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Maximal Lower Limb Strength in Patellar Tendinopathy: A Systematic Review With Meta-Analysis

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Objective: To investigate whether lower limb strength is reduced in people with patellar tendinopathy (PT) compared with asymptomatic control individuals or the asymptomatic contralateral limb.

Data Sources: MEDLINE, PubMed, Scopus, and Web of Science.

Study Selection: To be included in the systematic review and meta-analysis, studies were required to be peer reviewed, published in the English language, and case control investigations; include participants with a clinical diagnosis of PT and an asymptomatic control or contralateral limb group; and include an objective measure of lower limb maximal strength.

Data Extraction: We extracted descriptive statistics for maximal strength for the symptomatic and asymptomatic limbs of individuals with PT and the limb(s) of the asymptomatic control group, inferential statistics for between-groups differences, participant characteristics, and details of the strength-testing protocol. The risk of bias was assessed using the Joanna Briggs Institute critical appraisal tool for analytical cross-sectional studies.

Data Synthesis: Of the 23 included studies, 21 reported knee strength, 3 reported hip strength, and 1 reported ankle strength. Random-effects models (Hedges *g*) were used to calculate the pooled effect sizes (ESs) of muscle strength according to the direction of joint movement and type of contraction. The pooled ESs (95% CI) for maximal voluntary isometric contraction knee-extension strength, concentric knee-extension strength, and concentric knee-flexion strength were 0.54 (0.27, 0.80), 0.78 (0.30, 1.33), and 0.41 (0.04, 0.78), respectively, with all favoring greater strength in the asymptomatic control group. Researchers of 2 studies described maximal eccentric knee-extensor strength with no differences between the PT and asymptomatic control groups. In 3 studies, researchers measured maximal hip strength (abduction, extension, and external rotation), and all within-study ESs favored greater strength in the asymptomatic control group.

Conclusions: Isometric and concentric knee-extensor strength are reduced in people with PT compared with asymptomatic control individuals. In contrast, evidence for reduced eccentric knee-extension strength in people with PT compared with asymptomatic control individuals is limited and inconsistent. Although evidence is emerging that both knee-flexion and hip strength may be reduced in people with PT, more examination is needed to confirm this observation.

Key Words: knee, tendon, force, pathology, isokinetic strength

Key Points

- Patellar tendinopathy (PT) may be associated with muscle- and contraction-specific deficits in lower limb strength that could guide research and rehabilitation.
- Current evidence suggests that isometric and concentric but not eccentric knee-extension strength may be moderately reduced in people with PT.
- Concentric knee-flexion and hip-adduction, -extension, and external-rotation strength may be decreased in people with PT, but further assessment is needed.

Patellar tendinopathy (PT) is an overuse injury of the patellar tendon that is characterized by persistent tendon pain and dysfunction during repetitive mechanical loading, such as jumping, squatting, or resisted knee extension.[1,2] It occurs in both recreational (~8%) and elite (~14%) athletes,[3] although PT in the latter group is associated with prevalence rates as high as 32% and 45% in elite basketball and volleyball players, respectively.[3] Although the clinical diagnosis of PT is relatively straightforward, management of the associated tendon pain and dysfunction is challenging, with reported recurrence rates as high as 25%,[4] likely reflecting the complex pathophysiology and symptoms of tendinopathies.[5] Conservative management is the first-line treatment and focuses on appropriate load management and strengthening of the knee extensors to improve tendon symptoms, function, and structure.[6] Slow progressive heavy, moderate, or both types of resistance training, often with a focus on eccentric loading, remains the most common and recommended exercise therapy for the management of PT[7–10]; however, overall success rates for exercise therapy have been mixed and moderate at best,[6,11] including the emphasis on eccentric loading.[9]

Tendinopathies are associated with micro- and macrostructural changes that may contribute to altered tendon mechanical properties, morphological properties, or both,[12] which could affect the ability of the tendon to absorb or transfer muscular

force.[13] Although growing evidence shows that Achilles tendinopathy is associated with reduced tendon stiffness, studies of PT have revealed conflicting findings, with researchers reporting either decreased[14,15] or unchanged[16,17] tendon stiffness compared with control groups. Given that tendon stiffness is intrinsically linked to the strength of the muscle(s) in series,[18–20] it may be reasonable to assume that tendinopathies could be associated with changes in muscular strength such that reduced tendon stiffness would be associated with reduced muscle strength and vice versa. Alternatively, investigators[21] have suggested that a relative mismatch between muscle strength and tendon stiffness may be a risk factor for the development of tendinopathy in young athletes such that athletes with high muscle strength-to-tendon stiffness ratios may be at risk of tendon overload. To this end, the authors of a recent systematic review concluded that individuals with Achilles tendinopathy demonstrated deficits in maximal (slow concentric = 44%, fast concentric = 38%), reactive (16%–35%), and explosive (10%–21%) strength of the triceps surae compared with asymptomatic control individuals.[22] They suggested that the inadequate resolution of strength deficits in rehabilitation may contribute to the high recurrence rates and persistent symptoms in individuals with tendinopathy.[22] Importantly, this review only included studies of Achilles tendinopathy, so whether similar strength deficits of the quadriceps exist in people with PT is unknown.

The success of exercise therapy in managing long-term PT, therefore, may be determined by matching exercise loads to suit both tendon capacity and associated impairments in muscular fitness, including contraction- and velocity-specific strength and power. Reliance on generic slow, heavy resistance isotonic- or eccentric-only exercise therapy may overlook important deficits in neuromuscular function, including muscle size, composition, and activation, and affect long-term outcomes.[23] Thus, exercise rehabilitation that focuses on symptom resolution may be insufficient to restore muscle-tendon–unit structure and function.[23,24] Rehabilitation may require longer treatment times with more comprehensive exercise progressions[25,26] that extend beyond pain inhibition and symptom resolution[23,27] to include full neuroplastic and neuromechanical recovery.[25,28] Similarly, because the patellar tendon is viscoelastic, reduced tendon stiffness and modulus may contribute to velocity-dependent reductions in maximal quadriceps strength, as observed in Achilles tendinopathy,[22] which may not be identified using conventional maximal isometric strength testing. Identifying contraction-dependent deficits in maximal knee strength in people with PT will assist the development of tailored, patient-specific exercise rehabilitation programs.

Deficits in quadriceps strength may be expected in PT, whereas evidence from other tendinopathies indicated that neuromuscular deficits may be more widespread[28] and could include antagonist muscle groups and muscle groups proximal, distal, and contralateral to the affected muscle-tendon unit. For example, in a recent review of lateral elbow tendinopathy, Heales et al[29] found evidence of widespread strength deficits involving both the shoulder and axioscapular muscles of the affected and unaffected limbs. These findings and those of other studies of lower limb tendinopathies[30–32] highlight the importance of clinical examinations and rehabilitation programs that consider the whole kinetic chain: in particular, muscles proximal to the affected tendon.

The primary aim of our systematic review was to investigate whether PT was associated with changes in maximal lower limb strength compared with an asymptomatic control or an asymptomatic contralateral limb condition. We hypothesized that individuals with PT would demonstrate lesser maximal strength of the knee extensors and that such changes would be more pronounced during eccentric than during concentric and isometric contractions. We also sought to determine whether PT was associated with changes in knee-flexion strength and muscles both proximal and distal to the quadriceps. We hypothesized that, consistent with observations in upper limb tendinopathy,[29] concomitant strength deficits may be present in muscle groups proximal (eg, hip extensors) and distal (eg, ankle dorsiflexors) to the knee extensors.

METHODS

We followed the guidelines for Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)[33] and prospectively registered the research with PROSPERO (reference No. CRD42021282577).

