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High-Intensity Peripheral Magnetic Stimulation for Musculoskeletal Pain

NARRATIVE REVIEW

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Background: High-intensity peripheral magnetic stimulation (PMS), delivered as repetitive peripheral magnetic stimulation (rPMS) or functional magnetic stimulation (FMS), is increasingly used in rehabilitation, but interpretation is limited by unstable terminology, heterogeneous protocols, and frequent conflation with low-intensity electromagnetic field therapies.

Scope: This narrative review synthesizes peer-reviewed orthopedic, rehabilitation, pain, and translational literature on high-intensity PMS for musculoskeletal pain, including randomized trials, meta-analyses, mechanistic studies, feasibility studies, and adjacent evidence. Low-intensity exposure studies, non-musculoskeletal applications, commercial booklets, and non-indexed reports are discussed only to clarify scope, safety, or evidentiary boundaries.

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Findings: The evidence base comprises several meta-analyses and a set of controlled, mechanistic, pilot, and feasibility studies. In low back pain, pooled estimates suggest short-term pain reduction (SMD -1.16, 95% CI -1.56 to -0.76; very-low GRADE certainty; VAS MD -1.89, 95% CI -3.32 to -0.47; very-low-to-low GRADE certainty) and disability gains (ODI MD -6.55 to -8.39; very-low-to-low GRADE certainty), with partial primary-trial overlap between pooled syntheses. Myofascial trials show short-term analgesic signals at low certainty. Mechanistic studies using fNIRS and TMS suggest cortical reweighting beyond peripheral afferent recruitment and segmental modulation at very-low certainty. Pilot evidence in radicular pain and knee osteoarthritis is promising but not definitive, especially when PMS is tested as an adjunct rather than standalone therapy.

Conclusion: High-intensity PMS is a biologically plausible, noninvasive adjunct for selected musculoskeletal pain presentations when paired with rehabilitation that converts transient analgesia into movement and function. Its clinical role depends on adequately powered sham-controlled trials with standardized dose reporting, validated outcomes, durable follow-up, and embedded mechanistic endpoints

Keywords: *Magnetic Field Therapy*, *Musculoskeletal Pain*, *Low Back Pain*, *Myofascial Pain Syndromes*, *Rehabilitation*

Introduction

Musculoskeletal pain is a family of overlapping clinical states rather than a single diagnosis. Nociceptive tissue input, peripheral nerve irritability, nociplastic amplification, altered motor control, fear-driven avoidance, and deconditioning may coexist in the same patient. This complexity matters because interventions attractive at the level of physics or physiology can fail clinically when the target population, dose, comparator, and outcome window are poorly matched. The revised International Association for the Study of Pain definition frames pain as a sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage 1. This is particularly relevant in chronic musculoskeletal disorders, where tissue status and pain intensity frequently diverge.

High-intensity peripheral magnetic stimulation (PMS) sits at the intersection of neuromodulation and rehabilitation. Delivered as repetitive peripheral magnetic stimulation (rPMS), functional magnetic stimulation (FMS), or related high-intensity systems, it can induce electric fields in excitable tissues, depolarize peripheral axons, evoke muscle contraction, and generate proprioceptive input 2,3. The literature remains terminologically unstable: low-intensity pulsed electromagnetic field therapy, transcranial magnetic stimulation, rPMS, FMS, extracorporeal magnetic innervation, and branded high-intensity Tesla stimulation are often discussed under similar language despite different field strengths, targets, tissue effects, and clinical claims 4. A useful review must therefore separate exposure-level electromagnetic therapies from stimulation-level magnetic neuromodulation.

This review addresses high-intensity PMS for musculoskeletal pain as a structured expert synthesis rather than a systematic review, a format appropriate for a theory-rich, heterogeneous field with small trials, mixed comparators, incomplete dose reporting, and uneven mechanistic measurement. The goal is to define what can be concluded, what remains speculative, and what evidence would be required before high-intensity PMS could move from promising adjunct to reproducible component of musculoskeletal pain care.

