



Gluteal muscle inhibition: Consequences of patellofemoral pain?

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ABSTRACT

Muscle weakness is a common impairment in females with patellofemoral pain (PFP), clinically seen in both the quadriceps and gluteal muscles. These weaknesses have been suggested to result in poor movement patterns, which results in the clinical presentation of pain due to increased stress on the patellofemoral joint. While quadriceps weakness have been identified as a risk factor for the development of PFP, prospective studies have not found gluteal muscle weakness to be a risk factor. Therefore, gluteal muscle weakness may in fact be a consequence of PFP. This muscle weakness is often combated with traditional strengthening programs. These interventions improve both short-term subjective function and improve strength, yet their relationship to long-term outcomes are less than optimal. Strength training of the gluteals also does not transition to improved movement patterns in this population. The presence of hip muscle inhibition may be the explanation for both poor long-term function and altered movement patterns. Assessment methods for muscle inhibition have been studied in the quadriceps, commonly conducted with the superimposed burst technique to calculate an activation ratio. The use of this method may be used on the gluteus medius and gluteus maximus muscles to determine if inhibition of the hip musculature is present in females with PFP. This knowledge would provide clinicians with a more complete picture of muscle dysfunction and lead to a paradigm shift in clinical treatment strategies for this chronic condition. We hypothesize that muscle inhibition is present in the gluteal muscles of females with PFP compared to healthy controls and it is associated with both decreased subjective function and longer duration of symptoms.

Introduction

Patellofemoral pain (PFP) is one of the most common orthopedic conditions faced by sports medicine clinicians [1]. Incidence rates for PFP range between 9 and 15% across the general population, recreationally active, and military populations [2–5]. Females are 2.23 times more likely to develop PFP compared to their male counterparts [2]. PFP also accounts for 25% of treatment provided to patients attending a sports medicine clinic [1]. While the occurrence of PFP is common, the true etiology is unknown.

The clinical presentation of PFP is pain around or behind the patella during weight-bearing activities, which results in impairments in physical activity and a decrease in health related quality of life [6,7]. Additionally, PFP patients often present with muscle weakness [8–10], poor movement patterns in the frontal plane [11,12], and elevated levels of kinesiophobia [13,14]. One of the challenges with PFP is the longevity of pain and symptoms, up to 91% of PFP patients experience pain 16 years following the initial diagnosis [15]. This long-term impairment results in negative consequences across the lifespan [15]. PFP has also been suggested to be a precursor to the development of patellofemoral osteoarthritis (PFOA) [16,17].

Females with PFP often present with hip muscle weakness, specifically with the gluteus medius and gluteus maximus. This impairment occurs in both isometric and isokinetic strength assessment testing.

Females with PFP are approximately 26% weaker in abduction and 24–36% weaker in hip external rotation compared to healthy controls during isometric strength tasks [8,18]. They also present with weakness during eccentric and concentric testing for both hip abduction and external rotation [19–21]. The hip musculature plays an essential role in pelvic control, and weakness of the gluteus medius has been suggested to altered frontal plane movement patterns in females with PFP across multiple functional tasks [22–24]. This altered movement strategy (Fig. 1; A1) is concerning, as it is suggested to increase stress on the patellofemoral joint and be a potential factor in the patient's chronic pain [25]. (Fig. 1; A2) This pain also has been linked to alter hip muscle activity [26–28], and directly contribute to a feedback loop of altered movement during functional tasks [29–31]. (Fig. 1; A3, C1).

While hip weakness is a common impairment seen in those with PFP, these studies have been retrospective in nature. Prospectively, females who develop PFP do not present with differences in hip strength compared to females who do not [32–34]. Finnoff et al. identified that female runners who developed PFP had stronger hip strength during pre-injury testing and they developed muscle weakness compared to their pre-injury strength following injury [34]. An increased risk for developing PFP was also seen in young female athletes who had greater isokinetic hip abduction strength [33]. These findings suggest that hip muscle weakness may be a result of the pathology and not a factor associated with the PFP development. Additionally, two

