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Quadriceps Inhibition After Naturally Occurring Patellar Tendon Damage and Pain

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Context: After knee-joint injury, pain, effusion, and mechanoreceptor damage alter afferent signaling, which can result in quadriceps inhibition and subsequent weakness. The individual contributions of each factor to inhibition remain unclear due to confounding knee-joint injuries and indirect experimental models.

Objective: To characterize the influence of naturally occurring knee damage and pain on quadriceps neuromuscular function in individuals with patellar tendinopathy.

Design: Cross-sectional study.

Setting: Research laboratory.

Patients or Other Participants: Twenty participants who self-reported patellar tendinopathy (PT) and 10 healthy control individuals underwent ultrasonic tendon assessment. Injured participants were dichotomized by an orthopaedic surgeon into groups with (1) pain and structural tendon abnormality and (2) regional pain alone.

Main Outcome Measure(s): Quadriceps inhibition was assessed with the Hoffman reflex and the central activation ratio via the superimposed-burst technique. Normally distributed measures were analyzed using a 1-way analysis of variance and post hoc independent *t* tests. Kruskal-Wallis tests with post hoc Mann-Whitney *U* tests were used to analyze nonnormally distributed data. An a priori α level of $P \leq .05$ was set.

Results: Control participants presented with more spinal-reflex excitability (0.37 $\pm$ 0.23) than the PT (0.10 $\pm$ 0.06; $P = .03$) and regional-pain (0.18 $\pm$ 0.05; $P = .02$) groups. Knee-extension strength was greater in the control (3.37 $\pm$ 0.59 Nm/kg) than in the PT (2.41 $\pm$ 0.67 Nm/kg; $P = .01$) group but not the regional-pain group (3.05 $\pm$ 0.66 Nm/kg; $P = .24$). Control individuals presented with more quadriceps activation (97.93% $\pm$ 3.12) than the PT (84.44% $\pm$ 16.98; $P < .01$) and regional-pain (91.17% $\pm$ 10.56; $P = .01$) groups. No differences were present for any measures between the PT and regional-pain groups (P values $> .05$).

Conclusions: Deficits in spinal-reflex excitability, quadriceps activation, and strength were present in both the PT and regional-pain groups. A combination of pain and structural damage appeared to have the greatest negative effect on quadriceps function, as only the PT group presented with neuromuscular outcomes that failed to meet clinical thresholds.

Key Words: diagnostic imaging, muscle inhibition, tendinopathy, knee pain

Key Points

- Patellar tendon damage and pain often coexist, and a combination of these factors appears to be more potent than either one in isolation.
- Patellar tendon damage and pain alter the afferent signaling from the joint and, therefore, evaluating the extent of damage and pain can guide therapeutic prescriptions.

Quadriceps muscle weakness is a common neuromuscular impairment after knee injury and is hypothesized to result from neurologic changes within the joint.[1–3] The primary influences of these neural alterations are pain, effusion, and mechanoreceptor damage, which alter afferent signaling from the joint to the central nervous system.[1,4] Modified afferent signals trigger inhibitory processes that limit quadriceps volitional contractility and perpetuate weakness.[1,5–7] The multifaceted nature of knee injuries makes it difficult to identify the most significant contributors to this neurally mediated quadriceps weakness, as pain, effusion, and damage often coexist. This uncertainty has led the scientific community to rely on simulated-pain and-effusion models to untangle the neuromuscular consequences of knee injury.[4–6] However, the knee-joint insult in these controlled laboratory studies does not faithfully mimic natural injury or tissue damage, and thus, our understanding of quadriceps inhibition is limited. An alternative model of knee injury that allows for isolation of specific factors is needed to comprehensively explore the causes of quadriceps weakness after knee-joint injury.