Scope and search

This narrative review used a structured search and citation-audit process rather than a formal systematic-review protocol and was appraised with the Scale for the Assessment of Narrative Review Articles (SANRA); the completed SANRA self-rating is submitted as a separate file. PubMed/MEDLINE, the Cochrane Library, Google Scholar, publisher full-text pages, DOI registries, and reference lists of relevant reviews were searched from database inception through June 2026. No start-date restriction was applied. Non-English and non-indexed records were retained only when an English full text or verifiable indexed record could be obtained. The most recent citation audit was completed in June 2026, including independent verification by a second member of the author team for the recent indexed citations carrying the greatest interpretive weight. The structured evidence-selection process is summarized in Figure 1; because this is a narrative review, no screened-record deduplication count is implied. Search concepts combined intervention terms ("peripheral magnetic stimulation," "repetitive peripheral magnetic stimulation," "functional magnetic stimulation," "rPMS," "FMS," "high-intensity magnetic stimulation," "Tesla stimulation") with musculoskeletal and pain terms ("musculoskeletal pain," "low back pain," "lumbar radiculopathy," "myofascial pain," "trigger point," "neck pain," "knee osteoarthritis," "fibromyalgia," "neuropathic pain," "rehabilitation"). The evidence hierarchy used for interpretation is described below so that sham-controlled trials, add-on trials, pilot studies, and feasibility reports are not weighted equally.

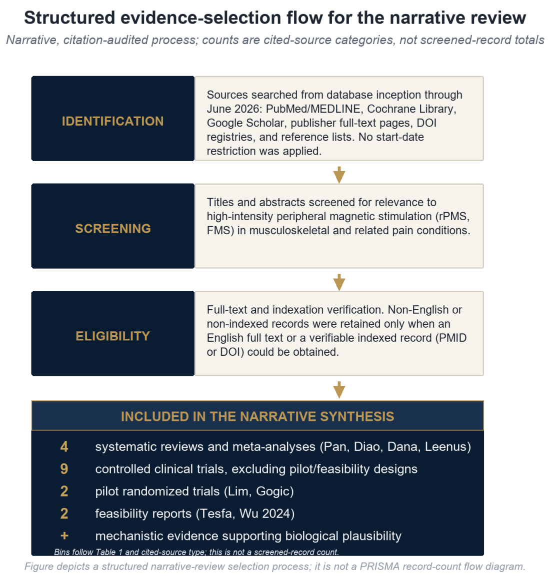

Figure 1. Structured evidence-selection flow for the narrative review. Sources were identified from database inception through June 2026 across PubMed/MEDLINE, the Cochrane Library, Google Scholar, publisher full-text pages, DOI registries, and reference lists; screened for relevance to high-intensity peripheral magnetic stimulation in musculoskeletal and related pain; and verified for full-text availability and indexation. Counts in the included box reflect cited sources by type, not screened-record totals. Controlled clinical trials include non-pilot sham-controlled, active-comparator, and add-on randomized trials listed in Table 1; pilot randomized trials and feasibility reports are counted separately according to study aim/design.

Eligible evidence included randomized trials, controlled clinical studies, meta-analyses, mechanistic human studies, and selected feasibility studies in musculoskeletal or closely adjacent pain populations. Studies were prioritized when they used high-intensity PMS capable of nerve stimulation or muscle contraction, reported clinical outcomes, or included mechanistic endpoints such as functional near-infrared spectroscopy, transcranial magnetic stimulation, ultrasound, electromyography, or validated pain and disability scales. Low-intensity pulsed electromagnetic field studies were used only to clarify the boundary between electromagnetic exposure and peripheral magnetic stimulation. Non-musculoskeletal studies were considered only when they informed safety, neuromuscular feasibility, or device-specific context.

Commercial booklets, end-user surveys, prepublication files, and non-indexed reports were treated as grey literature. They were not used as primary evidence of efficacy. When such sources identified a study or claim, the claim was independently checked against PubMed, DOI records, or full-text publisher sources whenever possible. This review is therefore narrative and expert-driven, not exhaustive; its strength is conceptual integration, while its main methodological limitation is the possibility of selection bias inherent to non-systematic synthesis.

Biological rationale and mechanistic plausibility

The core physical principle of PMS is electromagnetic induction. A rapidly changing magnetic field induces an electric field in tissue; when the induced current reaches threshold near a peripheral nerve, motor point, or intramuscular neural element, axonal depolarization occurs [2,3]. Unlike surface electrical stimulation, where current must cross skin and tolerability is often limiting, magnetic fields can stimulate deeper structures without direct electrical contact. The true biological dose, however, depends not only on field intensity in tesla but also on coil geometry, target depth, orientation, pulse width, train structure, frequency, duty cycle, session duration, and tissue conductivity.