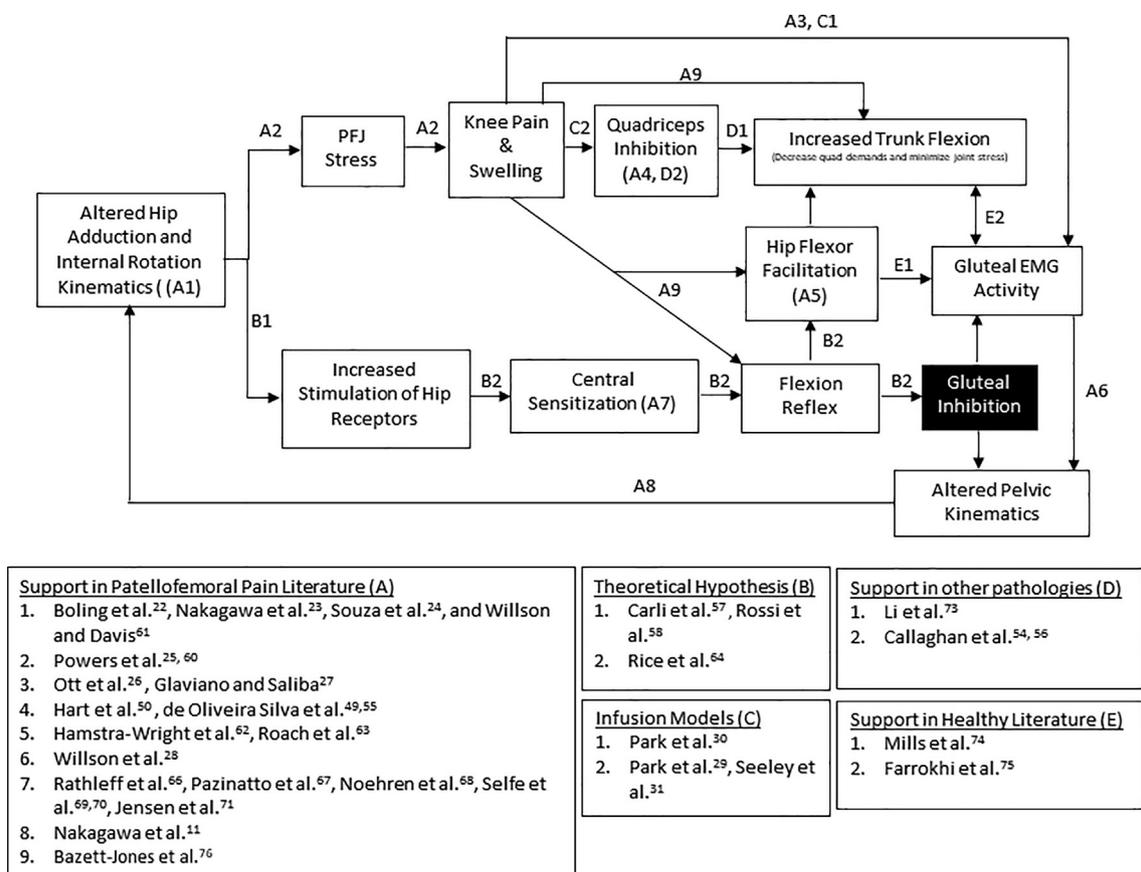
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References located in a box indicate support of that concept, references located on an arrow support the relationship between the two concepts. Letters are used to differentiate the type of supporting literature: A) Evidence in the PFP literature, B) Theoretical Model, C) Knee Infusion Model, D) Pathologies other than PFP, E) Healthy Population.

Fig. 1. Theoretical model for gluteal muscle inhibition in females with patellofemoral pain.

recent systematic reviews suggest that hip weakness is not a risk factor for developing PFP [10,35].

The presentation of hip muscle weakness has led to a paradigm shift in clinical treatment, with an increased emphasis on the hip muscles in addition to the quadriceps [36,37]. Hip-based programs have demonstrated improvement in pain and subjective function one week sooner compared to quadriceps based rehabilitation programs [36,38,39]. Combined strengthening programs that target both the quadriceps and hip muscles are the current recommendation for treating PFP [40]. However, the long-term subjective and objective outcomes are poor, with over 50% of patients reporting unfavorable results 5–8 years following treatment [41]. While these hip-based programs improve gluteus medius strength in females with PFP, there is inconsistent evidence that this translates to improved movement strategies in this population [42–44]. There is also a subset of individuals with PFP who do not improve their hip abduction strength, even at the conclusion of a 6-week proximal intervention targeting the hips [45]. This suggests additional underlying factors may be present, hindering both long-term outcomes and minimizing changes in frontal plane movement.