A feasible model for isolating the individual contributions to quadriceps weakness involves the comparison of participants with patellar tendinopathy (PT) with those who have pain in the patellar tendon region. A diagnosis of PT is best characterized as pain localized at the inferior pole of the patella during dynamic activity, with associated tendon thickening, areas of hypoechoic lesions, and the absence of edema,[8] whereas regional patellar tendon pain may exist in

Table. Participants' Characteristics

Variable	Group			*P* Value
	Patellar Tendinopathy	Regional Pain	Healthy Controls	
Demographics				
Sex, n (males/females)	5 (4/1)	15 (9/6)	10 (6/4)	NA
Age, y	21.20 ± 0.84	20.40 ± 2.90	22.20 ± 3.05	.29
Height, cm	178.82 ± 4.95	173.74 ± 9.35	173.50 ± 9.33	.48
Mass, kg	79.00 ± 6.64	70.35 ± 12.39	72.63 ± 14.25	.41
Subjective reports				
Victorian Institute for Sport Assessment, Patellar Tendon score	59.60 ± 14.36[a]	62.21 ± 13.97[a]	96.70 ± 4.27	<.01[b]
Pain at rest (10-cm VAS score)	0.88 ± 0.84[a]	0.94 ± 1.45	0.05 ± 0.07	.03[b]
Pain at maximal voluntary isometric contraction (10-cm VAS)	4.82 ± 2.53[a]	3.02 ± 2.45[a]	0.09 ± 0.11	<.01[b]
Tegner Activity Level Scale score (current)	7.80 ± 1.30	6.87 ± 2.13	7.40 ± 1.17	.54
Strength and inhibition				
Hoffman reflex	0.10 ± 0.06[a]	0.18 ± 0.05[a]	0.37 ± 0.23	.02[b]
Maximal voluntary isometric contraction, Nm/kg	2.41 ± 0.67[a]	3.05 ± 0.66	3.37 ± 0.59	.04[b]
Central activation ratio	84.44 ± 16.98[a]	91.17 ± 10.56[a]	97.93 ± 3.12	<.01[b]

Abbreviations: NA, not applicable; VAS, visual analog scale.

[a] Post hoc difference from healthy control group ($P < .05$).

[b] Group difference ($P < .05$).

the absence of structural tendon damage.[8] The similar locations of pain in both conditions provide the opportunity to explore possible individual contributions of pain and damage to quadriceps inhibition.

The complex relationship between corticospinal inhibition and pain has been preliminarily explored in patients with PT versus patellar tendon regional pain.[8,9] From this mechanistic angle, investigators[8,9] found that pain influenced corticospinal sources of quadriceps inhibition and strength. However, the afferent pathways that contribute to quadriceps inhibition resulting from naturally occurring pain and damage remain uncharacterized. Altered afferent signaling has been identified after traumatic joint injury as a possible precursor to central nervous system alteration and deficits in global quadriceps activation and strength,[10,11] but this relationship has yet to be observed after naturally occurring knee damage and pain.

Given the aforementioned gaps in and limitations of previous work, we sought to compare individuals with PT or pain in the patellar tendon region with healthy control participants to characterize the influence of naturally occurring knee pain and tendon damage on quadriceps neuromuscular function. We hypothesized that those with combined tendon damage and pain would exhibit greater *quadriceps inhibition,* as defined by reduced strength, spinal-reflex excitability, and global activation deficits, than healthy control participants or those with pain alone.

METHODS

Recreationally active college-aged individuals who self-reported PT and healthy control volunteers were recruited via informational flyer and word of mouth to participate in a single test session as part of a cross-sectional research study that was approved by the University of Connecticut's Institutional Review Board. Once each participant provided informed consent, he or she was asked to complete subjective assessments for pain, injury severity, and activity level. A 10-cm visual analog scale anchored by *no pain* and *worst possible pain* was used to objectively rate the tendon pain in the injured knee.[9] The visual analog scale captured 2 types of pain: (1) pain at rest (before spinal-reflex excitability testing) and (2) pain during maximum voluntary isometric contraction (MVIC). Symptom severity was assessed using the Victorian Institute of Sport Assessment, Patellar Tendon (VISA-P).[12] The scale consists of 8 visual analog questions on pain and functional ability that are rated from 0 (*nonfunctional*) to 100 (*fully functional*). Activity level was evaluated using the Tegner activity level scale from 0 (*sick leave or disability due to knee problems*) to 10 (*national elite competition*).[13] Next, participants underwent the following assessments: ultrasound analysis of the tendon, spinal-reflex excitability, quadriceps strength, and global quadriceps muscle inhibition. This testing order was designed to limit the potential influence of the evaluations on tendon neovascularization and afferent sensory spinal input. Each measure was assessed in the injured limbs of the PT and regional-pain groups and the right limbs of healthy control individuals.