Analgesic plausibility begins with afferent recruitment. High-intensity PMS can activate large-diameter sensorimotor fibers directly and can indirectly activate proprioceptive afferents through evoked muscle contraction [2]. Large-fiber input is compatible with gate-control physiology, in which non-nociceptive afferent traffic can attenuate dorsal horn transmission of nociceptive signals [5], though gate control remains a plausible explanatory model rather than a pathway proven uniquely for every PMS protocol.

Supraspinal mechanisms are increasingly important. Descending modulation through the periaqueductal gray and rostral ventromedial medulla provides a biologically plausible route by which repeated peripheral input could influence pain beyond the stimulated tissue [6]. More directly, mechanistic studies suggest cortical reweighting. Yan et al. used functional near-infrared spectroscopy in chronic non-specific low back pain (n=40) and found rPMS-related clinical improvement alongside changes in motor cortical and supplementary motor area activity [7]. Masse-Alarie et al. showed that multifidus rPMS combined with motor training influenced corticomotor excitability, anticipatory postural adjustments, pain, and disability, with the disability gain retained at one month (n=21) [8]. Wu et al. extended this signal in a two-center fNIRS study of non-specific neck and low back pain, reporting immediate analgesia and reduced pain-evoked dorsolateral prefrontal cortex activation after active rPMS, with the cortical reduction correlating with pain reduction [9].

These studies do not establish a single causal chain, but they make it unlikely that high-intensity PMS is merely a local muscle treatment. A more defensible model is distributed across the neuraxis: peripheral axonal depolarization and proprioceptive afferent input may modulate segmental nociceptive processing, motor network organization, cortical salience processing, and movement-related behavior (Figure 2). The supraspinal brainstem component should be interpreted as an inferential and least directly tested link, whereas the peripheral, segmental, and cortical levels are more directly supported. Evidence from non-pain populations supports feasibility of muscle recruitment but cannot be converted into musculoskeletal analgesic efficacy 10,11.

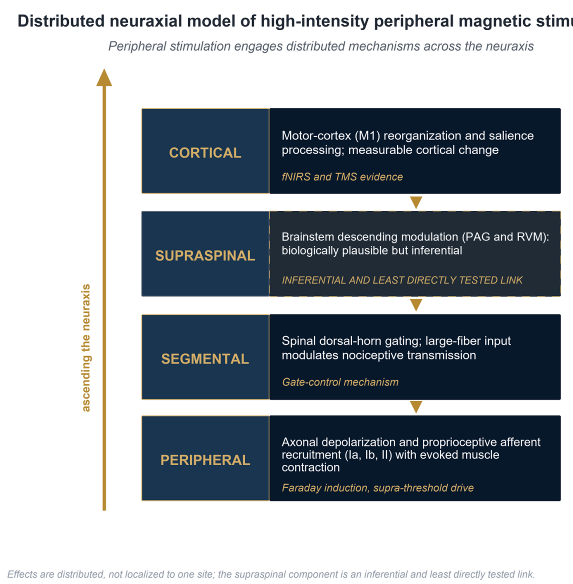

Figure 2. Distributed neuraxial model of high-intensity peripheral magnetic stimulation. Peripheral stimulation engages distributed mechanisms across the neuraxis: peripheral (axonal depolarization and proprioceptive afferent recruitment with evoked muscle contraction), segmental (spinal dorsal-horn gating of nociceptive transmission), supraspinal (brainstem descending modulation through the periaqueductal gray and rostral ventromedial medulla), and cortical (motor-cortex reorganization and salience processing supported by functional near-infrared spectroscopy and transcranial magnetic stimulation). The supraspinal component is marked as an inferential and least directly tested link, consistent with the current level of mechanistic evidence. The model is integrative rather than a single causal chain.

