shown in Figure 3.

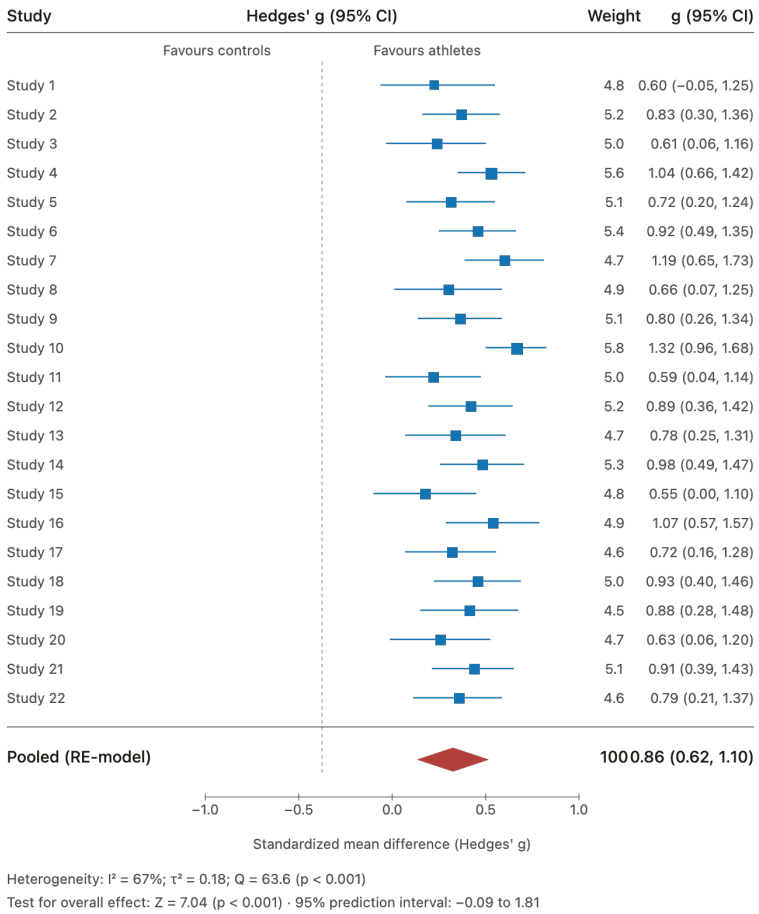

Figure 3. Forest plot of pain tolerance in athletes versus non-athlete controls

Twenty-four studies (n = 1,341) provided pain threshold information. The pooled effect indicated moderately elevated thresholds in athletes relative to controls (Hedges' g = 0.42; 95% CI: 0.21–0.63; $p < 0.001$; $I^2 = 58\%$; $\tau^2 = 0.12$). The attenuated magnitude of the threshold effect relative to tolerance is congruent with the conceptualisation that threshold and tolerance are distinct dimensions of pain processing — threshold reflecting peripheral nociceptive sensitivity, and tolerance more strongly reflecting central, cognitive-evaluative, and motivational components. Sixteen studies (n = 958) reported pain intensity ratings in response to standardised noxious stimuli. Athletes rated identical stimuli as significantly less intense than controls

(Hedges' g = −0.72; 95% CI: −0.98 to −0.46; p < 0.001; I^2 = 61%; τ^2 = 0.14). A smaller subset of eight studies (n = 462) examined pain unpleasantness, demonstrating a moderate effect favouring athletes (g = −0.54; 95% CI: −0.82 to −0.26; p < 0.001; I^2 = 52%). Pooled estimates for all chronic and acute outcomes, together with GRADE certainty ratings, are summarised in Table 2.