Procedures

Participant demographics are shown in the Table. For each person, a single trained clinician performed grayscale sonographic measurement (model 3200t with 4-cm linear transducer; Terason, Burlington, MA) of the patellar tendon to dichotomize injured participants into groups of those with (1) PT and (2) patellar tendon regional pain and to confirm that the healthy individuals were free of structural tendon abnormality. Inclusion criteria for the PT group were self-reported pain during physical activity and a hypoechoic lesion at the inferior pole of the patella.[8,9] *Patellar tendon regional pain* was defined as self-reported pain in the absence of tendon abnormality.[8] If bilateral symptoms were present, the knee with the greatest self-reported symptoms was tested.[8] Healthy participants were free of pain and structural tendon damage. Tendon structural damage was confirmed on the ultrasound images (Figure) by an orthopaedic surgeon blinded to participant status. Exclusion criteria for all groups were lower extremity injury within 6 months before testing (other than patellar tendon damage or pain or both in the injured

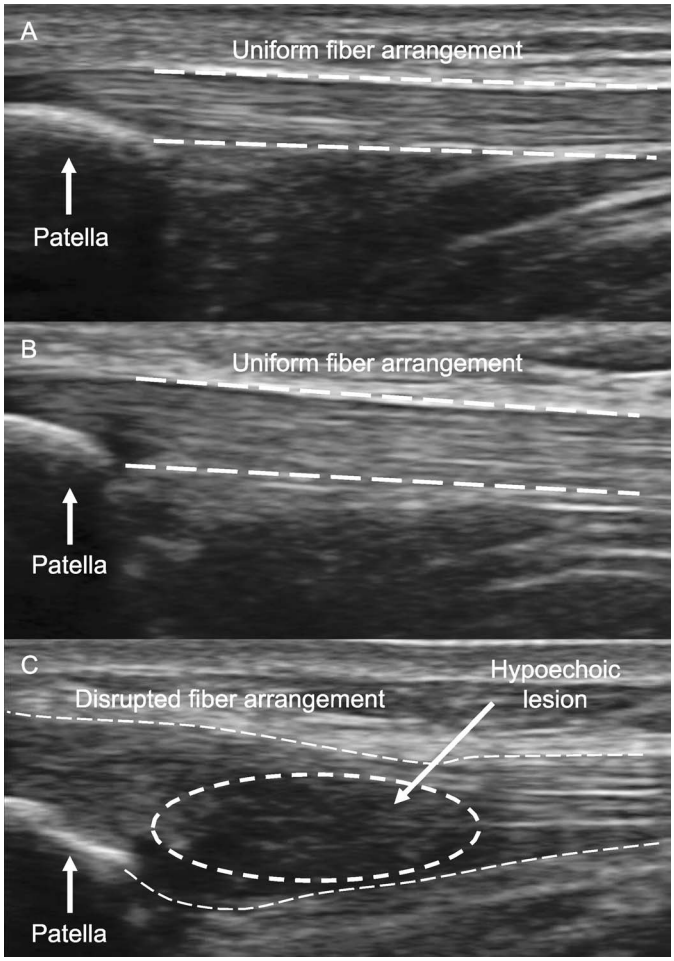

Figure. Representative ultrasound images of each group. A, Healthy control group. B, Regional-pain group. C, Patellar tendon group.

group), a history of lower extremity surgery, pregnancy, or a cardiac condition.

