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Unraveling the Mechanisms of Manual Therapy: Modeling an Approach

Manual therapy (MT) interventions are a preferred treatment for both health care professionals from a variety of disciplines[14,36,77,82] and patients with musculoskeletal pain conditions.[2,12,53,70] Despite the popularity of MT, systematic reviews only find small to modest effect sizes[43,73] or fail to recommend these interventions.[39,68] In fact, individual clinical practice guidelines for low back pain include differing recommendations for the use of spinal manipulation, indicating conflicting research support.[60] Such findings are not dissimilar to those for other interventions for pain and are attributed to substantial individual variability in treatment response.[32] Subsequently, the clinical decision-making process that guides the use of MT may be best directed at the individual patient on the provider level, rather than using a "one-size-fits-all" approach.[32]

Mechanistic-based approaches to treating individuals presenting with musculoskeletal pain conditions represent a rational targeted approach for personalizing treatment.[26,32,45] There are 2 prerequisites needed to properly implement this approach: first, a mechanism contributing to a clinical population or subpopulation (ie, a homogeneous subgroup) must be identified; second, the biological effects of a treatment should be established. When these 2 prerequisites are met, patients can be matched to an appropriate treatment, allowing for the targeted application of a specific intervention of known mechanisms to patients with presentations amenable to these mechanisms.[15,45] Mechanistic-based treatment approaches for MT necessitate identification of the key mechanisms through which MT works; however, the current understanding of these mechanisms is lacking, requiring additional and more optimally designed studies to answer this important question.

SYNOPSIS: Manual therapy interventions are popular among individual health care providers and their patients; however, systematic reviews do not strongly support their effectiveness. Small treatment effect sizes of manual therapy interventions may result from a "one-size-fits-all" approach to treatment. Mechanistic-based treatment approaches to manual therapy offer an intriguing alternative for identifying patients likely to respond to manual therapy. However, the current lack of knowledge of the mechanisms through which manual therapy interventions inhibit pain limits such an approach. The nature of manual therapy interventions further confounds such an approach, as the related mechanisms are likely a complex interaction of factors related to the patient, the provider, and the environment in which the intervention occurs. Therefore, a model to guide both study design and the interpretation of findings is necessary. We have previously proposed a model suggesting that the mechanical force from a manual therapy intervention results in systemic neurophysiological responses leading to pain inhibition. In this clinical commentary, we provide a narrative appraisal of the model and recommendations to advance the study of manual therapy mechanisms. *J Orthop Sports Phys Ther 2018;48(1):8-18. doi:10.2519/jospt.2018.7476*

KEY WORDS: *manipulation, mobilization, neurophysiology, pain, theory*

The Need for a Model of the Mechanisms of MT

The mechanistic approach to MT is complicated by the complex nature of MT interventions. While drug effects are often attributed to a specific and well-defined active ingredient, the mechanisms underlying complex interventions, such as those used for MT, are multifaceted and comprise specific and nonspecific factors related to the intervention, the patient, the provider, and the environment in which the intervention is provided. Subsequently, a single, well-defined mechanism of an MT intervention is unlikely, and resulting outcomes are probably related to varying inherent elements and contextual factors.[13,25,74] We believe that research focusing on individual mechanisms in isolation will always fall short of providing meaningful insight, because MT is a complex intervention involving

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multiple interactions of complementary mechanisms. As with other complex interventions, MT providers and researchers benefit from a theoretical model to both guide the design and assist in interpreting the results of mechanistic studies.

We published a model to begin to account for the multiple pain inhibitory mechanisms of MT.[6] The model postulates that the mechanical stimulus from an MT intervention results in neurophysiological responses within the peripheral and central nervous systems responsible for pain inhibition (**FIGURE 1**). Importantly, the model is applicable to different MT approaches (ie, joint mobilization, massage, neurodynamic interventions) and not intended to emphasize any single or specific approach. The model was designed to comprehensively account for the interacting mechanisms behind a complex MT intervention. Importantly, the model allows researchers (1) to consider and account for competing mechanisms when designing studies (ie, mechanisms related to biomechanical effects, peripherally mediated effects, spinal cord–mediated effects, and supraspinally mediated effects), and (2) to acknowledge the potential for alternative plausible explanations to their findings should the study not account for competing mechanisms.

This clinical commentary will address the current state of the MT mechanistic literature within the context of our model, as well as highlight key areas for advancing this area of research. For the model to continue to be relevant, specific issues related to its future application are considered. Importantly, this commentary is not intended to be a systematic review or complete appraisal of the original model. Rather, the commentary highlights areas that we believe are important considerations for progressing clinical and research perspectives.

Advancing the Understanding Through Appropriate Study Design

Mechanistic studies of MT are often performed in humans, which, unlike animal models, prohibit direct observation of the

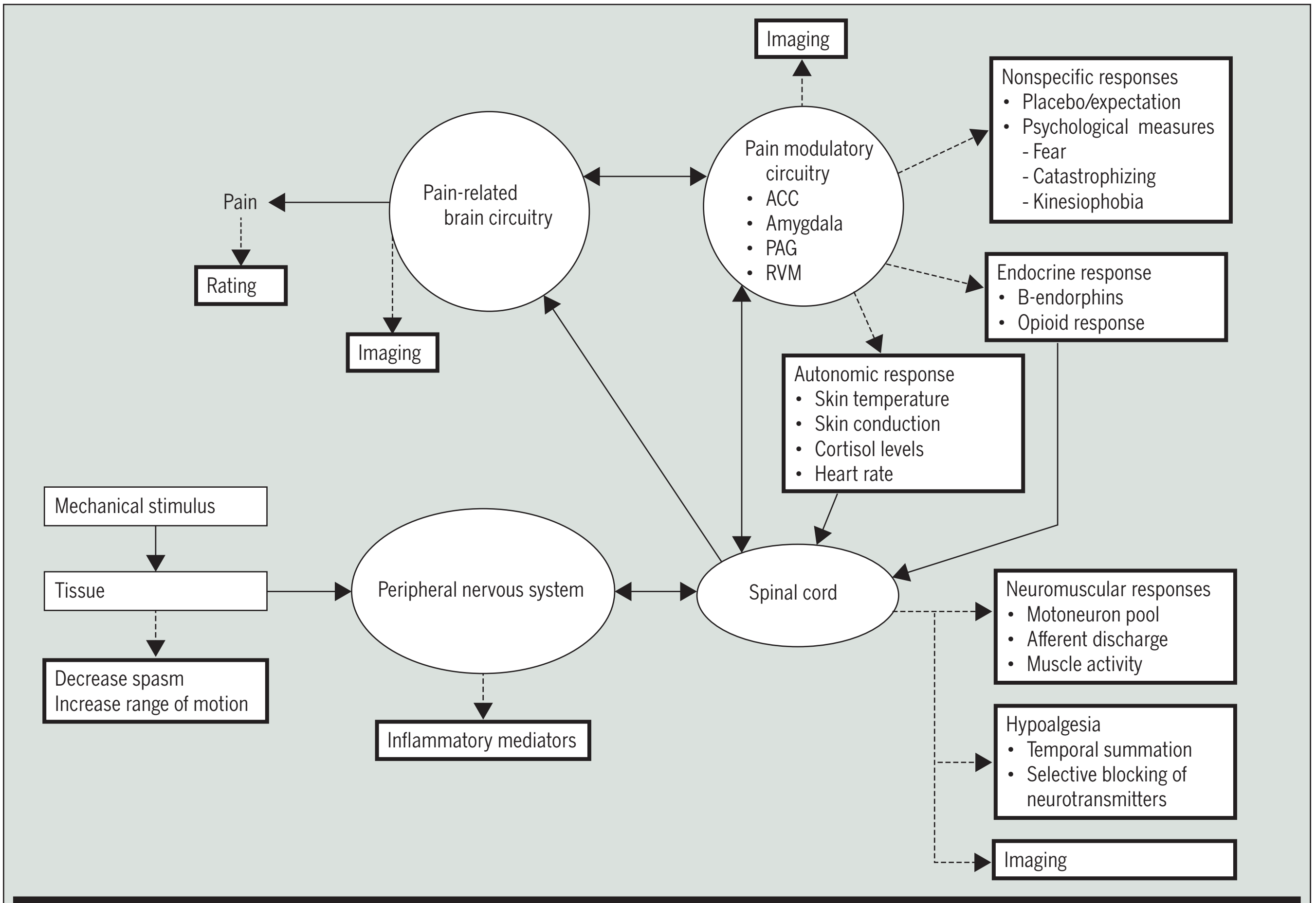

FIGURE 1. Comprehensive model of the mechanisms of manual therapy. The model suggests that a transient, mechanical stimulus to the tissue produces a chain of neurophysiological effects. Solid arrows denote a direct mediating effect. Broken arrows denote an associative relationship, which may include an association between a construct and its measure. Bold boxes indicate the measurement of a construct. Abbreviations: ACC, anterior cingulate cortex; PAG, periaqueductal gray; RVM, rostral ventromedial medulla. Reprinted from Bialosky et al,[6] with permission from Elsevier. ©2009 Elsevier.

nervous system. Our model based the assessment of nervous system responses to MT in humans on associated responses serving as behavioral correlates (ie, proxy measures) of underlying mechanisms. For example, changes in skin blood flow represent an indirect correlate of the sympathetic nervous system responses to MT,[104] while changes in the flexor withdrawal reflex may represent a spinal cord–mediated response to MT.[24] Numerous studies have provided evidence of immediate neurophysiological responses following MT; however, while serving as proof-of-concept work for more complex designs, single pre/post randomized controlled trials are not designed to determine the individual or combined influential factors of clinical improvement. Future studies must establish a link between these associated responses and clinical symptoms, as well as establish covariance of improvements between associated responses and clinical outcomes. Evaluating these multifactorial relationships requires complex study designs that are not always feasible to conduct in clinical settings. Cook[18] has highlighted the limitations of reliance on immediate assessment of either mechanistic or clinical outcomes, including similar findings in response to numerous interventions and the failure to relate these to long-term clinical outcomes. One strategy to address these concerns and to advance this line of research in future studies is to attempt to distinguish these immediate associated responses as treatment mediators and moderators. Mediators are variables measured during the course of treatment to evaluate for change and subsequent impact on outcome.[62,76] Mediators have been described as process variables that implicate possible mechanisms by which an intervention may be effective, especially when these variables represent a plausible construct that the treatment is intended to modify. Potential mediators of change establish how or why treatment effects occur and should be identified a priori and measured before, during, and after treatment to establish temporal precedence with an outcome. For example, spinal stiffness and lumbar multifidus recruitment were assessed at baseline and immediately following a spinal manipulative therapy intervention over 2 sessions, and then a week following the second session, in participants with low back pain.[38] Improvements in the Oswestry Disability Index were mediated by improved lumbar multifidus recruitment and decreases in stiffness.[38] Moderators are variables measured prior to treatment that interact with a specific intervention and influence an outcome of interest often identified in a randomized clinical trial.[62,76] For example, secondary analysis of the UK BEAM trial found that, although several baseline factors predicted overall outcome, none were predictive of response to a specific treatment (ie, spinal manipulation, exercise, or spinal manipulation followed by exercise), with only trends identified for the role of positive treatment expectations for those receiving combined treatment.[93] Identifying treatment-effect moderators provides information to establish "for whom and under what conditions" treatment is effective.[76]

Advancing the Understanding of the Mechanical Force

Clinical use of MT is traditionally driven by the assumption of a peripherally acting, mechanical mechanism,[10,33,52] for example, the application of a specific MT technique applied to a perceived dysfunctional vertebral segment identified through passive movement assessment or imaging. Our model acknowledges a mechanical force as an inherent element of any MT intervention and directs studies to account for mechanical force as a potential contributing mechanism. Based on the literature at the time, the model theorized that clinical outcomes were related to corresponding neurophysiological responses and occurred independent of the specific mechanical parameters of the force. Little has changed to support a mechanism related to the specific biomechanical parameters of the interventions since the model was originally published, and, in fact, more recent studies continue to refute a specific biomechanical mechanism. The clinical examination process for determining biomechanical dysfunction continues to be unreliable,[97] relates poorly to clinical outcomes,[85] and demonstrates a poor association with reliable and accurate mechanical measures[61] as well as with magnetic resonance imaging.[63]

Specific to clinical outcomes, significant within-group improvements are observed in response to MT interventions; however, between-group differences are not observed, confirming similar responses to techniques of varying mechanical parameters.[19,55,100] Furthermore, the clinical outcomes of MT interventions, whether based on clinical presentation or random allocation, are similar.[31,59] Collectively, this body of literature continues to support our initial assertion against an isolated and specific mechanical mechanism accounting for clinical outcomes in response to complex MT interventions.[6] Despite this evidence to the contrary, the clinical approach to MT based on a theorized specific biomechanical mechanism persists.[1,29,30,42,95] We believe that this perpetuation of dated modes of action for MT is both unsubstantiated and counterproductive.

Advancing the Understanding of MT-Related Pain Inhibition

Our model was designed to account for the mechanism of MT on pain inhibition.[6] Psychophysical testing, such as the application of standardized noxious thermal or mechanical forces, allows for the study of mechanisms related to changes in pain processing. Systematic reviews support a transient pain inhibitory effect of MT[21,40,69] on psychophysical measures, occurring both locally and remotely. Higher pain sensitivity, as determined by a lower pain threshold at the site of injury or pain, may reflect local sensitization in the peripheral (reduced receptor threshold) or central nervous system (specific somatosensory regions), while higher pain sensitivity at sites distant from the site of injury may reflect more general sensitization of the central nervous system. Changes in pain sensitivity are observed in response to MT both at the site of application and at distal sites, indicating the presence of a central mediating effect.[22,64,81] The approach of such studies is often limited to assessment of static measures of mechanical and thermal pain thresholds, providing little insight into individual pain modulation capacity.

Psychophysical testing protocols allow for the assessment of in vivo pain modulatory capacities and profiling of individuals based on response to nociceptive input. For example, conditioned pain modulation is characterized by a reduction of pain sensitivity at one site in response to nociceptive input at another site and reflects descending inhibition of pain through the spino-bulbar-spinal loop, representing a pain inhibitory process.[78,101] Temporal summation, characterized by an increase in pain sensitivity in response to repeated noxious stimulation, represents increased dorsal horn excitability[27,47] and reflects a pain facilitatory process.[45,102] Dynamic psychophysical testing allows for profiling of individuals. For example, those with augmented temporal summation or inefficient conditioned pain modulation are considered at risk for developing a pain condition, experiencing greater pain severity when a pain condition develops, and progressing from acute pain to chronic pain.[102] Conversely, those with blunted temporal summation or augmented conditioned pain modulation may be less likely to develop a pain condition, experience less pain severity when a pain condition develops, and be less likely to progress from acute to chronic pain.[102] Subsequently, pain modulatory profiles may be useful in identifying more homogeneous groups of patients.

Pain modulatory capacities are responsive to MT. For example, we have shown that temporal summation of heat pain is reduced immediately after the application of spinal manipulative therapy, and that these reductions are greater than those following exercise or carefully constructed sham interventions.[7-9,11] Improved pain modulatory capacity, as observed through changes in conditioned pain modulation, has been found to correspond to joint mobilization to the knee in participants with knee osteoarthritis.[23] Subsequently, favorable changes in pain modulatory capacity represent a potential biological effect of MT, possibly informing mechanistic-based treatment approaches. Such approaches have been undertaken in drug trials. For example, duloxetine, a drug that enhances descending inhibition of pain, is more effective in individuals who demonstrate diminished conditioned pain modulation.[103] Furthermore, ketamine, which inhibits temporal summation, is more effective for individuals presenting with heightened temporal summation.[46] A similar approach has not been adequately considered in the field of MT, necessitating further study and a future direction of studies of pain inhibition in response to MT.

Movement-evoked pain offers an alternative pain modulatory measure that should be considered in future mechanistic-focused MT studies. Movement-evoked pain often has a greater association with physical function decline and decreased quality of life than does resting/spontaneous pain.[87,89] For example, pain in response to a repeated lifting task accounted for significant and unique variance in disability beyond a measure of spontaneous pain in participants with whiplash-associated disorder.[66] Differences in magnitude and influence of pain types suggest that different mechanisms and MT effects may also differ between spontaneous and movement-evoked pain. Considering movement-evoked pain may better characterize the pain-relieving properties of interventions providing episodic relief. The literature on transcutaneous electrical nerve stimulation has incorporated paradigms that determine differential pain-relieving effects on movement-evoked pain.[83,94] Movement-evoked pain lessens following an MT intervention,[4,56,57] which suggests that future investigation should differentiate these findings from spontaneous pain, in terms of the magnitude of response as well as the relationship to clinical outcomes of importance to patients.

Advancing the Understanding of Supraspinally Mediated Mechanisms

Previous mechanistic models of MT incorporating nervous system responses took a "reflexive" route, meaning that neurological responses to MT were limited to physiologic or autonomic outputs.[75] Our model acknowledged such processes but advanced the pathway into regions of the nervous system not typically considered as having a "direct" response to MT. The timing of this focus was vital, because when the model was first proposed, limited evidence from human and animal research supported the assumption of MT altering sensory processing in supraspinal structures.[48,65,84]

The understanding of supraspinally mediated mechanisms of MT has progressed greatly since the model was originally published, including studies of MT-associated measures of cortical function through somatosensory-evoked potentials,[48,49] as well as neuroimaging advances through positron emission tomography[72] and functional magnetic resonance imaging (fMRI). Findings from these approaches have significantly advanced the understanding of MT-related changes in cortical function. For example, fMRI has been used to study the effects of MT in several complementary ways. First, fMRI has been used to investigate cortical responses during MT. For example, during the posterior-to-anterior mechanical force produced by MT, activation is observed in medial parts of the postcentral gyrus (S1) bilaterally, the secondary somatosensory cortex (S2), posterior parts of the insular cortex, different

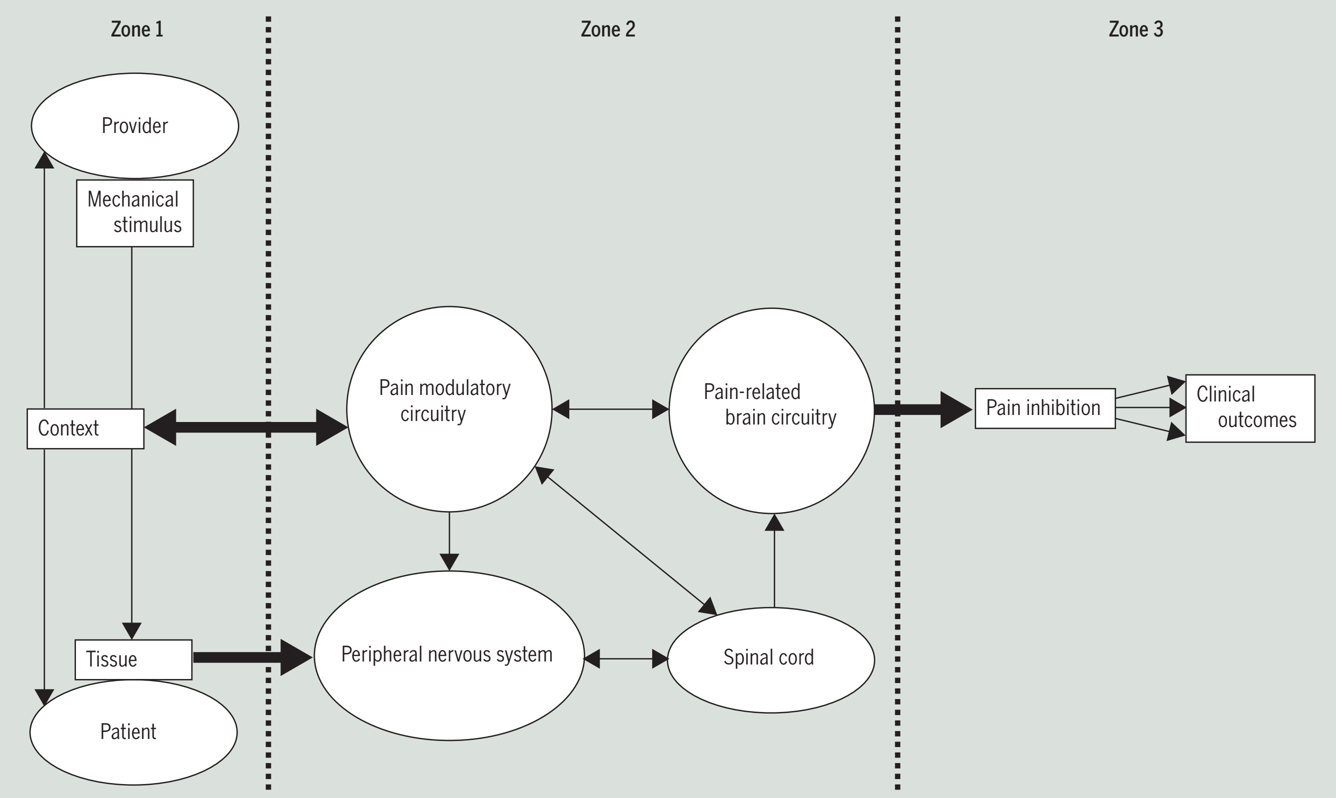

FIGURE 2. Updated comprehensive model of the mechanisms of manual therapy. The model suggests that a transient, mechanical stimulus to the tissue produces a chain of neurophysiological effects. Zone 1 represents the mechanical stimulus from the provider to the tissue, as well as the interaction between the patient and provider. Zone 2 represents potential nervous system responses to the mechanical stimulus, as well as the patient-provider interaction. Zone 3 represents the potential outcomes.

parts of the cingulate cortex, and the cerebellum.[67] Second, fMRI has been used to assess how MT alters the central nervous system responses to a noxious stimulus. For example, healthy volunteers underwent fMRI scanning while receiving noxious stimuli applied to the cuticle of the index finger. Participants then received a supine thrust manipulation directed to the mid thoracic spine and were immediately returned to the scanner for reimaging with a second delivery of noxious stimuli. The thrust joint manipulation was associated with hypoalgesia, as well as a significant reduction in activity in the sensory-motor cortices S1, S2, anterior cingulate cortex, cerebellum, and insular cortices, with reduction of cortical activity correlated to decreased pain perception.[86] Third, resting-state fMRI assessed the coupling of cortical activity between brain regions involved in the processing of nociception before and after MT. Healthy volunteers, who completed an exercise-injury protocol to induce low back pain, underwent resting-state fMRI. They were then randomized into 1 of 3 MT interventions: spinal thrust manipulation, spinal nonthrust mobilization, or therapeutic touch, and then underwent a second resting-state fMRI. Following MT, there was a reduction in experimentally induced low back pain, with no differences observed between types of MT. Common to all MT interventions, the coupling of cortical activity decreased between sensory discriminant and affective regions (primary somatosensory cortex and posterior insular cortex), while increases were observed between affective regions (posterior cingulate and anterior insular cortices) and affective and descending pain modulatory regions (insular cortex and periaqueductal gray).[41] The results of this study suggest that MT alters cortical interactions within nociceptive processing networks at rest, such that subsequent stimuli are received within the cortex in an altered state. Future studies should attempt to further clarify how MT disrupts maladaptive cortical patterns and functional connectivity associated with chronic pain.