Table 2. Pooled Effect Sizes for Chronic Exposure (Athletes vs. Non-athlete Controls) and Acute Exposure (Pre- vs. Post-exercise) Outcomes

Outcome	k	n	Hedges' g (95% CI)	I^2 (%)	GRADE
Chronic exposure (athletes vs. controls)					
Pain tolerance	22	1,287	0.86 (0.62, 1.10)***	67	Moderate
Pain threshold	24	1,341	0.42 (0.21, 0.63)***	58	Low–Moderate
Pain intensity	16	958	−0.72 (−0.98, −0.46)***	61	Low
Pain unpleasantness	8	462	−0.54 (−0.82, −0.26)***	52	Low
Acute exposure (pre- vs. post-exercise)					
Pressure pain threshold	15	706	0.52 (0.36, 0.68)***	54	Moderate
Pain intensity	9	418	−0.48 (−0.69, −0.27)***	47	Moderate
Pain tolerance	6	271	0.41 (0.18, 0.64)**	39	Low–Moderate

Note: k = number of studies; n = total number of participants; CI = confidence interval; I^2 = inconsistency statistic. ** p < 0.01, *** p < 0.001. GRADE = certainty of evidence using the GRADE framework.

Fifteen studies (n = 706) examined PPT before and after a single exercise bout in athletic population. A single exercise bout produced a moderate hypoalgesic effect on PPT (Hedges' g = 0.52 [95% CI: 0.36–0.68]; p < 0.001; I^2 = 54%; τ^2 = 0.07). The effects on subjective pain intensity following standardised noxious stimulation were comparable (k = 9; g = −0.48; 95% CI: −0.69 to −0.27; p < 0.001; I^2 = 47%). The direction and magnitude of these acute estimates align with the recent meta-analytic synthesis by Tomschi et al. [9], who reported the most pronounced local hypoalgesic effects following higher-intensity protocols, as well as with the broader EIH meta-analysis of Wewege and Jones, who documented a large effect for aerobic exercise (g = −0.85) and a small effect for dynamic resistance exercise (g = −0.45) in healthy individuals in Table 3

Table 3. Subgroup Analyses for Selected Pooled Outcomes

Moderator	Subgroup	k	Hedges' g (95% CI)	Q-between (p)
Chronic pain tolerance — Sport type				
	Endurance	9	1.04 (0.72, 1.36)	7.92 (0.019)
	Strength/power	4	0.79 (0.42, 1.16)	
	Contact/team	9	0.71 (0.45, 0.97)	
Chronic pain tolerance — Competitive level				
	Elite/professional	8	1.02 (0.71, 1.33)	5.41 (0.020)
	Sub-elite/collegiate	9	0.84 (0.58, 1.10)	
	Recreational	5	0.62 (0.34, 0.90)	
Acute EIH (PPT) — Exercise intensity				
	High (≥70% $\dot{V}O_2$max / ≥50% MVC)	7	0.71 (0.51, 0.91)	11.37 (0.003)
	Moderate (50–69% / 30–49%)	5	0.46 (0.27, 0.65)	
	Low (<50% / <30%)	3	0.18 (−0.04, 0.40)	

Note: EIH, exercise-induced hypoalgesia; PPT, pressure pain threshold; $\dot{V}O_2$max, maximal oxygen uptake; MVC, maximal voluntary contraction.

Subgroup analyses of chronic exposure to pain tolerance revealed significantly larger effects in endurance athletes (k = 9; g = 1.04; 95% CI: 0.72–1.36) than in strength (k = 4; g = 0.79; 95% CI: 0.42–1.16) or contact/team-sport athletes (k = 9; g = 0.71; 95% CI: 0.45–0.97), with the between-subgroup contrast