Data Sources and Searches

We searched MEDLINE, PubMed, Scopus, and Web of Science to identify all English-language studies published before October 2022. The search strategy encompassed key words related to the anatomic area (*patella* OR *patellar* OR *quadriceps* OR *jumper's knee*), clinical terminology (*tendinosis* OR *tendonopathy* OR *tendinitis* OR *tendinopathy*), and strength measures (*strength* OR *power* OR *force* OR *torque* OR *isokinetic* OR *concentric* OR *eccentric* OR *isometric*). The details of each database search are provided in Supplemental Table 1 (available online at https://dx.doi.org/10.4085/1062-6050-0662.22.S1). Where possible, additional limitations were applied to include journal articles on human participants published in English. One reviewer (S.J.O.) screened all titles and abstracts and then reviewed all potentially eligible full-text records against the a priori–determined eligibility criteria, with uncertainty resolved by discussion with a second reviewer (L.J.H.). Manual reference list screening was performed using forward and backward citation searches to identify additional investigations.

Study Selection

Studies were included if they met the following criteria: (1) participants had a clinical diagnosis of PT according to pain location (ie, localized to the patellar tendon) and pain with mechanical loading (eg, jumping and decline squat), with or without medical imaging; (2) maximal strength was objectively measured using isokinetic or hand-held dynamometry or both of any lower limb (ankle, hip, or knee) muscle group; and (3) a comparison with a control group was performed—for example, an asymptomatic control group, an asymptomatic contralateral limb, or a combination of both (mixed control group). The latter 2 comparators were included to further explore the presence or absence of bilateral deficits in people with unilateral tendinopathy, as had been reported previously[12,34]; however, these data were not included in the pooled meta-analyses. Reviews, case studies, abstracts only, and letters to the editor were excluded. Studies in which the diagnosis of PT was based on medical imaging alone were

excluded because structural tendon changes are common in individuals without clinical signs and symptoms of PT.[1]

Data Extraction

One reviewer (S.J.O.) completed data extraction, with all questions resolved by discussion with a second reviewer (L.J.H.). Descriptive statistics (mean, SD, median, and range) for maximal strength, where available, were extracted for the symptomatic and asymptomatic limbs of individuals with PT and limb(s) of the asymptomatic control group. Where available and required, inferential statistics (eg, T score and *P* value) for between-groups differences (tendinopathy versus control) were extracted. Participant characteristics (eg, demographics, duration of symptoms, and severity of symptoms using the Victorian Institute of Sport Assessment–Patellar tendon [VISA-P]) and details of the strength testing protocol (eg, instrumentation, joint position, angular velocity, and measurement units) were also extracted.

Risk-of-Bias Assessment

The risk of bias was assessed using the Joanna Briggs Institute (JBI) critical appraisal tool for analytical cross-sectional studies,[35] which includes the following criteria: (1) definition of the inclusion criteria, (2) description of the study participants and the settings, (3) valid and reliable measurements of the exposure, (4) objective and standard criteria for measurement, (5) confounding factors, (6) strategies for confounding factors, (7) valid and reliable measures of outcomes, and (8) statistical analysis used. Each criterion was scored as *yes*, *no*, *unclear*, or *not applicable*. For the current review, question 3 of the JBI tool was considered not applicable, as *exposure* could not be clearly defined in the context of PT. Two reviewers (S.J.O. and L.J.H.) independently rated each study using the checklist together with a brief reviewer's guide that was tailored to our research question and included studies (Supplemental Table 2). Any disagreements were to be moderated by a third reviewer (B.P.), but this step was not required. The JBI checklist was used to identify any possible sources of bias that might influence the interpretation of the meta-analyses and highlight areas for future research.

Data Synthesis and Analysis

For each investigation, the effect size (ES) expressing the difference in maximal strength between the PT and control groups was computed using the standardized mean difference (Hedges *g*) and corresponding 95% CIs.[36,37] For studies that included participants with bilateral PT in addition to an asymptomatic control group, data for the more symptomatic limb were used to compute the ES. For paired-group study designs (ie, those that compared symptomatic versus asymptomatic limbs), a conservative correlation value (*r*) of 0.7 was used to compute the within-study ES via a fixed-effects model.[38] A positive ES indicated greater strength in the asymptomatic control group or asymptomatic contralateral limb compared with the symptomatic limb of individuals with PT. The magnitude of the ES was interpreted as *small* (<0.5), *medium* (0.5–0.8), or *large* (>0.8).[39]

Where possible, a meta-analysis was performed using a random-effects model (Hedges *g*) and pooled according to the direction of joint movement (eg, knee extension and hip abduction) and contraction mode (eg, isometric and concentric). Although only ≥ 2 studies are required for a meta-analysis, we chose a more conservative minimum of 3.[40] The findings of researchers who reported multiple joint positions during isometric testing (eg, 60° and 90°) or multiple angular velocities during concentric or eccentric contractions (eg, 60°/s and 120°/s) were pooled using a fixed-effects model (Hedges *g*) before inclusion in the meta-analysis. Only studies involving an asymptomatic control group were considered for meta-analysis. Group mean differences between the PT and control groups were also expressed as a percentage. The meta-analyses were performed using the Excel (version 2016; Microsoft Corp) spreadsheets of Neyeloff et al.[41] The I^2 statistic was calculated to measure the heterogeneity of the pooled ES estimates and summarize the percentage of total variation across studies due to differences among studies rather than chance.[42] We broadly classified I^2 values of 25%, 50%, and 75% as *low*, *moderate*, or *high* thresholds, respectively.[42] The degree of heterogeneity across studies was also interpreted according to whether the individual study estimates showed the same direction of effect (ie, all >0 or <0), such that, if a pooled ES had a high I^2 (ie, $>75\%$) but was derived from studies all showing the same direction of effect (eg, ES $>$ 0), it was still considered appropriate for meta-analysis.[43] All ES estimates, including both within- and between-studies pooled estimates, are displayed in forest plots with corresponding 95% CIs.

RESULTS

Study Selection

The complete search results are shown in Figure 1. Electronic database searches identified 1379 records. A total of 328 duplicates were removed, and a further 953 records were excluded based on title and abstract screening. Of the remaining 98 studies, 77 were deemed ineligible based on the eligibility criteria. The reference lists of the 21 eligible studies were searched, and 2 additional studies were included.

Study Characteristics

The characteristics of the included research, including population characteristics, outcome measures, and summary results, are presented in Supplemental Table 3. Of the 23 studies, 21 described maximal knee strength*; 3, maximal hip strength[31,32,59]; and 1, maximal ankle strength.[31] Eighteen studies† supplied data for an asymptomatic control group, and 6 studies[44,46,48,50,52,55] provided data for the asymptomatic contralateral limb. Eighteen studies consisted of participants with unilateral PT,‡ with the remaining 5 studies involving a mix of unilateral and bilateral PT.[27,32,45,52,55] Sample sizes ranged from 5[27] to 59[59] and 7[31] to 133[59] for the PT and control groups, respectively. The mean age for the PT groups ranged from 18.3[59] to 37.6 years,[53] with many studies including mostly males. Symptom severity was reported in 16 studies§ using the VISA-P, with group scores ranging from 36 (median)[48] to 81 (mean)[46] out of 100 (lower scores indicate greater severity). The mean duration of symptoms was given

* *References 14–17, 27, 31, 44–55, 57–59.*
† *References 14–17, 31, 32, 44–47, 49, 51, 53, 54, 56–59.*
‡ *References 14–17, 31, 44, 46–51, 53, 54, 56–59.*
§ *References 14–17, 27, 31, 32, 44–49, 52, 58, 59.*

