Published in final edited form as:

Pain. 2019 April ; 160(4): 757–761. doi:10.1097/j.pain.0000000000001431.

Movement-evoked pain: transforming the way we understand and measure pain

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Introduction

Nearly 116 million Americans suffer from a persistent pain condition [17]. Persistent pain is most commonly attributed to musculoskeletal conditions like axial and joint pain, and involves an unpleasant sensory experience that interferes with overall quality of life. Indirectly, persistent musculoskeletal pain can also influence a person's health risks, and recent estimates implicate musculoskeletal pain as the global leader in disability [16]. Worse, current treatment pathways result in suboptimal outcomes potentially increasing health risks either directly (e.g. injections and surgery) or indirectly (e.g. consequences of failed opioid pharmacotherapy) [23; 50]. For these reasons, a high priority in public health research is to better understand mechanisms of persistent musculoskeletal pain and how these mechanisms influence subsequent management [46].

Clinical and mechanistic pain research bases its conclusions on assessment of particular pain outcomes. We surmise that *the pain outcome itself* has been a rate-limiting factor in understanding persistent musculoskeletal pain mechanisms. With musculoskeletal pain, the primary driver is often pain experienced during physical activity, commonly referred to as movement-evoked pain [11]. Yet, the most common used pain measures assess either spontaneous pain or pain recalled through the lens of questionnaires (Figure 1) [20]. Assessment of spontaneous pain, that does not involve physical activity, cannot discern pain related to movement. Similarly, questionnaires may inadvertently include movement-evoked pain measurement; however, they are dependent upon the individuals recall capacity and unable to differentiate pain experienced before, during, and after movement [15]. In recent years, a growing body of literature has demonstrated distinct differences between movement-evoked pain and pain at rest. In fact, multiple studies exploring the use of transcutaneous electrical nerve stimulation for treating pain in individuals with fibromyalgia have shown it to be effective in reducing movement-evoked pain, but not pain at rest [18;

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34]. In the context of these findings, the distinction between movement-evoked pain and resting or spontaneous pain is of critical importance, as the literature suggests they are likely driven by different underlying mechanisms.

The purpose of this topical review is to encourage standardized and specific measurement of movement-evoked pain in people experiencing musculoskeletal pain. Burgeoning research supports movement-evoked pain measurement [4; 6; 14], yet it remains slow to translate to clinical care [45]. Here, we will discuss recent calls to understand movement-evoked pain, potential mechanisms underlying movement-evoked pain, measurement gaps and opportunities for modern technology, and future directions for research (Table 1).

Call for Understanding Movement-Evoked Pain

The call to utilize movement-evoked pain measures arises from a recent transformation in our understanding of pain. Historically, our understanding of the relationship between pain and movement has been crude. One reason is that the primary drivers of pain—sensory, psychological, and motor factors—were commonly studied in isolation of each other. A recent conceptual model for studying pain *with* movement proposed an evolution of traditional methods by integrating *all three factors*, which would allow for a better understanding of how pain is interrelated with movement [4]. An important feature of this model is its focus on reciprocal relationships of pain and movement, such that pain affects movement, while movement affects pain. This focus may help in understanding the paradoxical relationship between the prescription of physical activity to treat pain, and participation in physical activity evoking pain [14]. Through the traditional top-down approach and individual focus on pain-related drivers, considerable work has been devoted to answering, “*How does movement affect pain?*” However, less attention has addressed the question, “*How does pain affect movement?*” Therefore, the increased use of movement-evoked pain measures may aid in our understanding of the bi-directional associations of pain and movement, and ultimately lead to a better understanding of the complex phenomenon of persistent pain.

The current review subscribes to similar ideas proposed by Hodges and Smeets, whose previous review provided a contemporary view of how movement changes in pain, the consequences of these changes, and the role of physical activity as treatment for pain [14]. A key premise of the review was that, regardless of whether movement changes are the cause or effect of tissue damage and/or pain, both *too little and too much movement* could be suboptimal for tissue health [25]. Surprisingly, the authors found the interaction between pain, movement, and injury is largely unclear, particularly with respect to intervention design. As a result, the review assigned a high priority for research to examine the dose response between physical activity and symptom exacerbation. The current review attempts to fill some of this gap with a specific call for more science in this area.

In a brief report of individual variation in response to pain, a theoretical model on fear-avoidance was proposed to guide research and management [52]. This model describes the cascade of events that occur after perceptually threatening pain stimulation and builds upon Moseley’s *Imprecision Hypothesis* that suggests persistent pain is a conditioned response to an initially neutral, yet simultaneously occurring, stimulus (e.g., movement) that becomes

associated with a repeated pain response [30]. According to the model, once the stimulus becomes conditioned to independently induce persistent pain, an individual then develops a pattern of pain-related fear and avoidance behavior to prevent or postpone any future encounter of the stimulus. In support of applying this hypothesis to movement, there are several studies demonstrating increased time sitting [13], reduced ambulatory movements (i.e., steps per day) [37], and overall slowness in individuals with persistent pain compared to those without [26]. Therefore, an individual with persistent pain may simply avoid certain patterns of movement to prevent their experience of pain.