Advances in more clinically relevant measures of hip function have recently identified deficits in hip power and dynamic strength (10 repetition maximum) in females with PFP for both hip abduction and extension [46,47]. Greater deficits were identified in hip power than isometric strength; however, the authors found no difference in gluteal muscle volume for these patients [46,47]. This suggests that isometric strength does not provide a complete picture of muscular capacity and that novel assessment methods are required to properly evaluate and treat patients with PFP. Neurophysiological assessments may provide this necessary information about muscle function in these patients [48].

Recent evidence has used neurophysiological measures to assist in the discrimination of PFP in clinical practice and found relationships in these measures to both pain and subjective function [49]. While the impairments of the gluteal muscles are common and a focus for intervention programs, the current neurophysiological measures have been limited to the quadriceps muscle.

Muscle inhibition and PFP

Muscle inhibition is reflexive response following result of injury, described by the inability to recruit motor units of any muscle surrounding an injured joint [50]. Clinically, it presents as muscle weakness; however is a result of altered afferent stimuli from receptors to the central nervous system (CNS) [50]. Burst superimposition is a common technique used to quantify the ability to voluntarily recruit motor units, often expressed as the central activation ratio (CAR) [50]. However, investigations of muscle inhibition using this technique are largely limited to the quadriceps [50–53]. Preliminary unpublished data have demonstrated the reliability of the CAR for the gluteal muscles in healthy individuals; however, this had yet to be applied to females with PFP.

Quadriceps muscle inhibition is a common deficit seen in both individuals with PFP and PFOA [50,54]. (Fig. 1; A4, D2) A 2010 systematic review identified that those with the greatest quadriceps inhibition, measured by the CAR, were individuals with PFP [50]. They presented with a 22% deficit in their volitional ability to contract their quadriceps muscle and demonstrated weakness bilaterally [50]. Quadriceps muscle inhibition has also been identified in this population when assessed by the Hoffmann reflex [49,55], which identifies altered

alpha motorneuron excitability at a spinal level. Greater inhibition of the quadriceps muscles is also directly linked to increased pain levels and decreased subjective function in this population [49]. This muscle dysfunction also presents with challenges for those with PFOA and clinicians who treat them, as increased muscle inhibition is associated with the most severe knee pain [49,54]. Most alarming, while interventions to overcome this inhibition have been evaluated in other knee pathologies, there are limited approaches to combat inhibition in the PFP population [56]. Treatment for muscle inhibition at the knee has focused on applications surrounding the knee joint tissue to alter the afferent information transmitted to the CNS [52].

Quadriceps muscle inhibition has been identified in individuals with PFP [49,55], yet evaluating the potential of hip muscle inhibition has yet to be examined. With the shift of focus on gluteal muscle function, assessing muscle function for inhibition may provide essential information to clinicians and researchers. Currently, researchers and clinicians do not understand why those diagnosed with PFP develop muscle weakness, why the long-term outcomes from rehabilitation programs are poor, and why strength training alone does not improve frontal plane motion during functional tasks. Current investigations assessing muscle inhibition are attempting to provide novel information about the pathophysiology of PFP [48] and exploring muscular inhibition of the gluteal muscles is essential for future research to truly understand this complex pathology.

Altered neural pathways

Previous models have suggested that hip joint afferents increase firing at maximum ranges of motion, most notably with frontal and transverse plane motion, which equates to inhibitory responses of the surrounding musculature [57]. Mechanoreceptors located in the posterior and superior capsule are stressed the most during femoral internal rotation and adduction [57,58]. These receptors, which include nociceptors [59], increase their neural output as hip adduction and internal rotation increases [58]. (Fig. 1; A5, B1) This neural output is greatest when maximal motion of the femur is reached along each axis in either isolation or in combination [58]. This is problematic for individuals with PFP for two main reasons, 1) females with PFP often present with greater amounts of hip adduction and internal rotation during functional tasks [23–25,60,61] and 2) this population often presents with limited hip range of motion [62,63]. This may result in reaching the maximal range of motion sooner, stimulating these receptors and altering the joint afferents.

Flexion reflex

Altering the discharge of these receptors has been linked to muscle inhibition through a variety in spinal pathways [64]. While these pathways have mainly been focused on the quadriceps muscle, they have yet to be examined in other muscle groups. One possible spinal pathway is the flexion reflex, which results in extensor inhibition (gluteal) and flexor facilitation (hip flexors). This flexion reflex has been previously suggested to occur in individuals with PFP, as bouts of pain could result in flexion facilitation and extension disfacilitation [65]. Pain experienced by these PFP patients was associated with increased polysynaptic pathway excitability, which could alter the central nervous system and pain thresholds [65].

