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Original Article

Kinematic and kinetic differences between military patients with patellar tendinopathy and asymptomatic controls during single leg squats

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Abstract

Background. Knee valgus alignment has been associated with lower-limb musculoskeletal injury. This case-control study aims to: assess biomechanical differences between patients with patellar tendinopathy and healthy controls.

Methods. 43 military participants (21 cases, 22 controls) were recorded using 3D-motion capture performing progressively demanding, small knee bend, single leg and single leg decline squats. Planned *a priori* analysis of peak: hip adduction, knee flexion, pelvic tilt, pelvic obliquity and trunk flexion was conducted using MANOVA. Kinematic and kinetic data were graphed with bootstrapped t-tests and 95% CI's normalised to the squat cycle. ANOVA and correlations in SPSS were used for exploratory analysis.

Findings. ~~Cases squatted to less depth (-6.62° , $p<0.05$), have greater abduction (valgus) angle with decreased abduction (valgus) moment, initiate and complete squatting with more trunk, hip and knee flexion associated with increased hip/knee extensor moment and hip/knee internal rotation moment ($p<0.05$).~~ On their symptomatic side cases squatted to less depth (-6.62° , $p<0.05$) than controls with exploratory curve analysis revealing a pattern of increased knee valgus collapse throughout the squatting movement ($p<0.05$). Greater patella tendon force was generated by: the eccentric than concentric phase of squatting (+30-43%, ES 0.52-1.32, $p<0.01$), declined (plantarflexed) compared to horizontal surface (+36-51%, ES 1.19-1.68, $p<0.01$) and deeper knee flexion angles ($F\geq 658.3$, $p<0.01$) with no difference between groups ($F\leq 1.380$, $p>0.05$). Cases experienced more pain on testing on decline board (ES=0.69, $p<0.01$). For symptomatic limbs pain ($r_s=0.458$ -0.641, $p\leq 0.05$), but not VISA-P (Victoria Institute of Sport Assessment) ($r_s=0.053$ -0.090, $p>0.05$), correlated with extensor knee moment.

Interpretation. Knee valgus alignment is a plausible risk factor for patellar tendinopathy. Conclusions relating to causation are limited by the cross-sectional study design. Increasing squat depth, use of a declined surface and isolating the

eccentric phase enable progression of loading prescription guided by pain. ~~Data is provided to assist in prescription of progressive loading programmes. Pain on activity is a useful guide to titrate load dosage.~~

1. Introduction

Patellar tendinopathy is a prevalent condition in physically active populations particularly those subjected to plyometric loads¹. As mechanical loading is considered the primary driver through a progressive pathological model² it is logical to look for ways to identify “excessive load” and modify this. Several studies using analysis of jumping describe loading associated with patellar tendinopathy³⁻⁷. In volleyball players increased forces in the knee extensor mechanism have been associated with patellar tendinopathy^{3,4}. Motion at other joints as shown in the ankle and trunk may contribute to excessive patella tendon forces⁵. Biomechanical differences may develop as a result of tendon pathology or represent aetiological factors. Limiting flexion at the knee during a jump has been seen in those with previous patellar tendinopathy⁶ and, in a multi-sport cohort, when fatigued⁷. These examples may indicate a protective mechanism.

~~Jumping is often too high a load for symptomatic patients to tolerate due to pain.~~ Patients with chronic degenerative patellar tendinopathy often find jumping or hopping painful⁸. The single leg squat (SLS) is a routine test for basic biomechanical assessment in clinic and has relevance to jumping mechanics⁹. Parameters looked for on clinical examination include pelvic obliquity, anterior pelvic tilt, hip internal rotation, peak knee flexion angle, peak knee valgus abduction (valgus) angle and movement variability¹⁰. Such factors have been included in functional movement screens (FMS) with the aim of identifying those at increased risk of injury¹⁰⁻¹². However, Bushman’s study of 2476 soldiers screened showed low predictive value of FMS at 6 months follow up¹³. This highlights the need to re-examine the relationship

between these factors and how they contribute to injury risk. Excessive anterior pelvic tilt with hip extension has been proposed to lead to greater knee flexion at footstrike increasing load of the knee extensor mechanism and potentially predisposing to patellar tendinopathy¹⁴. As anterior tilt is caused by weak external hip rotators and gluteal/abdominal muscles exercises to strengthen these muscles are commonplace. There is little evidence assessing kinematics and kinetics in patellar tendinopathy. The identification and quantification of parameters presenting with patellar tendinopathy will inform and enable health care professionals involved in exercise prescription. This has been proven to be effective in other conditions such as Patellofemoral Pain Syndrome (PFPS)¹⁵.