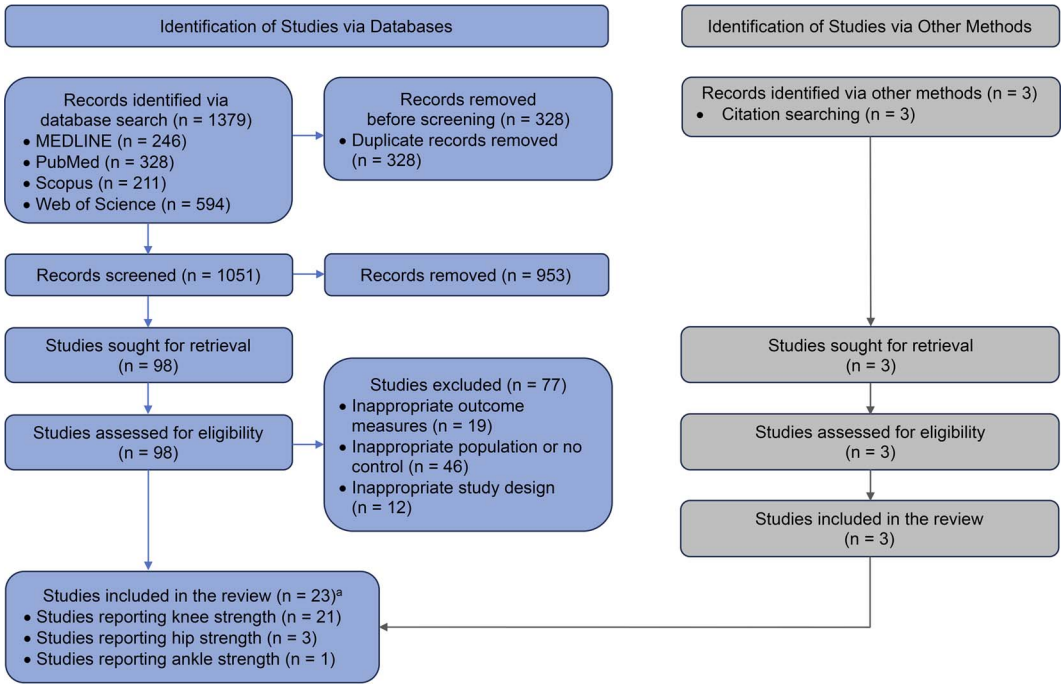

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram for study inclusion. [a] Some studies addressed >1 joint.

in 11 studies[14–17,32,45,47,49–51,56] and ranged from 9.9 months[47] to 8.9 years.[15]

Strength Assessment

Nineteen studies used an isokinetic dynamometer to measure maximal lower limb strength,[14–17,27,44–54,57–59] whereas 4 studies used a handheld dynamometer.[31,32,55,59] Fifteen studies reported maximal voluntary isometric contraction (MVIC) strength‖; 9, maximal voluntary concentric contraction (MVCC) strength[44,46,48–51,53,57,58]; and 3, maximal voluntary eccentric strength (MVEC).[50,51,53] Most studies (n = 17/23) described nonnormalized maximal strength as torque (N·m) or force (N).¶ Nine studies normalized strength to either body weight or height.[27,31,32,44–46,49,53,59]

The authors of 21 studies measured maximal knee-flexion or -extension strength or both[14–17,27,31,44–58]: MVIC knee extension most frequently (n = 13),# followed by MVCC knee extension (n = 9)[44,46,48–51,53,57,58] and MVCC knee flexion (n = 7).[44,46,49,50,53,57,58] Three studies reported MVEC knee extension[50,51,53]; 2, MVIC knee flexion[54,58]; and 1, MVEC knee flexion.[53] Three studies assessed maximal hip strength[31,32,59]: 2 evaluated MVIC hip abduction and external rotation[32,59] and 1, MVIC hip extension.[31] No researchers measured hip MVCC or MVEC. One study described MVIC ankle plantar flexion.[31]

Risk of Bias

A summary of the risk-of-bias assessment is provided in the Table. Most question areas were adequately addressed, with no studies receiving a score of *no* for any of the criteria. The most underreported area was the description of the sample population (question 2), with 11 of 23 studies receiving a score of *unclear*, mostly because the duration of symptoms or patient-reported functional outcomes (eg, VISA-P score) were not provided for the PT group.** Similarly, although all investigators supplied adequate descriptions of the inclusion and exclusion criteria for the PT group, that information for the control group was unclear in 5 studies.[14,17,46,49,54] Incomplete descriptions of the strength of the testing methods (question 7), including reliability and validity, were also common, with 8 studies receiving a score of *unclear*,[14,16,31,52–55,59] of which many used either custom devices or handheld dynamometry to measure strength.

Knee-Extension Strength

Of the 11 studies†† that compared MVIC knee-extension strength between the PT and asymptomatic control groups, all but 2[45,56] had a positive mean ES (Figure 2). Of the 9 remaining studies, 5 had ESs with lower CIs of >0,[15,16,27,45,47] favoring greater strength in the control group (Figure 2). Percentage differences in MVIC knee-extension strength between the PT and asymptomatic control groups ranged from −4.2%[57] to –39.8%,[27] with a mean difference of 14% across all studies. The pooled ES (random effects) for MVIC knee extension (PT: n = 181, control: n = 200) was 0.54 (95% CI = 0.27, 0.80), with low-to-moderate heterogeneity (I^2 = 30.5%) favoring greater strength in the control group. Three studies[47,52,55] reported MVIC knee-extension strength in the PT compared with an asymptomatic contralateral limb or mixed control group, with the ESs ranging from 0.10[55] to 0.51[52]; however, none had lower confidence limits of >0.

‖ *References 14–17, 27, 31, 32, 45, 47, 52, 54–56, 58.*
¶ *References 14–17, 44, 46–48, 50–55, 56–58.*
# *References 14–17, 27, 31, 45, 47, 52, 54, 55, 56, 58.*
** *References 27, 44, 46, 48, 51, 53, 54, 56, 57, 59.*
†† *References 14–17, 27, 31, 45, 47, 54, 56, 58.*

Table. Risk-of-Bias Assessment Using the Joanna Briggs Institute Checklist for Analytical Cross-Sectional Studies[35]

Study	1. Were the criteria for inclusion in the sample clearly defined?	2. Were the study participants and the setting described in detail?	3. Was the exposure measured in a valid and reliable way?	4. Were objective, standard criteria used for measurement of the condition?	5. Were confounding factors identified?	6. Were strategies to deal with confounding factors stated?	7. Were the outcomes measured in a valid and reliable way?	8. Was an appropriate statistical analysis used?
Chantrelle et al[44]	Yes	Unclear	N/A	Unclear	Yes	Yes	Yes	Yes
Crossley et al[45]	Yes	Yes	N/A	Yes	Yes	Yes	Yes	Yes
Dauty et al[46]	Unclear	Unclear	N/A	Yes	Yes	Yes	Yes	Yes
Davi et al[27]	Yes	Unclear	N/A	Yes	Yes	Yes	Yes	Yes
Fendri et al[47]	Yes	Yes	N/A	Yes	Yes	Yes	Yes	Yes
Frohm et al[48]	Yes	Unclear	N/A	Yes	Yes	Yes	Yes	Yes
Helland et al[14]	Unclear	Yes	N/A	Yes	Yes	Yes	Unclear	Yes
Kabacinski et al[49]	Unclear	Yes	N/A	Yes	Yes	Yes	Yes	Yes
Kaux et al[50]	Yes	Yes	N/A	Yes	Yes	N/A	Yes	Yes
Kim et al[51]	Yes	Unclear	N/A	Yes	Yes	Yes	Yes	Yes
Kongsgaard et al[52]	Yes	Yes	N/A	Yes	Yes	N/A	Unclear	Yes
Kongsgaard et al[16]	Yes	Yes	N/A	Yes	Yes	Yes	Unclear	Yes
Krauss et al[53]	Yes	Unclear	N/A	Yes	Yes	Yes	Unclear	Yes
Kujala et al[54]	Unclear	Unclear	N/A	Unclear	Yes	Yes	Unclear	Yes
Lee et al[17]	Unclear	Yes	N/A	Yes	Yes	Yes	Yes	Yes
Maffulli et al[55]	Yes	Unclear	N/A	Yes	Yes	Yes	Unclear	Yes
Mendonça et al[56]	Yes	Unclear	N/A	Unclear	Yes	Yes	Unclear	Yes
Rio et al[57]	Yes	Yes	N/A	Yes	Yes	Yes	Yes	Yes
Scattone Silva et al[31]	Yes	Yes	N/A	Yes	Yes	Yes	Unclear	Yes
Wiesinger et al[15]	Yes	Yes	N/A	Yes	Yes	Yes	Yes	Yes
Witvrouw et al[58]	Yes	Unclear	N/A	Yes	Yes	Yes	Yes	Yes
Yue et al[59]	Yes	Unclear	N/A	Yes	Yes	Yes	Yes	Yes
Zhang et al[32]	Yes	Yes	N/A	Yes	Yes	Yes	Yes	Yes