Ultimately, the growing body of literature on pain and movement supports the conceptual model for integrating pain with movement [4]. This model considers the Institute on Medicine's identification of persistent pain as a nervous system disease [17], linking it to motor control, and highlights the lack of existing literature focused on both nociception and motor control—a critical knowledge gap preventing advancement in the understanding of pain. As a result, the model encourages an integrated approach to research that is cognizant of the relationship between nervous system pain processing, movement adaptations, and recovery. In other words, this report calls for the transformation from studying pain *and* movement, to studying pain *with* movement [4].

To some degree, our shortcomings in understanding pain during movement may also arise from limitations in capturing such data. Ideally, pain should be captured during free-living conditions. For obvious reasons, this effort has been limited to recall questionnaires. However, recent technological advances may bridge this gap through enhanced networking capabilities of touchscreen mobile devices [32]. With respect to pain, a cloud-based system could use one device to collect pain data, via ecological momentary assessment, that is simultaneously collected with activity data [55].

Movement-Evoked Pain Mechanisms

The classification of movement-evoked pain generally falls into one of two primary categories of pain whose underlying mechanisms may differ: acute and persistent. Acute pain, characterized as a short-lasting response to tissue damage, serves a protective function that acts to prohibit further damage. In contrast, persistent pain is characterized as long-lasting pain that persists after healing has occurred, is often argued to serve no useful biological function, and can be associated with neuroplastic changes within the peripheral and central nervous systems [47]. While movement-evoked pain can certainly arise from both acute and persistent pain, the persistent form is where most issues with disability arise, as persistent pain is the most common cause of long-term disability [21]. Regardless, it is important to consider both peripheral and central mechanisms of movement-evoked pain in the context of both conditions.

Movement-evoked pain is often initiated in response to tissue damage through peripheral mechanisms of inflammation that can drive sensitization via maladaptive changes in the response properties of peripheral and/or central neurons. In overview, a cascade of events ensues locally to facilitate tissue healing and protection; including vascular changes and the release of neurotransmitters, cytokines, and chemokines [5; 43]. Inflammation occurs in the region of damage, which in turn alters nociceptor responsiveness and activity. A key

mechanism specific to movement-evoked pain is the ‘turning on’ of nociceptors previously ‘silent’; which can subsequently be activated in response to normally innocuous joint movement [27; 38]. This mechanism has been experimentally induced by injecting chemical irritant (e.g., carrageenan) directly into the muscle belly to produce inflammation, which in turn generated greater pain upon movement [1; 12]. Evidence suggests an increase of intramuscular nerve growth factor following muscle tissue damage triggers persistent upregulation of nociceptive receptors and neuropeptides that subsequently prime primary afferent neurons for enhanced responsiveness [9]. Other evidence showed inflammation following ischemia-reperfusion muscle injury produced hyperalgesia by sensitizing muscle afferents via upregulation of acid sensing ion channel 3 (ASIC3) —a proton-gated ion channel predominantly expressed in sensory neurons [36]. However, it is important to recognize that movement-evoked pain can also occur in the absence of tissue damage and inflammation. Indeed, in a preclinical model of delayed onset muscle soreness, mechanical hyperalgesia in the absence of tissue damage or inflammation, was mediated by the release of neurotrophic factors from muscle fibers and satellite cells that interact to trigger nociceptor activation [29]. Taken together, this evidence supports both inflammatory and non-inflammatory mechanisms in the peripheral generation of movement-evoked pain.

In addition to peripheral mechanisms, central nervous system pain pathways have been implicated in movement-evoked pain. Indeed, early animal studies documented the particularly strong ability of muscle afferents to generate central sensitization [53]. Additional evidence of central contributions to movement-evoked pain derive from studies of individuals with persistent musculoskeletal pain. For example, among patients with knee OA, quantitative sensory testing measures of central pain processing were associated with movement-evoked pain during functional tasks like the ‘timed up and go’ test and the six-minute walk test [35; 54]. Interestingly, however, these same measures were *not* associated with spontaneous pain [35; 40]. Motor imagery literature provides further evidence, where the mere thought of movement evoked painful symptoms among individuals with persistent arm pain [31]. Preclinical research implicates the involvement of brain pathways that modulate both motor and nociceptive responses. Specifically, Sluka and colleagues reported that activation of *N*-methyl-D-aspartate (NMDA) receptors—a type of glutamate receptor—in the medullary raphe nuclei mediated exercise-induced hyperalgesia [41]. Relatedly, movement perceived as threatening can affect both motor recruitment strategies and gross movement patterns, and is believed to be an underlying link involved in the transition from acute to persistent musculoskeletal conditions [14; 51]. A recent meta-analysis suggests that such threat perceptions likely impact central pain processing by engaging brain networks mediating cognitive and affective responses [22]. Further evidence continues to link psychological constructs, such as pain catastrophizing and/or fear of movement, with movement-evoked pain in persistent musculoskeletal pain conditions, including whiplash-associated disorder, persistent low back pain, and knee osteoarthritis [39; 48; 49; 54]. Even exercise-induced muscle pain, often used as an experimental control in persistent musculoskeletal pain studies, is substantively influenced by these psychological constructs [2; 33], including interactions between psychological factors and genetic predisposition [10]. Other evidence of central mechanisms comes from work exploring the paradoxical effect of movement on pain. Kosek and colleagues demonstrated that exercise activates unilateral,

