Journal of Pain Research

Dovepress
Taylor & Francis Group

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RESPONSE TO LETTER

A Proposed Diagnostic and Treatment Algorithm for the Management of Lumbar Discogenic Pain [Response to Letter]

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Dear editor

We appreciate the thoughtful comments by Drs. Joyal and Francio and thank them for their interest in our article, A Proposed Diagnostic and Treatment Algorithm for the Management of Lumbar Discogenic Pain.[1] We agree that multifidus dysfunction has emerged as an increasingly important contributor to chronic mechanical low back pain and acknowledge the growing body of literature supporting an association between lumbar degenerative changes and neuromuscular stabilization failure.

Importantly, omission of a dedicated multifidus pathway from our algorithm was not due to lack of awareness of this evolving field. During development of the discogenic pain algorithm, restorative neurostimulation and concepts related to neuromuscular instability were actively considered. Indeed, one of us previously participated in an International Society for the Advancement of Spine Surgery (ISASS) statement addressing restorative neurostimulation for chronic mechanical low back pain resulting from neuromuscular instability.[2] More recently, systematic review and meta-analytic evidence has strengthened support for multifidus dysfunction as a clinically relevant entity with diagnostic and therapeutic implications,[3] culminating in an updated ISASS guideline and coverage framework supporting restorative neurostimulation for multifidus dysfunction.[4]

The decision not to incorporate a multifidus pathway was therefore not an oversight, but rather a matter of intended scope and phenotype definition. The present algorithm was deliberately designed to identify and manage a specific anterior-column, disc-dominant pain phenotype using defined clinical findings, MRI characteristics, and treatment sequencing focused on lumbar discogenic pain.[1] Similar to the DISCS framework proposed in our original article, the purpose of the algorithm was to narrow the diagnostic aperture toward a distinct pain generator and guide phenotype-specific treatment selection. This approach aligns with recent efforts supporting mechanism-based classification and formal ICD-10-CM recognition of lumbar discogenic pain as a distinct diagnostic entity within the broader chronic low back pain landscape.[5]

While multifidus dysfunction frequently coexists with degenerative disc disease, coexistence should not be equated with diagnostic equivalence. Imaging evidence of multifidus fatty infiltration and atrophy demonstrates important associations with disc degeneration; however, these findings do not establish multifidus dysfunction as a defining criterion for discogenic pain itself. Likewise, the observation that substantial proportions of restorative neurostimulation cohorts exhibit concomitant degenerative disc disease likely reflects overlap among chronic low back pain populations rather than a singular pathophysiologic construct.

We fully endorse continued scientific attention to multifidus fatty infiltration and morphologic changes as emerging biomarkers of multifidus dysfunction, now recognized under ICD-10 code M62.85 (Dysfunction of the multifidus muscles, lumbar region). However, we would caution against conflating a frequently associated neuromuscular stabilization phenotype characterized by impaired spinal stability and motor control with the primary discogenic phenotype our

https://doi.org/10.2147/JPR.S623705
Received: 11 May 2026
Accepted: 24 May 2026
Published: 3 June 2026

algorithm was designed to pragmatically identify and treat. Disc degeneration and multifidus dysfunction may coexist, contribute to symptom persistence, or evolve sequentially; however, current evidence does not yet support automatic incorporation of restorative neurostimulation into a disc-first treatment pathway without phenotype-specific diagnostic confirmation.

We do agree with Drs. Joyal and Francio that future efforts may benefit from a broader phenotype-based framework for chronic low back pain incorporating multiple diagnostic entry points—including discogenic, vertebrogenic, and neuromuscular stabilization pathways. Within such an expanded framework, one could envision dedicated entry points for multifidus dysfunction incorporating clinical characteristics such as segmental mechanical pain, transitional movement intolerance, altered motor control, endurance deficits, and local multifidus abnormalities, in addition to imaging evidence of fatty infiltration or atrophy, neuromuscular instability assessments, functional markers of impaired spinal stabilization, and exclusion of major neurologic compression. Such an approach may ultimately allow patients to enter diagnostic and therapeutic algorithms through distinct but potentially overlapping mechanisms of pain generation. We appreciate the opportunity to advance this important discussion.

Disclosure

The authors report no conflicts of interest in this communication.

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https://doi.org/10.2147/JPR.S623705