Abbreviation: N/A, not applicable.

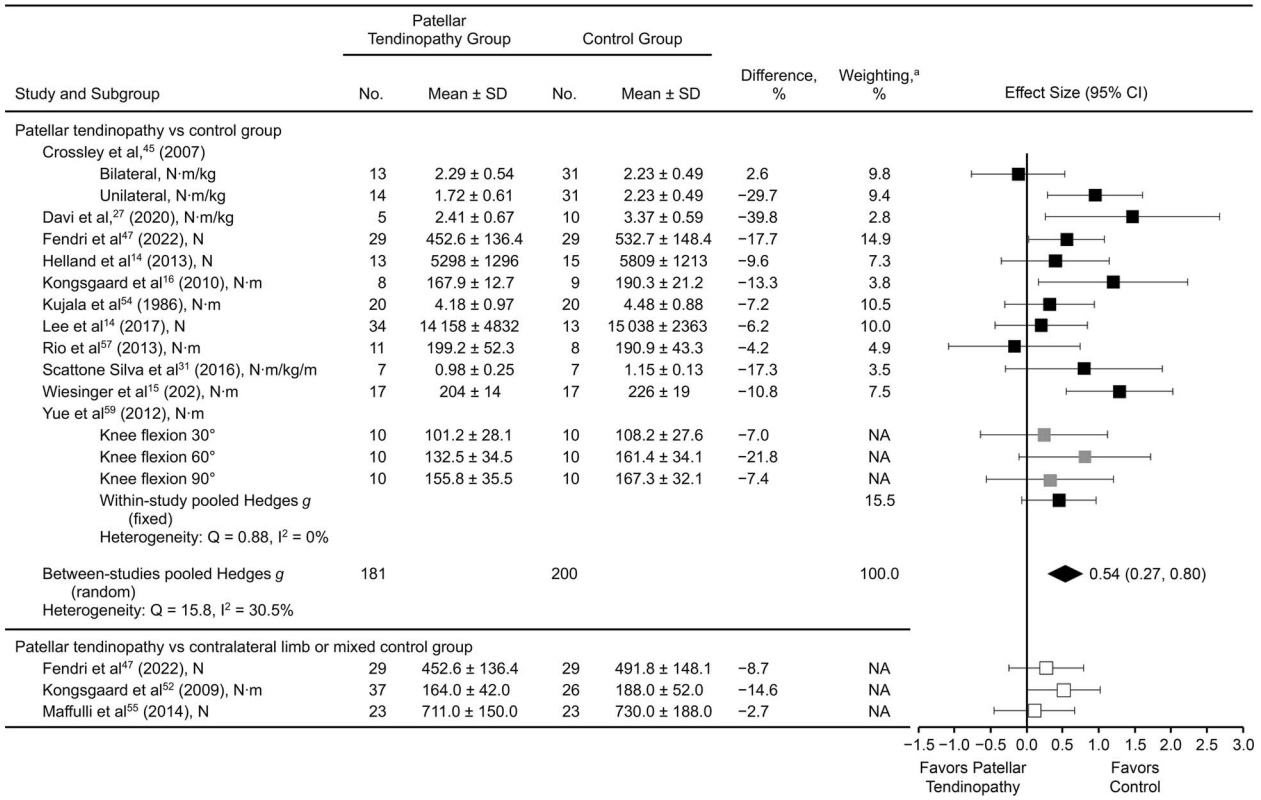

Figure 2. Meta-analysis of maximal voluntary isometric knee-extension strength. Gray squares indicate the within-study effect size (Hedges *g*) for each joint position; black squares, the single within-study effect size (or pooled fixed effect when multiple positions were reported); black diamond, the pooled effect size (random effects); white squares, the within-study single effect size for studies including the asymptomatic contralateral limb or mixed control (ie, a combination of the asymptomatic contralateral limb and asymptomatic control) group; and error bars, 95% CIs of the effect size.

Six studies compared MVCC knee-extension strength between the PT and asymptomatic control groups (Figure 3A).[44,49,51,53,57,58] Except for Kraus et al,[53] who reported only 1 angular velocity (60°/s), all researchers provided multiple angular velocities ranging from 60°/s to 300°/s. For these studies, ES estimates were pooled (fixed effects) to give an overall study ES. Except for 1 study (I^2 = 47.7%[49]), all within-study pooled ES estimates had low heterogeneity (all I^2 values = 0%), suggesting that the MVCC strength differences between the PT and asymptomatic control groups were similar across contraction velocities. Five[44,49,51,53,58] of the 6 studies had an overall positive ES, favoring greater strength in the control group, of which 3 had lower CIs of >0.[44,49,58] The pooled ES (random effects) for MVCC knee extension was 0.74 (95% CI = 0.30, 1.33), favoring greater strength in the control group with high heterogeneity (I^2 = 91.5%). As such, although 5 of 6 studies had a positive ES favoring greater MVCC knee-extension strength in the control group compared with the PT group, large variations existed in the size (not the direction) of the effect among studies (range = –0.05–1.92). The mean percentage difference in MVCC knee-extension strength between the PT and asymptomatic control groups was –14.8% and ranged from –42.1%[44] to 2.1%.[57] Of the 4 studies that compared MVCC knee extension in the PT group with the asymptomatic contralateral limb or mixed control group, all had positive ESs, favoring greater strength in the control group; however, only 2 studies had ES estimates with lower CIs of >0 (Figure 3B).[44,48] Percentage differences in MVCC knee-extension strength between the PT group and the asymptomatic contralateral limb or mixed control group ranged from –7.8%[50] to –18.7%.[46]

Two studies assessed MVEC knee-extension strength in the PT group compared with an asymptomatic control group (Figure 4).[51,53] Both studies had small positive ES estimates with CIs that crossed 0. Researchers of 1 study[50] compared the PT group with an asymptomatic contralateral limb or mixed control group, revealing a small positive ES favoring the control group, with a CI that crossed 0.