environments while also collecting movement data simultaneously –some platforms are currently aiming at this target [32; 55]. The future development of these measures is expected to provide important insights into pain experienced with movement to better characterize persistent pain attributed to musculoskeletal conditions. With movement-specific assessment, the study of persistent musculoskeletal pain would finally move beyond the rate-limiting factor of the pain outcome itself to allow for clear, focused attention on pain experienced during movement and would no longer be limited by recall capacity. Such progression would help distinctly identify factors associated with persistent musculoskeletal pain, enhance our ability to further adapt and validate relevant models, and ultimately advance our understanding and treatment of this devastating disease.

Acknowledgments

Dr. Duane Corbett was supported by the National Institutes of Health (NIH)/National Institute on Aging (NIA) (1T32AG049673-01). Dr. Todd Manini was partially supported by the NIH/NIA (R01AG042525) and by the Claude D. Pepper Older Americans Independence Centers at the University of Florida (1P30AG028740). Dr. Roger Fillingim was supported by the NIH/NIA (K07AG046371). The authors declare no conflicts of interest.

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PAIN ASSESSMENT CONTINUUM

Figure 1.

Movement-based dichotomy of pain assessment continuum. Assessment of pain is dichotomized according to pain at rest versus movement-evoked pain, and both types of pain assessment are depicted based on varying temporal frames of reference (i.e. past versus present). Assessment through Pain Recall Questionnaires captures past tense pain at rest, and less commonly movement-evoked pain. In contrast, Spontaneous Pain Assessment captures pain in the present and typically only captures pain at rest. Standardized methods for assessing present movement-evoked pain are needed.

Table 1.

Summary of Research Implications and Directions for Understanding Movement-Evoked Pain

Descriptions of Findings/Directions	References
<u><i>Applied Implications for Understanding Movement-Evoked Pain</i></u>	
•Musculoskeletal pain implicated as leading cause of disability worldwide	[16]
•Paradoxical relationship exists between persistent musculoskeletal pain and movement	[14]
•Traditional approach of studying pain <i>and</i> movement has led to lack of understanding of pain <i>with</i> movement	[4]
•Movement-evoked pain is a multisensory event that includes information specific to movement (e.g., proprioceptive information)	[30]
<u><i>Evidence of Unique Peripherally Influenced Mechanisms of Movement-Evoked Pain</i></u>	
•Experimentally-induced muscle inflammation yields greater pain upon movement	[1; 12]
•Preclinical animal studies describe potential pathways for movement to trigger nociceptor activation	[9; 36]
•Movement-evoked pain found to occur in absence of muscle fiber damage	[29]
<u><i>Evidence of Unique Centrally Influenced Mechanisms of Movement-Evoked Pain</i></u>	
•Treatments may differentially impact movement-evoked pain and pain at rest.	[8; 34]
•Measures of central pain processing associated with movement-evoked pain, but not pain at rest in people with musculoskeletal pain	[35; 40; 54]
•Psychological constructs (e.g., fear of pain) consistently associated with movement-evoked pain in acute and persistent musculoskeletal pain conditions	[2; 10; 33; 39; 48–49; 54]
•Preclinical animal studies describe potential pathways for movement to trigger nociceptor activation	[22; 29; 41; 53]
<u><i>Current Methodological Problems in Pain Assessment</i></u>	
•Activity avoidance may prevent traditional measures from capturing movement-evoked pain	[52]
•Spontaneous pain measures typically do not involve physical activity or consider movement	[20]
•Pain recall questionnaires are dependent on recall capacity and unable to differentiate pain experience before, during, and after movement	[15]
<u><i>Future Directions</i></u>	
•Standardize assessment of movement-evoked pain and establish reliability and validity	
•Examine sensory, psychological, and motor factors in parallel with movement-evoked pain	
•Consider modern technologies to simultaneously measure pain and other health-related outcomes in real-time and during free-living conditions	