Spinal-Reflex Excitability

The Hoffman reflex (H-reflex) normalized to maximal muscle response (H : M) of the vastus medialis was assessed to measure differences in quadriceps spinal-reflex excitability. The H-reflex is an electrically induced stretch reflex that quantifies the ability of the spinal cord to transform peripheral afferent information into a reflexive contraction.[11] Participants were positioned supine on a portable treatment table with their arms placed at their sides, head resting in neutral on a pillow, and knees flexed to 10° to 15° and supported by a half bolster.[11] During testing, participants were instructed to maintain constant head, eye, and hand positions. Two 10-mm, pregelled Ag-AgCl electromyography electrodes (model EL503; BIOPAC Systems, Inc, Goleta, CA) were placed 1.75 cm apart on cleaned and shaven sites over the medial vastus medialis muscle belly. An active 2-mm shielded disc-stimulating electrode (model EL2524S; BIOPAC Systems, Inc) was positioned over the femoral nerve and secured with hypoallergenic tape, and a 7- × 13-cm self-adhesive dispersive electrode was positioned over the hamstrings muscles. A stimulator module and a 200-V maximum stimulus adaptor (models STM100A and STMISOC, respectively; BIOPAC Systems, Inc) delivered a 1-millisecond square-wave stimulus to the femoral nerve.[11] Electromyography signals were bandpass filtered from 10 to 500 Hz and collected at 1024 Hz with a common mode rejection ratio of 110 dB.[11] Once each participant was settled, the stimulus intensity was applied in 0.2-V increments until a maximal H-reflex was obtained. A maximal H-reflex was the maximal peak-to-peak amplitude at which further increasing the stimulus would result in a decrease of amplitude. Next, the stimulus voltage was increased until no further increase in peak-to-peak amplitude of the muscle response (M-response) was measurable or the 20-V maximum was reached. The average of the 3 maximal H-reflexes was normalized to the average of the 3 maximal muscle responses to create the H : M outcome variable for analysis.

Global Quadriceps Inhibition

To quantify global quadriceps inhibition, the superimposed-burst technique was performed on an isokinetic dynamometer (model System 4; Biodex Medical Systems, Inc, Shirley, NY) to assess volitional muscle-activation failure. Each participant was seated in the dynamometer so that the hips and knees were flexed to 90°.[14] Two self-adhesive 7- × 13-cm stimulating electrodes (model Dura-Stick II; Chattanooga Group, Hixson, TN) were placed on the participant's thigh in locations that had been cleaned with isopropyl alcohol and were shaved free of hair. The superior electrode was placed over the vastus lateralis so that its medial border was in line with the anterior-superior iliac spine at the level of the greater femoral trochanter, while the inferior electrode was placed over the vastus medialis so that the lateral border aligned with the midpoint of the patella at a height of 3.8 cm above its superior pole.[14] Once the participants were properly positioned, we identified MVIC strength from an average of 3 knee-extension trials, which were performed until the increase in torque between trials was less than 10%.[2] Finally, an additional 3 trials were completed with the stimulus delivered through a custom-written computer program (LabVIEW version 8.5; National Instruments Corp, Austin, TX) once the knee-extension MVIC had been reached and dropped by 1 Nm (100-millisecond train of 10 stimuli, 100 pulses per second, 0.6-microsecond pulse duration, 0.01-millisecond pulse delay, 150 V).[14] We used a dual-output square-pulse stimulator and SIU8T isolation unit (model S48; Grass Technologies Corp, West Warwick, RI) to apply the superimposed bursts. Participants were given visual feedback and oral encouragement for maximal effort during each trial. Global quadriceps inhibition was quantified using the average central activation ratio (CAR) from 3 trials as shown[14]:

$$\mathrm{CAR} = \left(\frac{\mathrm{MVIC}}{\mathrm{MVIC} + \mathrm{Superimposed\ Burst}}\right) \cdot 100$$

Statistical Analyses

For the statistical analyses, we used a single independent grouping variable (PT, regional pain, or control). The main outcome measures of pain, VISA-P, H : M, MVIC, and

