



# Myofascial Pain Syndrome: Pathophysiology and Clinical Aspects

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WebLog Open Access Publications

Article ID : wjptr.2026.c1301

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Received Date: 15 Feb 2026

Accepted Date: 11 Mar 2026

Published Date: 13 Mar 2026

### Citation:

Pinheiro JP, Lourenço M. Myofascial  
Pain Syndrome: Pathophysiology and  
Clinical Aspects. WebLog J Phys Ther  
Rehabil. wjptr.2026.c1301. <https://doi.org/10.5281/zenodo.19268993>

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## Abstract

**Background:** Myofascial pain syndrome (MPS) is a highly prevalent yet frequently underdiagnosed musculoskeletal condition associated with significant functional impairment. Its clinical complexity and the absence of objective biomarkers contribute to diagnostic challenges.

**Aims:** To review the epidemiology, pathophysiology, clinical presentation and therapeutic management of MPS, with particular emphasis on its relevance to Physical and Rehabilitation Medicine (PRM).

**Methods:** A narrative review of key literature in PRM, rheumatology and pain medicine was conducted, focusing on trigger points, taut bands, nociception, and peripheral and central sensitisation mechanisms.

**Results:** MPS is characterised by regional pain associated with myofascial trigger points within taut muscle bands. Peripheral mechanisms involve motor endplate dysfunction, local ischaemia and the release of algogenic substances, while sustained nociceptive input may induce central sensitisation. Diagnosis is clinical, based on physical examination findings. Management is multimodal and function-oriented, integrating pharmacological, interventional and rehabilitative strategies.

**Conclusion:** MPS is a complex condition involving interconnected peripheral and central mechanisms. A biopsychosocial and rehabilitation-focused approach is essential for effective management. Further high-quality research is required to strengthen diagnostic criteria and therapeutic evidence.

**Keywords:** Myofascial Pain Syndrome; Trigger Points; Central Sensitisation; Rehabilitation

## Introduction

Pain is a multidimensional experience integrating sensory, emotional, cognitive and behavioural components, with clear implications for activity and participation. In PRM, pain is understood not merely as a symptom, but as a determinant of functional limitation and reduced quality of life.

MPS is a highly prevalent musculoskeletal disorder, frequently underdiagnosed, yet associated with considerable functional impact [4, 8, 9].

It is defined as a non-inflammatory musculoskeletal condition characterised by regional pain, restricted movement and the presence of hyperirritable trigger points (TrPs) located within skeletal muscle and surrounding fascia. Clinical presentation is heterogeneous and often overlaps with other pain syndromes, a difficulty compounded by the absence of objective biomarkers or specific imaging findings. A clear understanding of its pathophysiology is therefore essential for appropriate therapeutic decision-making and preventive strategies.

## Methods

This manuscript presents a narrative review based on the literature referenced in the course material, focusing on the epidemiology, pathophysiology, clinical features and therapeutic perspectives of MPS. Review articles, clinical studies and consensus papers addressing the concepts of trigger points, myofascial taut bands, nociception, peripheral and central sensitisation, and clinical frameworks were included. Emphasis was placed on publications within PRM, rheumatology and pain medicine to ensure clinical relevance and scientific rigour. This text was presented at the session for residents in the specialties of PRM and Orthopaedics (PAIO sessions) in January 2026.

## Results

To make this text more didactic, the results have been organized in terms of epidemiology, functional impact, pathophysiology, clinical presentation and diagnosis, and the role of PMR in the therapeutic approach.

### Epidemiology and Functional Impact

MPS is among the most common causes of musculoskeletal pain in clinical practice. Prevalence rates range from 30% to 93% among patients attending musculoskeletal services. It accounts for approximately 30% of cervical and lumbar pain syndromes, and in specialised pain centres this proportion may reach 85% of cases [4, 8].

The true incidence in the general population remains uncertain due to underdiagnosis and inconsistent—often subjective—diagnostic criteria. From a PRM perspective, MPS results in limitations in activities of daily living, occupational disability and restricted participation.

### Pathophysiology (peripheral and central mechanisms)

The most consistent pathological feature of MPS is the myofascial trigger point, a hyperirritable focus located within a palpable taut band of skeletal muscle.

Pathophysiological models describe motor endplate dysfunction with excessive acetylcholine release, leading to sustained sarcomere contraction and taut band formation [2, 9]. This state promotes local ischaemia, hypoxia and an “energy crisis” characterised by reduced ATP availability and impaired calcium reuptake. Accumulation of algogenic substances—including substance P, calcitonin gene-related peptide (CGRP), bradykinin and hydrogen ions—contributes to peripheral sensitisation and mechanical hyperalgesia [3].

Sustained nociceptive input from trigger points activates group III and IV muscle afferents, facilitating neuroplastic changes in the dorsal horn of the spinal cord [10]. In chronic MPS, this process promotes central sensitisation, resulting in persistent pain, referred pain and increased nociceptive responsiveness.

Functional neuroimaging studies demonstrate enhanced activation in somatosensory cortical regions and limbic structures, supporting the role of central mechanisms in pain chronification and in the amplification of activity and participation restrictions [2, 7].