The flexion reflex can occur from increased joint afferents from the mechanoreceptors, which can result in central sensitization, a common condition seen in individuals with chronic pain conditions [64]. (Fig. 1; A5, B2) Central sensitization often presents with symptoms such as hyperalgesia, sensitivity to touch and temperature, and increased psychosocial factors like anxiety. (Fig. 1; A7) Cohorts of PFP have been found to have altered pressure pain thresholds over their patella and lateral retinaculum [66–68] elevated threshold to sensing light touch to their patella [68], and increased reports of having “cold knees”

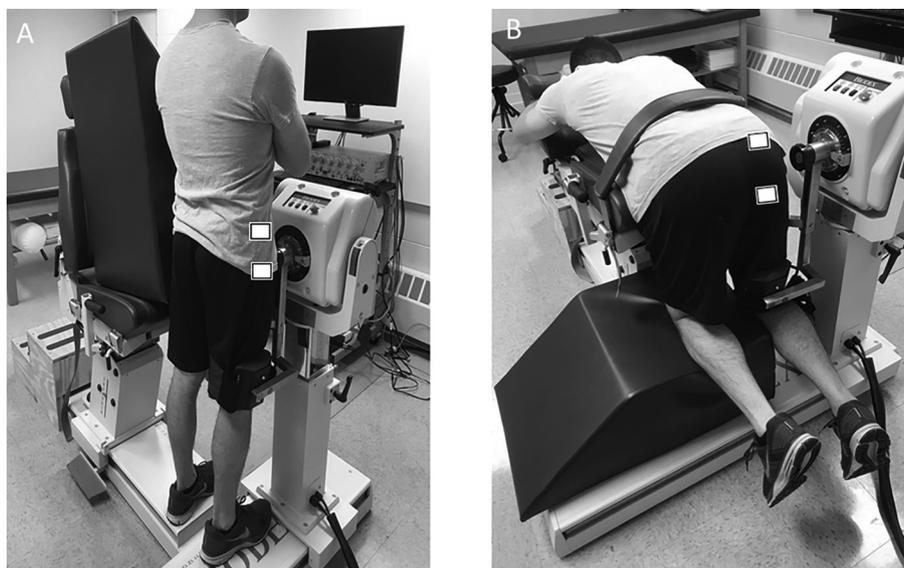
[69–71]. There is also recent evidence that individuals with PFP present with a variety of psychosocial factors, including anxiety [7]. It may be possible that individuals with PFP present with these factors due to central sensitization, resulting from altered neural input to the CNS, abnormal sensorimotor integration, and therefore altered motor response such as poor frontal and transverse biomechanics. A recent systematic review identified a moderate relationship for manifestation of pain sensitization in individuals with PFP, potentially due to repetitive activities that load the knee joint or result in localized inflammation [72]. Pain severity was also associated with greater pressure hyperalgesia at both localized and remote testing locations [72]. While these proposed mechanisms may play a role in the manifestation of pain sensitization, it is purely speculative at this current time, and additional research is required to determine the true mechanism for pain experienced by these patients.

The flexion reflexive pathway could also explain other findings within the PFP literature. One such finding is hip flexor tightness, which has been found within females with PFP [62,63]. Over activity of the hip flexors may also influence trunk motion during tasks. While it has been proposed that increased trunk flexion is a compensation strategy for quadriceps weakness [73] (Fig. 1; D1) and minimizing stress on the patellofemoral joint, it could be possible that the flexor facilitation may assist in this compensation strategy and result in increased trunk flexion (Fig. 1; A8). This increased muscle tightness, assessed by range of motion, in the hip flexors has found to alter gluteal muscle activation during functional tasks, albeit within a healthy population [74]. (Fig. 1; A6, E1) It has also been identified that changes in trunk flexion also contribute to changes in gluteal muscle function [75]. (Fig. 1; E2) It is well documented that females with PFP also present with altered gluteal muscle activity during a variety of functional tasks [9]. Additional research is needed to identify any relationship in hip flexor facilitation on gluteal muscle activation and kinematics during functional tasks in a PFP population.