Knee-Flexion Strength

Two studies described MVIC knee-flexion strength in the PT compared with the asymptomatic control group (Figure 5A).[54,58] Each study had a small positive ES (range = 0.01–0.13) favoring greater strength in the control group, even though the lower CIs were both <0. Of the 5 studies that evaluated MVCC knee-flexion strength in the PT compared with the asymptomatic control group,[44,49,53,57,58] only 2 had within-study pooled ESs with lower CIs of >0,[44,59] favoring greater strength in the control group (Figure 5A). Yue et al[58] observed MVCC knee-flexion strength across 4 velocities (60°/s, 120°/s, 180°/s, and 240°/s). The pooled within-study ES (95% CI) was 0.61 (0.15, 1.06) with moderate-to-high heterogeneity (I^2 = 56.4%), which was driven by large differences in strength at the 2 highest (180°/s = –23.2%; 240°/s = –31.9%) compared with the 2 lowest (60°/s = 2.2%; 120°/s = 0.01%) contraction velocities. Chantrelle et al[44] noted large differences in MVCC knee-flexion strength at both 60°/s (ES = 1.19) and 180°/s (ES = 0.88), with a within-study pooled ES

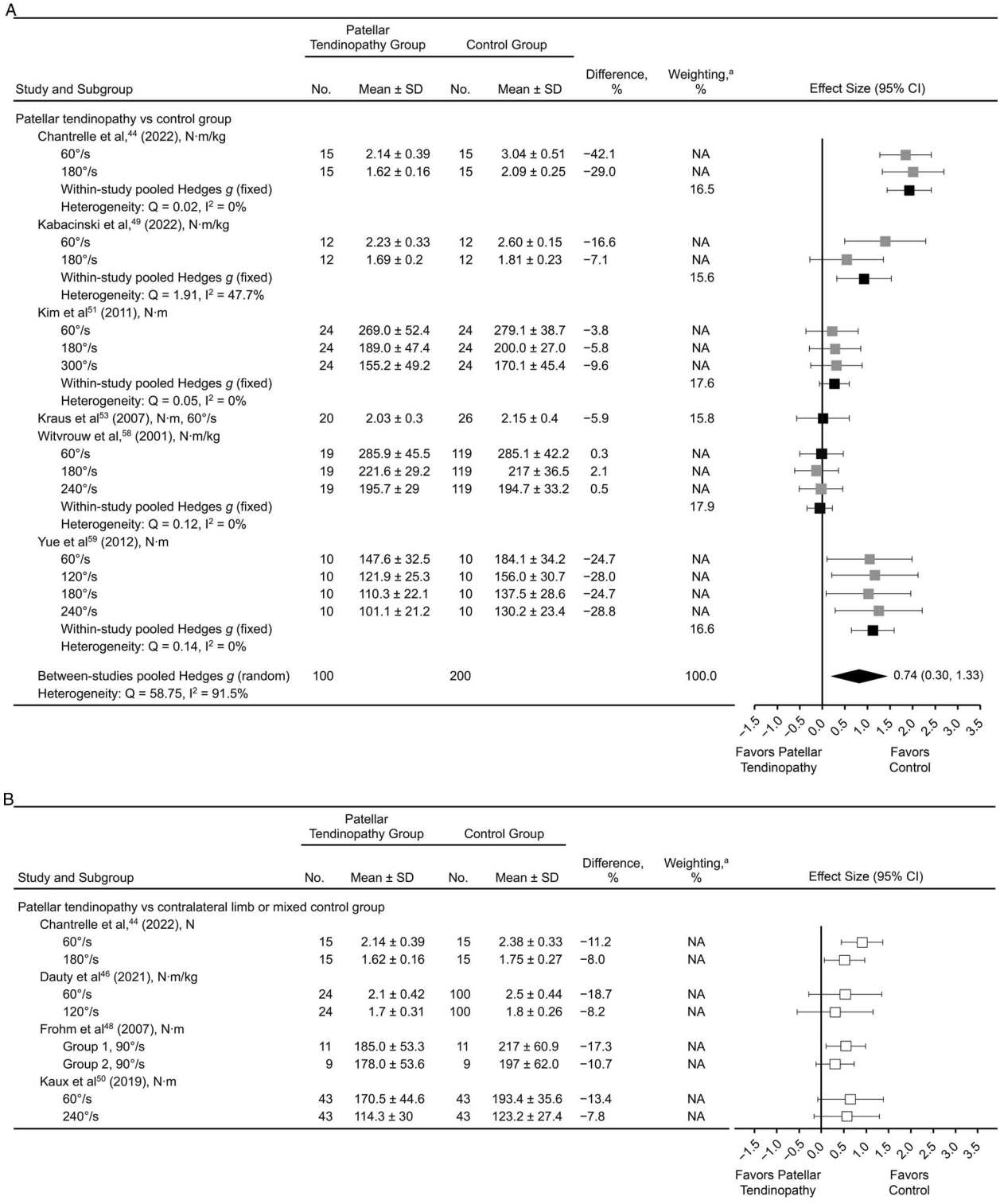

Figure 3. Meta-analysis of maximal voluntary concentric knee-extension strength. A, Patellar tendinopathy versus control group. Gray squares indicate the within-study effect size (Hedges *g*) for each joint position; black squares, the single within-study effect size (or pooled fixed effect when multiple positions reported); black diamond, the pooled effect size (random effects); and error bars, 95% CIs of the effect size. B, Patellar tendinopathy versus the contralateral limb or mixed control group. White squares indicate the within-study single effect size for studies including the asymptomatic contralateral limb or mixed control (ie, a combination of the asymptomatic contralateral limb and asymptomatic control) group, and error bars indicate 95% CIs of the effect size. [a] Random effects.

(95% CI) of 1.02 (0.58, 1.46). When pooled across all 5 studies, the ES (95% CI) for MVCC knee-flexion strength was 0.41 (0.04, 0.78), favoring greater strength in the asymptomatic control compared with the PT group. The only authors to measure MVEC knee-flexion strength[53] had a small ES (95% CI) of 0.16 (–0.48, 0.80) that was not different, favoring the control group (Figure 5A). Three studies compared MVCC knee-flexion strength between the PT group and the

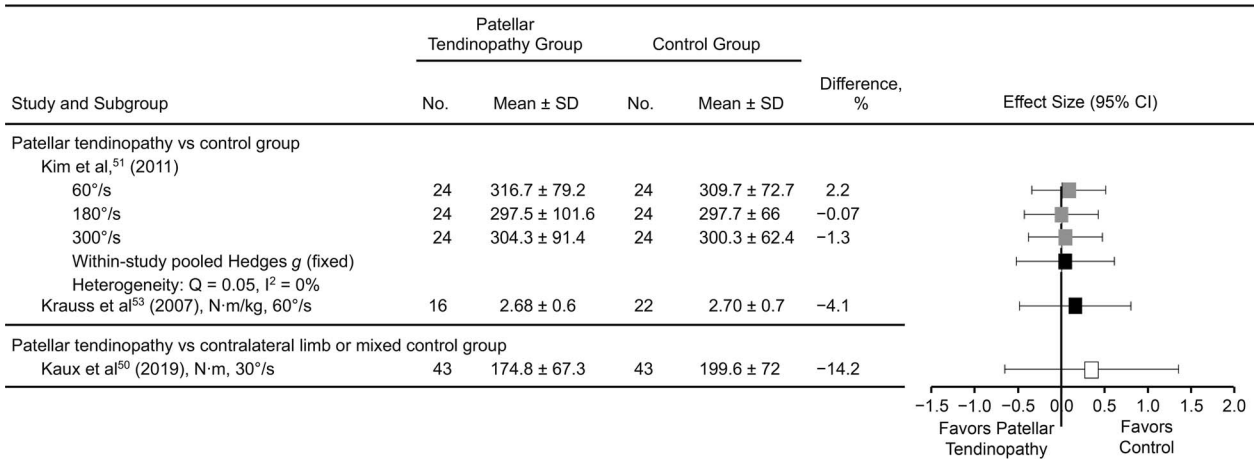

Figure 4. Meta-analysis of maximal voluntary eccentric knee-extension strength. Gray squares indicate the within-study effect size (Hedges *g*) for each joint position; black squares, the single within-study effect size (or pooled fixed effect when multiple positions reported); black diamond, the pooled effect size (random effects); white squares, the within-study single effect size for studies including the asymptomatic contralateral limb or mixed control (ie, a combination of the asymptomatic contralateral limb and asymptomatic control) group; and error bars, 95% CIs of the effect size.

asymptomatic contralateral limb or mixed control group (Figure 5B).[44,46,50] The ES for each study was positive, even though all lower confidence limits crossed 0, suggesting no difference in MVCC knee flexion.