Clinical evidence by indication

Table 1 summarizes indication-level evidence, reporting quantitative findings available from each source. Low back pain is the most developed clinical indication. Pan et al. synthesized eight randomized trials (n=177) across chronic musculoskeletal pain conditions and reported a favorable pooled effect on pain (standardized mean difference -1.16; 95% confidence interval -1.56 to -0.76) and disability (mean difference -6.55; 95% CI -10.27 to -2.82), with very-low GRADE certainty 12. Diao et al. focused on low back pain across six randomized trials (n=139) and found short-term reductions in pain (VAS mean difference -1.89; 95% CI -3.32 to -0.47) and disability (Oswestry Disability Index mean difference -8.39; 95% CI -13.65 to -3.12), at very-low-to-low certainty 13. Because the two syntheses partially share primary low-back trials, these pooled estimates should be read as overlapping signals rather than independent confirmation. The direction of effect is encouraging, but small samples, heterogeneous stimulation parameters, short follow-up, and variable comparator quality make the pooled estimates vulnerable to inflation.

Table 1. Table 1. Evidence summary by indication for high-intensity peripheral magnetic stimulation in musculoskeletal pain

Indication	Study	Design	n	Comparator	Main outcome / direction (quantitative where reported)	Certainty	Follow-up
Chronic musculoskeletal pain	Pan et al. 12	Systematic review / meta-analysis	8 RCTs, n=177	Sham / other therapy	Pain SMD -1.16 (95% CI -1.56 to -0.76); disability MD -6.55 (-10.27 to -2.82); kinesiophobia NS	Very low	Short-term

Indication	Study	Design	n	Comparator	Main outcome / direction (quantitative where reported)	Certainty	Follow-up
Low back pain	Diao et al. 13	Systematic review / meta-analysis	6 RCTs, n=139	Sham / other therapy	VAS MD -1.89 (95% CI -3.32 to -0.47); ODI MD -8.39 (-13.65 to -3.12); kinesiophobia NS	Very low to low	Short-term
Chronic non-specific low back pain	Yan et al. 7	Double-blind sham-controlled mechanistic RCT	40	Sham coil + core training	Clinical improvement with motor/SMA cortical activity changes on fNIRS; left M1 activation inversely correlated with pain reduction (r=-0.537)	Single trial (not GRADE-rated)	Post-intervention (no durable follow-up)
Low back pain / motor control	Masse-Alarie et al. 8	Randomized controlled trial (motor-training paradigm)	21	Sham + motor training	Influenced pain, disability, corticomotor excitability, and anticipatory postural adjustments	Single trial (not GRADE-rated)	1 month (disability gain retained)
Acute low back pain	Lim et al. 14	Pilot RCT	26	Sham rPMS	VAS 63.75 to 27.08; ODI 50.98 to 22.74 over 10 sessions; superior to sham at sessions 5 and 10	Low / pilot	Within treatment course
Lumbar radiculopathy	Lytras et al. 17	Add-on RCT	40	Manual therapy alone	Group x time p<0.01 all outcomes; pain, RMDQ, S-LANSS exceeded MCID; SLR exceeded MDC; only FMS arm S-LANSS <12	Single trial (not GRADE-rated)	3 weeks
Lumbar radiculopathy	Savulescu et al. 15	EMG-guided prospective randomized study	41	rPMS plus physiotherapy vs rPMS alone / physiotherapy context	Pain improved in both arms; EMG functional recovery was superior with rPMS plus physiotherapy	Low / small randomized study	Post-treatment
Chronic back pain	Tesfa et al. 16	Feasibility study	17	Usual care / feasibility context	Feasibility-level signal in veterans with chronic back pain; not designed to establish efficacy	Feasibility-level	Post-treatment
Localized myofascial pain	Pujol et al. 19	Sham-controlled RCT	30	Sham	101-point pain score -59% (real) vs -14% (sham), p=0.001; relief persisted several days	Low	Several days
Trapezius myofascial pain	Smania et al. 2003 20	Sham-controlled trial	18 (9 per arm)	Placebo	Significant VAS, NPDVAS, algometry, ROM, and trigger-point gains persisting at 1 month	Single trial (not GRADE-rated)	1 month
Trapezius myofascial pain	Smania et al. 2005 21	Three-arm RCT	53	TENS; placebo	rMS superior to both TENS and placebo	Single trial (not GRADE-rated)	Short/ medium-term
Neck myofascial pain	Mahisanun and Saengsuwan 22	Sham-controlled crossover RCT	27	Sham	Greatest difference at day 4: VAS -24.1; NDI -8.5 vs sham (single 10-min session, 3,900 pulses, 5-10 Hz)	Single trial (not GRADE-rated)	7 days