Limitations

Methodological approaches to measurement are one of the primary limitations to the study of MT mechanisms, as many techniques described in the model to evaluate nervous system processing are not direct or are isolated measures of nervous system activity. The model is based on associated neurophysiological responses and not direct observation of nervous system activity. Subsequently,

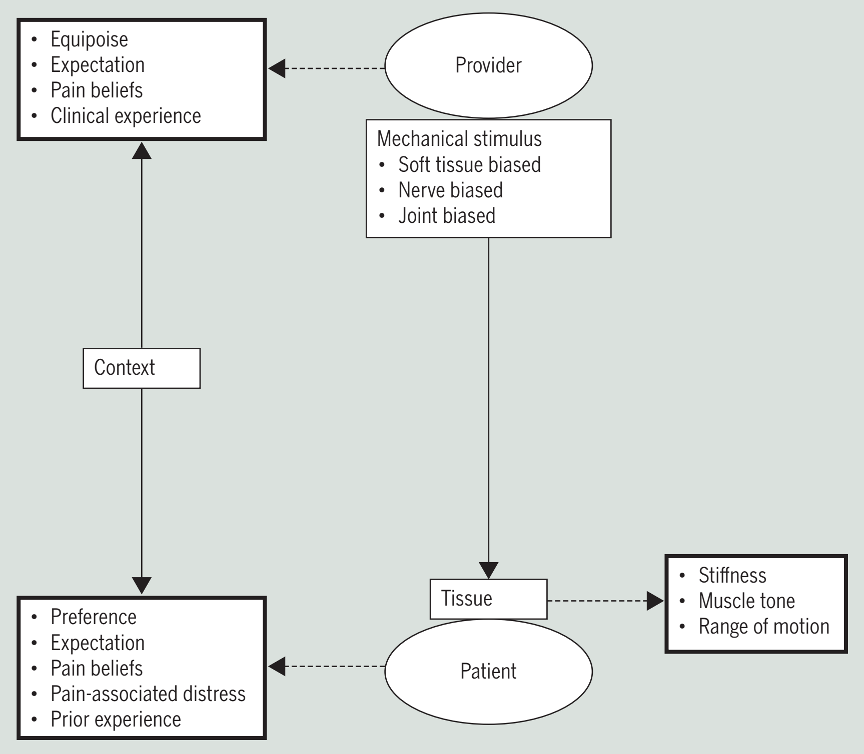

FIGURE 3. Zone 1 of the model, encompassing the interaction between the provider and patient, as well as the mechanical stimulus to the targeted tissue. Solid arrows denote a direct mediating effect. Broken arrows denote an association between a construct and its measure. Bold boxes provide examples of measurable constructs for consideration.

the observed responses are suggestive of specific nervous system activity (generally based on findings from animal studies); however, these assumptions are not directly confirmable in humans, as conducting such studies would introduce valid ethical concerns. The model considers associated neurophysiological responses and attempts to provide direct relationships to clinical outcomes. Importantly, neurophysiological responses to MT are beneficial in furthering our understanding of why MT is effective; however, the gold standard for determining whether MT is effective is patient self-report.[80] The model can be used to guide and account for nervous system responses to MT as a plausible explanation for observed clinical outcomes; however, neurophysiological responses must be linked to patient self-report outcomes and should not be interpreted as a replacement for determining clinically effective interventions.

Advancing the Model

We have modified the model since its initial development to represent some of the key changes in understanding MT mechanisms. For simplicity, we present the revised model in its entirety (**FIGURE 2**) and by individual zones, with zone 1 (**FIGURE 3**) representing the provider, mechanical force, and targeted tissue; zone 2 (**FIGURE 4**) representing patient nervous system responses; and zone 3 (**FIGURE 5**) representing clinical outcomes. The personal attributes of the MT provider (ie, the clinician) comprise one element omitted from the original model. Clinical equipoise is the lack of a preference for an intervention. Equipoise is desirable in clinical trials to avoid bias[20]; however, a lack of equipoise may be desirable in practice, as provider preferences for an intervention have been associated with clinical outcomes. For example, a study comparing the use of spinal thrust manipulation to nonthrust mobilization for participants with low back pain observed no group-dependent differences in pain, disability, total visits, days in care, or rate of recovery; however, a significant association was observed between the treating therapist's lack of equipoise (ie, preference for thrust versus nonthrust mobilization) and subsequent outcomes.[19] Moreover, provider expectations can also influence patient outcomes. For example, baseline physician expectations are predictive of changes in pain and physical function in response to acupuncture in individuals with chronic pain[99] and in return to work following an acute episode of low back pain.[58] Furthermore, pain relief in response to a placebo intervention was significantly greater for a group of individuals following third molar surgery when the provider was aware of the chance of administering an active medication, as compared to when the provider knew that no active drug would be administered.[44] Collectively, provider preference and expectations have strong potential to influence MT outcomes; therefore, we have revised the model to account for both the potential role of provider characteristics in the mechanical force, as well as the potential influence on patient-reported outcomes through a supraspinally mediated effect.

Finally, the model was designed to account for the mechanisms of MT in pain inhibition. However, complete reliance on this aspect of MT may result in limited conclusions and failure to acknowledge overall clinical effectiveness, which is yet another multifactorial construct. More recently, reliance on the sensory aspect of pain as a primary outcome has been discouraged in the case of chronic pain conditions.[90] Core outcome domains for pain have been suggested, including factors such as physical function, emotional function, sleep, and satisfaction with treatment.[91,92] Patients seeking physical therapy care attach importance to improvement in constructs

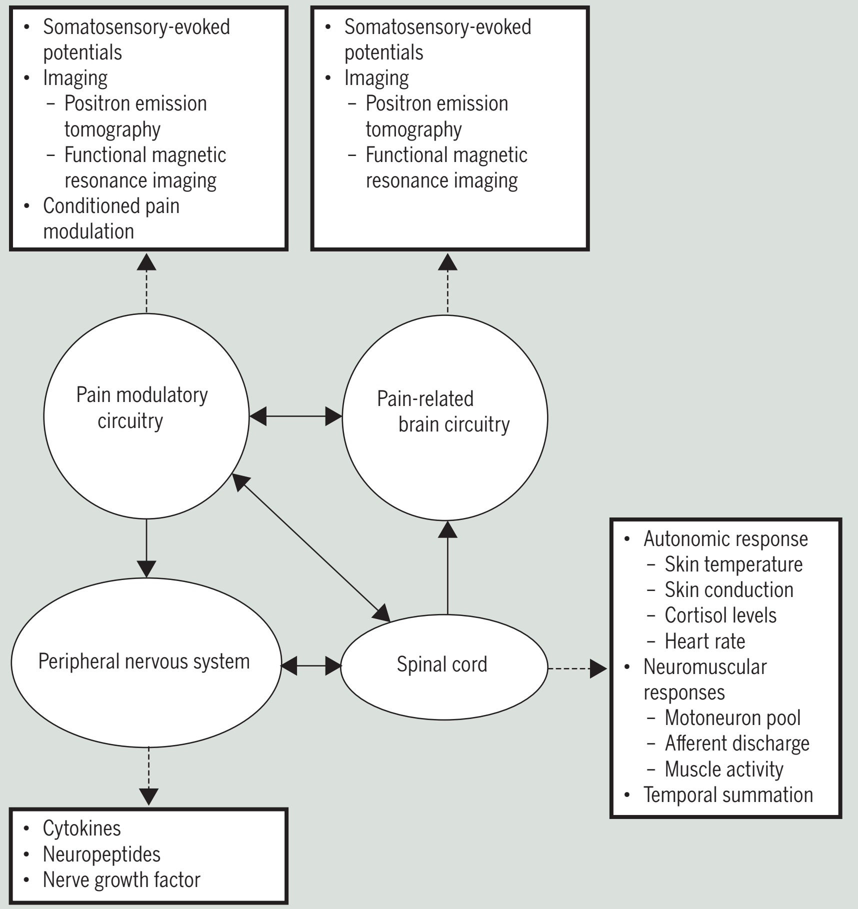

FIGURE 4. Zone 2 of the model, encompassing the nervous system response of the patient to receiving a manual therapy intervention. Solid arrows denote a direct mediating effect. Broken arrows denote an association between a construct and its measure. Bold boxes provide examples of measurable associated responses suggestive of a direct nervous system response.

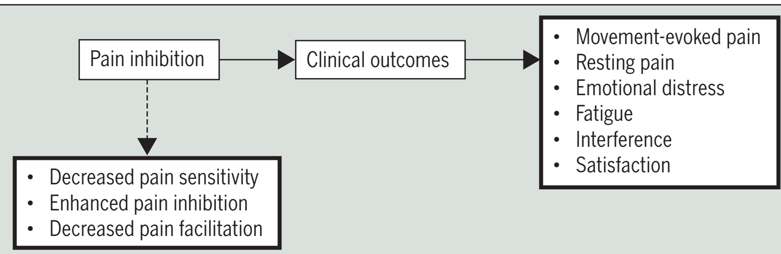

FIGURE 5. Zone 3 of the model, encompassing the outcomes of the model for which related mechanisms of manual therapy may account. Solid arrows denote a direct mediating effect. Broken arrows denote an association between a construct and its measure. Bold boxes provide examples of measurable constructs for consideration.

beyond the sensory aspect of pain,[105] and MT is effective in altering outcomes beyond the sensory aspect of pain.[53,98] We believe that a continued emphasis of the model of the mechanisms of MT in pain inhibition is warranted, because (1) other domains are not mechanistic, precluding a similar approach to study; and (2) pain inhibition is an important precursor to the other domains. However, mechanistic studies should be designed to link MT-related pain inhibition to core outcome domains that are valued from a patient perspective.

CONCLUSION

The implementation of effective MT depends on many factors, including a thorough understanding of the underlying multifactorial mechanisms through which these interventions exert their effectiveness. Determining the mechanisms of MT would both strengthen the best available research and enhance clinical practice through a personalized treatment approach, perhaps resulting in better agreement between clinical judgment, patient preferences, and the available literature. Clinical prediction rules are one approach to stratification initially embraced by MT providers and researchers. Many clinical prediction rules purported to identify key signs and symptoms suggestive of patients with musculoskeletal pain who are likely to benefit from MT.[3,16,28,34,37,54,71,79,96] Despite the initial enthusiasm, the methodology of these approaches has been questioned and cautious interpretation recommended, as initial results may represent spurious findings or a generally favorable prognosis rather than one specific to the effects of MT.[5,51,88] Furthermore, derivation studies require validation, and the vast majority of derived clinical prediction rules lack additional study or have failed attempted validation studies.[17,35,50] While a noble effort, the current state of clinical prediction rules suggests that this approach may not be optimal for identifying MT responders. Subsequently, a different approach is necessary, and mechanistic-based approaches may provide a more robust method.

Study of the mechanisms of MT is made difficult by the complex nature of these interventions, resulting in the interaction of multiple complementary mechanisms. We have published a model that served as the basis for studies to further our understanding of aspects of modulation of pain sensitivity, as well as to guide studies of supraspinal effects of

MT. Recent work suggests limitations to the original model that can be improved by the inclusion of provider factors, the inclusion of movement-evoked pain, and linking findings to a broader spectrum of pain-related outcome domains. Moving forward, we believe that the traditional emphasis on solely biomechanical mechanisms of MT is misguided in focus and limited in scope. Subsequent efforts should focus on a broader understanding of how MT alters processing of nociception to impact the entire pain experience. Specifically, greater consideration of pain modulatory capacity, as determined by dynamic measures of psychophysical testing, consideration of neurophysiological responses to MT, and studies better designed to account for potential mediators and moderators of treatment outcomes will better inform knowledge of this important topic. ◉

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[VIEWPOINT]

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Benefits and Threats to Using Social Media for Presenting and Implementing Evidence

J Orthop Sports Phys Ther 2018;48(1):3-7. doi:10.2519/jospt.2018.0601

In 1995, Darrell Berry wrote a paper suggesting that the internet would evolve from a static archive of documents into a network of users engaging with each other, a term he defined as *social media spaces*.[4,5] Since the publication of his paper 22 years ago, those engaging with social media platforms have increased exponentially, and include approximately 1.94 billion Facebook, 1 billion Whatsapp and Facebook Messenger, 600 million Instagram, and 284 million Twitter users.[20] These numbers do not include users who blog[29] or share resources or opinions through professional social media sites such as LinkedIn (approximately 500 million users).[20]

Berry's idea of a "hybrid or augmented reality" has come to fruition. Over the last decade, social media use for obtaining news has markedly increased in adults.[3] In the United States, 62% of adults acquire news through social media, most commonly Facebook.[15] Concurrent with this increase in use is the ubiquity of source amnesia bias,[28] which relates to giving a high level of credence to information without remembering the quality of the source. In mainstream media, this has led to the growth of "fake news" and "echo chambers"/"filter bubbles," situations in which individuals become insulated from contrary perspectives, even those that are truthful and meaningful.[3]

As a potential high-yield tool for disseminating information that can reach many people,[30] social media is transforming how clinicians, the public, and policy makers are educated and find new knowledge associated with research-related information.[8] It might offer some partial solutions to managing the sheer wealth of information one has to sift through—2.5 million academic publications each year of the possible 50 million or more in existence.[19] Social media is available to all who access the internet, reducing selected barriers to acquiring original source documents such as journal articles or books and potentially improving implementation—the process of formulating a conclusion and moving on that decision. The use of social media for evidence dissemination/implementation of research has both benefits and threats. It is the aim of this Viewpoint to provide a balanced view of each.

Benefits of Social Media for Research Dissemination

Social Media Provides a Quick Method of Information Dissemination and, Potentially, Implementation It is well known that there is a delay in practice implementation once information is published. Traditionally, it takes 17 years to implement new knowledge into clinical

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practice.[25] Reasons for the delay are likely multifactorial, one of which being the slow dissemination of new information to clinicians. To address the traditional delays associated with publishing, most journals have adopted "online, ahead of print" publication options to precipitate the availability of information. However, this strategy still requires the end user to access the journal's website or to conduct a formal search to identify the article. Social media may improve the dissemination of information by reducing the barriers associated with traditional information gathering, through the sharing of information in social media communities.

Social Media Offers a Broader Scope of Potential End-Point Users (Patients, Clinicians, Researchers, and Policy Makers) The online and open nature of social media provides individuals from low-resource regions with improved access, reducing geographical restrictions,[18] though it should be noted that this benefit is not universal. For example, at the writing of this Viewpoint, access to Twitter is blocked in North Korea, China, and Iran. As conceptualized by Berry,[5] social media can create communities in which the creators of the information are accessible to users of the information. This mechanism expands one's potential audience, allowing individuals who once had no voice (eg, patients, clinicians) to now engage on perspectives that used to be controlled by a select few.[10] This serves as a democratizing process that can enrich conversations about evidence and its links to practice.

Social media plays an important role in direct public engagement[10] and, potentially, public policy stimulus.[21] Researchers and policy makers have very different goals and barriers when disseminating information to the public.[16] A goal of research is to provide steady, long-term contributions to knowledge within a spectrum of scholarly oversight. Researchers are interested in traditional publication metrics, peer acceptance, and perhaps enhancing their personal visibility and profile. Public policy makers are interested in timely research that is both relevant and derived from trusted sources, and that can meet the needs of their constituents.[16] Engaging researchers, clinicians, patients, and policy makers is a potentially useful benefit of social media, one that could hypothetically meet the goals of all groups. Examples of effective use of social media for disseminating information to the public include information on diabetes care,[1] sports injury/concussion management,[2] and the management of procedural pain in infants.[9]

Social Media Can Be Used as a Mechanism for Postpublication Review Social media is a useful platform to discuss the relevance of publication findings[11] and provides an additional channel for authors to expand beyond the confines of publication. Social media also provides an opportunity for critiquing publications that are accepted in mainstream publishing. Peer review is an imperfect system. Traditional peer-review processes have at times led to the publication of papers with nonreproducible results, incomplete or poorly described methodologies, or manipulated/potentially fraudulent data.[31] Social media provides a way to debate findings with a pace and immediacy that would be simply impossible through a journal's website or in a formal medium such as a letter to the editor. Open peer review has already been implemented (https://f1000.com/), allowing transparent reviews and free access to viewership.

Threats of Social Media for Research Dissemination

Social Media Has the Potential for Proliferation of Echo Chambers and Filter Bubbles Social media works best for health professionals when it is used to combat misinformation as vigorously as propagating information.[10] This requires an environment in which users respectfully debate information to challenge one another's thoughts and to correct misrepresentation. Unfortunately, there is a tendency to follow those on social media who have similar thoughts and beliefs.[10,22] Doing so can lead to tribal exchanges among self-segregated groups that filter the information people receive so that it largely supports their existing opinions.

There Is Presently a Void of Knowledge on the Utility of Social Media for Implementation to End Users There is no known evidence to show whether the high-yield options of social media lead to better learning, better patient care, or improved dissemination.[30] Traditional means of information incorporate safety mechanisms such as errata and retractions of papers, providing formal processes to alert and inform end users of newly discovered errors in published work. Because of the fluid nature of social media, there is no formal mechanism to retract an inaccurate statement.[10] This can lead to false information competing equally with correct information. Furthermore, although dissemination may be improved, we may find that social media has no effect on implementation of findings.

Those Who Disseminate Evidence Via Social Media May Not Always Be the Best Qualified to Do So In 2014, Hall[17] created the Kardashian Index (K-index). The K-index is the discrepancy between a scientist's social media profile and publication record in peer-reviewed journals. The index was created because it was well recognized that those who had the largest online following were frequently not those who generated the evidence that was being discussed. Contextually, the K-index[17] defines categories of disseminators:

(A) True positive: someone with a highly visible social media profile and high publication record (gold standard)
(B) False positive: someone with a highly visual social media profile but low publication record
(C) False negative: someone with a low visual social media profile but high publication record
(D) True negative: someone with a low visual social media profile and low publication record (not pertinent to our discussion)

In the social media worlds, the false negative voice is ignored and/or infrequently heard, whereas the false positive voice is well represented. This is worth noting, because the cascade size (proliferation of social media buzz) is highly dependent on the initial source of the information.[23] When the source lacks understanding, the information may be presented incorrectly, which can directly influence implementation by the end user. It is especially problematic when public figures (eg, athletes and actors) interpret and endorse scientific issues about which they have little knowledge.

The misrepresentation of research findings and "spin" are substantial problems in the research world.[6,14,24] Even experienced researchers may find it difficult to interpret, share, or present their results carefully and impartially. The situation worsens when the presenting individuals lack understanding of the research and their intentions are injudicious. Indeed, many social media pioneers have used this platform for personal gain; social media has been known to markedly enhance one's professional image.[22]

Social Media Is an Environment in Which Poor Professionalism Goes Unchecked Another concerning element of disseminating evidence through social media is professionalism.[8] Social media platforms, such as Twitter, have been used to bully or to disseminate racist, sexually inappropriate, or sexually explicit material.[7] Further, the inability to identify nonverbal behaviors and other features so important in messaging of information can lead to misinterpretation of information, especially when humor or sarcasm is an element of a post.[22] On character-limited platforms such as Twitter, important detail and nuance, often vital to communicate complex positions, are omitted. On the other side of this coin, critics can use objections about the tone of a message as an excuse to disengage from it.[33] Where the line lies between legitimate robust, challenging, and lively discussion and bullying or unprofessional behavior is notoriously unclear, and this issue plays to a wider one regarding censorship and freedom of expression.

RECOMMENDATIONS

We recognize that social media may contribute to and possibly revolutionize the dissemination/implementation of research evidence and propose the following thoughts and recommendations.

Social Media Buzz Isn't Always Associated With Traditional Measures of Information (Publication) Quality

Traditionally, the importance of research is measured through its citation (impact factor) and its incorporation into clinical practice guidelines. Social media buzz is measured through metrics such as Altmetrics importance measurements, which are derived from social websites and are increasingly advocated and used as early indicators of article influence.[32] However, at present, the results are mixed. Outside of Twitter, social media metrics for publishing are nominal. When comparisons have occurred, some have found social media buzz to predict future citations,[13] whereas others have found low to no correlations between social media and traditional publishing metrics.[12]

We suggest that disseminators of research who use social media should provide a PubMed link (or equivalent) to the social media report[11] to allow the end users to verify findings and judge the quality of the work themselves. This could combat exaggerated claims generated from inadequately powered, poorly designed studies with a high risk of bias. We also suggest that one never blindly accept a polarizing recommendation from a social media disseminator without reading the original article first, assessing the risk of bias of the study through appropriate means, and considering its place within the wider context of the literature. We would also suggest a further step: before engaging with, and judging the worth and importance of, a given paper, take a moment to reflect on one's own personal biases, motivations, and tribal affiliations.