Hip Strength

Group mean and ES data for hip strength are shown in Figure 6. Researchers of only 1 study examined MVIC hip-extension strength,[31] demonstrating a 37.6% reduction in the PT compared with the asymptomatic control group, with a corresponding ES (95% CI) of 1.2 (0.16, 2.29). In 2 studies, investigators supplied MVIC hip-abduction strength.[32,59] Zhang et al[32] noted a 28.2% reduction in MVIC strength in the PT compared with the asymptomatic control group, corresponding to an ES (95% CI) of 1.09 (0.56, 1.63). In contrast, Mendonça et al[59] found a negligible difference in MVIC hip-abduction strength (−0.7%), corresponding to an ES (95% CI) of 0.27 (–0.04, 0.57). Two studies that assessed MVIC hip external rotation revealed large differences (−25.0%[32] and −16.7%[59]), favoring the asymptomatic control group, with corresponding ESs (95% CIs) of 0.83 (0.31, 1.35) and 5.0 (4.4, 5.6), respectively.

Ankle Strength

Researchers of only 1 study examined ankle strength in the PT and asymptomatic control groups.[31] The MVIC ankle plantar-flexion strength was reduced by 15%, which was not different in the PT compared with the asymptomatic control group, with a corresponding ES (95% CI) of 0.52 (–0.47, 1.52).

DISCUSSION

In this systematic review, we identified 23 studies in which the authors observed maximal lower limb strength in a PT group compared with either an asymptomatic control group or an asymptomatic contralateral limb or mixed control group. Although many researchers underreported key sample characteristics, such as symptom duration and severity, we detected no clear bias that prevented pooling of the data. Meta-analyses involving the asymptomatic control group indicated decreased knee-extension MVIC and MVCC and knee-flexion MVCC in the PT group, with medium-to-large ESs. In contrast, evidence was limited for differences in knee-extension MVEC or knee-flexion MVIC and MVEC between the PT and asymptomatic control groups. Similarly, no clear and consistent evidence for differences in any knee-extension or -flexion strength measure between the symptomatic and asymptomatic contralateral limb of those with unilateral PT was present. Finally, some evidence from individual studies suggested that hip strength may be reduced in those with PT compared with asymptomatic control individuals, although more examination is needed to enable pooled analyses in order to confirm these results. Overall, we found evidence for contraction-specific deficits in maximal knee-extension and -flexion strength, which currently do not include deficits in maximal eccentric strength. Exercise therapy for PT that prioritizes eccentric quadriceps exercises over concentric or eccentric-concentric exercises does not appear to be justified based on deficits in maximal eccentric strength. In contrast, exercise therapy that emphasizes the recovery of both MVIC and MVCC quadriceps strength is supported by evidence of moderate-to-large deficits in maximal strength in people with PT compared with asymptomatic control individuals. Regardless of the loading program, exercise therapy for PT rehabilitation should address the recovery of isotonic and isometric quadriceps strength, as both variables are linked to clinical outcomes.[6,16,52]

Knee-Extension Strength

Knee-extension MVIC was the most frequently reported strength variable. The pooled ES of 0.54 (95% CI = 0.27, 0.80) was based on 11 studies with a total of 181 participants who had PT and 200 asymptomatic control individuals and included 5 studies with within-study ES measures[15,16,27,45,47] that were different, all favoring greater knee-extension MVIC strength in the control group. Similar percentage strength deficits were also seen in the 3 studies comparing the symptomatic limb with an asymptomatic control limb group, mixed control group, or both (range = –2.7% to –14.6%),[47,52,55]

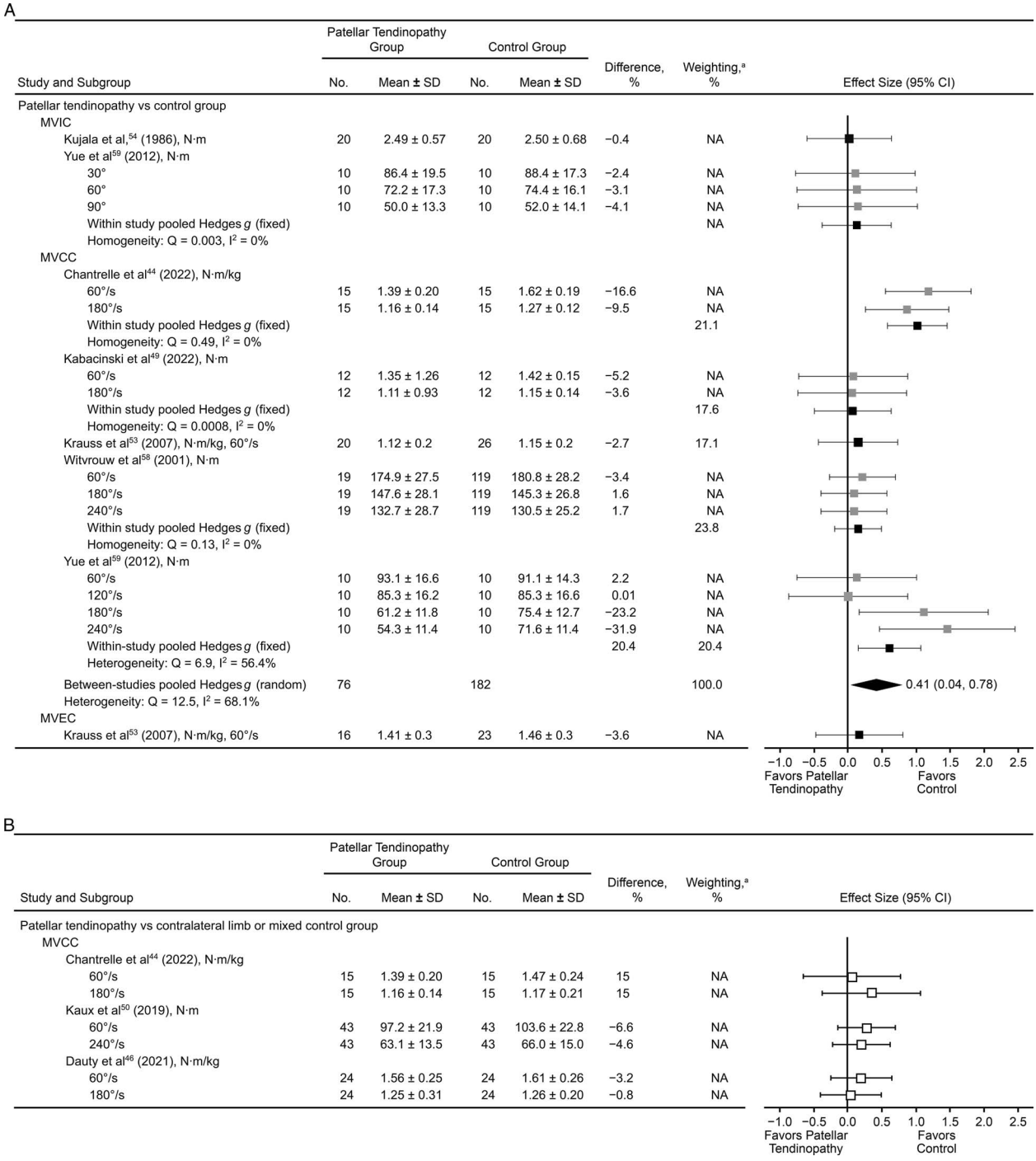

Figure 5. Meta-analysis of maximal voluntary knee-flexion strength. Maximal voluntary isometric contraction (MVIC), maximal voluntary concentric contraction (MVCC), and maximal voluntary eccentric contraction (MVEC) knee-flexion strength in the patellar tendinopathy versus control group. Gray squares indicate the within-study effect size (Hedges *g*) for each joint position; black squares, the single within-study effect size (or pooled fixed effect when multiple positions reported); black diamond, the pooled effect size (random effects); and error bars, 95% CIs of the effect size. B, Maximal voluntary concentric contraction knee-flexion strength in the patellar tendinopathy versus the contralateral limb or mixed control group. White squares indicate the within-study single effect size for studies including the asymptomatic contralateral limb or mixed control (ie, a combination of the asymptomatic contralateral limb and asymptomatic control) group, and error bars indicate 95% CIs of the effect size.[a] Random effects.