Indication	Study	Design	n	Comparator	Main outcome / direction (quantitative where reported)	Certainty	Follow-up
Upper-trapezius pain (post-needling)	Vearasilp and Sukareechai 23	Double-blind RCT	40 (20 per arm)	Sham	PPT increase p=0.002; NRS improvement p<0.05 vs sham	Single trial (not GRADE-rated)	48 hours
Knee osteoarthritis	Gogic et al. 25	Pilot RCT (unblinded)	30	Interferential current + kinesiotherapy	FMS + kinesiotherapy improved VAS pain more (1 T, 30 Hz)	Hypothesis-generating	No durable follow-up
Fibromyalgia (adjacent)	Leenus et al. 26	Systematic review / meta-analysis	6 trials, n=279	Sham	Pain MD -1.86 NRS (95% CI -2.85 to -0.87), I^2 = 68%; 16-55% reduction at 1-3 months; not sustained at 3 months or later	Low	1-3 months
Low back pain (device feasibility)	Wu et al. 27	Feasibility case series	3	None (single-arm)	Feasibility of ultrasound-guided injection plus rPMS; not efficacy	Low / feasibility	Pre/post (no durable follow-up)

Individual low-back studies illustrate why design determines interpretive weight. Yan et al. provide a useful sham-controlled mechanistic trial in chronic non-specific low back pain (n=40) because a credible inactive coil isolates the specific effect of stimulation from expectancy, and fNIRS links clinical improvement to measurable cortical change 7. Masse-Alarie et al. studied multifidus rPMS embedded in motor training; this is clinically relevant to rehabilitation use, but not a pure test of standalone stimulation (n=21) 8. Lim et al. reported positive pilot data in acute low back pain (n=26), with VAS improving from 63.75 to 27.08 and ODI from 50.98 to 22.74 over ten sessions 14. Savulescu et al. found significant pain improvement in both arms of an EMG-guided randomized study (n=41) with superior functional recovery in the rPMS-plus-physiotherapy group, while Tesfa et al. provided feasibility-level inference in seventeen veterans with chronic back pain 15,16. Thus, apparently uniform "positive" low-back results conceal very different evidentiary strengths.

Radicular pain is mechanistically attractive because it involves peripheral neural structures that may respond to stimulation. Lytras et al. randomized forty patients with MRI-confirmed lumbar disk herniation and radiculopathy to manual therapy with or without FMS over ten sessions across three weeks; significant group-by-time interactions favored the combined arm for all outcomes (p<0.01), with reductions in lumbar and leg pain, Roland-Morris disability, and S-LANSS scores exceeding established minimal clinically important difference thresholds and straight-leg-raise gains surpassing published minimal detectable change values, and only the FMS arm fell below the neuropathic-pain cutoff (S-LANSS <12) [17]. The result is clinically meaningful as an incremental add-on signal, but the absence of a sham magnetic control and the short follow-up, with outcomes measured only at week three, prevent conclusions about isolated efficacy or durability. The broader neuropathic-pain literature, including the systematic review and meta-analysis by Dana et al., supports plausibility but remains heterogeneous across diagnoses and protocols [18].

Myofascial pain has the longest controlled evidence trail, and individual trials should be read separately rather than pooled by impression. Pujol et al. randomized thirty patients to a single forty-minute session over the tender body region and found real stimulation reduced a 101-point pain score by 59% versus 14% for sham (p=0.001), with relief for several days 19. The two Smania trials are distinct: the 2003 placebo-controlled trapezius study reported improvement in VAS, disability VAS, algometry, trigger-point characteristics, and range of motion at one month (n=18) 20, while the 2005 three-arm trial (n=53) found rMS superior to TENS and placebo 21. Recent small sham-controlled studies also support short-term benefit in neck or upper-trapezius myofascial pain 22,23. Renner et al. add trigger-point stimulation data in migraine-related neck and shoulder targets, but the headache-centered endpoint limits direct musculoskeletal inference 24.

Joint pain is less mature. Gogic et al. reported a pilot randomized trial in knee osteoarthritis using an Iskra Tesla Care device at 1 T and 30 Hz, in which FMS plus kinesiotherapy improved VAS pain more than interferential current plus kinesiotherapy [25]. The result is promising but hypothesis-generating: the trial included only thirty participants, lacked blinding, used VAS as the sole endpoint, had no durable follow-up, and tested stimulation within an active cointervention package.