Consider the Source of the Information and the Potential Conflicts of Interest

There is an unfortunate commonality of conflicts of interest in research. Good research is costly and often cannot occur without the assistance of external funding. One example of external subsidy is industry funding. Although strikingly underreported in the peer-review publication process,[27] there is a strong association between improved outcomes and the funding source of the study reporting the outcomes.[26] Social media is not immune to these problems. Many individuals who promote research via social media have conflicts of interest, and choose to endorse study findings that support their own monetary interests.[7] Examples of conflicts of interest may vary markedly, but can include endorsing a clinical philosophy that is tied to one's continuing education platform, clinical practice, and livelihood, or supporting a concept that leads to the purchase of products in which one has a financial interest.

Traditional peer-review publishing leans heavily on the Committee on Publication Ethics and the International Committee of Medical Journal Editors guidelines to vet conflicts of interest, but no such means are available for social media. Conference platform presentations require a slide associated with disclosures before presenting one's works. We propose a similar disclosure of conflicts of interest when discussing content on social media.

Seek Balance on Important Issues: Avoid Echo Chambers and Seek Out Sites That Deliberately Provide a Point/Counterpoint Atmosphere

A hallmark of social media is that it is designed to provide representation from all individuals within a community. This community allows individuals to participate in balanced, thoughtful, professional, and accurate exchange and follow-up. The environment is designed to embrace quality and transferability of information among all participants[10] in a setting that fosters an open, respectful dialog. As discussed in this Viewpoint, at their worst, social media sites have become echo chambers in which clinicians often provide polarizing information without a careful vetting of the evidence. We propose the importance of upskilling in evidence literacy. Upskilling allows clinicians to better understand the strengths and weaknesses of the information they evaluate. Individuals with increased knowledge can participate in a more balanced debate.

CONCLUSION

How, if at all, can we include a diverse range of voices and styles while navigating the complex waters of social media? How can we maintain an acceptable level of professionalism without being hypersensitive to how messages are packaged? Improving our ability in these respects will require all participants to genuinely reflect on whether they are personally on the right side of an undoubtedly difficult line to define. Both sides, those who send and those who receive, need to evaluate carefully the information being shared. Although imperfect, social media can be a useful platform for presenting and assimilating evidence. ◉

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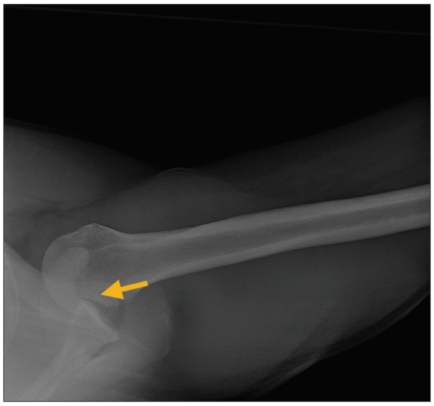

FIGURE 1. An axillary radiograph of the left shoulder, demonstrating an anterior dislocation of the glenohumeral joint and a large Hill-Sachs impaction fracture wedged on the anterior rim of the glenoid (arrow). Immediately after reduction, a second portable axillary radiograph demonstrated successful reduction.

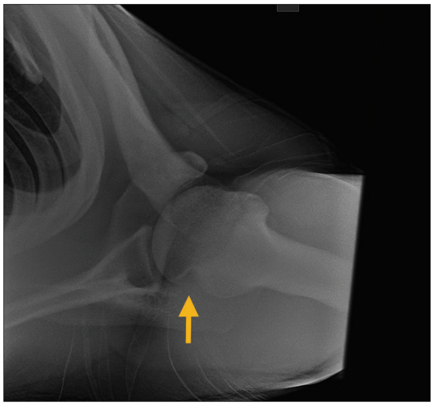

FIGURE 2. An axillary radiograph of the left shoulder, demonstrating a successfully reduced glenohumeral joint. The arrow indicates a large Hill-Sachs impaction fracture deformity, that may engage the glenoid and predispose the patient to recurrent dislocation.

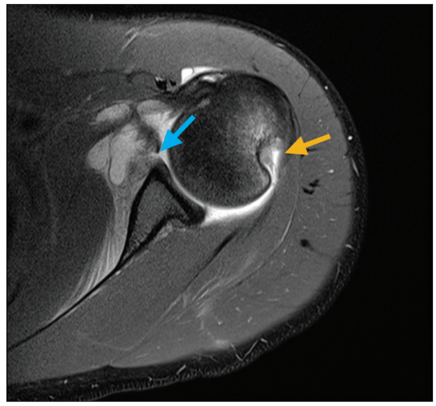

FIGURE 3. Axial, proton density–weighted, noncontrast magnetic resonance image showing a large, trough-like Hill-Sachs impaction fracture (orange arrow). This lesion may engage the glenoid, predisposing the patient to future instability dislocations. An extensive anterior glenoid labral tear is also visible on this image (blue arrow).

Glenohumeral Dislocation With Engaging Hill-Sachs Lesion

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A 20-YEAR-OLD MILITARY CADET injured his left shoulder after landing in an abducted and externally rotated arm position while participating in a mandatory obstacle course. He presented to on-site physical therapists immediately following the event with a complaint of severe pain. Initial observation revealed anterior shoulder fullness, loss of lateral deltoid contour, and adduction of the left arm that indicated anterior dislocation of the glenohumeral joint. Following a negative neurovascular examination, an attempted reduction using the FARES (Fast, Reliable, Safe) method failed.[2]

The individual was taken to the emergency department, where radiographs confirmed an anterior dislocation of the glenohumeral joint and a large Hill-Sachs lesion wedged on the glenoid (**FIGURES 1** and **2**). Following glenohumeral intra-articular injection,[3] successful reduction was achieved using the traction-countertraction method.[2] Results of noncontrast magnetic resonance imaging (**FIGURE 3**) and a computed tomography scan (**FIGURE 4**, available at www.jospt.org) for surgical-planning purposes confirmed the presence of a large Hill-Sachs lesion. Furthermore, the radiologist commented on the presence of a potentially "engaging" impaction fracture.

Hill-Sachs lesions have a greater likelihood of engaging the glenoid rim when the shoulder is in an abducted and externally rotated position, increasing the likelihood of recurrent glenohumeral dislocations.[3] Individuals with structural bone deficits, including engaging Hill-Sachs lesions, are not candidates for isolated arthroscopic labral repair due to the procedure's high failure rate in these individuals.[1] Therefore, this individual underwent arthroscopic remplissage and Bankart labral repair. At the 6-month follow-up, the cadet had returned to full activity, including performing push-ups, without pain or symptoms of instability.[1] ◉ *J Orthop Sports Phys Ther 2018;48(1):50. doi:10.2519/jospt.2018.7609*

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The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of The United States Military Academy, the US Army, or the Department of Defense.

[RESEARCH REPORT]

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Hip and Knee Strengthening Is More Effective Than Knee Strengthening Alone for Reducing Pain and Improving Activity in Individuals With Patellofemoral Pain: A Systematic Review With Meta-analysis

STUDY DESIGN: Systematic review with meta-analysis.

BACKGROUND: The addition of hip strengthening to knee strengthening for persons with patellofemoral pain has the potential to optimize treatment effects. There is a need to systematically review and pool the current evidence in this area.

OBJECTIVE: To examine the efficacy of hip strengthening, associated or not with knee strengthening, to increase strength, reduce pain, and improve activity in individuals with patellofemoral pain.

METHODS: A systematic review of randomized and/or controlled trials was performed. Participants in the reviewed studies were individuals with patellofemoral pain, and the experimental intervention was hip and knee strengthening. Outcome data related to muscle strength, pain, and activity were extracted from the eligible trials and combined in a meta-analysis.

RESULTS: The review included 14 trials involving 673 participants. Random-effects meta-analyses revealed that hip and knee strengthening decreased pain (mean difference, –3.3; 95% confidence interval [CI]: –5.6, –1.1) and improved activity (standardized mean difference, 1.4; 95% CI: 0.03, 2.8) compared to no training/placebo. In addition, hip and knee strengthening was superior to knee strengthening alone for decreasing pain (mean difference, –1.5; 95% CI: –2.3, –0.8) and improving activity (standardized mean difference, 0.7; 95% CI: 0.2, 1.3). Results were maintained beyond the intervention period. Meta-analyses showed no significant changes in strength for any of the interventions.

CONCLUSION: Hip and knee strengthening is effective and superior to knee strengthening alone for decreasing pain and improving activity in persons with patellofemoral pain; however, these outcomes were achieved without a concurrent change in strength.

LEVEL OF EVIDENCE: Therapy, level 1a–.
J Orthop Sports Phys Ther 2018;48(1):19-31. Epub 15 Oct 2017. doi:10.2519/jospt.2018.7365

KEY WORDS: *anterior knee pain, muscle strength, patellofemoral pain syndrome, rehabilitation*

Patellofemoral pain is a chronic condition characterized by retropatellar and/or peripatellar pain that worsens with squatting, sitting, climbing stairs, and running.[44] Although the annual incidence and true prevalence are still unknown, it has been described as one of the most common musculoskeletal conditions presenting to general practice and sports medicine clinics.[45,48] The pain and disability resulting from patellofemoral pain not only limit short-term performance in daily and physical activities, but also have the potential to interfere with long-term social participation, as 90% of patients report pain lasting up to 4 years after the onset of symptoms and 25% report significant symptoms lasting up to 20 years.[29,48]

Although the etiology of patellofemoral pain is not fully understood, the condition is thought to be multifactorial, including both local and nonlocal factors.[11,22,30,32,34] Local factors are related to the patellofemoral joint and surrounding tissues, such as altered mechanics of the joint and impaired quadriceps function.[9,13] Nonlocal factors are related to the mechanics of the distal and proximal joints, such as increased foot pronation and increased hip adduction and

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medial rotation during weight-bearing tasks.[24,42,46] Theoretically, weakness of the hip abductors, lateral rotators, and extensors is thought to lead to excessive hip adduction and medial rotation, which contributes to altered tibiofemoral and patellofemoral joint kinematics and patellofemoral joint stress.[23]

Traditionally, rehabilitation protocols for treating persons with patellofemoral pain have focused exclusively on local factors, such as the use of knee orthoses (eg, patellar taping and bracing) and strengthening of the quadriceps muscles.[5,6,8,41] Although there is a lack of evidence on the use of knee orthoses,[41] knee strengthening increases patellofemoral joint contact area[6] and reduces pain intensity.[5,15,44] It has been suggested that strengthening of the hip abductors, lateral rotators, and extensors, associated or not with knee strengthening, may reduce excessive hip adduction and medial rotation during weight-bearing activities and decrease patellofemoral joint stress. This suggestion is supported by the reported associations among increased hip adduction and medial rotation and weakness of the hip abductors, lateral rotators, and extensors, a deficiency commonly demonstrated by individuals with patellofemoral pain.[35,42] In fact, recent prospective studies have demonstrated that increased peak hip medial rotation angle during a landing task[5] and greater peak hip adduction angle in recreational runners[30] are risk factors for the development of patellofemoral pain. Therefore, the addition of hip strengthening for the treatment of persons with patellofemoral pain has the potential to reduce pain and improve performance of activities of daily living.

To date, 4 systematic reviews have examined the effects of exercise interventions in individuals with patellofemoral pain.[4,37,39,44] The first review suggested that hip strengthening had a positive effect on pain reduction, with effect sizes ranging from 0.54 to 0.62.[4] The second review found that the addition of hip strengthening decreased pain during activity (mean difference, −2.2; 95% confidence interval [CI]: −3.8, −0.6) and usual pain (mean difference, −1.8; 95% CI: −2.8, −0.8), but did not change functional ability (standardized mean difference [SMD], 0.6; 95% CI: −0.4, 1.6) in comparison to knee strengthening alone.[44] However, the findings of this review were based on 4 clinical trials with substantial statistical heterogeneity (I^2 = 82%-90%). The third review found hip strengthening to be effective for improving pain and patient-reported function, with moderate-to-strong effect sizes. However, the absolute values were not provided, and the inclusion of a nonrandomized trial might have introduced bias into the results.[37] The fourth review included 7 randomized clinical trials and concluded that hip strengthening was effective in reducing pain and improving functional capabilities, without changes in strength, compared to no intervention, placebo intervention, or any other type of treatment.[39] A quantitative description of the results was provided, without the benefit of a meta-analysis.[39]

Given that different trials have been examined in different reviews and that previous reviews have included a few studies with substantial statistical heterogeneity or did not pool the results from different trials, a meta-analysis of the current evidence is warranted. The aim of this systematic review was to examine the efficacy of knee strengthening, associated or not with hip strengthening (from now on referred to as hip and knee strengthening), to increase strength, reduce pain, and improve activity in individuals with patellofemoral pain. The specific research questions were:

1. Does hip and knee strengthening increase strength, reduce pain, and improve activity in individuals with patellofemoral pain? Are any benefits maintained beyond the intervention period?
2. Is hip and knee strengthening more effective than knee strengthening alone for increasing strength, reducing pain, and improving activity in individuals with patellofemoral pain? Are any benefits maintained beyond the intervention period?

To make recommendations based on a high level of evidence, this systematic review included only randomized and/or controlled trials.

METHODS

Identification and Selection of Trials

The review was registered at PROSPERO (CRD42015027762). Searches were conducted in the Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, MEDLINE, PsycINFO, and Physiotherapy Evidence Database (PEDro) databases for relevant studies, without date or language restrictions. The search strategy was registered at PubMed/MEDLINE, and the authors received notifications with potential papers related to this systematic review. Search terms included words related to patellofemoral pain and randomized, quasi-randomized, or controlled trials, and words related to strength training (**APPENDIX A**, available at www.jospt.org). Titles and abstracts were displayed and screened by 2 reviewers to identify relevant studies. Full paper copies of peer-reviewed, relevant papers were retrieved and their reference lists screened to identify further relevant studies. The Methods section of the retrieved papers was extracted and independently reviewed by 2 reviewers using predetermined criteria (**TABLE 1**). Both reviewers were blinded to authors, journal, and results. Disagreement or ambiguities were resolved by consensus.

Assessment of Trial Characteristics

Quality The quality of included trials was assessed by extracting the PEDro scale scores from PEDro (www.pedro.org.au). The PEDro scale is an 11-item scale designed for rating the methodological quality (internal validity and statistical information) of randomized trials. Each item, except for item 1, contributes 1 point to the total score (range,

TABLE 1	Inclusion Criteria
Criterion	**Description**
Design	Randomized and/or controlled trials
Participants	Individuals with patellofemoral pain
Intervention	Experimental intervention is strengthening, in order to increase strength of the posterolateral hip muscles (ie, hip abductors, extensors, and/or lateral rotators)
Outcome measures	Measures of strength, pain intensity, or activity
Comparisons	Hip and knee strengthening versus nothing/placebo Hip and knee strengthening versus knee strengthening alone

0-10 points). Reliability of the total score is 0.68 (95% CI: 0.57, 0.76) for consensus ratings.[26] When a trial was not included in the PEDro database, it was scored by a reviewer who had completed the PEDro scale training tutorial.

Participants Studies had to include individuals with patellofemoral pain. Patellofemoral pain was defined as retropatellar pain (behind the patella) or peripatellar pain (around the patella), mostly occurring when load was put on the knee extensor mechanism, such as when climbing stairs, squatting, running, cycling, or sitting with flexed knees. Studies including participants with other knee conditions, such as Hoffa's syndrome, Osgood-Schlatter syndrome, Sinding-Larsen-Johansson syndrome, iliotibial band friction syndrome, tendinopathies, neuromas, intra-articular pathology (including osteoarthritis and rheumatoid arthritis), traumatic injuries (eg, injured ligaments, meniscal tears, patellar fractures, and patellar luxation), plica syndromes, and more rarely occurring pathologies, were not included.[22,44] The number of participants and their age, level of physical activity, and baseline pain intensity were extracted to assess the similarity of the subject populations among studies.

Intervention The experimental intervention had to consist of a hip- and/or knee-strengthening program using body weight, free weights, machines, or elastic resistance. The intervention had to be of a dose that would be expected to improve strength (ie, it had to involve repetitive and/or effortful muscle contractions), and it had to be stated or implied that the purpose of the intervention was strengthening.[2,40] Session duration, session frequency, program duration, and characteristics of the strength training (ie, muscles, type of exercises, setting, load, and progression) were recorded to assess the similarity of the interventions among the studies. The control intervention was defined according to each research question: (1) to examine the efficacy of hip and knee strengthening, the control intervention could be nothing, placebo, or any other non–lower-limb intervention; (2) to examine the effect of hip and knee strengthening compared with knee strengthening alone, the control intervention could be a single-joint resistance training applied to the knee muscles only.

Outcome Measures Three outcome measures were of interest: strength, pain, and activity. The strength measurement had to be reported as peak force/torque generation and representative of maximum voluntary contraction (eg, manual muscle test or dynamometry). When multiple measures of strength were reported, only measures obtained from the trained muscle(s) were used. If it was appropriate to use the measures from several different muscles targeted in the intervention, then the means and SDs of the individual measurements were summed.[1,28]

The pain measurement had to be reported as pain intensity and based on validated self-reporting methods (eg, visual analog scale or numeric rating scale). When multiple measures of pain intensity were reported in 1 study (eg, pain at rest, worst pain, or pain during activity), the means and SDs of the individual measurements were averaged. Questionnaires examining multiple aspects of pain (eg, pain duration and/or pain frequency) were included when pain intensity was separately reported.

The activity measurement had to be a direct measure of capacity or performance. When multiple measures of activity were reported in 1 study, the measure used to calculate the sample size or the measure that combined more activities was used. Questionnaires examining multiple outcomes (eg, Western Ontario and McMaster Universities Osteoarthritis Index) were used if they were the only available measure of activity. The timing of the measurements of outcomes and the procedure used to measure the different outcomes were recorded to assess the appropriateness of combining studies in the meta-analysis.

Data Analysis

Information about the method (ie, design, participants, intervention, measures) and results (ie, number of participants and mean ± SD of outcomes of interest) was independently extracted by 2 reviewers. Disagreement or ambiguities were resolved by consensus. Where information was not available in the published trials, details were requested from the corresponding author.

The postintervention scores and/or change scores were used to obtain the pooled estimate of the effects of the intervention, immediately postintervention and in the long term (ie, after a period of no intervention), using the fixed-effects model. In the case of significant statistical heterogeneity (I^2>40%),[18] a random-effects model was applied. Post hoc sensitivity analysis was planned when the result of the random-effects model was different from that of the fixed-effects model. The analyses were performed using Review Manager Version 5.3 (The

Nordic Cochrane Centre, Copenhagen, Denmark). For all outcome measures, the critical value for rejecting H_0 was set at a level of .05 (2 tailed). The pooled data for each outcome were reported as the weighted mean difference (95% CI) or SMD (95% CI) between the groups. Standardized mean differences were interpreted as small (less than 0.4), moderate (0.4-0.7), or large (greater than 0.7).[18] Where data of trials could not be included in a pooled analysis, the between-group result was reported.

RESULTS

Flow of Trials Through the Review

The electronic search strategy identified 5053 papers (excluding duplicates). After screening titles, abstracts, and reference lists, 52 potentially relevant full papers were retrieved. Forty failed to meet the inclusion criteria and 2 papers were found after the search update. Therefore, 14 papers were included in this systematic review. One of the papers[17] reported a trial with 3 arms (hip and knee strengthening, knee strengthening, and nonintervention group); therefore, 15 relevant comparisons were reported among the 14 included trials. **FIGURE 1** shows the flow of papers through the review.

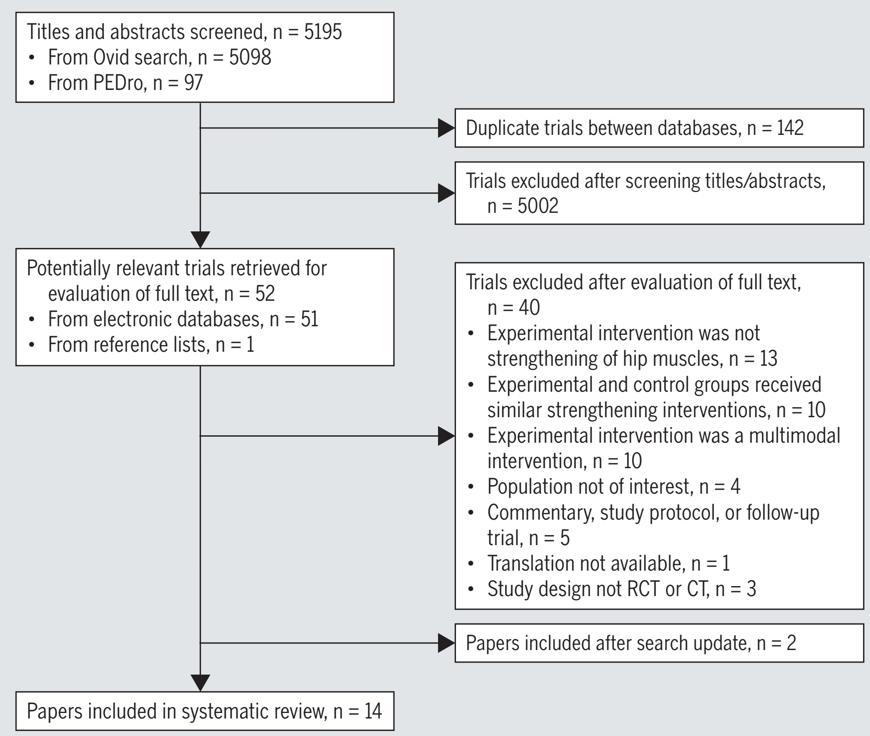


FIGURE 1. Flow of studies through the review. Trials may have been excluded for failing to meet more than 1 inclusion criterion. Abbreviations: CT, controlled trial; PEDro, Physiotherapy Evidence Database; RCT, randomized clinical trial.