although only 1 study had a within-study ES that was different.[52] Compared with the asymptomatic control group, deficits in knee-extension MVIC in the PT group ranged between 4.2% and 39.8% and tended to be larger in studies that provided ratio-normalized strength measures[27,31,45] and those that included participants with more severe and prolonged PT symptoms.[15,27,52] For example, the 2 studies[15,45] with the largest ESs for MVIC extension consisted of participants with mean symptom durations of 2.8 and 3.2 years and VISA-P scores of 60 and 56.6 (out of 100), respectively. In comparison, Helland et al[14] found a medium ES that was not different, still favoring the control group, but included participants with

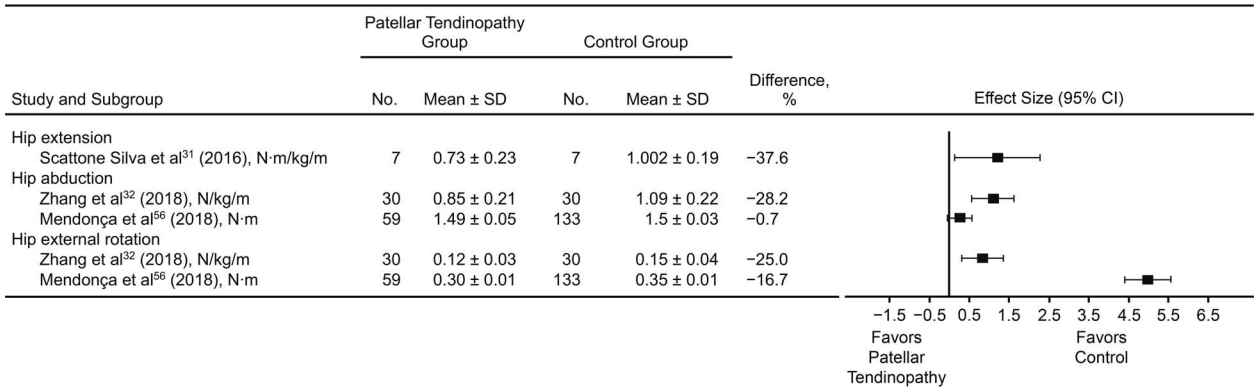

Figure 6. Forest plot of the within-study effect size (Hedges *g*) for maximal voluntary hip strength. Black squares indicate the within-study effect size (Hedges *g*) for each joint position, and error bars indicate 95% CIs of the effect size.

PT who had relatively mild-to-moderate symptoms (VISA-P = 76); however, these researchers did not supply symptom duration. Although more severe and prolonged PT pain may increase the likelihood of disuse-related quadriceps atrophy and other neurophysiological adaptations (eg, reduced neural drive) that would affect muscular strength,[27,28,60,61] further work is needed to elucidate this relationship. Nevertheless, the evidence is consistent for a small-to-moderate reduction (mean deficit of 14%) in MVIC knee-extension strength in people with PT compared with asymptomatic control individuals. This finding contrasts that in a recent review of Achilles tendinopathy,[22] in which strength differences between PT and control groups were mixed and ranged from –12%[62] to 8.5%[63]; nonetheless, this review was based on only 4 studies compared with our review of 12. Therefore, even though these differences might reflect the unique functional requirements and adaptation of each tendon to loading and injury,[12] more investigation is required to confirm whether deficits in isometric strength in lower limb tendinopathies are unique to the patellar tendon.

Some evidence supported a moderate reduction in MVCC knee-extension strength in the PT group compared with an asymptomatic control limb group, asymptomatic contralateral limb group, or mixed control group. The pooled ES for MVCC knee extension using an asymptomatic control group was 0.74 (95% CI = 0.30, 1.33) and was based on 6 studies (100 participants with PT, 200 without PT), of which 3 had within-study ESs that were different and favored the control group.[44,49,58] In the remaining 3 studies, researchers described negligible differences in knee-extension MVCC strength, of which 2 favored the control group[51,53] and 1 favored the PT group.[57] Although no meta-analysis was performed, the MVCC strength results of explorations including an asymptomatic contralateral limb group, a mixed control group, or both[44,46,48,50] broadly mirrored those comparing PT and asymptomatic control groups. Thus, even though evidence of bilateral sensorimotor changes in people with unilateral tendinopathy exists,[34] data from individual studies suggest that knee-extension strength deficits in PT may be confined to the symptomatic limb. The largest group differences in knee-extension MVCC strength were demonstrated by Chantrelle et al[44] and Yue et al,[58] in which the within-study pooled ESs (fixed effects) were 1.92 (95% CI = 1.44, 2.41) and 1.12 (95% CI = 0.64, 1.59), respectively, corresponding to mean differences of 35.6% and 26.6%, respectively, favoring the control group. These differences are comparable with those found in individuals with Achilles tendinopathy: MVCC strength at slow and fast contraction velocities was, on average, 44% (ES = 0.52) and 38% (ES = 0.61) lower, respectively than those in asymptomatic control individuals.[22] Furthermore, we identified no clear evidence that MVEC strength was reduced in the PT group compared with an asymptomatic control[51,53] or an asymptomatic contralateral limb group.[50] Although investigators in all 3 studies observed higher MVEC in the control group, the corresponding ESs were small and not different. Moderate-to-large reductions in maximal concentric and small reductions in eccentric strength in PT are consistent with data reported for Achilles tendinopathy,[22] in which maximal eccentric strength, particularly at slow contraction velocities, appeared to be preserved in people with lower limb tendinopathy. Therefore, although eccentric loading remains an important component of PT rehabilitation,[7–10] its inclusion based solely on perceived deficits in eccentric strength is not supported by our results. Furthermore, in the absence of evidence for contraction-specific adaptation (at an equivalent exercise load and velocity) in muscle[64] or tendon strength[6] or exercise-induced hypoalgesia,[65] together with the potential effect of symptom limitation on load potential of eccentric exercises in people with tendon pain,[6] the mechanisms underpinning the success of some, but not all, eccentric-only programs remain unclear. To this end, despite the popularity of eccentric-focused training regimes, surprisingly, few authors have measured maximal eccentric knee-extension strength in people with PT.