CAR were the dependent variables. One participant in the patellar tendon regional-pain group dropped out after completing H-reflex testing due to discomfort from the stimulus and, thus, those data were not included in the MVIC and CAR analyses. Normality of all main outcome measures was assessed. The outcome measures that were normally distributed across groups were analyzed using 1-way analysis of variance to identify group mean differences in pain score during MVIC, Tegner score, or strength. The Kruskal-Wallis test was used to analyze nonnormally distributed data, which included the VISA-P score, pain score at rest, H : M, and CAR. Pairwise comparisons were further analyzed using independent t tests or Mann-Whitney U tests for outcome measures that were normally or nonnormally distributed, respectively, when significant group main effects were identified. Means and standard deviations for all demographic and main outcome variables are shown in the Table. The 95% confidence interval (CI) of the mean difference and effect sizes were calculated for all main outcome measures as appropriate.[15] Medians and ranges were reported for all variables (Appendix). Effect sizes of normally distributed variables were reported as Cohen d, while nonnormally distributed variables were reported as Cohen r.[15] Significance was set a priori at an α level of $P < .05$, and all statistical analyses were performed using SPSS (version 25; IBM Corp, Armonk, NY).

RESULTS

No differences in participant demographic information were found between groups (P values $> .05$; Table). Differences were identified in VISA-P scores ($P < .01$). Healthy control participants reported better knee function than both the PT ($P < .01$, Cohen $r = 0.81$) and regional-pain ($P < .01$, Cohen $r = 0.84$) groups. No differences were present for VISA-P score between the PT and regional-pain groups ($P = .74$, Cohen $r = 0.07$). Finally, at the time of testing, all groups were functioning at similar activity levels (Tegner score: $F = 0.629$, $P = .54$). These results indicate that our inclusion criteria successfully captured a cohort of knee-injured individuals who were closely related in measures of pain, injury severity, and activity level before data collection.

Differences in pain at rest were observed for all groups ($P = .03$). Those with PT had more pain than healthy control individuals ($P < .01$, Cohen $r = 0.74$) but pain similar to that of participants with regional pain alone ($P = .40$, Cohen $r = 0.19$). Pain at rest did not differ between the regional-pain and healthy control groups ($P = .06$, Cohen $r = 0.38$). Differences were also present between pain scores with MVIC ($F = 11.01$; $P < .01$); the healthy control group reported less pain with MVIC than both the PT ($P < .01$; 95% CI = –6.39, –3.07, d = 2.64) and regional-pain ($P = .01$; 95% CI = –4.56, –1.32, d = 1.23) groups, but the PT and regional-pain groups did not differ ($P = .18$, d = 0.72).

Spinal-reflex excitability differences were present between groups (H : M: $P = .02$). The healthy control group demonstrated more spinal-reflex excitability compared with both the PT ($P = .03$; 95% CI = 0.04, 0.51, d = 1.36) and regional-pain ($P = .02$, Cohen $r = 0.46$) groups. No difference in spinal-reflex excitability was present between the PT and regional-pain groups ($P = .49$, Cohen $r = 0.16$). These results indicate that afferent signaling was altered after knee damage and pain.

Normalized-strength differences were identified between groups (MVIC: $F = 3.73$, $P = .04$). The healthy control group presented with greater knee-extension strength than the PT group ($P = .01$; 95% CI = 0.23, 1.68, d = 1.51) but not the regional-pain group ($P = .24$, d = 0.50). No differences in strength occurred between the PT and regional-pain groups ($P = .08$, d = 0.96).

Between-groups differences were demonstrated for global quadriceps activation (CAR: $P < .01$). The healthy control group had greater quadriceps activation than both the PT ($P < .01$, Cohen $r = 0.70$) and regional-pain ($P = .01$, Cohen $r = 0.58$) groups. No differences were identified between those with PT and regional pain ($P = .23$, Cohen $r = 0.28$). Thus, those groups experiencing PT and regional pain both had a reduced ability to volitionally contract the quadriceps.