Characteristics of Included Trials

The 14 trials involved 673 participants and investigated the effects of hip and knee strengthening for increasing strength (n = 9), reducing pain (n = 14), and improving activity (n = 12) in people with patellofemoral pain (**TABLE 2**). Four trials compared hip and knee strengthening with nothing/placebo, providing data to answer the first study question.[7,17,21,25] Eleven trials compared hip and knee strengthening with knee strengthening alone, providing data to answer the second study question.[3,10,12,14,16,17,19,20,27,36,38] Additional information on 8 papers was requested from the authors.

Quality The mean PEDro score of the trials was 5.8 (range, 3-8) (**TABLE 3**). The majority of trials randomly allocated participants (93%), had similar groups at baseline (86%), had less than a 15% dropout rate (71%), had blinded assessors (57%), and reported between-group differences (86%) and point estimate and variability (93%). However, the majority of trials did not report concealed allocation (57%), and half did not report an intention-to-treat analysis (50%). No trials blinded participants or therapists.

Participants The mean age of the participants ranged from 21 to 35 years across trials. The majority of trials (72%) included participants who reported pain duration of greater than 3 months, with a mean pain intensity ranging from 3 to 8 out of 10 across trials. Four trials included active participants, 6 included sedentary participants, and 4 trials did not report whether the included participants were active or sedentary.

Intervention In all trials, the experimental intervention was strengthening of the hip muscles. In the majority of trials (79%), hip strengthening was accompanied by knee strengthening. The main hip muscle groups targeted in the experimental groups were the lateral rotators (13 trials), abductors (12 trials), and extensors (4 trials). One trial[25] delivered hip strengthening exclusively via functional exercises. Participants undertook training mostly 2 or 3 times per week (9 trials) for an average ± SD of 6 ± 2.5 weeks. Detailed information regarding the type of exercises, load, setting, and progression is provided in **TABLE 2**. The control group received no intervention or placebo intervention in 4 trials, and knee strengthening alone in 11 trials. Four trials delivered additional therapy to both experimental and control groups.

Outcome Measures Measures of strength consisted of maximum voluntary force production obtained during isometric contractions in 4 trials, concentric contractions in 1 trial, eccentric contractions

TABLE 2 Characteristics of Included Trials (n = 14)*

Study	Design	Participants	Intervention: Frequency and Duration	Intervention: Parameters	Outcome Measures
Avraham et al[3]	RCT	n = 20 Age, 35 y Pain duration not reported Pain intensity not reported Activity level not reported	EG: hip and knee strengthening, 30 min, twice per week for 3 wk CG: knee strengthening, 30 min, twice per week for 3 wk Both: TENS and stretching	Muscles: hip lateral rotators and knee muscles Load: not reported Type: body weight Setting: not reported Progression: not reported	Pain: VAS (0-10 cm) Activity: scoring of patellofemoral disorders scale (0-100) Timing: 0, 3 wk
de Marche Baldon et al[10]	RCT	n = 31 Age, 22 ± 3 y Pain duration, 44 mo (range, 3-180 mo) Pain intensity (0-10), 6.4 ± 1.5 Active	EG: hip and knee strengthening, 90-120 min, 3 times per week for 8 wk CG: knee strengthening, 75-90 min, 3 times per week for 8 wk	Muscles: hip abductors, lateral rotators, extensors, and knee and trunk muscles Load: 20%-75% of 1RM Type: body weight, free weights, machines, and elastic resistance Setting: clinics Progression: resistance and/or repetitions increased according to participants' capacity	Pain: VAS (0-10 cm) Strength: dynamometry, Nm/kg Activity: LEFS (0-80) Timing: 0, 8, 20 wk
Clark et al[7]	RCT	n = 27 Age, 28 ± 7 y Pain duration, >3 mo Pain intensity (0-10), 8.0 ± 4.2 Activity level not reported	EG: hip and knee strengthening, 7 times per week for 12 wk CG: nothing Both: education	Muscles: hip abductors, lateral rotators, extensors, and knee muscles Load: body weight Type: body weight Setting: home Progression: difficulty of exercise increased every day	Pain: VAS (0-10 mm) Strength: dynamometry, kgf Activity: WOMAC (0-96) Timing: 0, 12, 48 wk
Dolak et al[12]	RCT	n = 27 Age, 26 ± 6 y Pain duration, 32 ± 34 mo Pain intensity (0-10), 4.4 ± 2.4 Activity level not reported	EG: hip strengthening, 3 times per week for 4 wk CG: knee strengthening, 3 times per week for 4 wk	Muscles: hip abductors, lateral rotators Load: 3% of body weight Type: body weight, free weights Setting: home and clinics Progression: resistance increased every week until 7% of body weight	Pain: VAS (0-10 cm) Strength: dynamometry, Nm/kg Activity: LEFS (0-80) Timing: 0, 4, 12 wk
Ferber et al[14]	RCT	n = 199 Age, 29 ± 7 y Pain duration, 28 ± 35 mo Pain intensity (0-10), 5 ± 1.6 Active	EG: hip strengthening, 3 times per week for 6 wk CG: knee strengthening, 3 times per week for 6 wk	Muscles: hip abductors, lateral rotators, and core muscles Load: 10 maximal repetitions Type: elastic resistance Setting: clinics Progression: sets, repetitions, and/or duration of exercises increased according to participants' feedback and symptoms	Pain: VAS (0-10 cm) Strength: dynamometry, Nm/kg Activity: AKPS (0-100) Timing: 0, 6 wk
Fukuda et al[17]	RCT	n = 64 Age, 25 ± 7 y Pain duration, >3 mo Pain intensity (0-10), 4.8 ± 2.3 Sedentary	EG: hip and knee strengthening, 3 times per week for 4 wk CG 1: nothing CG 2: knee strengthening, 3 times per week for 4 wk	Muscles: hip abductors, lateral rotators, and knee muscles Load: 70% of 1RM or 10RM Type: free weights, machines, and elastic resistance Setting: clinics Progression: resistance adjusted to 70% of maximal strength every week	Pain: NPRS (0-10) Activity: LEFS (0-80) Timing: 0, 4 wk
Fukuda et al[16]	RCT	n = 49 Age, 23 ± 3 y Pain duration, 22 ± 18 mo Pain intensity (0-10), 6.3 ± 1.2 Sedentary	EG: hip and knee strengthening, 3 times per week for 4 wk CG: knee strengthening, 3 times per week for 4 wk	Muscles: hip abductors, lateral rotators, extensors, and knee muscles Load: 70% of 1RM Type: body weight, free weights, machines, and elastic resistance Setting: clinics Progression: resistance adjusted to 70% of maximal strength every week	Pain: NPRS (0-10) Activity: LEFS (0-80) Timing: 0, 12, 24 wk

Table continues on page 24.

TABLE 2 Characteristics of Included Trials (n = 14)*(continued)

Study	Design	Participants	Intervention: Frequency and Duration	Intervention: Parameters	Outcome Measures
Ismail et al[19]	RCT	n = 32 Age, 21 ± 3 y Pain duration, >1.5 mo Pain intensity (0-10), 4.9 ± 1.7 Activity level not reported	EG: hip and knee strengthening, 3 times per week for 6 wk CG: knee strengthening, 3 times per week for 6 wk	Muscles: hip abductors, lateral rotators, and knee muscles Load: not reported Type: body weight and elastic resistance Setting: clinics Progression: not reported	Pain: VAS (0-10 cm) Strength: dynamometry, Nm/kg Activity: scoring of patellofemoral disorders scale (0-100) Timing: 0, 6 wk
Khayambashi et al[21]	RCT	n = 28 Age, 30 ± 6 y Pain duration, >6 mo Pain intensity (0-10), 7.3 ± 1.9 Sedentary	EG: hip strengthening, 30 min, 3 times per week for 8 wk CG: placebo	Muscles: hip abductors, lateral rotators Load: elastic tubing color Type: elastic resistance Setting: gym Progression: resistance increased every 2 wk	Pain: VAS (0-10 cm) Strength: dynamometry, N/kg Activity: WOMAC (0-96) Timing: 0, 8, 24 wk
Khayambashi et al[20]	CT	n = 36 Age, 28 ± 7 y Pain duration, >6 mo Pain intensity (0-10), 7.3 ± 1.7 Sedentary	EG: hip and knee strengthening, 30 min, 3 times per week for 8 wk CG: knee strengthening, 30 min, 3 times per week for 8 wk	Muscles: hip abductors, lateral rotators Load: elastic tubing color Type: elastic resistance Setting: gym Progression: resistance increased every 2 wk	Pain: VAS (0-10 cm) Activity: WOMAC (0-96) Timing: 0, 8, 24 wk
Lun et al[25]	RCT	n = 64 Age, 35 ± 11 y Pain duration, 9 ± 6 mo Pain intensity (0-10), 4.6 ± 2.9 Active	EG: hip and knee strengthening not reported CG: nothing Both: patellar brace	Muscles: hip and knee muscles via squats Load: not reported Type: body weight Setting: home Progression: exercises changed every 5 d	Pain: VAS (0-10 cm) Activity: knee function scale (0-53) Timing: 0, 3, 6, 12 wk
Nakagawa et al[27]	RCT	n = 14 Age, 24 ± 6 y Pain duration, >1 mo Pain intensity (0-10), 4.6 ± 2.8 Active	EG: hip and knee strengthening, 5 times per week for 6 wk CG: knee strengthening, 5 times per week for 6 wk	Muscles: hip abductors, lateral rotators, and knee and transversus muscles Load: not reported Type: body weight and elastic resistance Setting: home and clinics Progression: resistance increased every 2 wk	Pain: VAS (0-10 cm) Strength: dynamometry, Nm/kg Timing: 0, 6 wk
Razeghi et al[36]	RCT	n = 32 Age, 23 ± 3 y Pain duration, >1 mo Pain intensity (0-10), 6.5 ± 1.4 Sedentary	EG: hip and knee strengthening for 4 wk CG: knee strengthening for 4 wk	Muscles: hip abductors and adductors, lateral and medial rotators, flexors and extensors, and knee muscles Load: not reported Type: not reported Setting: not reported Progression: resistance increased according to McQueen progressive resistive technique	Pain: VAS (0-10 cm) Strength: dynamometry, % Timing: 0, 4 wk
Şahin et al[38]	RCT	n = 50 Age, 34 ± 6 y Pain duration, >3 mo Pain intensity (0-10), 3 (3-4) Sedentary	EG: hip and knee strengthening, 30 sessions, 5 times per week for 6 wk CG: knee strengthening, 30 sessions, 5 times per week for 6 wk Both: education	Muscles: hip abductors, lateral rotators, and knee muscles Load: 10 maximal repetitions Type: elastic resistance Setting: clinics Progression: not reported	Pain: VAS (0-10 cm) Strength: dynamometry, Nm/kg Activity: AKPS (0-100) Timing: 0, 6, 12 wk

Abbreviations: 1RM, 1-repetition maximum; 10RM, 10-repetition maximum; AKPS, Anterior Knee Pain Scale; CG, control group; CT, controlled trial; EG, experimental group; LEFS, Lower Extremity Functional Scale; NPRS, numeric pain-rating scale; RCT, randomized clinical trial; TENS, transcutaneous electrical nerve stimulation; VAS, visual analog scale; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.
**Groups and outcome measures listed are those that were analyzed in this systematic review; there may have been other groups or measures in the paper.*

in 2 trials, or concentric and eccentric contractions in 1 trial. One trial[7] did not report the type of contraction used to measure strength. Measures of pain intensity were based on validated self-reporting methods obtained using a numeric rating scale (0-10) in 2 trials and a visual analog scale (0-10) in 12 trials. Pain intensity was reported as "worst pain" in 4 trials, "pain in activity" (eg, ascending stairs or walking) in 4 trials, or "pain in different situations" (eg, pain at rest, worst pain, and pain in activity) in 4 trials. Two trials[3,36] did not report the characteristics of pain measurement. Measures of activity were always based on questionnaires that reflected performance in activities of daily living. The

TABLE 3 PEDro Criteria and Scores for the Included Papers (n = 14)

Study	Item* 1	2	3	4	5	6	7	8	9	10	Total (0-10)
Avraham et al[3]	Y	N	N	N	N	Y	N	N	Y	N	3
de Marche Baldon et al[10]	Y	Y	Y	N	N	N	Y	Y	Y	Y	7
Clark et al[7]	Y	N	Y	N	N	Y	Y	Y	Y	Y	7
Dolak et al[12]	Y	N	Y	N	N	Y	N	Y	Y	Y	6
Ferber et al[14]	Y	Y	Y	N	N	N	N	Y	Y	Y	6
Fukuda et al[17]	Y	Y	Y	N	N	Y	Y	N	Y	Y	7
Fukuda et al[16]	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8
Ismail et al[19]	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8
Khayambashi et al[21]	Y	N	Y	N	N	N	Y	N	Y	Y	5
Khayambashi et al[20]	N	N	Y	N	N	N	Y	N	Y	Y	4
Lun et al[25]	Y	N	Y	N	N	N	N	N	N	Y	3
Nakagawa et al[27]	Y	Y	Y	N	N	Y	Y	Y	N	Y	7
Razeghi et al[36]	Y	N	N	N	N	N	Y	N	Y	Y	4
Şahin et al[38]	Y	N	Y	N	N	Y	Y	N	Y	Y	6

Abbreviations: N, no; PEDro, Physiotherapy Evidence Database; Y, yes.
**Scored items: 1, Random allocation; 2, Concealed allocation; 3, Groups similar at baseline; 4, Participant blinding; 5, Therapist blinding; 6, Assessor blinding; 7, less than 15% dropout rate; 8, Intention-to-treat analysis; 9, Between-group difference reported; 10, Point estimate and variability reported.*

specific instruments used in each trial are listed in **TABLE 2**.

Effect of Hip and Knee Strengthening

Strength The overall effect of hip and knee strength training on strength was examined by pooling postintervention data from 2 trials (n = 70)[7,21] with a mean PEDro scale score of 6. There was substantial statistical heterogeneity (I^2 = 82%), indicating that the variation between the results of the trials was above the variation expected by chance. When a random-effects model was applied, hip and knee strengthening did not significantly change strength compared with no strengthening/placebo (SMD, 0.8; 95% CI: –0.4, 2.1) (**FIGURE 2**). No trials examined the effect of intervention beyond the intervention period.

Pain The effect of hip and knee strengthening on pain was examined by pooling postintervention/change score data from 3 trials (n = 112)[7,17,21] with a mean PEDro scale score of 6.3. There was substantial statistical heterogeneity (I^2 = 81%), indicating that the variation between the results of the trials was above the variation expected by chance. When a random-effects model was applied, hip and knee strengthening significantly reduced pain by 3.3 points out of 10 (95% CI: –5.6, –1.1) compared with no strengthening/placebo (**FIGURE 3**). The maintenance of benefits beyond the intervention period was examined in 1 trial (PEDro scale score, 7/10).[7] The mean difference between groups after 1 year was –3.9 points out of 10 (95% CI: –7.4, –0.4) in favor of the experimental group.

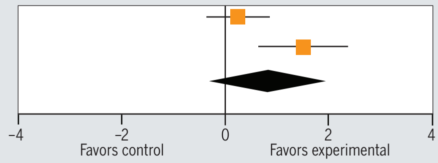
Activity The effect of hip and knee strengthening on activity was examined by pooling postintervention data from 3 trials (n = 114)[7,17,21] with a mean PEDro scale score of 6.3. There was substantial statistical heterogeneity (I^2 = 90%). When a random-effects model was applied, hip and knee strengthening significantly improved activity, with an effect size of 1.4 (95% CI: 0.03, 2.8), compared with no strengthening/placebo (**FIGURE 4**). The maintenance of benefits beyond the intervention period was examined in 1 trial (PEDro scale score, 7/10).[7] The mean difference between groups after 1 year was –12.0 out of 96 (95% CI: –24.7, 0.7) in favor of the experimental group.

Effect of Hip and Knee Strengthening Compared With Knee Strengthening Alone

Strength The effect of hip and knee strengthening, compared with knee strengthening alone, on strength was examined by pooling postintervention data from 6 trials (n = 359)[10,12,14,19,27,38] with a mean PEDro scale score of 6.7. Hip and knee strengthening did not significantly change strength compared with knee strengthening alone (SMD, 0.2; 95% CI: –0.1, 0.4; I^2 = 0%) (**FIGURE 5**). One trial[36] did not provide viable data to be included in the meta-analysis. The effect of intervention beyond the intervention period was examined in 2 trials.[12,38] No significant change was found in strength of the hip and knee muscles between the groups 4 weeks beyond the intervention period (mean difference, 0.4 Nm/kg; 95% CI: –0.4, 1.3)[12] or 6 weeks beyond the intervention period (mean difference, –2 Nm/kg; 95% CI: –10, 6).[38]

Hip/Knee Strengthening Versus Nothing/Placebo—Immediate: Strength

	Experimental Group		Control Group			
Study	Mean ± SD	Total, n	Mean ± SD	Total, n	Weight	SMD IV, Random (95% Confidence Interval)
Clark et al[7]	314 ± 178	20	269.3 ± 157.3	22	53.0%	0.26 (−0.35, 0.87)
Khayambashi et al[21]	13.5 ± 2.6	14	9.5 ± 2.5	14	47.0%	1.52 (0.67, 2.38)
Total		34		36	100.0%	0.85 (−0.38, 2.09)

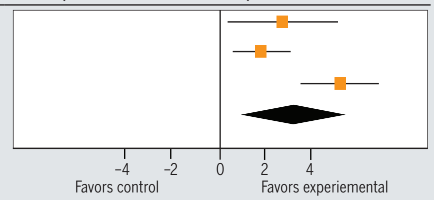


Abbreviations: IV, independent variable; SMD, standardized mean difference.

FIGURE 2. Standardized mean differences of the effect of hip and knee strengthening versus nothing/placebo on strength, immediately after the intervention (n = 70).

Hip/Knee Strengthening Versus Nothing/Placebo—Immediate: Pain

	Experimental Group		Control Group			
Study	Mean ± SD	Total, n	Mean ± SD	Total, n	Weight	MD IV, Random (95% Confidence Interval)
Clark et al[7]	5.8 ± 3.8	22	3.0 ± 3.9	20	28.7%	2.80 (0.47, 5.13)
Fukuda et al[17]	4.55 ± 2.40	23	2.65 ± 1.65	21	37.2%	1.90 (0.69, 3.11)
Khayambashi et al[21]	6.7 ± 2.5	14	1.4 ± 1.9	14	34.1%	5.30 (3.66, 6.94)
Total		59		55	100.0%	3.32 (1.07, 5.56)

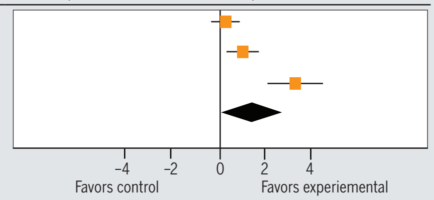


Abbreviations: IV, independent variable; MD, mean difference.

FIGURE 3. Mean differences of the effect of hip and knee strengthening versus nothing/placebo on pain intensity (0-10 scale), immediately after intervention (n = 114).

Hip/Knee Strengthening Versus Nothing/Placebo—Immediate: Activity

	Experimental Group		Control Group			
Study	Mean ± SD	Total, n	Mean ± SD	Total, n	Weight	SMD IV, Random (95% Confidence Interval)
Clark et al[7]	13.8 ± 15.8	22	10.0 ± 11.8	20	35.3%	0.27 (−0.34, 0.87)
Fukuda et al[17]	65.7 ± 13.5	21	51.2 ± 15.1	21	35.0%	0.99 (0.35, 1.64)
Khayambashi et al[21]	59.9 ± 12.6	14	10.7 ± 16.1	14	29.7%	3.30 (2.11, 4.50)
Total		57		55	100.0%	1.42 (0.03, 2.82)



Abbreviations: IV, independent variable; SMD, standardized mean difference.

FIGURE 4. Standardized mean differences of the effect of hip and knee strengthening versus nothing/placebo on activity, immediately after intervention (n = 112).

Pain The effect of hip and knee strengthening, compared with knee strengthening alone, on pain intensity was examined by pooling postintervention/change score data from 10 trials (n = 517)[10,12,14,16,17,19,20,27,36,38] with a mean PEDro scale score of 6.3. There was substantial statistical heterogeneity (I^2 = 82%). When a random-effects model was applied, hip and knee strengthening significantly reduced pain by 1.5 points out of 10 (95% CI: −2.3, −0.8) compared with knee strengthening alone (**FIGURE 6**).

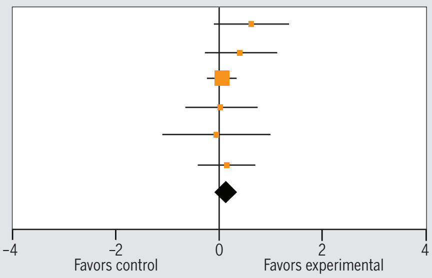
Reduction of pain beyond the intervention period was examined by pooling postintervention/change score data from 5 trials[10,12,16,20,38] (n = 191). Hip and knee strengthening resulted in a significant decrease in pain intensity of 1.9 points out of 10 (95% CI: −3.1, −0.7; random effects) compared with knee strengthening alone 12.0 ± 5.7 weeks beyond the intervention period (**FIGURE 7**).

Activity The effect of hip and knee strengthening, compared with knee strengthening alone, on self-reported activity level was examined by pooling postintervention data from 8 trials[10,12,14,16,17,19,20,38] (n = 471) with a mean PEDro scale score of 6.5. There was substantial statistical heterogeneity (I^2 = 87%). When a random-effects model was applied, hip and knee strengthening significantly improved activity, with an effect size of 0.7 (95% CI: 0.2, 1.3), compared with knee strengthening alone (**FIGURE 8**).

