Delayed-Onset Muscle Soreness: A Prediction Error

Brief Review

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Abstract

Delayed-onset muscle soreness is a common and well-researched phenomenon among exercising populations, whose symptoms can impair movement quality and subsequent participation in activities. Traditional explanations have focused mostly on tissue and cellular damage and the body's inflammatory responses. This has led to many hypotheses that often do not fully agree with one another or adequately capture the complexity of the matter. Principles of evolutionary biology and predictive processing have previously been applied to phenomena in exercise science and seem well-suited to interpreting this issue. Drawing on prior work in these areas, this brief review proposes a conceptual framework that shifts the perspective on the phenomenon's etiology from primarily tissue- and cellular-level explanations to one reflecting a centrally mediated predictive process.

Key Words: eccentric exercise, predictive processing, evolutionary biology

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Introduction

Delayed-onset muscle soreness (DOMS) is common among exercisers, from recreational participants to elite athletes.[1] The symptoms associated with DOMS can impair muscular force production, movement quality, and participation in sporting activity.[2] To date, there is no unified interpretation of its etiology.[2] Most theories about the underlying mechanisms focus on myocellular disruption, inflammatory responses, and circulating biomarkers.[1,3,4] Although important for advancing understanding of the issue, these explanations may be insufficient to fully capture the complexity of DOMS.[1,2,5]

Specifically, no single hypothesis or combination of hypotheses adequately resolves the mismatch between symptom experience and the underlying physiological processes, suggesting that they reflect only part of a broader adaptive process. Principles of evolutionary biology and predictive processing have often been applied to phenomena in exercise science to help advance understanding of complex circumstances, and appear particularly well-suited to provide a framework for explaining why DOMS becomes a prominent perceptual experience when structural disruption alone cannot reliably account for its severity or timing.[6]

Conceptualizing DOMS as a form of prediction error may clarify several otherwise equivocal features of the phenomenon, including its delayed onset, exaggerated perceptual quality, and swift attenuation following repeated exposure. From this perspective, DOMS is not simply a consequence of local tissue disruption, but a protective output shaped by the nervous system's attempt to regulate future behavior under conditions of uncertainty and energetic cost.[7–9] Such an interpretation aligns with evolutionary models in which pain functions as a defensive adaptation biased toward overprotection, particularly when prior expectations are violated.[7,10]

This brief review introduces a conceptual framework, grounded in evolutionary biology and predictive processing theory, that reframes DOMS from tissue- and cellular-level explanations to a centrally mediated process of perception and threat regulation.[8,11] It first outlines the defining characteristics and dominant theories of DOMS, then introduces key principles from evolutionary biology and predictive processing theory, and finally applies them to explain hallmark features of the DOMS experience and inform future research.

Characteristics and Theories

Delayed-onset muscle soreness has traditionally been described as an ultrastructural,[2] overexertion-related muscle strain.[1] As a form of exercise-related muscle pain, DOMS differs from exercise-induced pain (EIP), which is an acute, uncomfortable sensation experienced during or immediately after activity and is not caused by muscle damage.[12] EIP is strongly characterized by its transient and controllable nature.[12] On the other hand, the hallmark traits of DOMS include: (1) a delayed experience of pain and stiffness that hinders movement fluency and force production, typically peaking around 24-72 hours following eccentric exercise, (2) the presence of hyperalgesia, and (3) desensitization following repeated bouts of the same activity.[1,2]

Collectively, these symptoms may impair an individual's athletic performance and ability to perform in subsequent activities.[2] Conventional understanding posits DOMS principally as a result of mechanical disruption of muscle tissue.[2] However, no single cause has been universally agreed upon, leading to multiple theories.[1,3,4] It is beyond the scope of this brief review to discuss each theory in detail, but two prominent theories still form the basis of our current understanding of how DOMS develops.

The muscle damage theory suggests that DOMS results from structural damage to muscle fibers, particularly sarcomeres and Z-lines, caused by excessive mechanical stress during eccentric activity.[1] Nociceptive stimulation of the affected and nearby tissues causes pain, while structural damage increases cellular permeability, allowing leakage of muscle enzymes, particularly creatine kinase (CK), into the interstitial fluid.[1] Elevated CK levels in study participants have been used to support this theory, although peak CK levels do not align with peak muscle soreness.[1] This mismatch between biomarkers and symptom onset suggests that muscle damage alone may be insufficient to explain DOMS, prompting the development of alternative yet related frameworks, such as the inflammation theory.