Knee pain and gluteal inhibition

Knee infusion models have also provide some insight into the role of anterior knee pain on hip muscle function (Fig. 1; C1, C2). Neural effect of the knee pain could provide some addition insight into why females with PFP often present with hip muscle weakness. Park et al. [30] identified that experimental knee pain resulted in a 10% decrease in gluteus medius activity during a jumping task as well as a 10% increase during the landing phase of the same task. This may suggest that knee pain may have a relationship with gluteal muscle activation, specifically motor unit availability to complete the task. Therefore, those available motor units would need to activate at a greater demand in an effort to provide lower limb control of the hip during the eccentric phase of the task, which is supported by the findings of Park et al. [30]. They also found that an inverse relationship between the gluteus maximus and hamstring muscle function during the pain model, where gluteus maximus activity decreased by 20% and hamstrings increased by 10% [30]. These findings demonstrate altered gluteal muscle function in the presence of anterior knee pain, suggesting the need for additional measures of gross neuromuscular function of the gluteal muscles within a population who is experiencing anterior knee pain. Additionally, both prospective and retrospective studies would need to be conducted to evaluate the influence of pain on motor unit recruitment of the gluteal muscles and their relationship during functional tasks.

There is also some evidence of altered gluteal function when increasing pain experienced in individuals with PFP. Bazett-Jones et al. [76] evaluated the effect of an exhaustive run on trunk and lower extremity running kinematics in individuals with PFP. Those with PFP had a significant increase in their knee pain, 2.26 on the visual analog scale, and demonstrated increased knee flexion, hip flexion, and anterior pelvic tilt following the run [76] (Fig. 1; A9). Additionally, there



Testing position for gluteus medius (a) and gluteus maximus (b) testing. White boxes represent location of electrode placement for testing

Fig. 2. Participant set-up for gluteus medius and gluteus maximus superimposed burst technique assessment.

was no change in the amount of knee flexion, hip flexion, or anterior pelvic tilt seen in the healthy population [76]. The authors also found an increase in hip extension moments following the exhaustive run [76]. They suggested this increased hip extension moment to be a compensation strategy to decrease the load placed on the knee and theorized increased hamstring and adductor activity to allow completion of the running task [76]. While they did not collect EMG activity in their study to evaluate this hypothesis, this is similar to the increase hamstring activity found by Park et al. [30] in the knee infusion model.

Hypotheses

While muscle inhibition has been identified in the quadriceps for those with PFP [49,55], there are no studies that have evaluated if gluteal muscle inhibition is also present in this population. Since altered kinematics may change joint afference and lead to muscle inhibition, and those with PFP present with impairments in their gluteal function, it is possible that those with PFP have inhibition to their gluteal muscles. Evaluating for underlying muscle inhibition in the gluteus medius and gluteus maximus muscles can be performed by conducting the superimposed burst technique assessments of both muscles and calculate an activation ratio. These measures have been found to be reliable and accurately recruit the motor units of both gluteal muscles in a healthy population. The next step will be using this assessment method on females with PFP. We propose the following hypotheses:

1. Females with PFP will present with muscle inhibition of both their gluteus medius and gluteus maximus compared to matched healthy females.
2. Females with greater gluteal muscle inhibition will also present with greater levels of pain and decreased subjective function.
3. A weak relationship between gluteal muscle strength and gluteal muscle inhibition will be seen in females with PFP.

Evaluation of hypotheses

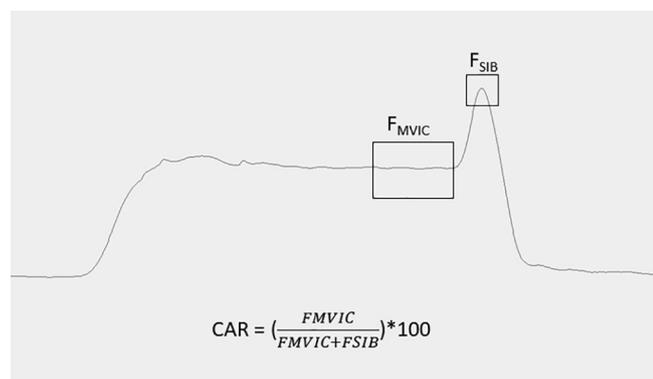
In order to improve interventions when treating this chronic condition, novel evaluation methods must be conducted to provide insight into gluteal muscle function. We intend to perform the superimposed burst technique on the gluteus maximus and gluteus medius muscles in

females with PFP to identify if muscle inhibition is present in this population. Additionally, we will be evaluating if there are any relationships with subjective function or duration of symptoms to gluteal activation ratios.