Maintenance of activity beyond the intervention period was examined by

Hip/Knee Strengthening Versus Knee Strengthening Alone—Immediate: Strength

Study	Experimental Group Mean ± SD	Experimental Group Total, n	Control Group Mean ± SD	Control Group Total, n	Weight	SMD IV, Random (95% Confidence Interval)
de Marche Baldon et al[10]	1.77 ± 0.20	15	1.6 ± 0.3	16	8.3%	0.65 (−0.08, 1.37)
Dolak et al[12]	5.2 ± 1.2	17	4.6 ± 1.5	16	9.1%	0.43 (−0.26, 1.12)
Ferber et al[14]	2.66 ± 0.95	111	2.59 ± 1.02	88	55.5%	0.07 (−0.21, 0.35)
Ismail et al[19]	2 .00 ± 0.75	16	1.92 ± 1.90	16	9.1%	0.05 (−0.64, 0.75)
Nakagawa et al[27]	160.2 ± 45.2	7	161.7 ± 35.6	7	4.0%	−0.03 (−1.08, 1.01)
Şahin et al[38]	48.5 ± 15.6	25	45.9 ± 15.7	25	14.1%	0.16 (−0.39, 0.72)
Total		191		168	100.0%	0.16 (−0.05, 0.37)

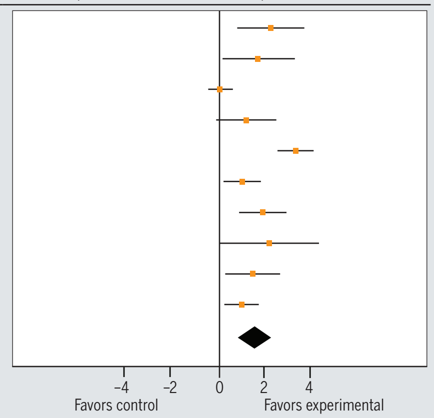


Abbreviations: IV, independent variable; SMD, standardized mean difference.

FIGURE 5. Standardized mean differences of the effect of hip and knee strengthening versus knee strengthening alone on strength, immediately after intervention (n = 359).

Hip/Knee Strengthening Versus Knee Strengthening Alone—Immediate: Pain

Study	Experimental Group Mean ± SD	Experimental Group Total, n	Control Group Mean ± SD	Control Group Total, n	Weight	MD IV, Random (95% Confidence Interval)
de Marche Baldon et al[10]	5.2 ± 1.6	16	3 ± 2.4	15	8.7%	2.20 (0.75, 3.65)
Dolak et al[12]	4.1 ± 2.5	16	2.4 ± 2.0	17	8.3%	1.70 (0.15, 3.25)
Ferber et al[14]	1.99 ± 2.05	88	1.96 ± 1.92	111	12.2%	0.03 (−0.53, 0.59)
Fukuda et al[17]	2.4 ± 2.3	20	1.25 ± 1.90	21	9.3%	1.15 (−0.14, 2.44)
Fukuda et al[16]	4.6 ± 1.6	24	1.3 ± 1.1	25	11.5%	3.30 (2.53, 4.07)
Ismail et al[19]	3.2 ± 0.9	16	2.2 ± 1.3	16	11.4%	1.00 (0.23, 1.77)
Khayambashi et al[20]	5.5 ± 1.6	18	3.6 ± 1.4	18	10.6%	1.90 (0.92, 2.88)
Nakagawa et al[27]	3.00 ± 2.65	7	0.80 ± 0.95	7	6.4%	2.20 (0.11, 4.29)
Razeghi et al[36]	4.81 ± 1.79	16	3.37 ± 1.50	16	10.0%	1.44 (0.30, 2.58)
Şahin et al[38]	2.0 ± 1.1	25	1.0 ± 1.5	25	11.6%	1.00 (0.27, 1.73)
Total		246		271	100.0%	1.54 (0.80, 2.27)

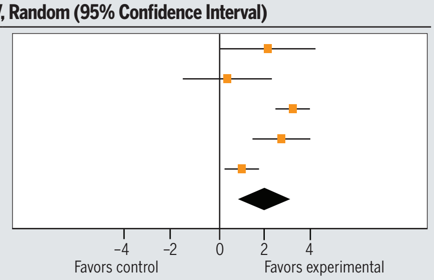


Abbreviations: IV, independent variable; MD, mean difference.

FIGURE 6. Mean difference of the effect of hip and knee strengthening versus knee strengthening alone on pain intensity (0-10 scale), immediately after intervention (n = 517).

Hip/Knee Strengthening Versus Knee Strengthening Alone—Beyond Intervention Period: Pain

Study	Experimental Group Mean ± SD	Experimental Group Total, n	Control Group Mean ± SD	Control Group Total, n	Weight	MD IV, Random (95% Confidence Interval)
de Marche Baldon et al[10]	5.7 ± 2.3	16	3.6 ± 3.3	15	15.1%	2.10 (0.09, 4.11)
Dolak et al[12]	2.4 ± 2.3	11	2.1 ± 2.5	14	15.9%	0.30 (−1.59, 2.19)
Fukuda et al[16]	4.15 ± 1.40	24	0.95 ± 1.00	25	24.3%	3.20 (2.52, 3.88)
Khayambashi et al[20]	5.64 ± 1.99	18	2.92 ± 1.72	18	20.7%	2.72 (1.50, 3.94)
Şahin et al[38]	2.0 ± 1.1	25	1.0 ± 1.5	25	24.0%	1.00 (0.27, 1.73)
Total		94		97	100.0%	1.95 (0.75, 3.14)

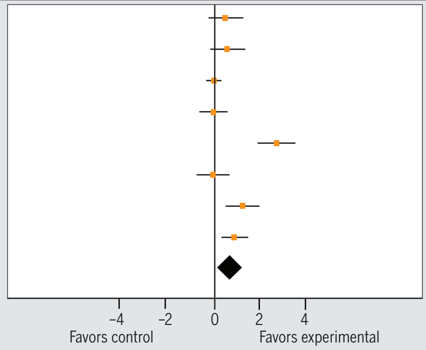


Abbreviations: IV, independent variable; MD, mean difference.

FIGURE 7. Mean difference of the effect of hip and knee strengthening versus knee strengthening alone on pain intensity (0-10 scale), beyond the intervention period (n = 191).

Hip/Knee Strengthening Versus Knee Strengthening Alone—Immediate: Activity

	Experimental Group		Control Group			
Study	Mean ± SD	Total, n	Mean ± SD	Total, n	Weight	SMD IV, Random (95% Confidence Interval)
de Marche Baldon et al[10]	74.3 ± 4.6	15	70.6 ± 8.0	16	12.1%	0.55 (−0.17, 1.27)
Dolak et al[12]	67 ± 11	17	59 ± 14	16	12.2%	0.62 (−0.08, 1.32)
Ferber et al[14]	88.0 ± 11.2	111	87.7 ± 10.5	88	14.4%	0.03 (−0.25, 0.31)
Fukuda et al[17]	65.7 ± 13.5	21	65.6 ± 14.5	20	12.7%	0.01 (−0.61, 0.62)
Fukuda et al[16]	74.1 ± 5.6	25	49.4 ± 11.2	24	11.5%	2.76 (1.96, 3.56)
Ismail et al[19]	85.1 ± 6.2	16	85.0 ± 6.7	16	12.2%	0.02 (−0.68, 0.71)
Khayambashi et al[20]	21.9 ± 16.5	18	6.2 ± 3.9	18	12.0%	1.28 (0.56, 2.01)
Şahin et al[38]	85.4 ± 5.8	25	79.1 ± 7.6	25	12.9%	0.92 (0.33, 1.50)
Total		248		223	100.0%	0.74 (0.17, 1.31)

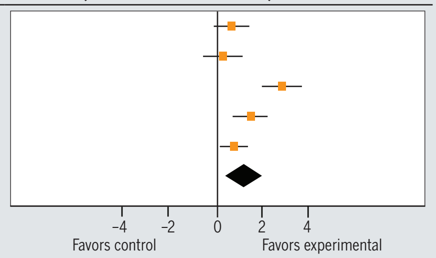


Abbreviations: IV, independent variable; SMD, standardized mean difference.

FIGURE 8. Standardized mean differences of the effect of hip and knee strengthening versus knee strengthening alone on activity, immediately after intervention (n = 471).

Hip/Knee Strengthening Versus Knee Strengthening Alone—Beyond Intervention Period: Activity

	Experimental Group		Control Group			
Study	Mean ± SD	Total, n	Mean ± SD	Total, n	Weight	SMD IV, Random (95% Confidence Interval)
de Marche Baldon et al[10]	74.9 ± 3.9	15	70.4 ± 8.4	16	20.1%	0.66 (−0.06, 1.39)
Dolak et al[12]	70 ± 10	12	67 ± 11	10	19.1%	0.28 (−0.57, 1.12)
Fukuda et al[16]	72.4 ± 6.1	25	47.7 ± 10.5	24	19.4%	2.85 (2.03, 3.66)
Khayambashi et al[20]	23.16 ± 14.15	18	6.94 ± 5.70	18	20.0%	1.47 (0.72, 2.22)
Şahin et al[38]	83.0 ± 6.8	25	77.9 ± 6.6	25	21.4%	0.75 (0.17, 1.32)
Total		95		93	100.0%	1.19 (0.37, 2.01)



Abbreviations: IV, independent variable; SMD, standardized mean difference.

FIGURE 9. Standardized mean differences of the effect of hip and knee strengthening versus knee strengthening alone on activity, beyond the intervention period (n = 188).

pooling postintervention data from 5 trials[10,12,16,20,38] (n = 188). Hip and knee strengthening resulted in a significant improvement in activity, with an effect size of 1.2 (95% CI: 0.4, 2.0; random effects), compared with knee strengthening alone 12 ± 5.7 weeks beyond the intervention period (**FIGURE 9**).

DISCUSSION

This systematic review provides evidence that hip and knee strengthening is effective in reducing pain and improving activity in individuals with patellofemoral pain. The review also indicated that hip and knee strengthening results in greater decrease in pain and improvement in activity compared to knee strengthening alone. Importantly, benefits were maintained beyond the intervention period. Interestingly, the meta-analyses indicated that hip and knee strengthening did not significantly change strength when compared with no exercise/placebo intervention or with knee strengthening alone.

The nonsignificant change in strength found in this review may be explained by the fact that the strengthening interventions were not of sufficient duration and/or intensity. Although the literature indicates a rapid increase in neurological activation of the motor units during the initial phases of strength training, most of the muscle adaptations occur after 8 to 12 weeks of training.[32] The average duration of the strength training in this review was 6 weeks. Only 3 trials,[7,10,21] which investigated 8 to 12 weeks of hip and knee strengthening, provided data regarding strength measures, and their results were considerably higher (SMD, 0.8; 95% CI: 0.1, 1.4; random effects) compared with the pooled effects found in the present review.

Although the strengthening interventions outlined in the reviewed trials were characterized as progressive, they were not administered at the intensity recommended by the American College of Sports Medicine.[2] For example, 1 trial[12] investigated a strengthening program with a load equivalent to 3% of the participant's body weight, when the American College of Sports Medicine guidelines suggest a load of 60% to 70% of 1-repetition maximum for novices.[2] In addition, 5 trials did not report the load applied during strength training. Unfortunately, the majority of trials (9 trials) did not report the duration of the intervention sessions, which could reflect important training properties such as volume of training, contraction velocity, or rest intervals. In summary, the current evidence is insufficient to support or refute the efficacy of strength training to increase muscle strength in people with patellofemoral pain. Further randomized clinical trials, with appropriate training duration and intensity as well as appropriate sample sizes, are warranted.

Despite the lack of strength increases, hip and knee strengthening exercises significantly decreased pain intensity and improved activity in people with patellofemoral pain, with results being maintained beyond the intervention period. The meta-analyses indicated that hip and knee strengthening decreased pain intensity by 3.3 points compared with no exercise/placebo, and by 1.5 points compared with knee strengthening alone. According to Ostelo et al,[31] the cutoff value for minimal important change in pain is 1.5 points (or 30% improvement from baseline). Because the average ± SD pain intensity of the participants in the present review was 5.3 ± 2.5 points, the changes after intervention represent, respectively, 60% and 30% decreases in pain intensity, which are sufficient to be considered clinically meaningful.[31] The meta-analyses also indicated that hip and knee strengthening had a large positive effect on self-reported activity (SMD, 1.4) compared with no exercise/placebo, and a moderate positive effect (SMD, 0.7) compared with knee strengthening alone.

Improvements in pain and activity could be related to the inclusion of weight-bearing exercises (eg, squats), which might have had positive effects on other variables related to patellofemoral syndrome, such as lower-limb pattern of motion[47] and ankle flexibility.[33] In addition, the strength training also may have increased hip and knee muscle endurance, as training intensity and repetitions, in the majority of the trials, were delivered according to the recommended parameters for endurance training.[2] A recent study demonstrated that people with patellofemoral pain exhibit diminished hip muscle endurance compared with healthy controls.[43] However, these hypotheses are speculative at this point, and further research is needed to better understand the effects of strengthening exercises on strength outcomes.

The results of our review are in accordance with a previous Cochrane meta-analysis[44] that demonstrated that hip and knee strengthening decreased pain intensity (mean difference, −1.8; 95% CI: −2.8, −0.8), and add evidence regarding the efficacy of strengthening on self-reported activity. Therefore, this review provides additional evidence on the effect of hip and knee strengthening, as the conclusions are based on meta-analyses of 13 randomized trials and 1 controlled trial of reasonable quality. Furthermore, the results indicate that the decrease in pain intensity and improvements in activity were maintained beyond the intervention period, with moderate-to-large effect sizes, suggesting that benefits were incorporated into daily life.

This systematic review has some limitations. Given that a score of 8 was likely to be the maximum achievable PEDro scale score, owing to the difficulty in blinding therapists or participants, the mean PEDro scale score of 5.8 for the 14 included trials represents moderate quality, suggesting that the findings were credible. Other sources of bias were lack of reporting concealed allocation and whether an intention-to-treat analysis was undertaken. Additionally, the number of participants per group (mean, 24; range, 7-100) was quite low, opening the results to small-trial bias. It is recommended that future randomized clinical trials provide appropriate sample-size calculations so that further systematic reviews can plan sensitivity analyses based on the number of participants.

The current meta-analyses included studies that provided hip strength training and hip and knee strength training to the experimental group, which could be considered a confounding factor. However, the exclusion of the 2 studies[12,14] that provided hip strengthening alone did not change the effects on strength (SMD, 0.2; 95% CI: −0.1, 0.6), pain intensity (mean difference, −1.8; 95% CI: −2.4, −1.1), and activity (SMD, 0.9; 95% CI: 0.2, 1.6). At this time, there is insufficient evidence to indicate that hip strengthening alone is more effective than knee strengthening. Therefore, it is suggested that clinicians provide both hip and knee strengthening to decrease pain and improve activity in people with patellofemoral pain.

Another confounding factor could be the inclusion of 3 studies[10,14,27] that provided trunk muscle training (eg, transversus abdominis). However, the exclusion of these studies from the meta-analyses, again, did not change the effects on strength (SMD, 0.2; 95% CI: −0.2, 0.6), pain intensity (mean difference, −1.6; 95% CI: −2.4, −0.9), and activity (SMD, 0.9; 95% CI: 0.2, 1.7). Based on this information, further systematic reviews should plan subgroup analyses.

Apart from the above-noted limitations, this systematic review has several strengths. Heterogeneity among the trials pooled in the meta-analyses, based on a random-effects model, was low. Overall, the included trials were similar in their clinical characteristics. Most of the trials included adults with moderate-to-high levels of pain intensity, lasting for more than 3 months. Although most of the trials failed to report the session duration, they provided similar session frequencies (mean ± SD, 3.5 ± 1.4 per week) and program durations (mean ± SD, 6.0 ± 2.5 weeks). In addition, this systematic review included 4 recent randomized trials since the last review was published,[39] and also investigated whether the benefits of intervention are maintained beyond the intervention period.

CONCLUSION

This systematic review with meta-analyses provides evidence that hip and knee strengthening is not only effective, but also superior to knee strengthening alone, for decreasing pain intensity and improving activity in people with patellofemoral pain. The results of the meta-analyses, based on 14 trials, indicated that strength training of the hip muscles, accompanied by strengthening of the knee muscles, 3 times a week for 6 weeks can be expected to decrease pain and improve activity in people with moderate-to-high levels of patellofemoral pain. The training benefits are maintained beyond the intervention period. Future studies, with appropriate training duration and intensity, are recommended to elucidate the effects of hip and knee strengthening on increasing strength. ◉

KEY POINTS

FINDINGS: Hip and knee strengthening is not only effective, but is also superior to knee strengthening alone for decreasing pain intensity and improving activity in people with patellofemoral pain. These results were maintained beyond the intervention period.

IMPLICATIONS: Strength training of the hip muscles, accompanied by strengthening of knee muscles, should be included in clinical management of individuals with patellofemoral pain in order to reduce pain and improve activity.

CAUTION: Strengthening interventions were not of sufficient duration and/or intensity, and there is insufficient evidence to support or refute their efficacy in improving muscle strength.

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APPENDIX A

SEARCH STRATEGY

Databases: Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, Ovid MEDLINE, PsycINFO, PEDro.

1. exp patellofemoral pain syndrome/ (549)
2. patella/ or exp knee joint/ or knee/ (62198)
3. arthralgia/ or pain/ (142584)
4. anterior knee pain.tw. (1127)
5. ((patell* or femoropatell* or femoro-patell* or retropatell*) adj2 (pain or syndrome or dysfinction)).tw. (1869)
6. ((lateral compression or lateral facet or lateral pressure or odd facet) adj2 syndrome).tw. (25)
7. ((chondromalac* or chondropath* or chondrosis) adj2 (knee*1 or patell* or femoropatell* or femoro-patell* or retropatell*)).tw. (534)
8. chondromalacia patellae/ (66)
9. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 (202018)
10. randomized controlled trial.mp. or exp randomized controlled trial/ (487814)
11. random allocation.mp. or exp random allocation/ (107923)
12. double-blind method.mp. or exp double-blind method/ (235921)
13. single-blind method.mp. or exp single-blind method/ (33399)
14. randomized controlled trials.mp. (128290)
15. clinical trial.mp. or exp clinical trial/ (931625)
16. exp$ clinical trials.mp. (814)
17. (clinic$ adj trial$).mp. (945789)
18. ((singl$ or doubl$ or treb$ or tripl$) adj (blind$ or mask$)).mp. (388475)
19. exp clinical trials as topic/ or placebo.mp. or exp placebo effect/ or exp placebos/ (623702)
20. (randomised controlled trial or randomised clinical trial).mp. (31292)
21. randomly allocated.mp. (35345)
22. 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 (1640886)
23. exp exercise therapy/ or exercise.mp. or exp exercise/ (341453)
24. rehabilitation.mp. or exp rehabilitation/ (327345)
25. (physical therapy or physiotherapy).mp. (51147)
26. resistance training.mp. or exp resistance training/ or exp weight lifting/ (12190)
27. strength$.mp. (341202)
28. (eccentric or concentric or isometric).mp. (51502)
29. 23 or 24 or 25 or 26 or 27 or 28 (985812)
30. 9 and 22 and 29 (5151)
31. limit 30 to human [Limit not valid in CCTR,CDSR; records were retained] (5098)

PEDro

<u>Abstract and Title</u>

Search 1: knee anterior pain + hip + strengthening
Search 2: knee pain + hip muscles
Search 3: knee pain + hip + strength
Search 4: patellofemoral pain syndrome
When searching: match all search terms (AND)

APPENDIX B

EXCLUDED PAPERS

Study	Reasons for Exclusion*						
	1	2	3	4	5	6	7
Almeida et al 2015							✓
Bakhtiary and Fatemi 2008			✓				
Bolgla et al 2015							✓
Balci et al 2009			✓				
Collins et al 2009	✓						
Coppack et al 2011				✓			
Crossley et al 2002					✓		
Crossley et al 2003						✓	
Cowan et al 2002					✓		
Denton et al 2005			✓				
Dursun et al 2002					✓		
Halabchi et al 2015					✓		
Harrison et al 1999	✓				✓		
Herbst et al 2015							✓
Hott et al 2015						✓	
Kannus et al 1999	✓						
Karakus et al 2014			✓				
Kim et al 2013				✓			
Linschoten et al 2009					✓		
Mazloum and Rahnama 2014		✓					
Motealleh et al 2016	✓						
Moyano et al 2013	✓						
Osteras et al 2013a			✓				
Osteras et al 2013b						✓	
Palmer et al 2015				✓			
Qiu et al 2006	✓		✓				
Rathleff et al 2012					✓		
Rathleff et al 2016					✓		
Roush et al 2000					✓		
Scheider et al 2001	✓						
Song et al 2009	✓						
Thomas et al 2002				✓			
Thomas et al 2005	✓						
Vicenzino et al 2008						✓	
Whittingham et al 2004			✓				
Witrouw et al 2000	✓		✓				
Witvrouw et al 2003	✓		✓				
Witvrouw et al 2004	✓		✓			✓	
Yilmaz et al 2015	✓						
Yip et al 2006					✓		

**(1) Experimental intervention was not strengthening or did not include hip muscles (abductors, lateral rotators, or extensors); (2) Translation of paper was not available; (3) Both experimental and control groups received similar strengthening interventions; (4) Population was not composed of participants with patellofemoral pain syndrome; (5) Experimental intervention was a multimodal intervention; (6) Paper was a commentary, study protocol, or follow-up trial; (7) Design was not a randomized or controlled trial.*

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Individuals Post Achilles Tendon Rupture Exhibit Asymmetrical Knee and Ankle Kinetics and Loading Rates During a Drop Countermovement Jump

Achilles tendon rupture is a common injury in adults, with a current incidence of 12 to 55 per 100 000 person-years.[14] Approximately 73% of these injuries occur in sports-related activities, when a player changes direction, accelerates, or lands from a jump.[17] Achilles tendon ruptures typically involve either rapid and forceful ankle dorsiflexion or a push-off movement with the knee extended.[2]

STUDY DESIGN: Cross-sectional laboratory study.