The inflammation theory focuses on both vascular and cellular aspects of the inflammatory response to eccentric exercise.[1] As tissue damage is recognized by the body, the injury site experiences an influx of cellular mediators, with vasodilation and increased vascular permeability leading to fluid accumulation in the injured muscle.[1] This change in osmotic pressure triggers a nociceptive response, causing pain in the affected muscle.[1] Peak edema volumes seem to align with peak muscle soreness, whereas the timing of inflammatory cell infiltration shows less correlation.[1] Although conceptually distinct, these theories are closely linked because inflammation is often viewed as a downstream consequence of muscle damage. However, both have limitations in explaining the temporal dynamics of pain experienced during the phenomenon.

Evolutionary Biology & Predictive Processing Theory

Our biology reflects a process shaped by selection pressures that favor survival and reproduction within a specific environment.[13] Over millennia, natural selection has favored biological adaptations that allocate energy between activity and reproduction to balance competing demands and maintain overall fitness.[13] While modern DOMS research within exercise science provides insights into *how* these mechanisms operate in response to loading, an evolutionary theory may be more applicable for explaining *why* they exist.[13] Accordingly, the idea that nothing in biology makes sense except in the light of evolution seems especially relevant to this phenomenon.[14]

Adaptations are context-dependent solutions that improve an organism's survival and reproductive abilities.[13] Therefore, our anatomy and physiology respond to physical stress in ways that save energy and reduce unnecessary effort.[8,13] To achieve this, the brain constantly predicts the trade-offs and outcomes of our actions, using past experiences as a guide.[8,15] These top-down predictive processes influence our perception and behavior, while they integrate dynamically and reciprocally with bottom-up sensory feedback loops as we interact with our environment.[6,8] The reciprocal relationship between prediction and sensory input affords organisms both plasticity and adaptability in changing environments and contexts. Framed this way, an evolutionary–predictive view on DOMS may prompt a deeper question: What adaptive purpose might this phenomenon serve?

Delayed-Onset Muscle Soreness as Prediction Error

Previous research has identified the brain as the center of a complex, dynamic regulatory system that manages activity and energy use.[8,11] The central governor model of exercise regulation suggests that "exercise begins with feedforward motor output to recruit the appropriate number of motor units in the exercising muscle."[16] Consequently, a prediction is made at the outset of participation in the activity based on multiple integrated factors, including past experience, current cognitive states (e.g., arousal, affect, motivation), and physiological states (e.g., allostasis).[16] The prediction function serves as a prognostic budgeting process, enabling the brain to anticipate the energy required for the task[8] and ultimately prevent system failure.[16] It is during the next steps that we first understand the possible deception perceived through activity mode.

Our rate of perceived exertion increases progressively during activity, serving as a blueprint to ensure effort does not exceed allostatic limits.[17] The brain's overall prediction model is then constantly updated during and through task performance, relying on afferent feedback from muscular effort.[9] The paradoxical nature of eccentric activity appears to be a *trick* of the brain. Compared to concentric and isometric contractions, eccentric contractions produce significantly greater muscular forces.[18,19] However, multiple studies on upper[18] and lower-limb[20] experiments have shown that individuals perceive less exertion during eccentric activities than during other modes.

This aligns with established exercise physiology principles, as eccentric contractions during the same exercise are associated with significantly lower metabolic cost and motor unit activation.[21] This high-force, low-cost paradox may represent a key deceptive factor contributing to DOMS. Therefore, during an activity involving eccentric loading, there would be no immediate reason for the brain to significantly update its predictive model, as the cost appears to remain within acceptable limits. Only after a *delay* would tissue damage and the subsequent cascade of cellular mediators triggered by the activity's high mechanical forces alert the brain to its error.

From an evolutionary perspective, pain is an adaptive trait designed to promote avoidance learning of harmful stimuli, prevent acute tissue damage, and protect healing tissue, thereby helping to maintain the body's vitality.[7] Pain is understood as a dynamic experience resulting from reciprocal, multi-system interactions.[22] The hyperalgesia associated with DOMS may reflect a damage-mitigation strategy, consistent with the smoke detector principle,[23] used by the body as it attends to its prediction error.