DISCUSSION

The primary purpose of our study was to compare individuals experiencing PT or pain in the patellar tendon region and healthy control participants to characterize the influence of naturally occurring knee pain and tendon damage on quadriceps neuromuscular function. Although we found differences in all measures between individuals with knee symptoms and those who were healthy, only 5 participants demonstrated structural damage via ultrasound. The preliminary insight our work offers is that compared with persons who had regional patellar tendon pain, those with structural damage demonstrated worse function across all neurologic measures. These data represent the first report on reduced spinal-reflex excitability and global quadriceps activation in individuals experiencing patellar tendon damage and pain.

Pain alters afferent signaling from the knee joint and can reduce quadriceps volitional and reflexive contractions.[4,16–18] The PT and regional-pain groups scored similarly for subjective pain at rest, pain at MVIC, and injury severity on the VISA-P. As such, our chosen models of knee injury allowed us to investigate individual contributions to quadriceps inhibition by recruiting people who had similar clinical findings but differed in the extent of true patellar tendon damage. Patellar tendon regional pain presents with generalized pain over or around the patellar tendon during physical activity in the absence of structural tendon abnormality. Patellar tendinopathy presents with pain localized to the inferior pole of the patella during physical activity in conjunction with tendon degeneration. Similar locations and onsets of pain with different tendon structural properties permitted comparisons of quadriceps inhibition between individuals with pain alone and those with pain plus structural damage.

Pain may alter the capacity for reflexive muscular contraction through a variety of pre- and postsynaptic mechanisms. Deleterious changes in afferent information originating at the joint can result in presynaptic inhibition,[19] Renshaw cell dysfunction,[19] or alterations in the γ-motoneuron loop,[20] all of which reduce muscular function. Individuals from both the PT and regional-pain groups presented with reduced H : M ratios compared with the healthy group, which indicates a reduced ability to

reflexively contract the quadriceps. Reduced spinal-reflex excitability has been associated with lower extremity strength deficits after anterior cruciate ligament injury and in knee-effusion models.[5,10,11,21] To our knowledge, we are the first to observe alterations in afferent signaling pathways that result in spinal-reflex–mediated quadriceps inhibition, specifically related to patellar tendon pain and damage. Similarly, compared with healthy control individuals, both the PT and regional-pain groups exhibited global quadriceps inhibition. The central activation ratio quantifies the availability of the α-motoneuron pool for voluntary recruitment, but this measure does not identify specific underlying causes of quadriceps dysfunction. Two major contributors to global quadriceps inhibition are spinal-reflex and corticospinal inhibition.[7] Rio et al[8] reported corticospinal inhibition in individuals with PT compared with the healthy control group, while the regional-pain group did not differ from either the healthy or PT group. Further, corticospinal inhibition was effectively treated with pain-reducing modalities in a small cohort of individuals.[9] Our data, in combination with those of Rio et al,[8,9] may offer insight into a paradigm similar to that for traumatic knee injury in which quadriceps inhibition is initiated by alterations to afferent signaling that eventually result in long-term neuroplastic changes to corticospinal pathways.[11,22,23] These underlying neural changes ultimately could lead to global quadriceps inhibition and weakness. Importantly, the proposed progressive mechanism of inhibition is based on work performed after traumatic joint injury and not simulated injury, pain, or effusion.[11,23] Additional investigation of the proposed paradigm after PT and regional pain is needed to provide critical information on the global neuromuscular consequences of knee injury.