BACKGROUND: Asymmetrical knee loading during jogging and hopping has been reported in individuals who have ruptured their Achilles tendon. No studies have examined knee loads in individuals post Achilles tendon rupture during high-demand tasks, such as single-limb landings.

OBJECTIVES: We sought to determine whether individuals post Achilles tendon rupture demonstrated asymmetrical knee loads and impact forces during drop countermovement jumps (CMJs).

METHODS: Achilles tendon length and the single-leg heel-rise test for endurance were assessed in 34 individuals (31 male) 6.1 ± 2.0 years post Achilles tendon rupture. Movement patterns were assessed during a drop CMJ. Data were analyzed via repeated-measures analyses of variance, with comparisons between limbs and prior treatment history (surgery versus nonsurgery).

RESULTS: An 8.6% longer Achilles tendon ($P<.001$) was found in the involved limb. During the single-leg heel-rise test, the involved limb demonstrated 22.4% less endurance and 14.6% lower heel-rise height (all, $P<.001$). During the landing phase of the drop CMJ, the involved limb exhibited 39.6% greater loading rate ($P<.001$), 16.8% greater eccentric knee power ($P = .048$), but 21.6% lower eccentric ankle power ($P<.001$). During the take-off phase, the involved limb exhibited 12.1% lower jump height and 19.9% lower concentric ankle power (both, $P<.001$).

CONCLUSION: Elevated eccentric knee joint power and higher loading rates during a drop CMJ in individuals who experienced Achilles tendon rupture several years earlier may be a compensation pattern for reduced plantar flexor function. This movement pattern may place individuals who have had an Achilles tendon rupture at greater risk for knee injuries. *J Orthop Sports Phys Ther 2018;48(1):34-43. Epub 26 Oct 2017. doi:10.2519/jospt.2018.7684*

KEY WORDS: *ankle, biomechanics, jumping, knee, tendon*

Permanent impairments in plantar flexor performance are common in individuals who have ruptured an Achilles tendon.[29,44,49] Achilles tendon elongation is prevalent in individuals after Achilles tendon rupture,[39] resulting in active insufficiency of the plantar flexors.[46] Active insufficiency of the plantar flexor musculature contributes to diminished plantar flexor force production and endurance,[19,49] as well as reduced ability to perform tasks that require rapid force production in end-range plantar flexion.[29,44,50]

As the majority of Achilles tendon ruptures occur in athletes, it is not surprising that many who experience this injury aim to resume sport activities.[52] To date, relatively few studies have examined performance during high-demand tasks in patients post Achilles tendon rupture.[25,28,29] Nilsson-Helander and colleagues[25] found significant side-to-side (12%-24%) deficits in maximal jump height during a single-leg drop countermovement jump (CMJ) in individuals 6 and 12 months post Achilles tendon rupture. Olsson and colleagues[30] found similar side-to-side deficits during a drop CMJ at 6 and 12 months post rupture,

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which continued to persist at 24 months post injury. Thus, it appears that deficits in jumping performance are a long-term consequence of Achilles tendon ruptures. This reduction in the ability to perform explosive single-limb movements may help explain the overall decline in player performance that is common when athletes who have had an Achilles tendon rupture return to certain sports.[1,31]

Not surprisingly, after Achilles tendon rupture, individuals have demonstrated altered lower extremity movement patterns during functional activities. For instance, large (5%-20%) side-to-side deficits in ankle joint power production during walking, repetitive hopping, and jogging have been observed in individuals long after Achilles tendon rupture.[47,50] In these low- to moderate-demand activities, side-to-side ankle power deficits become more evident as the demand on the ankle plantar flexors increases via higher ground reaction forces and/or angular velocities. For instance, individuals post Achilles tendon rupture walk with a 15% deficit in ankle power production, and an 18% deficit during the higher-demand task of hopping.[50]

There is also evidence that a past Achilles tendon rupture alters lower extremity movement patterns beyond the ankle, likely as a compensation for reduced plantar flexor performance.[18,45,50] Asymmetrical and elevated knee loads in the involved limb during jogging and repetitive hopping have been observed in individuals 6 years post Achilles tendon rupture, regardless of surgical or nonsurgical treatment.[50] Over time, a pattern of elevated knee loads may place individuals who have had an Achilles tendon rupture and engage in jogging and hopping activities at greater risk for knee injuries.

To date, little is known regarding knee and ankle joint loads during high-demand sport-related tasks, such as single-limb landing. This type of landing closely replicates the mechanism of many Achilles tendon ruptures[17] and can be simulated via a drop CMJ maneuver.[9,41] A drop CMJ imparts considerably higher external loads to the lower limb compared with lower-demand tasks (eg, jogging). For instance, vertical ground reaction forces (vGRFs) in healthy individuals are progressively higher during a single-leg drop CMJ (approximately 3.0 to 3.5 body weights [BW])[40] compared with either jogging or single-leg hopping (1.7-2.5 BW).[13,37] As external loads increase, the compromised plantar flexors in individuals following Achilles tendon rupture would likely be challenged to a greater extent. Thus, it can be surmised that even greater asymmetry of knee kinetics would be present during a drop CMJ in individuals post Achilles tendon rupture when compared with low- or moderate-demand activities. Asymmetrical sagittal plane knee kinetics and loading rates of the vGRF during jump landings are associated with an increased risk of traumatic and overuse knee injuries.[4,5,33] Over time, an elevated loading rate of the vGRF may also be detrimental to the articular cartilage of the knee,[23,24] resulting in an eventual diminished ability to tolerate loads.[36]

We sought to determine whether greater knee joint powers and increased loading rates of the vGRF are present in the involved limb during a drop CMJ in individuals several years after Achilles tendon rupture. We hypothesized that there would be increased knee loads and loading rates in the involved limb as compared with the uninvolved limb.

METHODS

Participants

Prior to study initiation, the research protocol (058-14) was approved by the Regional Ethical Review Board in Gothenburg, Sweden. Participants were recruited from 2 previously conducted randomized controlled trials (combined n = 201) comparing outcomes in individuals who were treated surgically versus nonsurgically for an acute Achilles tendon rupture.[25,30] Recruitment was based on recovery of single-leg heel-rise ability at 1 year post rupture. Heel-rise ability was operationally defined as the limb symmetry index (LSI) between the heel-rise heights during a single-leg standing heel-rise test.[42] Heel-rise-height LSI was calculated as (involved-limb heel-rise height/uninvolved-limb heel-rise height) × 100 and expressed as a percent. All individuals were ranked according to their heel-rise-height LSI and consecutively recruited from the top and bottom of the ranked recruitment pool. There were an equal number of surgically and nonsurgically treated individuals in the top and bottom thirds of the sample. Any individuals with a bilateral Achilles tendon rupture, rerupture, or who did not participate in the 1-year follow-up assessment were not included in this study. Regardless of surgical or nonsurgical management, postinjury rehabilitation emphasized early and progressive loading.[25,30]

Clinical Tests

To assess level of self-reported ankle function at the time of enrollment, the Achilles tendon Total Rupture Score[26] and the Foot and Ankle Outcome Score sport subscale[38] were administered. In addition, all participants completed the single-leg heel-rise test for endurance.[42] The single-leg heel-rise test for endurance is a valid assessment of plantar flexor function in individuals post Achilles tendon rupture.[8] To complete this test, individuals were asked to perform a maximal number of single-leg heel rises on a 10° incline at a 0.5-Hz rate, while a linear encoder (MUSCLELAB; Ergotest Innovation AS, Porsgrunn, Norway) assessed heel-rise performance. Total cumulative work (product of body mass and cumulative heel-rise height across the heel-rise test) was calculated across the heel-rise trial, and the best heel-rise height of each limb (centimeters) was retained for analysis.

Side-to-side differences in Achilles tendon length were assessed by an experienced evaluator (A.B.) using extended field-of-view ultrasound imaging, as previously described in the literature.[43]

Achilles tendon length was measured from the calcaneal osteotendinous junction to the gastrocnemius musculotendinous junction, with the patient positioned in prone with both feet off the examination table in a neutral position. Extended-field-of-view ultrasound imaging was performed (LOGIQ e; GE Healthcare, Waukesha, WI) using a linear probe with a wide-band array (5.0-13.0 MHz), in brightness mode, at a sampling rate of 10 MHz, and at a depth of 3 cm. These methods have previously been shown to have excellent day-to-day reliability (intraclass correlation coefficient [$ICC_{2,3}$] = 0.895).[43] In addition, the between-limb reliability has been shown to be excellent (ICC = 0.940), indicating that the uninjured side can be used for comparison for determining tendon elongation due to injury.[43]

FIGURE 1. Lower extremity marker set used for analysis of the single-leg drop countermovement jump.

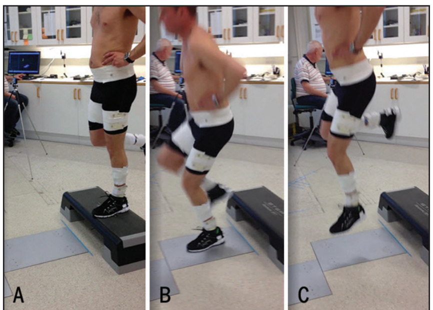

FIGURE 2. Biomechanical analysis of the single-leg drop countermovement jump. (A) After starting the single-leg drop countermovement jump on a 20-cm platform, (B) loading rate of the vertical ground reaction force and ankle and knee joint powers were analyzed during the landing and take-off phases, and (C) peak jump height was analyzed.

Biomechanical Analysis of the Drop CMJ For the biomechanical analysis of the drop CMJ, participants wore a standard laboratory shoe (OMEGA; Bagheera AB, Avesta, Sweden). A lower extremity and trunk marker set, consisting of 55 retroreflective markers, was used to define the individual segments of the participants.[34] A static calibration trial was collected, as marker trajectories (200 Hz) and ground reaction forces (1600 Hz) were sampled with a 12-camera motion-capture system (Oqus 4; Qualisys AB, Gothenburg, Sweden) and a multi–force plate configuration (Kistler Holding AG, Winterthur, Switzerland), respectively. Anatomical coordinate systems were then established for the trunk, pelvis, thighs, lower legs, and feet via markers placed on the proximal and distal ends of each body segment. The hip joint center was determined by an anatomically based algorithm via markers placed on the bilateral anterior superior iliac spines, the level of the posterior superior iliac spines, greater trochanters, and lower-limb length.[11] The knee and ankle joint centers were determined by the centroids of markers placed on the medial and lateral femoral condyles and malleoli, respectively. Tracking markers consisted of clusters of markers affixed to the pelvis, bilateral thighs, lower legs, and feet.

Next, participants completed a series of drop CMJs as 3-D lower extremity mechanics were sampled. Participants were asked to assume single-leg stance on a 20-cm-high box, fall forward, and land on the same leg, followed immediately by a CMJ for maximal height while minimizing time on the ground. Participants practiced the drop CMJ at least 5 times per side, or until they were comfortable with the testing procedures, before data were recorded. A total of 5 drop CMJs were collected per limb, with the uninvolved limb tested first. See **FIGURES 1** and **2** for drop CMJ testing procedures.

Data Processing

Data were processed using Motion-Monitor software (Innovative Sports

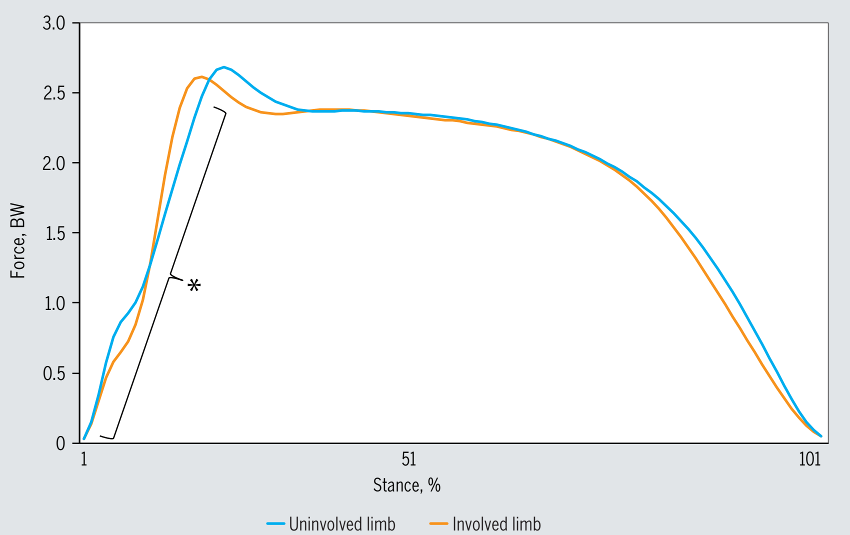

FIGURE 3. Vertical ground reaction force during the landing phase of the drop countermovement jump. These are group mean data across the entire sample. *Initial loading phase where instantaneous vertical loading rate (BW per second) was determined. Abbreviation: BW, body weight.

Training, Inc, Chicago, IL) and custom-written LabVIEW software (National Instruments, Austin, TX). Motion trials were examined for data fidelity, and any invalid trials were discarded. Next, raw ground reaction force data were filtered using a fourth-order, low-pass Butterworth filter with a 50-Hz cutoff. The initial single-leg landing and subsequent take-off phases were the periods of interest during the drop CMJ. To isolate the stance phase of each trial, a 20-N threshold of the vGRF was used.

Next, the loading rate of the vGRF curve was determined using the first central difference method[7,51] between foot strike and the initial peak of the vGRF curve (**FIGURE 3**). The peak instantaneous loading rate was thus determined for each landing phase and retained for analysis. Jumping height was determined by the estimated vertical displacement of the participant's center of mass during the CMJ phase of the drop CMJ, as previously described and validated.[10]

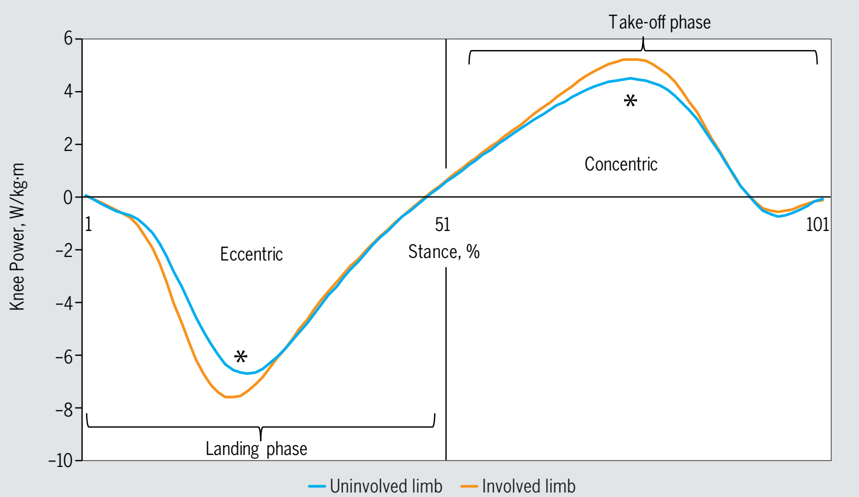

FIGURE 4. Knee joint power across the landing and take-off phases of the drop countermovement jump. These are group mean data across the entire sample. *Significantly different between limbs.

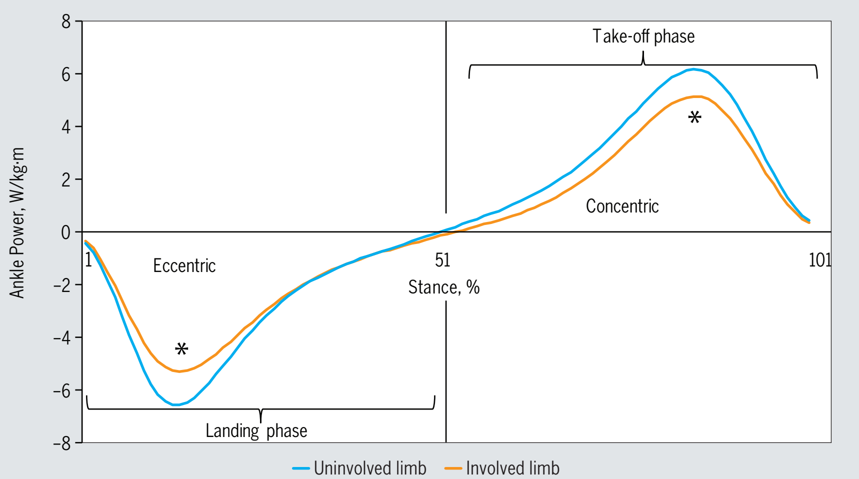

FIGURE 5. Ankle joint power across the landing and take-off phases of the drop countermovement jump. These are group mean data across the entire sample. *Significantly different between limbs.

Ankle and knee sagittal plane joint powers were then calculated. First, raw ground reaction force data and marker data were filtered using identical 15-Hz frequency cutoffs via a fourth-order, low-pass Butterworth filter. This filtering routine was chosen to reduce nonphysiological signal artifacts in knee and ankle moment data that are often observed in high-velocity activities such as jumping.[20] Estimated segmental inertial parameters[12] were used in the subsequent inverse dynamics routine to calculate internal joint moments, which were expressed in the proximal coordinate system. Eccentric and concentric joint powers for the ankle plantar flexors and knee extensors were calculated as the instantaneous product of the sagittal plane angular velocity and internal joint moment for the respective joints. Ankle and knee joint powers were normalized to subject height and mass and expressed in watts per kilogram times meters. Eccentric and concentric powers corresponded with the landing and take-off phases of the drop CMJ maneuver, respectively. The most negative and positive instantaneous values represented the peak eccentric and concentric joint powers, respectively, for each trial (**FIGURES 4** and **5**).

Statistical Analysis

An a priori power analysis was conducted, utilizing previously collected pilot data detailing knee and ankle biomechanics measured during an athletic task post Achilles tendon rupture.[45] In order to detect at least a moderate difference (α = .05, β = .90) in lower extremity biomechanics between limbs, at least 13 participants were required to adequately power the present investigation. Data were analyzed via separate 2-by-2 (group [surgical, nonsurgical] by limb [involved, uninvolved]) repeated-measures analyses of variance (ANOVAs) using SPSS Version 20 (IBM Corporation, Armonk, NY). When the assumptions of the ANOVA were violated, a Greenhouse-Geisser adjustment was applied. For the clinical measures, the variables of interest were patient demographics, Achilles tendon Total Rupture Score scores, heel-rise work and height, and Achilles tendon length. A Bonferroni adjustment was used (α = .008) to reduce the risk of familywise error for multiple tests for the biomechanical variables. When the ANOVA was significant, post hoc pairwise comparisons were conducted. Limb symmetry index values were calculated for biomechanical variables to provide a clinical reference of between-limb differences. Biomechanical discrete variables of interest were analyzed during the respective phases of the drop CMJ: (1) landing phase: loading rate of the vGRF and eccentric ankle and knee powers, and (2) take-off and flight phase: concentric ankle and knee joint powers and maximal jump height.

TABLE 1 DEMOGRAPHICS OF PARTICIPANTS*

	Surgical	Nonsurgical	Composite (n = 34)	*P* Value
Age, y	48.6 ± 9.5	47.9 ± 12.4	48.3 ± 10.9	.84
Treatment type, n	17	17	34	1.00
Time since rupture, y	6.5 ± 2.3	5.8 ± 1.7	6.1 ± 2.0	.11
Range post rupture, y	4.2-10.6	4.2-9.2	4.2-10.6	.11
Sex, n				.07
Male	14	17	31	
Female	3	0	3	
Height, cm	177.2 ± 10.9	180.2 ± 6.2	178.7 ± 8.9	.37
Mass, kg	87.6 ± 14.1	83.7 ± 11.3	85.7 ± 12.8	.47
Body mass index, kg/m²	27.9 ± 3.7	25.7 ± 2.7	26.8 ± 3.4	.11
ATRS (0-100)	90.0 ± 13.5 94 (5.8)†	87.9 ± 12.3 93 (17)†	89.0 ± 12.7 93.5 (14.5)†	.86
FAOS sport subscale (0-100)	91.7 ± 11.1 100 (20)†	87.3 ± 14.9 95 (20)†	89.5 ± 13.1 95 (20)†	.85

Abbreviations: ATRS, Achilles tendon Total Rupture Score; FAOS, Foot and Ankle Outcome Score.
**Values are mean ± SD unless otherwise indicated.*
†Values are median (interquartile range).

RESULTS

Demographics

In total, 34 participants were recruited from 2 previous randomized controlled trials. Mean ± SD heel-rise-height LSI at the 1-year follow-up assessment post Achilles tendon rupture was 78% ± 19%, with a median heel-rise-height LSI of 78% and a range of 37% to 109%. Participants were recruited at a mean ± SD of 6.1 ± 2.0 years after Achilles tendon rupture. See **TABLE 1** for demographics of the sample.

Clinical Tests

In the present investigation, no significant group-by-limb interactions or main effects of treatment group (surgical, nonsurgical) were found for any of the clinical measures (P>.05) (**TABLE 2**). However, differences between involved and uninvolved limbs were seen in both heel-rise height (P<.001; LSI, 85.4%; 95% confidence interval [CI]: 80.0%, 90.8%) and heel-rise work (P<.001; LSI, 77.6%; 95% CI: 70.1%, 85.2%) during the single-leg heel-rise test. Differences between limbs were also seen in Achilles tendon length (P<.001; LSI, 108.6%; 95% CI: 105.6%, 111.7%).