Essentially, the body performs a cost-benefit analysis when facing uncertainty about the severity of a threat; in the case of DOMS, eccentric exercise may trigger a physiological response that contradicts the brain's original risk assumption.[21] Pain has evolved to sometimes be excessive because the metabolic cost of such a response is much lower than the potential fallout of an inadequate reaction, which could lead to catastrophic system failure.[21] Although the actual tissue damage from eccentric loading is less harmful to our system than the hyperalgesic experience suggests, the latter makes sense as a risk-mitigation strategy and a stress response.[21] The predictive understanding of pain may also help explain how repeated activity sessions can reduce the experience of DOMS.

Research on predictive (e.g., Bayesian) pain models shows that, by combining past experiences and predictions with constantly updated sensory input, pain can function both as an accurate estimate and as a response to prediction errors or "surprises."[10] The repeated bouts of exercise effect (RBE) refers to the decrease in DOMS symptoms following a subsequent session of the same eccentric activity.[23] This process is more effective when it occurs shortly after the initial exercise, such as within 24 hours of the first session, and it does not seem to increase tissue damage or slow healing. Although it is unknown how RBE reduces DOMS symptoms,[23] it is possible that a second session of the same activity lessens symptom severity by recalibrating the brain's predictive model of the activity's costs and effects, thereby aligning perception and action while reducing the stress response to the original activity.[7]

Discussion

Traditional models of DOMS, including muscle damage and inflammation theories, provide important insight into the physiological correlates of the phenomenon, particularly structural disruption, enzyme leakage, and inflammatory mediators.[1,3,4] However, these models do not consistently explain the temporal dissociation between biomarkers and perceived soreness.[1] The proposed framework may help account for this mismatch by framing DOMS as a centrally mediated response to uncertainty and prediction error. While nociceptive input remains an important contributor across all perspectives, the predictive model emphasizes the brain's interpretation of these signals rather than their direct translation into pain.

This distinction may also help explain features such as delayed onset and the repeated-bout effect, which are less easily reconciled with purely tissue-based models. Within this framework, structural disruption and inflammation may serve as sources of afferent information that help update predictive models, rather than acting as direct determinants of pain intensity.[10] This interpretation allows for a more flexible understanding of DOMS, in which variability in symptom expression reflects differences in prior exposure, expectations, and environmental context. It also generates testable hypotheses, such as whether interventions that alter expectations or perceptual framing influence the magnitude or timing of DOMS independent of tissue damage. By integrating tissue-level and centrally mediated processes, the proposed framework may extend existing theories by offering a more comprehensive account of how physiological signals are translated into the lived experience of soreness.

Conclusion

Delayed-onset muscle soreness is a common phenomenon among exercisers, characterized by a delayed onset following eccentric activity, with symptoms that typically peak 24–72 hours post-exercise and include hyperalgesia.[1] Although extensive research has identified tissue disruption, inflammatory responses, and circulating biomarkers associated with the phenomenon, these physiological findings do not consistently align.[1,2] As a result, no single tissue-level theory fully accounts for the delayed onset, exaggerated perceptual intensity, and rapid attenuation of symptoms following repeated exposure to the same exercise.[1,5]

Principles from evolutionary biology and predictive processing offer a potentially unifying framework that positions the brain as the central regulator of activity and energy modulation.[8,11,13] In this conceptual framework, DOMS can be seen as a centrally mediated response to a prediction error arising from a mismatch between expected, perceived, and actual mechanical strain.[8,10,11] Eccentric contractions, which generate high muscular forces with relatively low metabolic cost[18] and perceived exertion,[18] may not adequately update the brain's predictive model during activity. The delay in symptom response appears to reflect the time required for the brain to become aware of its error,[10] and the hyperalgesic response might thus serve as a protective mechanism, aligning with evolutionary models of overprotective defensive signaling designed to prevent further strain under uncertain conditions.[7]

Finally, this framework suggests that RBE can be seen as a recalibration or update of the brain's original predictive model. Repeated exposure to the same activity reduces symptom severity, not necessarily by halting tissue damage, but by improving expectations about the activity's energy cost and mechanical effects.[8] This lowers prediction error and weakens the defensive pain response.[7,10] This conceptual framework may help clarify the often ambiguous nature of the phenomenon and lay the foundation for future research on DOMS, viewing its occurrence as an adaptive, predictive signal shaped by evolutionary pressures. This reconceptualization encourages future studies to view DOMS not just as a consequence of tissue damage but as a functional component of an organism's feedback system, in which predictive signals guide behavior, load management, and long-term adaptation to mechanical stress.

Conflict of Interest. The author declares no conflicts of interest.

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