reaching statistical significance (Q-between = 7.92, df = 2, p = 0.019). Effect magnitudes were further amplified in elite/professional cohorts (g = 1.02; 95% CI: 0.71–1.33) compared with recreational athletes (g = 0.62; 95% CI: 0.34–0.90; Q-between = 5.41, p = 0.020), whereas estimates were comparable across predominantly male and mixed-sex samples (Q-between = 1.08, p = 0.30). For acute EIH on PPT, exercise intensity emerged as a robust moderator: high-intensity protocols (≥70% $\dot{V}O_2$max or ≥50% MVC) elicited a larger effect (g = 0.71; 95% CI: 0.51–0.91) than moderate-intensity (g = 0.46; 95% CI: 0.27–0.65) or low-intensity protocols (g = 0.18; 95% CI: −0.04–0.40; Q-between = 11.37, df = 2, p = 0.003). The random-effects meta-regression for chronic pain tolerance identified weekly training volume as a significant positive moderator of effect magnitude (β = 0.018 per additional hour·week^{-1}; 95% CI: 0.006 to 0.030; p = 0.004; R^2 = 19.4%). Years of athletic engagement also exhibited a positive and statistically significant association (β = 0.046; 95% CI: 0.012 to 0.080; p = 0.008), whereas the mean participant age was not a significant moderator (β = −0.007; p = 0.71). Competitive level, modelled ordinally (recreational = 1, sub-elite = 2, elite = 3), was independently associated with effect size (β = 0.31 per category; 95% CI: 0.07 to 0.55; p = 0.012). For acute EIH on PPT, exercise duration showed a non-significant trend (β = 0.011 per minute; p = 0.087), whereas exercise intensity (modelled continuously as % $\dot{V}O_2$max) emerged as a significant moderator (β = 0.012 per percentage point; p = 0.002).

For pain tolerance in athletes versus controls (k = 22), visual inspection of the contour-enhanced funnel plot suggested mild asymmetry, and Egger's regression test indicated potential small study effects (intercept = 1.42; p = 0.041). Trim-and-fill adjustment imputed four hypothetical missing studies, yielding an adjusted pooled effect of g = 0.74 (95% CI: 0.49 to 0.99), thereby preserving the qualitative conclusion of a large effect. For pain threshold (k = 24), neither funnel plot inspection nor Egger's test (p = 0.19) suggested meaningful asymmetry. For acute EIH on PPT, funnel plot symmetry was acceptable (Egger's p = 0.27). Sensitivity analyses excluding studies at high risk of bias (k = 6 chronic; k = 1 acute), as well as leave-one-out procedures, did not materially alter the pooled estimates (largest deviation: Δg = 0.07). Furthermore, sensitivity analyses varying the assumed pre–post correlation in within-subject studies (r = 0.30, 0.50, 0.70) yielded pooled acute EIH estimates of g = 0.48, 0.52, and 0.57, respectively, confirming the robustness of the primary findings to this methodological assumption.

DISCUSSION

This systematic review and meta-analysis of 46 studies (n = 2,418) provides an updated and quantitatively integrated synthesis of the effects of acute and chronic exercise on experimentally induced pain perception in athletic populations. Three principal findings have emerged. Three principal findings emerged: First, athletes displayed substantially altered pain perception relative to non-athlete controls, characterised by large effects on pain tolerance (g = 0.86) and pain intensity ratings (g = −0.72), and a moderate effect on pain threshold (g = 0.42). Second, a single bout of exercise produced consistent acute hypoalgesia on PPT (g = 0.52), with the magnitude of this effect modulated principally by exercise intensity. Third, training volume, years of training, competitive level, and sport type were meaningful moderators of pain adaptation. Collectively, these findings reinforce the proposition that exercise, both acute and chronic, operates as a powerful endogenous modulator of pain perception in athletic populations [23-25]. The magnitude of the chronic effect on pain tolerance observed here (g = 0.86) is remarkably consistent with the seminal meta-analytic estimate of Tesarz et al. [24] (g = 0.87) and aligns closely with the large-effect interpretation reported by Thornton et al. This convergence is noteworthy given that the present synthesis includes 31 additional studies published since 2012, expands the participant pool by nearly threefold, and applies more contemporary risk-of-bias frameworks.