Biomechanical Assessment

Four of the participants were excluded from the biomechanical analysis of the drop CMJ: 1 surgical participant and 1 nonsurgical participant were unable to perform the drop CMJ task due to knee pain on the uninvolved limb, and 1 surgical participant and 1 nonsurgical participant had faulty marker data. Thus, 30 participants (15 surgical, 15 nonsurgical) were retained for analysis of the biomechanical variables. Identical to the clinical measures, no significant group-by-limb interactions or main effects of treatment group (surgical versus nonsurgical) were found for any of the biomechanical measures (P>.05). Thus, only significant main effects of limb (involved versus uninvolved) were found for biomechanical measures during the drop CMJ (**TABLE 3**). During the landing phase, the involved limb experienced a 39.6% greater loading rate of the vGRF (P<.001; LSI, 139.6%; 95% CI: 122.7%, 156.5%), 21.6% less eccentric ankle joint power (P<.001; LSI, 78.4%; 95% CI: 70.6%, 86.2%), and 16.8% greater eccentric knee joint power (P = .048; LSI, 116.8%; 95% CI: 106.8%, 126.9%) compared with the uninvolved limb. During the take-off and flight phases, participants jumped 12.1% lower with the involved limb compared with the uninvolved limb (P<.001; LSI, 87.9%; 95% CI: 83.0%, 92.8%). There was a 19.9% deficit in concentric ankle joint power (P<.001; LSI, 80.1%; 95% CI: 74.7%, 85.7%), but there was no difference found for concentric knee power (P>.05; LSI, 110.1%; 95% CI: 101.0%, 119.3%).

DISCUSSION

The main findings of this study were that the involved limb in individuals 6.1 ± 2.0 years post Achilles tendon rupture demonstrated elevated impact loading rate and eccentric knee power and reduced ankle power and maximal jump height during a single-limb drop CMJ, compared with the uninvolved limb. We also found long-term deficits in plantar flexor function, via the single-leg heel-rise test for endurance, and an elongated Achilles tendon.

There were significant differences between involved and uninvolved limbs for the clinical measures of Achilles ten-

TABLE 2	Clinical Measures of Achilles Function*				
Measure/Limb	**Surgical**	**Nonsurgical**	**Composite**	**ANOVA**	***P* Value**
Heel-rise height, cm					
Involved	11.4 (10.5, 12.4)	10.7 (9.7, 11.7)	11.1 (10.4, 11.8)	Interaction	.740
Uninvolved	13.7 (12.9, 14.4)	12.4 (11.8, 13.0)	13.0 (12.5, 13.5)	Main effect of limb†	<.001
LSI, %	83.8 (77.9, 89.7)	86.9 (77.9, 95.9)	85.4 (80.0, 90.8)	Main effect of group	.844
Heel-rise work, J					
Involved	2355.2 (1937.8, 2772.6)	2135.5 (1683.0, 2588.0)	2241.6 (1936.2, 2546.9)	Interaction	.599
Uninvolved	3068.2 (2663.4, 3473.0)	2722.8 (2351.6, 3094.0)	2889.6 (2613.1, 3166.0)	Main effect of limb†	<.001
LSI, %	76.7 (66.7, 90.3)	78.4	77.6 (70.1, 85.2)	Main effect of group	.320
Tendon length, cm					
Involved	21.7 (19.1, 24.3)	23.8 (21.2, 26.4)	22.7 (20.9, 24.6)	Interaction	.619
Uninvolved	20.0 (17.6, 22.5)	21.9 (19.5, 24.3)	21.0 (19.3, 22.7)	Main effect of limb†	<.001
LSI, %	107.8 (103.2, 112.3)	109.5 (105.3, 113.6)	108.6 (105.6, 111.7)	Main effect of group	.246

Abbreviations: ANOVA, analysis of variance; LSI, limb symmetry index.
**Values are mean (95% confidence interval) unless otherwise indicated.*
†Significant (P<.05).

don length and plantar flexor function. We found an average of a 1.7-cm longer Achilles tendon on the affected side in both surgically and nonsurgically treated individuals. This difference exceeds the small amount of normal asymmetry (0.1 cm) of Achilles tendon length seen in healthy individuals.[44] Our sample was between 4.2 and 10.6 years post rupture, which indicates that the elongated tendon is a long-term structural complication in these individuals. Further clinical evidence that an elongated Achilles tendon was present in this sample was found via the single-leg heel-rise test for endurance. This clinical test is a validated predictor of the presence of Achilles tendon elongation[44] and may be an important clinical prognosticator of ankle biomechanics after Achilles tendon rupture. In fact, heel-rise deficits at 1 year predict long-term ankle biomechanics after Achilles tendon rupture, whereas surgical or nonsurgical treatment does not.[9] Overall, participants reported minimal to moderate ankle limitations at 4.2 to 10.6 years after their injury, suggesting that they had largely adapted to their Achilles tendon rupture.

Despite relatively high reported function, we found moderate to large deficits in interlimb mechanics during the drop CMJ. The drop CMJ maneuver is a challenging activity, requiring the absorption of high external loads during the landing phase. While not analyzed statistically, our participants experienced an average peak vGRF of approximately 2.75 BW of force during landing from the elevated platform. Thus, external forces during the drop CMJ exceeded the peak vGRF commonly noted during other tasks that have been evaluated in individuals post Achilles tendon rupture, including walking, jogging, and single-leg hopping (1.7-2.5 BW of the vGRF).[13,37,47,50] During the initial landing phase, the involved limb experienced considerably higher (LSI, 139.6%) loading rates of the vGRF, indicating greater impact loading. Higher loading rates of the vGRF during a jump landing may place an individual at greater risk for certain lower extremity injuries. For instance, higher loading rates during a jump-landing task have been reported in volleyball players with a previous history of patellar tendinopathy[5] and in runners with a history of patellar tendinopathy.[16] In addition, loading rates during a jump-landing task have been suggested to increase the risk of anterior cruciate ligament rupture.[32] Over time, a pattern of higher impact forces may also increase the risk of knee osteoarthritis[23,27] by degrading the articular cartilage matrix of the tibiofemoral joint.[36]

Greater (LSI, 116.8%) eccentric knee joint power was also noted during the landing phase of the drop CMJ, perhaps as a compensation for the 21.6% reduction in eccentric power at the ankle. This suggests that the energy not absorbed at the ankle may have consequences for the knee during the eccentric phase of this high-demand activity. The pattern of greater eccentric knee joint power may increase risk for mechanical overload of the knee during single-limb landing maneuvers.[21]

During the take-off and flight phases, participants did not compensate for reduced concentric ankle power (LSI, 80.1%) with greater concentric knee power, resulting in a 12.1% lower jump height. The involved Achilles tendon was found to be elongated, and participants had lower single-leg heel-rise height and work during the heel-rise test for endurance. These clinical measures indicated a compromised plantar flexor musculotendinous unit. Previous work also suggests that a ruptured Achilles tendon heals with less tendon stiffness[15,22] and

TABLE 3 Results of ANOVA for Biomechanical Variables of Interest*

Measure/Limb	Drop CMJ Surgical	Drop CMJ Nonsurgical	Drop CMJ Composite	ANOVA	*P* Value†
Average jump height, cm					
Involved	9.5 (7.3, 11.6)	10.4 (8.3, 12.6)	10.0 (8.4, 11.5)	Interaction	.784
Uninvolved	10.9 (8.8, 13.0)	12.0 (9.9, 14.1)	11.5 (10.0, 12.9)	Main effect of limb‡	<.001
LSI, %	85.7 (78.6, 92.8)	90.0 (83.2, 97.0)	87.9 (83.0, 92.8)	Main effect of group	.466
Instantaneous loading rate of vGRF, BW/s					
Involved	144.5 (119.1, 169.9)	118.4 (95.0, 141.7)	131.4 (113.8, 149.0)	Interaction	.434
Uninvolved	105.0 (85.7, 124.4)	90.2 (79.0, 101.5)	97.6 (86.3, 109.0)	Main effect of limb‡	<.001
LSI, %	147.4 (120.3, 131.8)	131.8 (111.5, 152.1)	139.6 (122.7, 156.5)	Main effect of group	.126
Eccentric ankle joint power, W/kg·m					
Involved	−5.3 (−6.5, −4.2)	−6.0 (−6.9, −5.1)	−5.7 (−6.4, −5.0)	Interaction	.995
Uninvolved	−6.9 (−7.5, −6.3)	−7.5 (−8.1, −6.8)	−7.2 (−7.6, −6.7)	Main effect of limb‡	<.001
LSI, %	77.2 (63.8, 90.6)	79.6 (71.1, 88.1)	78.4 (70.6, 86.2)	Main effect of group	.211
Concentric ankle joint power, W/kg·m					
Involved	5.2 (4.4, 6.1)	5.8 (4.9, 6.6)	5.5 (4.9, 6.1)	Interaction	.605
Uninvolved	6.5 (5.8, 7.2)	7.1 (6.3, 8.0)	6.8 (6.3, 7.3)	Main effect of limb‡	<.001
LSI, %	80.5 (70.7, 90.4)	79.9 (74.5, 85.3)	80.1 (74.7, 85.7)	Main effect of group	.275
Eccentric knee joint power, W/kg·m					
Involved	−8.2 (−9.3, −7.1)	−9.2 (−10.1, −8.4)	−8.7 (−9.4, −8.0)	Interaction	.65
Uninvolved	−7.1 (−8.1, −6.1)	−8.3 (−9.1, −7.6)	−7.7 (−8.4, −7.1)	Main effect of limb‡	.048
LSI, %	120.7 (105.0, 136.4)	113.0 (100.3, 125.7)	116.8 (106.8, 126.9)	Main effect of group	.120
Concentric knee joint power, W/kg·m					
Involved	5.2 (4.4, 6.0)	6.4 (5.6, 7.2)	5.8 (5.2, 6.4)	Interaction	.545
Uninvolved	4.6 (4.0, 5.3)	6.1 (5.2, 7.1)	5.4 (4.8, 6.0)	Main effect of limb	.516
LSI, %	120.7 (105.0, 136.4)	112.3 (100.0, 124.9)	110.1 (101.0, 119.3)	Main effect of group	.060

Abbreviations: ANOVA, analysis of variance; BW, body weight; CMJ, countermovement jump; LSI, limb symmetry index; vGRF, vertical ground reaction force.
**Values are mean (95% confidence interval) unless otherwise indicated.*
†Values are Bonferroni corrected.
‡Significant (P<.05) with Bonferroni correction applied.

loses more energy to tendon hysteresis[35,48] during maximal, rapid plantar flexor contractions compared with a healthy Achilles tendon. Thus, a reduction in concentric ankle power during the take-off phase was not unexpected. Other factors that might have contributed to reduced maximal jump height include high-speed strength deficits[49] and the presence of kinesiophobia,[28] both of which have been reported in individuals post Achilles tendon rupture. The findings of reduced jump height in the involved limb may help explain the reduced player performance in athletes post Achilles tendon rupture who attempt a return to sports that place a premium on jumping ability.[52] For instance, lower player performances in the form of fewer rebounds, steals, and blocks were noted in National Basketball Association players with a past Achilles tendon rupture compared with matched controls.[1]

At the knee, the present study only found greater eccentric power in the involved limb during the single-leg drop CMJ, whereas previous reports found both increased eccentric and concentric knee joint powers during jogging and repetitive hopping in the long term post Achilles tendon rupture.[50] Discrepancies between studies may be due to methodological differences associated with the nature of the tested tasks. In this study, the patients were instructed to perform a maximal jump during the propulsive phase of the drop CMJ task. In contrast, the previous studies involved either jogging[45,50] or submaximal hopping,[50] which are both repetitive tasks. A post hoc analysis found that limb asymmetries in maximal jump height did not explain the lack of differences in concentric knee joint power between limbs. Therefore, it is not clear why this investigation did not find differences in concentric knee power during the take-off phase. Nevertheless, involved-limb eccentric (mean, −8.7 W/kg·m) and concentric (5.8 W/kg·m) knee joint powers found during the drop CMJ were both considerably greater than joint powers previously reported during jogging and hopping (eccentric, –3.5 to –5.0 W/kg·m; concentric, 3.0 to 4.1 W/kg·m) in the same sample.[50] Provided sufficient repetition, activities that involve single-leg landings may result in greater risk of knee injury in individuals with a past Achilles tendon rupture compared with less strenuous activities, such as jogging.

We did not find differences between surgically and nonsurgically treated individuals for any of the variables in this investigation. While this study was not powered to detect differences between treatment approaches, LSIs noted in each group were similar. The lack of biomechanical differences during the drop CMJ between groups agrees with our previous report that also found no differences between groups in the lower-demand tasks of walking, jogging, or hopping.[50]

Clinically, our findings suggest that jumping athletes post Achilles tendon rupture may require a rehabilitation component that addresses more than just ankle function. Several studies have indicated that knee joint loads and vGRF during jump landings can be reduced with a movement re-education program.[3,6] If reductions in loading rates and knee joint loading are not achieved through movement re-education, then athletes may benefit from counseling to reduce their overall participation in sports that require single-leg jumping in favor of activities with lower knee joint loads, such as jogging or cycling.

While this study indicated higher knee joint loading and impact forces in the involved limb, we did not assess patient-reported knee function or pain. Future studies should investigate whether overuse and traumatic knee injuries are more prevalent in individuals post Achilles tendon rupture. This study had a cross-sectional, observational design. A longitudinal study is necessary to determine whether lower-limb mechanics change over time in individuals post Achilles tendon rupture. Additionally, comparisons were made between limbs, and we did not include matched, healthy control participants. However, between-limb differences may be of smaller magnitude in individuals with Achilles tendon injuries than when compared with healthy individuals.[47] Thus, it is possible that our present investigation is a conservative assessment of lower-limb mechanics in individuals post Achilles tendon rupture.

CONCLUSION

Overall, these data indicate a pattern of greater lower extremity impact loading rate and greater eccentric knee power, perhaps as a compensation for reduced ankle power, during a drop CMJ maneuver. Our findings suggest that overall movement patterns should inform guidance on activity participation in the long term after Achilles tendon rupture, rather than whether the patient was treated surgically or nonsurgically. Individuals post Achilles tendon rupture may benefit from strategies to reduce knee joint loading and loading rates if a return to jumping sports is desired.

KEY POINTS

FINDINGS: Individuals in the long term post Achilles tendon rupture demonstrated greater knee powers and impact forces during a drop countermovement jump, while also demonstrating reduced plantar flexor function.

IMPLICATIONS: These findings suggest that jumping athletes who are recovering from an Achilles tendon rupture may benefit from rehabilitation programs that address knee powers and impact forces, in addition to ankle function, during jump-landing tasks.

CAUTION: These results should be interpreted with caution, as participants sustained an Achilles tendon rupture several years prior and the study design was cross-sectional in nature.

ACKNOWLEDGMENTS: *Lotta Falkheden Henning, PT assisted with data collection.*

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The Challenge of Sharing New Information

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J Orthop Sports Phys Ther 2018;48(1):1-2. doi:10.2519/jospt.2018.0101

In their timely Viewpoint in this issue of *JOSPT*, Dr Cook and his colleagues[6] from around the world express the shared challenges of keeping up to date with the vast and fast-growing information relevant to orthopaedic and sports physical therapy clinical practice—and do so while avoiding the traps and hazards of an internet world, in which information may be misinterpreted, misrepresented, or, even worse, misused. Clinical journals are part of this complex equation of providing new information in an accessible, meaningful, and trustworthy manner. The Viewpoint by Dr Cook et al[6] is a reminder of this responsibility, as I officially assume the role of Interim Editor-in-Chief of *JOSPT* to start the year 2018.

Having worked behind the scenes with the Editorial Board over the past 5 months and overseen the review of material to be published beginning with the January 2018 issue of *JOSPT*, I am inspired again by the talented researchers and clinicians in the physical therapy profession. While the road to publication is never as straightforward as one would want or imagine, a review process that allows for examination of the strengths and weaknesses of submitted articles contributes to the growth and maturity of the involved parties and, ultimately, of the profession.

Warden et al,[13] in their brief report in this issue of the *Journal*, provide evidence of the continued evolution of orthopaedic and sports physical therapy research through the higher rate of conversion of conference abstracts to peer-reviewed publications achieved in more recent years. From a look at that more recent work, it is clear that some advances can be attributed to continued sophistication in research training, research methods, and technology itself.[7,9] However, and perhaps more subtly, there has been a progressive shift or expansion in the profession's research paradigms, with an increased focus on better understanding and quantifying the human experience associated with injury/pain/dysfunction/disability and the related rehabilitation/recovery process.

Foremost in our minds when thinking of the patient experience is the now nearly ubiquitous use of patient-reported outcome measures in publications of clinical trials. Hopefully, these outcome measures are widely implemented in daily clinical practice to rightfully supplement information gained from the clinical examination and measurement of impairments.[1,11]

Paramount to our research progress has also been the enormous effort devoted to gaining a better understanding of the pain experience and how our interventions affect this experience.[2,8,10] As exemplified by some of the work recently published in *JOSPT*, clinical trials and outcomes research are beginning to show a concerted effort to incorporate such factors as patients' and therapists' beliefs and preferences, placebo, the influence of verbal and other forms of communications, and patient expectations and other psychosocial factors in clinical research.[3-5,12] That work promises to impact many aspects of physical therapy clinical practice.

Returning to *JOSPT* as Interim Editor-in-Chief after a 2-year hiatus also affords me the opportunity to work closely with a talented Editorial Board. Please see the masthead in this copy of the *Journal* or online at www.jospt.org for a complete list of continuing and new members. While the majority of the Editorial Board returns for 2018, it is my pleasure to formally announce that Dr Josh Cleland and Dr Steve Kamper will take on new roles as editors, while Dr Marcie Harris-Hayes, Dr Rasmus Nielsen, Dr Jean-Sébastien Roy, and Dr Arianne Verhagen will assume the role of associate editors.

We also welcome Dr Joaquin Barrios, Dr Paula Beckenkamp, Dr Kristin Briem, Dr Rogelio Coronado, Dr Patrick Grabowski, Dr Cara Lewis, Dr Amee Seitz, Dr Tiê Yamato, and Dr Chris Williams as new members of the International Editorial Review Board. I welcome all of these fine researchers and clinicians to this critical role of core reviewers for *JOSPT*.

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Progress in the Full-Text Publication Rate of Orthopaedic and Sports Physical Therapy Abstracts Presented at the American Physical Therapy Association's Combined Sections Meeting

Professional meetings are an important forum for disseminating advances in physical therapy. However, work presented in abstract form does not undergo rigorous peer review, contains limited methodological details, and is often preliminary in nature. In terms of the latter, there are often major discrepancies between data presented in abstract form and subsequent full-text publication,[3,10,15,16] confirming that therapists should not make evidence-based practice decisions based solely on information presented in an abstract.

STUDY DESIGN: Descriptive study.

BACKGROUND: Professional meetings, such as the American Physical Therapy Association's (APTA's) Combined Sections Meeting (CSM), provide forums for sharing information. However, it was reported that only one quarter of Orthopaedic and Sports Physical Therapy Sections abstracts presented at the CSM between 2000 and 2004 went on to full-text publication. This low conversion rate raises a number of concerns regarding the full dissemination of work within the profession.

OBJECTIVES: The purpose of this study was to determine the full-text publication rate of work presented in abstract form at subsequent CSMs and investigate factors influencing the rate.

METHODS: A systematic search was undertaken to locate full-text publications of Orthopaedic and Sports Physical Therapy Sections abstracts presented at CSMs between 2005 and 2011. Eligible publications were published within 5 years following abstract presentation. The influences of year of abstract presentation, APTA section, presentation type, institution of origin, study design, and study significance were assessed.

RESULTS: Over one third (38.6%) of presented abstracts progressed to full-text publication. Odds of full-text publication increased when the abstract was presented as a platform presentation, originated from a doctorate-granting institution, reported findings of an experimental study, or reported a statistically significant finding.

CONCLUSION: The full-text publication rate for Orthopaedic and Sports Physical Therapy Sections abstracts presented at recent CSMs has increased by over 50% compared to that reported for the preceding period. The rate is now in the range of that reported in comparable clinical disciplines, demonstrating important progress in the full dissemination of work within the profession. *J Orthop Sports Phys Ther 2018;48(1):44-49. Epub 26 Oct 2017. doi:10.2519/jospt.2018.7581*

KEY WORDS: *bibliometrics, information dissemination, peer review, publishing, sports medicine*

Full-text publication forms the cornerstone of knowledge dissemination. It requires complete disclosure of work of a certain standard in order to pass the rigors of peer review, while publishing in indexed journals facilitates retrievability within the broader community. The full-text publication and retrieval of work previously presented in abstract form provide useful metrics for the quality of work performed and the extent to which it is fully disseminated.

We previously reported that one quarter (25.4%, 209/823) of abstracts presented within the Orthopaedic and Sports Physical Therapy Sections at the American Physical Therapy Association's (APTA's) Combined Sections Meeting (CSM) between 2000 and 2004 went on to full-text publication within 5 years following presentation.[15] This is a low conversion rate, considering that between one and two thirds of abstracts presented in comparative clinical disciplines progress to full-text publication.[3,6,7,9-11,17]

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To explore whether the publication rate of abstracts in physical therapy has more recently improved, the current study investigated the publication rate of abstracts presented within the Orthopaedic and Sports Physical Therapy Sections at the APTA's CSM in the succeeding years (2005-2011). Factors contributing to full-text publication and the features of the publications were also explored.

METHODS

Abstract Data Extraction

Abstracts presented between 2005 and 2011 within the Orthopaedic and Sports Physical Therapy Sections at the APTA's CSM were entered into a database. Authors' names, abstract title, year of presentation, presentation type (platform/poster), section in which the abstract was presented (Orthopaedic/Sports Physical Therapy), institution of origin, study design, and study significance were recorded.

Institution of origin was determined from the primary/first-listed affiliation on the abstract, and was categorized as "doctorate granting," "non–doctorate granting," or "special focus," according to the basic classification in the 2015 edition of the Carnegie Classification of Institutions of Higher Education.[4] Nonlisted institutions were categorized as either "international" (ie, outside the United States) or "other" (ie, non–degree-granting institutions).