### Clinical Features and Diagnosis

Clinically, MPS presents with regional pain, frequently accompanied by characteristic patterns of referred pain corresponding to specific myotomal distributions.

Core diagnostic criteria include a palpable taut band, a hypersensitive trigger point within the band, reproduction of the patient’s typical pain upon palpation, a local twitch response and restricted regional mobility [1, 4].

Although virtually any muscle may be involved, the condition most commonly affects cervical and scapular stabilisers, lumbar paravertebral muscles, quadratus lumborum, and the gluteal and pelvirochanteric muscle groups.

Diagnosis is essentially clinical, as laboratory and imaging investigations are typically unremarkable. Differential diagnosis with fibromyalgia is important, although overlap between tender points and trigger points may occur. Inconsistencies in terminology within the literature may further complicate this distinction [5].

### The Role of PRM

Management of MPS in PRM is multimodal and oriented towards analgesia and functional restoration [6]. Pharmacological treatments include non-opioid analgesics, non-steroidal anti-inflammatory drugs, muscle relaxants and antidepressants, primarily for symptomatic relief. However, pharmacological therapy alone is rarely sufficient. Interventional procedures, such as dry needling and trigger point injections (local anaesthetics with or without corticosteroids), as well as mesotherapy, are frequently employed to reduce local and regional nociceptive input.

Physical and kinesiological interventions include manual therapies (myofascial release and trigger point pressure techniques), therapeutic exercise focusing on static stretching, muscle strengthening and motor re-education. Patient education is increasingly recognised as a fundamental therapeutic component.

Physical modalities such as heat therapy, transcutaneous electrical nerve stimulation (TENS) and ultrasound are considered adjunctive rather than primary treatments.

Long-term preventive strategies emphasise postural correction, ergonomic adaptation, management of biomechanical overload and prescription of low-impact aerobic exercise.

## Discussion and Conclusion

Current evidence indicates that MPS is a complex pain condition involving interrelated peripheral and central mechanisms. Although trigger points originate within muscle tissue, persistent nociceptive input may drive central sensitisation and pain chronification.

This mechanism may explain the limited efficacy of isolated peripheral interventions and highlights the need for comprehensive biopsychosocial management strategies.

Despite its high prevalence and clinical burden, the overall quality of evidence supporting available treatments remains limited, underscoring the need for well-designed clinical trials and the development of more objective diagnostic tools.

In summary, MPS is a highly prevalent and disabling musculoskeletal condition frequently encountered in PRM. It results from complex interactions between peripheral muscular dysfunction and central sensitisation, leading to persistent pain and functional limitation.

Diagnostic challenges, overlap with other chronic pain syndromes and the lack of objective serum or imaging biomarkers contribute to its under recognition. A biopsychosocial and function-oriented approach remains central to quality care. Further advances in understanding trigger point physiology, taut band formation, nociceptive processing and associated neuroplastic changes, supported by robust evidence, will contribute meaningfully to clinical practice.

## References

1. Cummings M & Baldry P. Regional myofascial pain: Diagnosis and management. *Best Practice & Research Clinical Rheumatology*, 2007; 21(2), 367–387.
2. Fernández-de-Las-Peñas C & Dommerholt J. Myofascial trigger points: Peripheral or central phenomenon? *Current Rheumatology Reports*, 2023; 25(2), 43–52.
3. Fernández-de-Las-Peñas C, Nijs J & Simons D. G. Pathophysiology of

- myofascial trigger points: An integrative perspective. *Journal of Pain Research*, 2022; 15, 311–324.
4. Gerwin R. D. Diagnosis of myofascial pain syndrome. *Physical Medicine and Rehabilitation Clinics of North America*, 2021; 32(3), 403–415.
  5. Kuncewicz E & Samborski W. Tender points and trigger points: Differences and similarities. *Reumatologia*, 2009; 47(2), 77–82.
  6. Lam C, Francio V. T, Gustafson K, Carroll M, York A & Chadwick A. L. Myofascial pain — A major player in musculoskeletal pain. *Best Practice & Research Clinical Rheumatology*, 2024; 38, 101944.
  7. Niddam D. M, Chan R. C, Lee S. H, Yeh T. C & Hsieh J. C. Central representation of hyperalgesia from myofascial trigger point. *NeuroImage*, 2007; 39(3), 1299–1308.
  8. Rosen N. The confusing nomenclature of muscle pain syndromes. *Physical Medicine and Rehabilitation Clinics of North America*, 1993; 4, 347–359.
  9. Shah J. P, Thaker N, Heimur J, Aredo J. V, Sikdar S & Gerber L. Myofascial trigger points then and now: A historical and scientific perspective. *PM&R*, 2015; 7(7), 746–761. <https://doi.org/10.1016/j.pmrj.2015.01.024>
  10. Windhorst U, Kirmayer D, Soibelman F, Misri A & Rose R. Effects of neurochemically excited group III–IV muscle afferents on motoneuron afterhyperpolarisation. *Neuroscience*, 1997; 76(3), 915–92.