As previously stated, quadriceps inhibition is likely an instigator of protracted weakness and has been repeatedly demonstrated in both clinical and laboratory models of lower extremity injury.[1–7,10,11,23,24] The PT group presented with quadriceps weakness (2.41 Nm/kg) during the isometric knee-extension exercise, which was well below the 3.0 Nm/kg that is reported for optimal patient-reported outcomes after other knee injuries.[25] Weakness is a common symptom of PT[26–30] that results in functional deficits. In patients with severe PT, this weakness is known to influence an individual's decision to cease activity participation.[31] In previous work with experimentally induced knee pain and effusion, Palmieri-Smith et al[4] found that the interaction between the 2 variables did not result in a larger deficit of quadriceps inhibition and weakness than either variable alone. Our data support those of Palmieri-Smith et al in that the strength of the PT and regional-pain groups did not differ despite differences in structural damage to the patellar tendon. To our knowledge, no discrete data exist regarding the association between isolated structural knee damage and muscle function because structural damage cannot be ethically induced through laboratory measures. Thus, the importance of investigating pain and damage occurring through natural means becomes apparent and speaks to the clinical applicability of the PT and regional-pain model.

Our study had limitations that may be addressed by future researchers to provide additional insight into the contributions of damage and pain to quadriceps function. First, the PT sample size was small due to limited availability of participants presenting with structural damage; therefore, our findings can offer only preliminary insight into the differences in these conditions. Our post hoc power analyses ($1–\beta = 0.80$, $\alpha = .05$) indicated that a sample size of 13 individuals per group would provide sufficient power to detect strength differences between the PT and regional-pain groups. Second, a limitation inherent to the study of naturally occurring pain is the inability to control for pain variability. Experimentally induced pain allows for continued modulation of pain until a predetermined level is met. As such, differences in the effect of naturally occurring versus laboratory-induced pain are difficult to control without adequate sample sizes for various levels of pain. Further, discrepancies between self-reported pain (using the visual analog scale) and symptom severity (according to the VISA-P) were likely due to our inability to assess participants during true physical activity. Investigators should consider quantifying pain while participants are engaged in activity. Third, we were unable to control for the specific sources of pain reported by injured individuals without structural tendon damage. A wide variety of local and generalized sources have been proposed as capable of generating pain in the patellar tendon region.[8,32] Despite variability in the underlying source(s) of painful stimuli, our results are comparable with those of experimentally induced pain,[4] which we believe speaks to the similarity of afferent pain pathways, independent of the source. Finally, all participants were of college age and recreationally active. As such, we were unable to determine how more severe cases of PT and regional patellar tendon pain affected quadriceps strength and inhibition. Future authors should seek to identify severity-dependent changes to inhibitory pathways.

In conclusion, individuals with PT or patellar tendon regional pain demonstrated alterations to spinal-reflex excitability and global quadriceps inhibition compared with healthy control participants. This work, when considered with previous evaluations of Rio et al,[8,9] outlines a possible framework for quadriceps dysfunction resulting from PT and regional pain. It appears that the combination of pain with structural tendon damage may result in the largest influence on quadriceps dysfunction, as only the PT group presented with clinical weakness. Given the available evidence, an in-depth longitudinal evaluation of quadriceps inhibition resulting from structural tendon damage and pain could assist in the development of a symptom-specific rehabilitation approach that targets primary functional deficits, such as tissue repair and analgesic effects.

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Appendix. Main Outcome Variables

Variable	Group, Median (Range)		
	Patellar Tendinopathy	Regional Pain	Healthy Controls
Subjective reports			
Victorian Institute for Sport Assessment, Patellar Tendon score	62 (36–71)	62 (30–80)	100 (90–100)
Pain at rest	0.50 (0.10–2.10)	0.20 (0–4.80)	0 (0–0.20)
Pain at maximal voluntary isometric contraction	5.50 (1.00–7.30)	3.15 (0–8.10)	0 (0–0.30)
Tegner Activity Level Scale score (current)	8 (6–9)	7 (3–9)	7 (5–9)
Strength and inhibition			
Hoffman reflex	0.11 (0.03–0.17)	0.13 (0.02–0.66)	0.31 (0.09–0.80)
Maximal voluntary isometric contraction, Nm/kg	2.33 (1.74–3.46)	2.98 (1.95–4.10)	3.36 (2.49–4.16)
Central activation ratio	91.47 (55.32–97.72)	95.90 (63.05–98.99)	99.04 (89.21–99.62)