Study design was classified by 2 independent investigators as (1) meta-analysis/systematic review, (2) randomized controlled trial (RCT), (3) validation of tests and measures, (4) nonexperimental, or (5) other experimental and observational. Randomized controlled trials included a statement that study participants were randomly allocated into groups or that the repeat test conditions were introduced in random order. Validation of tests and measures included abstracts reporting on the reliability and validity or diagnostic accuracy of tests and measures. Nonexperimental abstracts presented case studies/series, outcomes research, expert opinion, or general reviews/overviews. Other experimental and observational included cohort, prospective case-control, and cross-sectional studies. Discrepancies in study design were resolved by consensus.

Abstracts with an RCT or other experimental and observational design were categorized as significant by 2 independent investigators when a statistically significant finding for the primary outcome variable was reported. Abstracts not reporting statistical results (ie, *P* values or whether statistical significance was obtained) were classified as not significant. Discrepancies in determining significance were resolved by consensus.

Full-Text Publication Search

A systematic search of Google Scholar was conducted by 2 independent investigators to establish whether the work presented in abstract form had been

TABLE 1 Characteristics of Abstracts Presented Within the Orthopaedic and Sports Physical Therapy Sections of the APTA's 2005 to 2011 Combined Sections Meetings*

Characteristic	Presented
Year of abstract presentation	
2005	223 (15.0)
2006	205 (13.8)
2007	178 (12.0)
2008	176 (11.8)
2009	258 (17.3)
2010	234 (15.7)
2011	215 (14.4)
APTA section	
Orthopaedic	1187 (79.7)
Sports Physical Therapy	302 (20.3)
Presentation type	
Platform	606 (40.7)
Poster	883 (59.3)
Institution of origin	
Doctorate granting	729 (49.0)
Non–doctorate granting	319 (21.4)
Special focus	75 (5.0)
International institution	36 (2.4)
Other institution	330 (22.2)
Study design	
Meta-analysis/systematic review	33 (2.2)
Randomized controlled trial	183 (12.3)
Validation of tests and measures	169 (11.3)
Nonexperimental	407 (27.3)
Other experimental and observational	697 (46.8)
Study finding†	
Significant	649 (73.8)
Not significant	231 (26.2)

Abbreviation: APTA, American Physical Therapy Association.
**Values are n (%). Abstracts (n = 57) published prior to and more than 5 years after conference presentation are not included.*
†Randomized controlled trial and other experimental and observational study designs only.

published in full text in the 5 years following presentation. Google Scholar was chosen because it has been shown to find twice as many relevant articles as PubMed when performing short clinical queries, and provides greater retrieval of open-access and non–English-language articles.[14] A 5-year window after abstract presentation was chosen, as timely dissemination of work presented in abstract form is essential to maintaining relevance, and previous studies indicate that most (greater than 95%) abstracts that ultimately go on to full-text publication are published within 5 years following presentation.[13]

Each full-text publication was reviewed by 2 independent investigators to verify that it represented the work presented in the earlier abstract, with discrepancies resolved by consensus. The date of full-text publication and name and impact factor of the publishing journal were recorded. Impact factors were obtained from the Journal Citation Reports Science Edition for 2014.[1]

Statistical Analyses

Analyses were performed using SPSS (Version 23; IBM Corporation, Armonk, NY), with a level of significance set at .05. The influences of year of abstract presentation, APTA section, presentation type, institution of origin, study design, and study significance on the odds of work progressing to full-text publication were assessed using multiple logistic regression, with outcomes expressed in odds ratios and 95% confidence intervals (CIs). The areas under the receiver operating

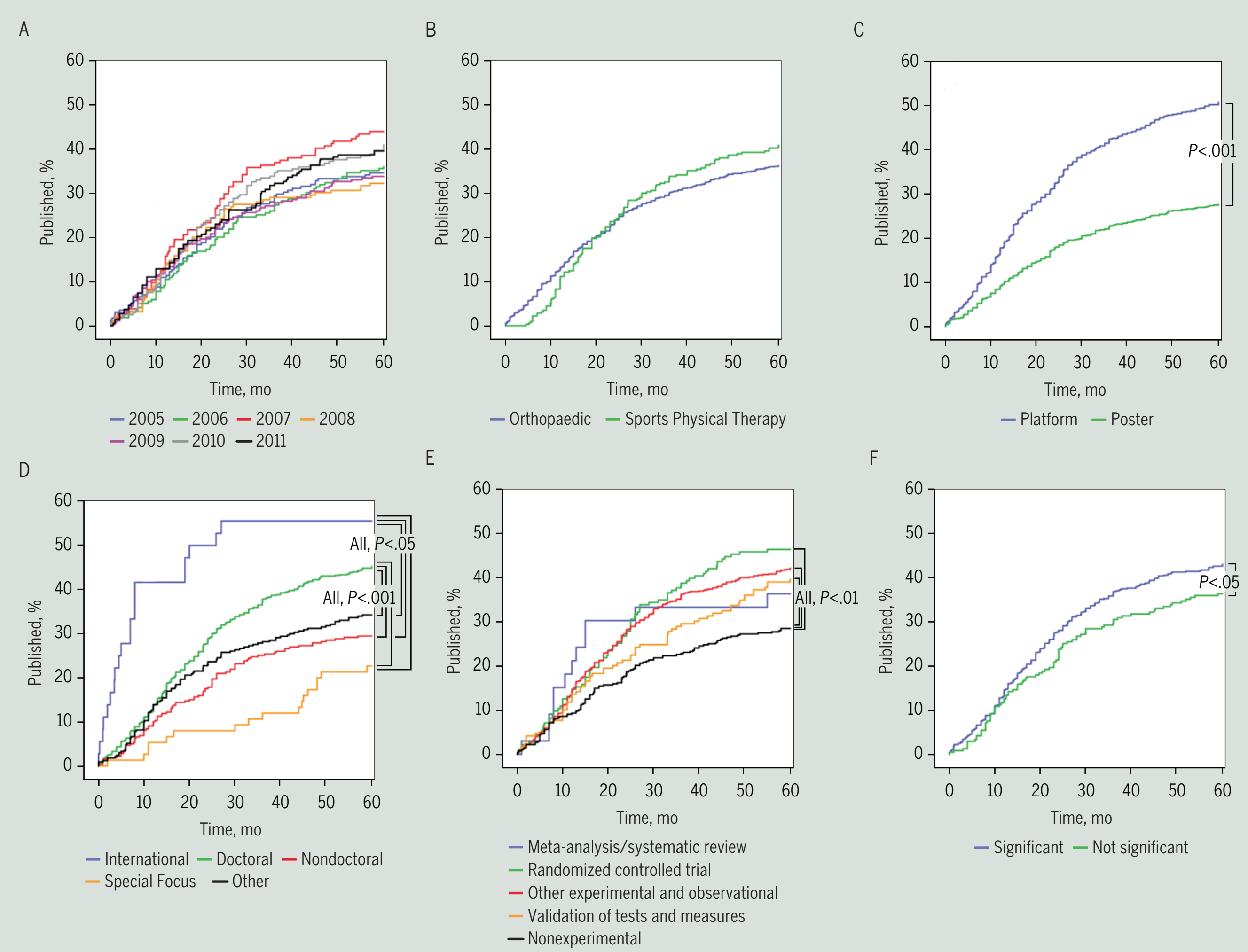

FIGURE. Influence of (A) year of presentation, (B) APTA section, (C) presentation type, (D) institution of origin, (E) study design, and (F) study significance on the full-text publication rate during the 5 years following abstract presentation at the Combined Sections Meeting. Odds of full-text publication increased when the abstract was presented as a platform presentation, originated from a doctorate-granting institution, reported findings of a randomized controlled trial, was a validation of test and measures or other experimental and observational study, or reported a statistically significant finding, as determined by multiple logistic regression. The APTA section and year of presentation had no effect on the odds of full-text publication. Data are presented in one-minus-survival plots, which graph the percent of abstracts that progressed to full-text publication as a function of time. Abbreviation: APTA, American Physical Therapy Association.

characteristic curves were determined to indicate the ability to discriminate full-text publication. Mann-Whitney *U* or Kruskal-Wallis 1-way analysis-of-variance tests were used to establish the influence of abstract features on journal impact factor.

RESULTS

Full-Text Publication Rate

Fifty-seven abstracts (3.7%) were published outside the 5-year publication window and were removed from analyses. Over one third (38.6%, 575/1489) of remaining abstracts (**TABLE 1**) were published in full text in the 5 years following presentation, with a mean ± SD time to publication of 21.3 ± 14.9 months (median [interquartile range], 17.9 [9.9-30.0] months).

Factors Contributing to Full-Text Publication

Occurrence of full-text publication at 5 years was the outcome of interest; however, Kaplan-Meier one-minus-survival plots were generated for data visualization (**FIGURE**). These plots graph the percentage of abstracts that progressed to full-text publication as a function of time.

Presentation type, institution of origin, study design, and study significance all independently increased the odds of full-text publication when adjusting for the other factors (all, $P<.05$) and combined to explain 15.5% of the variance in full-text publication ($P<.001$; Nagelkerke R^2). Year of abstract presentation and APTA section did not impact full-text publication ($P = .25$ and .73, respectively) (**FIGURE** panels **A** and **B**).

Platform presentations were 2.8 (95% CI: 2.2, 3.5) times more likely to be published in full text than were abstracts presented as a poster (**TABLE 2**, **FIGURE** panel **C**). Abstracts from international and doctorate-granting institutions were between 1.6 and 3.5 times more likely to be published in full text than abstracts from non–doctorate-granting, special focus, and other institutions (**TABLE 2**, **FIGURE** panel **D**). There were no differences in publication rate between abstracts from international and doctorate-granting institutions ($P = .36$) or between abstracts from non–doctorate-granting, special focus, and other institutions (all, $P = .11$-.54).

Abstracts presenting RCT, validation of tests and measures, and other experimental and observational data were between 1.6 and 2.3 times more likely to be published in full text than were nonexperimental abstracts (**TABLE 2**, **FIGURE** panel **E**). There were no differences in publication rate between abstracts presenting RCT, validation of tests and measures, and other experimental and observational data (all, $P = .30$-.59). Abstracts presenting meta-analyses/systematic reviews did not statistically differ in their full-text publication rate from abstracts presenting any other study design (all, $P = .27$-.88), likely as a result of insufficient statistical power due to the former's low number (less than 3% of presented abstracts).

TABLE 2 Factors Influencing the Odds of Full-Text Publication and Their Ability to Discriminate Full-Text Publication for Abstracts Presented Within the Orthopaedic and Sports Physical Therapy Sections of the American Physical Therapy Association's 2005 to 2011 Combined Sections Meetings

	Odds Ratio*†	AUC†‡
Presentation type		
Platform versus poster	2.8 (2.2, 3.5)	0.63 (0.60, 0.66)
Institution of origin		
Doctoral versus nondoctoral	1.8 (1.4, 2.5)	0.57 (0.54, 0.60)
Doctoral versus special focus	2.5 (1.4, 4.5)	0.54 (0.50, 0.58)
Doctoral versus other	1.6 (1.2, 2.1)	0.55 (0.51, 0.58)
International versus nondoctoral	2.6 (1.2, 5.4)	0.56 (0.49, 0.62)
International versus special focus	3.5 (1.5, 8.6)	0.66 (0.55, 0.77)
International versus other	2.2 (1.1, 4.6)	0.54 (0.48, 0.60)
Study design		
Randomized controlled trial versus nonexperimental	2.3 (1.6, 3.4)	0.59 (0.54, 0.64)
Validation of tests and measures versus nonexperimental	1.6 (1.1, 2.3)	0.55 (0.50, 0.60)
Other experimental and observational versus nonexperimental	1.7 (1.2, 2.2)	0.57 (0.53, 0.60)
Study finding		
Significant versus not significant	1.4 (1.1, 1.9)	0.53 (0.49, 0.57)

Abbreviation: AUC, area under the curve.
**Determined via multiple logistic regression.*
†Values in parentheses are 95% confidence interval.
‡Derived from receiver operating characteristic curves.

Abstracts presenting statistically significant data were at 1.4 (95% CI: 1.1, 1.9) greater odds of full-text publication than were abstracts presenting data that were not significant (**TABLE 2**, **FIGURE** panel **F**).

Full-Text Publication Features

Publications resulting from work previously presented in abstract form were published in 119 different journals. Twelve journals published over half (56%, 322/575) of the publications, with each journal publishing data from 12 or more abstracts (**TABLE 3**).

Four hundred sixty-eight (81.4%) of the full-text publications were published in journals possessing an impact factor. The median (interquartile range) impact factor of these journals was 2.56 (1.94-3.01). There was no influence of APTA section, year of abstract presentation, presentation type, institution of origin, study design, or study significance on impact factor (all, P = .12-.70).

DISCUSSION

Over one third (38.6%) of abstracts presented within the Orthopaedic and Sports Physical Therapy Sections at the APTA's CSMs between 2005 and 2011 were published in full text during the 5 years following presentation. This represents a 52% increase (P<.001, chi-square analysis) compared to the preceding period of 2000 to 2004.[15] The newly observed publication rate is within the range reported in comparative orthopaedic and musculoskeletal clinical disciplines[3,6,7,9-11,17] and demonstrates important progress in the full dissemination of work within the profession.

A number of factors might have contributed to the recent greater full-text publication rate. Study methodology might have contributed, with our current and former studies using different databases to identify publications (Google Scholar versus PubMed/CINAHL/Evidence-Based Medicine Reviews). To determine the impact of the database searched, we used Google Scholar to reperform the search for abstracts presented during the previous study period (2000-2004). An additional 18 full-text publications were located, resulting in a revised full-text publication rate of 27.6% (227/823 abstracts). Despite the small increase in the retrieval of full-text publications when searching with Google Scholar, the 38.6% publication rate in the current study period (2005-2011) remains 40% greater (P<.001, chi-square analysis).

The greater full-text publication rate during the current study period could be explained by an increase in the number of venues in which to publish work and, in particular, by the ever-growing number of open-access journals. However, the proportion of full-text publications in open-access journals during the current study period accounted for only 2.6% of publications (15/575), compared to 0.5% (1/209) during the previous study period. The lack of an impact of publishing in open-access journals may be due to an unwillingness or inability of authors to pay open-access publication fees and due to confusion and concerns generated by the bevy of predatory journals within the open-access domain that offer a gold (author pays) model with limited peer review or editorial oversight.[2,12]

We speculate that the greater full-text publication rate in the current study period reflects the cumulative influences of a progressive change within the profession toward being more evidence based and an increase in both the quantity and quality of work being performed. There has been an increase in the number of doctorally trained researchers within the profession, with the percentage of core faculty within academic physical therapy departments holding a terminal research degree (ie, PhD) rising from 36% in 2000 to 52% in 2011.[5] These faculty have requirements from the professional accrediting body and institutional promotion and tenure committees to disseminate peer-reviewed scholarly products, with full-text publications being viewed favorably.

As a reflection of the embedding of PhD-trained faculty in academic institutions, the proportion of abstracts presented at CSMs from doctorate-granting

TABLE 3 Journals Publishing Full-Text Publications of Work Presented in Abstract Form Within the Orthopaedic and Sports Physical Therapy Sections of the American Physical Therapy Association's 2005 to 2011 Combined Sections Meetings*

Journal	Publications
Journal of Orthopaedic & Sports Physical Therapy	131 (22.8)
Physical Therapy	43 (7.5)
Archives of Physical Medicine and Rehabilitation	22 (3.8)
American Journal of Sports Medicine	18 (3.1)
Clinical Biomechanics	18 (3.1)
Journal of Manual & Manipulative Therapy	15 (2.6)
Journal of Athletic Training	14 (2.4)
North American Journal of Sports Physical Therapy	13 (2.3)
International Journal of Sports Physical Therapy	12 (2.1)
Manual Therapy	12 (2.1)
Physiotherapy: Theory and Practice	12 (2.1)
Spine	12 (2.1)
Other†	253 (44.0)

*Values are n (%).
†Includes 107 individual journals publishing data from between 1 and 9 presented abstracts.

institutions increased by 14% between the previous (42.9%, 353/823 abstracts) and current (49.0%, 729/1489 abstracts) study periods (P<.01, chi-square analysis). The current study confirms our previous finding that the abstracts originating from doctorate-granting institutions are more likely to progress to full-text publication than are abstracts from all other noninternational institutions.

Progressive changes within the profession toward more evidence-based practice and an increase in the number of doctoral-trained researchers correspond to an increase in both quantity and quality of work being performed.[8] The net result is an increase in the competitiveness and subsequent quality of work selected for presentation at the CSM, and a greater likelihood that the presented work will ultimately meet the rigors of peer review for publication.

Competitiveness for platform presentation at the CSM has gradually risen due to increased numbers of abstracts being submitted annually for a steady number of platform presentations. Platform presentations were 2.8 times more likely to progress to full-text publication than were poster presentations, suggesting greater quality of information selected for the former. As the number of platform presentations at the CSM over the years has remained constant due to meeting logistics, the increase in submitted abstracts has likely led to downstream increases in the quality of abstracts selected for poster presentation. As a result, the current study period observed significant increases in the publication rate of both platform (53.5%, 324/606 abstracts versus 34.8%, 139/400 abstracts) and poster (28.3%, 250/883 abstracts versus 16.5%, 70/423 abstracts) presentations compared to the previous study period (all, P<.01; chi-square analysis).

CONCLUSION

In summary, the current study found that over one third of the abstracts presented within the Orthopaedic and Sports Physical Therapy Sections at the APTA's CSMs from 2005 to 2011 were published as full-text manuscripts within 5 years of presentation. This publication rate is substantially higher than reported for the preceding period (2000-2004), demonstrating important progress in the full dissemination of work within the profession. As scholarly research continues to advance within academic and clinical physical therapy settings, we anticipate that the breadth and rigor of data gathered will also rise, leading to further increases in the rate of full-text publication and greater dissemination of knowledge to physical therapy consumers. ◉

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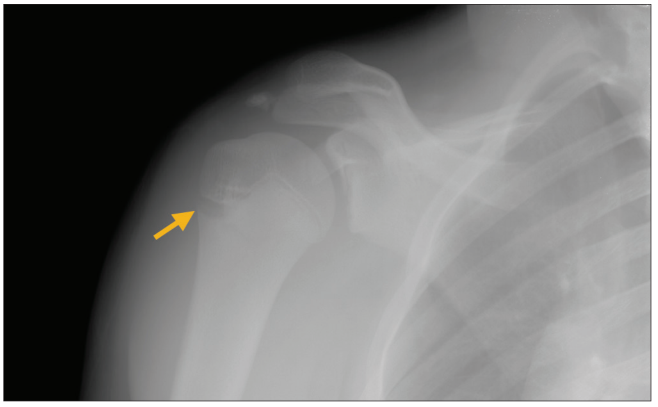

FIGURE 1. Internally rotated right shoulder radiograph showing lateral physeal widening and separation of the epiphysis from the diaphysis (arrow), known as humeral epiphysiolysis.

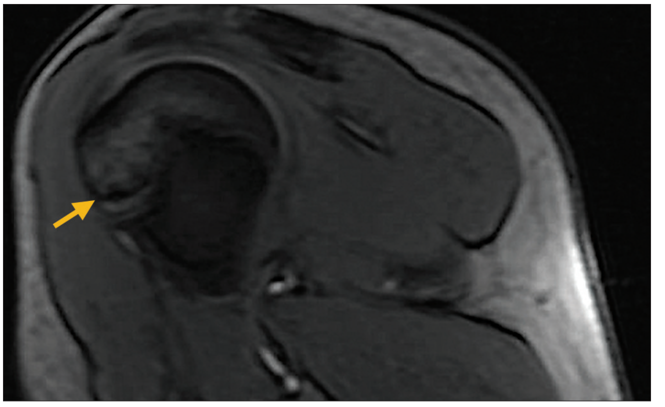

FIGURE 2. Sagittal proton-density, fat-saturated magnetic resonance imaging showing a widening of the lateral physis (arrow) with adjacent edema.

Little League Shoulder in a 15-Year-Old Male Baseball Pitcher

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A 15-YEAR-OLD, RIGHT-HAND-DOMINANT baseball pitcher presented to physical therapy with a 1-week history of acute right shoulder pain experienced during the acceleration phase of throwing. The rotational range of motion of the patient's right shoulder was from 140° of external rotation to 5° of internal rotation, and that of his left shoulder was from 90° of external rotation to 70° of internal rotation. The patient's horizontal adduction on the right was 15° less than that on the left. Subscapularis tendinopathy was suspected, based on pain with active internal rotation and positive special tests (bear hug, belly press, and lift-off).

Due to age, level of activity, and focal pain over the proximal humerus, particularly the lateral aspect,[3] humeral epiphysiolysis was also suspected. Treatment addressing acute symptoms was initiated, with concurrent referral for imaging. The initial plan of care included soft tissue mobilization, modalities, rotator cuff strengthening, scapular stabilization, and a structured return-to-throwing program.

The week after physical therapy evaluation, the patient returned with radiographs and magnetic resonance imaging results that revealed lateral physeal widening with adjacent edema (**FIGURES 1** and **2**). Additionally, a nondisplaced labral tear was noted (**FIGURES 3** and **4**, available at www.jospt.org). The labral tear was considered a subsidiary finding in this case, as humeral epiphysiolysis was the more critical finding to direct the course of treatment. Subsequently, the plan of care shifted to complete cessation of throwing for 3 months.[1] The patient returned to throwing 3 months later without exacerbation of symptoms. In this case, imaging was necessary for an accurate diagnosis of lateral physeal widening, commonly referred to as "Little League shoulder."[2] The diagnosis significantly altered the original plan of care, highlighting the importance of early imaging referral when necessary to help guide physical therapy treatment. ◉ *J Orthop Sports Phys Ther 2018;48(1):51. doi:10.2519/jospt.2018.7369*

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