To calculate an activation ratio of the gluteal muscles, we aim to conduct similar methods of the superimposed burst technique [51], but to the gluteus medius and gluteus maximus muscles. To complete this task, the function of each muscle and the pennate angles that contribute to their function must be understood. This is essential for two reasons: 1) participants must be situated in a way to allow for the calculation of their maximal voluntary contractions within a standard dynamometer and 2) ensure proper electrode positioning to deliver the stimulus to accurately assess the motor neuron pool of the muscle of interest.

Participant positioning for gluteus medius testing will be conducted in a standing position to minimize the influence of gravity. The dynamometer's axis of rotation will be aligned to the center of the anterior hip and the attachment arm will be secured superior to the knee joint for the test limb. The chair of the dynamometer will be in the forward position so it is in contact with the participant's contralateral hip and a foam block will be placed between the chair and participant's entire lateral trunk to minimize aberrant trunk movement. During testing, participant will stand on their non-test limb, place their arms across their chest, and abduct their test hip into the dynamometer attachment arm. Electrode position will be superior to the greater trochanter and across the center of the most superior aspect of the muscle, just distal to the iliac crest. This electrode position will be to minimize direct stimulation to the anterior and posterior fibers of the muscle, which are responsible for hip internal rotation and hip external rotation, respectively (Fig. 2a).

Gluteus maximus testing will require the participant to be prone on the dynamometer with the hip flexed to 90 degrees and knee flexed to 90 degrees. The axis of rotation will be at the greater trochanter and the attachment arm will be on the posterior distal thigh, just superior to the knee joint. Stabilization straps will be placed over the trunk and hips to minimize movement. Participants will perform maximal voluntary contractions by extending their hip. The gluteus maximus has two distinct portions, the superior and inferior fibers, with the latter contributing to hip extension. Electrode position for the gluteus maximus will be just inferior to the posterior gluteal line of the ilium and medial to the greater trochanter along the line of its insertion to the iliotibial



CAR: Central Activation Ratio; FMVIC: Force, Maximal Voluntary Isometric Contraction; FSIB: Force, Superimposed Burst Technique

Fig. 3. Central activation ratio calculation.

band (Fig. 2b).

Participants will perform a warm-up period with submaximal contractions to allow for familiarization. During testing, participants will provide a maximal voluntary contraction during hip abduction or extension (F_{MVIC}), depending on the muscle being tested. The F_{MVIC} will be normalized to body mass (Nm/kg). The researcher will evaluate the data real-time and when a visible plateau in torque data is observed an electrical stimulus will be manually applied. The stimulus will be a 100 ms train of 10 square-wave pulses at an intensity of 125 V, delivered by a Grass S88 dual-output square-pulse stimulator with a Grass SIU8T stimulator isolation unit. This stimulus will result in an increase in torque, termed the superimposed burst (F_{SIB}). The central activation ratio is calculated by comparing the participant's F_{MVIC} , 100 ms prior to the stimulus, to the total torque of the F_{MVIC} and F_{SIB} stimulus $CAR = \left(\frac{F_{MVIC}}{F_{MVIC} + F_{SIB}} \right) * 100$ (Fig. 3).

Clinical implication

Quadriceps muscle weakness and inhibition are commonly identified in individuals with PFP [49–51,53,55]. When examining other impairments commonly seen in this population, gluteal muscle weakness has also been identified. However, no studies have evaluated the presence of gluteal muscle inhibition in those with PFP. It is important to evaluate if muscle inhibition is present in gluteal muscles and determine if this measure has a relationship to pain and subjective function in those with PFP. Additionally, evaluating how strong the relationship between strength and muscle activation will provide researchers and clinicians insight into if inhibition provides unique information when assessing muscle function in this population. If muscle inhibition of the gluteal muscles is present, a shift in both the current evaluation and treatment of PFP is required. These findings would also allow future research to evaluate treatment methods to combat this muscle inhibition, and allow for recommendation to clinicians who treat this condition. Therefore, to improve clinical care in patients with PFP, it is essential to identify if muscle inhibition is present in this population by conducting novel assessment methods. The outcomes of this study would provide future direction for serial testing following PFP diagnoses to understand the neuromuscular natural history, from pathology to inhibition and muscle weakness. It may also provide the ability to help diagnose PFP, which is currently a very difficult pathology to define in both clinical practice and clinical research. The outcomes would also suggest the need to identify interventions to overcome this impairment and evaluate its effectiveness to improve the outcomes for individuals suffering from PFP.

Conflict of interest

The authors would like to disclose that there are no known conflicts of interest associated with this publication.

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