Fibromyalgia and nociplastic pain should be treated as adjacent rather than directly interchangeable evidence. Leenus et al. meta-analyzed six randomized trials (n=279) and found that PMS significantly reduced pain within one to three months (mean difference -1.86 on the Numeric Rating Scale; 95% CI -2.85 to -0.87; I^2 = 68%; four trials, 154 participants; low certainty), with reported reductions ranging from 16% to 55%, but the effect was not sustained beyond three months and functional improvement was not significant 26. Fibromyalgia, however, is not focal myofascial pain or osteoarthritis; it is a generalized nociplastic syndrome with distinct pathophysiology and outcome needs. These data support broader neuromodulatory plausibility but should not be used to claim efficacy for focal musculoskeletal disorders.

Evidence hierarchy and certainty

The apparent consistency of favorable results should not be confused with high certainty; the evidence hierarchy is uneven. Meta-analyses in low back and chronic musculoskeletal pain provide the broadest quantitative signal, but their conclusions inherit the limitations of the underlying trials: small samples, heterogeneous treatment parameters, short follow-up, and variable sham credibility [12,13]. Sham-controlled trials provide the cleanest test of specific efficacy, yet they are few and generally underpowered. Active-comparator and add-on trials are closer to clinical practice but more difficult to interpret mechanistically. Feasibility studies and non-indexed reports can identify hypotheses, not establish treatment effects.

This hierarchy changes how the literature should be read. A positive sham-controlled myofascial pain trial supports a specific analgesic signal in that population. A positive add-on radiculopathy trial supports the incremental value of adding FMS to manual therapy under the tested conditions [17]. A pilot knee osteoarthritis trial supports further research, not clinical adoption as a disease-specific standard [25]. A commercial survey may describe user experience but cannot adjudicate efficacy, and a non-musculoskeletal FMS study may inform safety or neuromuscular feasibility but cannot serve as evidence that musculoskeletal pain improves.

The strongest current conclusion is therefore conditional: high-intensity PMS has credible short-term analgesic and functional signals in selected musculoskeletal conditions, with the best replicated evidence in low back and myofascial pain, but the field has not yet resolved dose, target, durability, responder phenotype, or comparative value. This conclusion is deliberately narrower than many promotional claims and stronger than a purely dismissive reading, recognizing that immature evidence can still be clinically important when the mechanism is plausible, the safety profile is acceptable, and the research agenda is clear.

Device-specific evidence and grey literature

Device specificity matters because commercial implementation affects coil design, available programs, field strength, applicator cooling, ergonomics, and target selection, yet a scientific review should not allow brand identity to substitute for evidence. The Iskra Tesla family is one named implementation of high-intensity magnetic stimulation, but its musculoskeletal evidence base is narrow.

Within musculoskeletal pain, the directly relevant named-Iskra evidence includes the Gogic knee osteoarthritis pilot RCT, the Wu ultrasound-guided lumbar injection plus Tesla Stym feasibility study, and the Radakovic sciatica report 25,27,28,29. These studies answer different questions. Gogic et al. provide the strongest controlled device-specific musculoskeletal signal, though still at pilot level 25. Wu, Chang, and Ozcakar demonstrate feasibility of combining ultrasound-guided facet/multifidus injections with rPMS in three patients, not efficacy 27. The 2025 perspective by Wu et al. is useful for research framing but is not primary efficacy evidence 28. Radakovic and Radakovic should be treated as non-indexed preliminary evidence because of major design and reporting limitations 29.

Non-musculoskeletal Iskra studies can inform platform breadth or safety context only when labeled as such. Braga et al. evaluated 3-T FMS for urinary incontinence, and Skocir et al. used Tesla Stym for quadriceps stimulation in critically ill patients 10,30. These data do not prove musculoskeletal pain efficacy. Commercial booklets, surveys, and continuing-education materials are best treated as claim inventories and citation maps, not load-bearing efficacy evidence.

Dose, protocol heterogeneity, and outcome selection

The largest practical barrier to interpretation is not whether PMS can stimulate tissue, but whether published protocols describe stimulation reproducibly. Many reports emphasize nominal field strength or branded programs while underreporting the variables that determine the induced electric field and biological dose. A compact minimum reporting checklist is therefore proposed in Box 1.