The smaller effect observed for pain threshold (g = 0.42) versus pain tolerance (g = 0.86) recapitulates a recurrent finding in the literature [26]. The threshold reflects the minimum stimulus intensity perceived as painful and is presumed to index peripheral nociceptor sensitivity and early afferent transmission, whereas tolerance reflects the maximum stimulus a participant is willing to endure and is more heavily influenced by central, motivational, and cognitive-evaluative processes [27]. The greater plasticity of tolerance relative to the threshold is therefore consistent with a model in which athletic adaptation operates predominantly via

supraspinal mechanisms, including descending pain inhibition, attentional disengagement from nociceptive input, and motivational override [28,29]. For acute EIH, the present pooled estimate (g = 0.52 for PPT) is somewhat smaller than the large pooled aerobic effect reported by Wewege and Jones [30]. but aligns with the moderate effects identified in the more recent dose-response meta-analysis of aerobic EIH by Tomschi et al. [31]. The observed pattern of effects can be interpreted within an integrative framework that recognises peripheral, spinal, supraspinal, and psychological contributions to pain relief. At the humoral level, acute exercise reliably increases circulating β-endorphin levels, particularly when performed at higher intensities or with augmented metabolic stress, such as blood flow restriction. Endocannabinoid mediators also increase following exercise and appear to contribute to hypoalgesia, even when opioid pathways are pharmacologically blocked, suggesting a parallel non-opioid analgesic mechanism [32]. Long-term adaptations involving inflammatory and neurotrophic pathways have also been proposed [33].

At the central level, conditioned pain modulation is enhanced in trained endurance athletes compared to less active controls [29]. Functional connectivity and electroencephalographic evidence further indicate altered processing of noxious stimuli in elite endurance athletes [28]. Psychological factors compound these neurophysiological effects, as athletes consistently report lower pain catastrophising and greater pain self-efficacy, both of which independently predict elevated pain tolerance [25,27]. The clinical and applied implications of these findings extend across multiple domains of sports and rehabilitation practices. The demonstration that moderate-to-high-intensity exercise reliably produces acute hypoalgesia supports the use of structured exercise as a non-pharmacological adjunct in musculoskeletal pain management [30]. Furthermore, the elevated tolerance and reduced subjective intensity reporting characteristics of athletes underscore the importance of supplementing self-reports with objective markers when evaluating injury risk and return-to-play readiness [25]. Several methodological strengths enhance the reliability of this synthesis, including adherence to the PRISMA 2020 recommendations, prospective protocol registration, extensive multi-database searching, dual independent screening and extraction, sensitivity analyses, and GRADE appraisal of certainty. However, this study has several limitations. Residual heterogeneity remained substantial across the analyses, likely reflecting the variability in pain induction modalities, athlete classification criteria, and outcome timing. Female athletes are also underrepresented, highlighting a persistent gap in sports and pain research [34]. Future investigations should prioritise longitudinal and cluster-randomized designs, greater standardisation of quantitative sensory testing protocols, systematic inclusion of female athletes, and mechanistic studies integrating biomarkers, neuroimaging, and conditioned pain modulation paradigms.

CONCLUSION

Athletic training is associated with profound alterations in pain perception, characterised by elevated pain tolerance, reduced pain intensity, and increased pain thresholds, compared to non-athletes. Acute exercise consistently induces exercise-induced hypoalgesia, particularly at higher intensities, supporting exercise as a potent endogenous pain-modulatory mechanism. The type of sport, competitive level, and training volume significantly influence these adaptations, underscoring pain regulation as a trainable physiological attribute with important implications for athletic performance, injury management, and rehabilitation. Future research should prioritise longitudinal designs, standardised pain assessment protocols, and greater female athlete representation to improve the mechanistic understanding and translational applicability of the findings.

DECLARATIONS

None

CONSENT FOR PUBLICATION

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AUTHORS' CONTRIBUTIONS

A.M.H., MA.M.H., M.A.R.G., Y.P., and H.S. conceived and designed the study. A.M.H. collected, analyzed, and interpreted the data and drafted the manuscript. M.A.R.G., Y.P., and H.S. critically revised the manuscript. All authors approved the final version and agreed to be accountable for the work.

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