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Peer Review

Review of: "The "High-Pressure Pain Cavity" Hypothesis: A Pressure-Volume Model of Myofascial Trigger Points Redefining the Local Twitch Response as an Event of Energy Resupply"

Md Toukir Ahmed[1]

1. Dhaka Medical College and Hospital, Dhaka, Bangladesh

This is a highly creative and intellectually refreshing paper. The author tries to move away from purely biochemical models of Myofascial Trigger Points (MTrPs) and looks at them through structural mechanics instead. Modeling the perimysium as a high-pressure, sealed 'micro-compartment' is an excellent conceptual exercise to explain the physical hardness of these nodules. However, the hypothesis runs into severe, fatal conflicts with foundational muscle biophysics and established clinical data.

The Dealbreakers

- **Muscle physiology doesn't work this way:** The paper argues that an immediate flood of ATP into a high-calcium environment triggers a synchronized muscle twitch (LTR) without a nerve impulse. This fundamentally misinterprets actomyosin kinetics. In a true rigor/ischemic state (where ATP is depleted), introducing fresh ATP causes immediate detachment of the myosin heads from actin, not a sudden contraction stroke. Furthermore, fluid-driven ATP diffusion would happen along a gradient, leading to slow, uneven relaxation—not a sharp, simultaneous 'twitch.'
- **Absolute ischemia means tissue death:** If the trigger point were a completely 'sealed' capsule under catastrophic, escalating fluid pressure as described, the local capillaries would be completely crushed, causing total ischemia. Skeletal muscle cells cannot survive long-term absolute ischemia; they would undergo localized necrosis and turn into scar tissue within days. We simply do not see widespread focal necrosis in chronic MTrPs clinically.

doi.org/10.32388/HPOWFJ

- **The 'prediction' has already been disproven:** The author attempts to rule out the spinal reflex model by suggesting a proximal nerve block experiment (Prediction #4). The problem is, this work has already been done by other researchers. Multiple historical studies have conclusively shown that proximal nerve blocks, spinal anesthesia, and general anesthesia successfully abolish or heavily blunt the LTR. The LTR is inherently tied to the nervous system, invalidating the paper's core premise that it is a purely local mechanical-chemical event.

Summary Recommendation

I appreciate the outside-the-box thinking, but a scientific hypothesis cannot stand when its mechanisms violate fundamental biophysics and ignore existing literature that explicitly disproves its core prediction. To make this viable, the author needs to completely reframe the paper: instead of arguing that the mechanical cavity replaces the nervous system, they should look at how this high-pressure compartment interacts with and hyper-sensitizes local nerve endings to trigger the reflex twitch.

Declarations

Potential competing interests: No potential competing interests to declare.