Box 1. Proposed minimum reporting checklist for high-intensity peripheral magnetic stimulation studies

1. Device manufacturer, model, applicator/coil, cooling method, coil geometry, and nominal field strength in tesla.

2. Target anatomy, target-localization method, coil orientation, coil-to-tissue distance, coupling, and estimated target depth.

3. Frequency, pulse width or waveform when available, train duration, inter-train interval, duty cycle, session duration, and total pulses.

4. Intensity calibration anchor, motor threshold or sensory threshold, contraction criterion, progression rule, and stopping rule.

5. Number of sessions, interval between sessions, total program duration, cointerventions, rehabilitation protocol, and concomitant care.

6. Comparator or sham description, sham fidelity, blinding-credibility checks, timing of outcomes, follow-up duration, adverse-event monitoring, and prespecified responder phenotype.

The difference between standalone and adjunctive use is equally important. A sham-controlled standalone trial asks whether the magnetic intervention has a specific effect beyond expectancy and time; an add-on trial asks whether adding stimulation to active rehabilitation improves outcomes beyond that program alone. Both are useful but answer different questions. The Lytras radiculopathy and Gogic knee osteoarthritis trials are pragmatic because they test PMS inside active care and should not be read as proof that the magnetic field alone is sufficient [17,25], whereas sham-controlled trials in low back and myofascial pain offer cleaner estimates of specific effect despite small samples [7,20,21,22,23].

Outcome selection should also mature, as pain intensity is necessary but insufficient. In low back pain, disability instruments (Oswestry Disability Index, Roland-Morris Disability Questionnaire), neuropathic screening for radicular features, analgesic use, global perceived effect, and durable follow-up matter more than immediate pain reduction. In knee osteoarthritis, WOMAC, KOOS, physical performance, rescue medication, and adverse events should supplement VAS. In myofascial pain, pressure-pain threshold, trigger-point irritability, range of motion, and recurrence are relevant. For mechanistic trials, fNIRS, quantitative sensory testing, surface EMG, ultrasound muscle morphology, and TMS-indexed corticomotor excitability should be prespecified explanatory endpoints rather than decorative biomarkers.

The field would also benefit from responder-oriented designs. A patient with focal trigger-point sensitivity, motor inhibition, or radicular irritability may not respond like one with widespread nociplastic pain or advanced structural joint disease, yet current trials are too small to test phenotype-treatment interaction. Future studies should define responder hypotheses before enrollment (neuropathic features, pressure-pain sensitivity, baseline disability, cortical excitability, paraspinal inhibition, fear-avoidance, or prior response to electrical stimulation), so the literature stops producing favorable average effects while leaving clinicians uncertain about whom to treat.

Safety, implementation boundaries, and trial standards

High-intensity PMS is usually described as well tolerated, and most small musculoskeletal trials report few serious adverse events, but this does not justify complacency. Relevant safety issues include implanted electronic devices, magnetically sensitive implants, stimulation near vulnerable hardware, protocol- and history-dependent seizure risk, pregnancy policies, inability to communicate discomfort, and local heat accumulation under high-duty-cycle stimulation. The ICU quadriceps trial by Skocir et al. is a useful cautionary example: mild thermal skin injuries occurred early and were prevented after applicator-positioning changes [10]. Although ambulatory patients differ from critically ill ones, the lesson generalizes: applicator heat, cable contact, tissue compression, skin inspection, and patient feedback should be part of protocolized safety monitoring.

Blinding deserves special attention because active PMS can be audible, visible, and somatically distinctive. A sham coil that fails to reproduce acoustic cues, vibration, pressure, or expectation may exaggerate between-group effects, so trials should report participant and assessor blinding credibility, not only the sham device. Without credibility checks a trial may be nominally double-blind yet functionally unblinded, especially when outcomes are subjective and immediate, as pain intensity often is.

Comparator choice should match the clinical question. A sham comparator is essential for estimating specific efficacy; an active comparator such as interferential current tests pragmatic relative value but may obscure mechanism; usual-care or exercise comparators test incremental value in clinical pathways. Head-to-head trials against TENS, manual therapy, dry needling, injections, or structured exercise should not be read as equivalence or superiority trials unless adequately powered for that question, and small pilots cannot establish practice-changing comparative effectiveness.