Overall, consistent evidence indicated that MVIC and MVCC knee-extension strength was moderately reduced in people with PT compared with asymptomatic control individuals. These findings may be explained by several factors. First, pain-related adaptation of quadriceps neural control could limit maximal muscle activation during strength testing.[28,66] For example, both Davi et al[27] and Rio et al[56] noted neurophysiological changes in PT consistent with reduced neural drive to the quadriceps muscle, although interestingly, Rio et al[56] did not report differences in MVIC strength between the PT and control groups. As such, although reduced quadriceps neural drive may be a feature of PT and reflect a protective mechanism to reduce knee-joint power and work and thus the rate or magnitude of tendon load,[67,68] it may be insufficient or absent in some populations (eg, high-level athletes) to effect a change in maximal volitional muscle strength.[56] Without concomitant measures of pain and muscle-activation patterns during MVIC testing, the influence of symptom severity and associated

quadriceps inhibition on maximal strength cannot be determined but remains an important consideration in rehabilitation.[23] Second, changes in tendon morphological and mechanical properties in PT could compromise quadriceps force transmission during maximal contractions.[13] For example, Wiesinger et al[15] described higher VISA-P scores associated with lower patellar tendon stiffness and modulus and reduced MVIC knee-extension strength in athletes of various activity backgrounds and volumes with moderate-to-severe PT (mean VISA-P = 56.6) compared with asymptomatic control individuals. In contrast, Helland et al,[14] despite showing less tendon stiffness and modulus in elite volleyball players with mild-to-moderate severity PT (mean VISA-P = 76), did not see a concomitant difference in knee-extension MVIC compared with asymptomatic control individuals. Similarly, despite a small correlation between patellar tendon stiffness and pain ($r = 0.30$, $P = .09$) during a single-legged decline squat, Lee et al concluded that neither tendon stiffness nor MVIC knee-extension strength differed between the PT and control groups.[17] Therefore, although changes in tendon morphological and mechanical properties in PT have the potential to influence maximal strength, the associations between tendon structure and function remain inconsistent[69] and may only be evident in people with severe and long-term PT who have pronounced tendon and neurophysiological adaptations.[23] Furthermore, currently, no consistent evidence suggests that tendon stiffness is reduced in PT.[12] In fact, the authors of several intervention studies found reductions in patellar tendon stiffness in patients with PT[16,52,70] after exercise that increased tendon stiffness in healthy populations[71–74] and provided symptomatic relief in those with PT (eg, heavy-load isotonic resistance exercise).[6,26,75] As such, the relationships among tendon pain, structure, and function in PT remain complex and areas of ongoing debate.[12,26,76–80] Finally, we cannot discount the role of disuse atrophy in explaining decreased knee-extension strength in patients with PT, particularly in studies of nonelite athletes with severe and long-term PT.[15,45] Disuse atrophy in PT would not be limited to the quadriceps muscles and could explain the small-to-moderate reductions in MVCC knee-flexion strength identified by some researchers[44,58]; however, this may not explain the contraction-specific deficits in knee-extension strength.

Knee-Flexion Strength

Maximal concentric strength was the knee-flexion strength variable evaluated most often. The pooled ES was 0.41 (95% CI = 0.04, 0.78), based on 5 studies, involving 76 participants with PT and 182 asymptomatic control individuals. Greater strength was present in all the control groups, although within-study ESs were different in only 2 studies (1.02[44] and 0.61[58]). Interestingly, Yue et al[58] only reported differences in MVCC knee flexion at contraction velocities of 180°/s and 240°/s, with no differences between the PT and control groups at 60°/s and 180°/s. We did not perform subgroup analyses based on contraction velocity, but Yue et al[58] were the only researchers with data indicating velocity-dependent differences in MVEC or MVCC knee strength between the PT and asymptomatic control groups. Velocity-dependent differences in muscle strength have been observed between individuals with and those without Achilles tendinopathy,[22] yet the available data do not support a similar deficit in people with PT for either maximal knee-extension or -flexion strength. For example, with respect to knee-extension strength, researchers of only 1 study[49] characterized velocity-dependent differences in MVCC strength between the PT and control groups at 60°/s but not at 180°/s. Given that the rate and magnitude of muscular force development are directly related to tendon stiffness[13,81] (among other factors), the absence of clear velocity-dependent differences in knee-extension strength in PT, in contrast to Achilles tendinopathy,[22] could reflect changes in stiffness unique to each tendon.[12] However, considering that we did not perform a subgroup analysis based on contraction velocity, our observations are speculative, and further work is needed.

Hip and Ankle Strength

In a recent review on lateral elbow tendinopathy, Heales et al[29] concluded that deficits in muscle strength in chronic tendinopathy may exist in muscles either proximal or distal to the affected tendon. In support, we identified 3 studies[31,32,59] that indicated maximal hip strength in the PT group compared with an asymptomatic control group, and 1 study of ankle strength[31]; the latter study showed no difference between the PT and asymptomatic control groups. Authors who evaluated hip strength detected less maximal hip extension,[31] abduction,[32] and external rotation[32,59] in the PT group than in an asymptomatic control group. The largest differences were for external-rotation strength, with mean differences of −25.0%[32] and −16.7%,[59] favoring the control group. These differences are comparable with those for shoulder external rotation in individuals who had lateral elbow tendinopathy (range = 26%–10%) compared with asymptomatic control individuals.[29] Proximal muscle strength deficits in tendinopathy could reflect disuse atrophy associated with reduced physical activity due to pain but also systemic biochemical and neurophysiological changes associated with chronic pain that may extend beyond the affected muscle-tendon unit.[28,29] Irrespective of whether local or global motor-control changes in tendinopathy reflect cause or epiphenomenon,[28] evidence is growing to support the inclusion of clinical assessments and rehabilitation strategies that extend beyond the affected tendon and encompass the entire kinetic chain.

Limitations

Several limitations must be considered when interpreting the findings of our review. First, we restricted our review to studies in which researchers reported maximal lower limb strength measured using instrumented dynamometry, so we did not include studies in which investigators assessed other aspects of muscle-tendon performance, such as the rate of force development or jump-landing biomechanics (for a review of this topic, see Tayfur et al[67]). Future studies, therefore, are needed to determine whether deficits in maximal knee strength in patients with PT measured during isolated contractions translate to changes in performance during dynamic whole-body tasks. Second, most studies were cross-sectional, so the causal relationship between strength and the development of PT was not explored. Furthermore, incomplete reporting of symptom duration, severity, and tendinopathy stage and the relative timing of strength measures hindered the interpretation and clinical application of the findings. Third, we performed meta-analyses when ≥3 studies had equivalent data, which omitted 3 strength

measures from the pooled analyses (hip-abduction, hip external-rotation, and knee-flexion MVIC). These variables should be examined in future cross-sectional studies to better elucidate these relationships and to facilitate pooled analyses in systematic reviews. Fourth, we pooled within-study data by using a fixed-effects model when multiple joint angles (MVIC) or multiple velocities (MVCC) were used to measure maximal strength. Although the heterogeneity scores for each within-study pooled ES were generally very low ($I^2 = 0\%$ for 10/12), suggesting minimal variation in the ES data, this approach may have overlooked relevant angle- or velocity-dependent effects of PT on maximal strength. Separate subgroup meta-analyses based on joint angle or angular velocity were beyond the scope of our review and may require further consideration. Also, investigators in most studies (n = 18/23) included either all male participants or more male than female participants, so the results may not represent both sexes and highlight the potential "gender void" of tendinopathy research.[82] Finally, underreporting in the included studies precluded the exploration of the influence of PT symptom severity and duration on lower limb strength but warrants further consideration.

CONCLUSIONS

In this review, we provided evidence for contraction-specific deficits in knee-extensor strength in people with PT compared with asymptomatic control individuals. The findings support the inclusion of progressive isometric and concentric resistance exercise for the rehabilitation of patients with PT, with the aim of restoring deficits in maximal muscle strength and promoting tendon adaptation. In contrast, although progressive eccentric resistance exercise remains a recommended component of PT rehabilitation, evidence is inconsistent to support its inclusion based on concurrent deficits in maximal knee-extension eccentric strength, which appear limited and variable. Finally, evidence is emerging that both knee-flexion strength and hip strength may be reduced in those with PT, although more research is needed to confirm this observation.

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SUPPLEMENTAL MATERIAL

Supplemental Table 1. Database search strategies and results.
Supplemental Table 2. Guidance criteria for the Joanna Briggs Institute checklist for analytical cross-sectional studies.
Supplemental Table 3. Characteristics of included studies.
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