The next research phase should be multicenter, adequately powered, and phenotype-aware. Priority populations include chronic non-specific low back pain, lumbar radicular pain with neuropathic features, upper-quarter myofascial pain, and knee osteoarthritis with pain-related quadriceps inhibition. Core outcomes should include pain, disability, function, global perceived effect, medication use, adverse events, and follow-up at one, three, and six months. Mechanistic substudies should test whether cortical activation, sensory thresholds, EMG recruitment, or muscle morphology predict durability and guide selection.

Synthesis and clinical implications

The clinical signal for high-intensity PMS merits serious investigation but not broad therapeutic claims. It may be considered an adjunctive neuromodulatory and neuromuscular tool for selected patients, particularly with altered motor control, myofascial sensitivity, proprioceptive disturbance, or radicular features. The evidence does not support using the modality as a replacement for education, graded activity, exercise therapy, pharmacologic care, injections, or surgery when indicated.

The most defensible clinical model is a rehabilitation window, not passive analgesia. If PMS reduces pain or threat and improves tolerance to contraction or movement, that window should be paired with graded loading, motor-control retraining, gait or task practice, and self-management. This interpretation fits the frequent use of PMS as an add-on and mechanistic evidence suggesting cortical and motor-system involvement [7,8,9,17].

This positioning protects the field from a common translational error: treating a device response as equivalent to a patient-centered recovery response. A short-term reduction in pain becomes clinically meaningful only if it changes what the patient can do: walk farther, tolerate loading, sleep better, reduce rescue medication, or participate more effectively in exercise. PMS should therefore be embedded in a plan that defines the functional behavior expected to change after each treatment block, and the device should be judged not by contraction intensity but by whether it helps unlock a clinically relevant progression.

The same logic applies to nonresponse. A patient with transient analgesia but no functional carryover may need a different target, dose, or rehabilitation pairing; one who cannot tolerate evoked contraction, has diffuse pain amplification without a focal target, or expects a passive cure may be a poor candidate. Routine outcome tracking prevents open-ended treatment courses: define baseline pain and function, reassess after a short induction series, and continue only when improvement exceeds a prespecified symptom and activity threshold. For researchers, the urgent task is multicenter sham-controlled trials with credible blinding, standardized dose reporting, mechanistic stratification, and follow-up at one, three, and six months.

Limitations

This review is narrative and structured, not systematic. Although the search strategy, SANRA self-appraisal, citation audit, and evidence hierarchy were explicit, study selection was not performed with dual independent screening, formal risk-of-bias adjudication, or meta-analytic reanalysis. Selection bias is therefore possible. Independent verification by a second member of the author team was added for the recent indexed citations carrying the greatest interpretive weight, but this does not replace dual screening of the full evidence base. The pooled meta-analyses also overlap in their underlying low-back trials, so their estimates should not be read as independent confirmation.

The evidence base imposes further limitations: small samples, heterogeneous protocols, variable targets, inconsistent sham procedures, incomplete blinding checks, and short follow-up. Several contemporary studies cited here are indexed publications with assigned PMID and DOI records, but they still await independent replication. Mechanistic studies suggest cortical and afferent contributions, yet most trials were not designed to test mediation. Non-musculoskeletal and non-pain studies support feasibility of muscle recruitment or platform use, not clinical analgesia. Grey literature and commercial materials were considered only as context.

Conclusion

High-intensity PMS is a plausible, noninvasive adjunct for selected musculoskeletal pain presentations, with the strongest short-term evidence in low back and myofascial pain and early pilot signals in radicular pain and knee osteoarthritis. Its value appears most defensible when stimulation is integrated into rehabilitation rather than delivered as a passive standalone procedure.

The field now needs adequately powered sham-controlled trials that define dose, target, comparator, responder phenotype, safety procedures, and durable outcomes. Until then, high-intensity PMS should be presented as promising and mechanistically credible, but not established as a definitive musculoskeletal pain therapy.

Author track declaration

This manuscript is submitted under the Standard track. A senior author is named on the separate Title Page and accepts responsibility for the integrity and scientific content of the work.

AI tools used

In accordance with ICMJE 2025 guidance, the authors disclose that generative AI-assisted tools were used for language refinement, formatting support, and reference-organization support. AI tools were not used to generate scientific conclusions, interpret data, or determine the certainty ratings. The authors independently reviewed and verified all scientific content, all quantitative values, and every citation against primary sources, and take full responsibility for the final manuscript.

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