Squats are also effective in treating patellar tendinopathy^{16 17} and can be modified as part of a progressive loading programme. Additional use of a decline board increases tendon forces¹⁸ and strain¹⁹. This study aims to assess kinematic and kinetic differences between patients with patellar tendinopathy and healthy controls when performing increasingly demanding variations of the SLS. It is hypothesised that cases with patellar tendinopathy will exhibit greater anterior pelvic tilt, pelvic obliquity, hip adduction, hip internal rotation, knee valgus and reduced knee flexion in comparison to both their asymptomatic side and a group of healthy controls.

2. Methods

A total of 24 controls and 25 cases were screened. Inclusion criteria for controls were males aged 18-55 and exclusion criteria musculoskeletal injury in the preceding 6 months or occupational restrictions concerning physical activity. For cases inclusion criteria, assessed by a Sport and Exercise Medicine Consultant (PB) and physiotherapist (JW), were patellar tendinopathy diagnosis of at least 6 months

chronicity with minimal improvement following best practice management including eccentric loading, heavy slow resistance and extra-corporeal shockwave therapy aged 18-55, ultrasound evidence of chronic degenerative patellar tendinopathy including thickening, hypoechogenic changes and neovascularisation. All cases assessed experienced pain on attempting to run or jump and were independently certified as medically unfit for their full military role by an Armed Forces General Practitioner prior to referral. Exclusion criteria were females and patients with a concurrent alternate lower limb diagnosis. The Ministry of Defence Research Ethics Committee approved the study (684/MODREC/15) and all participants gave written informed consent. Participants undertook movements described below.

For the small knee bend (SKB) verbal instruction was given:

“Stand on one leg with your foot pointing forward. Place the unsupported foot behind you by bending your knee 90°. While keeping your body upright, keeping your pelvis and heel in position, bend your knee so that your knee is in line with your 2nd toe and moves past it until you can no longer see the tape line.”¹⁰

5 repetitions were tested²⁰ allowing 2-3 practice repetitions immediately prior to testing^{21 22}. There was one minute of rest between trials^{22 23}.

Squat movements were standardised and 2 further tests the SLS and with the addition of a 25° decline board (see figure1)¹⁸ the single leg decline squat (SLDS) performed. Participants were instructed for SLS and SLDS to squat to 60° knee flexion using metronome pacing over a 4 second cycle^{22 24}. The non-stance leg was flexed at the knee to 90°²². Again 5 repetitions were captured following 2-3 practice squats with a minute's rest between trials.

FIGURE 1 INSERTED HERE – Caption below

Figure 1 – Decline board with example of foot placement

The testing sequence was SKB followed by SLS then SLDS. Tests were conducted bilaterally with the first leg to be tested decided by coin toss in controls and asymptomatic limb tested prior to the symptomatic limb in cases. Leg dominance according to which leg a participant would kick a ball with was recorded. Pain caused by the squats after each trial was monitored with a 100mm visual analogue scale.

For kinematic and kinetic data acquisition and processing 8 body segments (feet, shank, thigh, pelvis and trunk) were defined using retro-reflective markers placed on anatomical landmarks by the same operator (RBD) as previously described by this laboratory²⁵ (~~article submitted, Barker-Davies 2017~~). Joint moments were normalised to participant mass.

A VICON (Oxford, UK) 10-camera motion capture system and one AMTI (Boston, USA) force plate captured data at 120Hz and 1200Hz respectively. Following static and range of motion calibration trials participants performed the squats as described above.

All squat trials were trimmed to the corresponding 2nd, 3rd and 4th repetitions. Data was labelled in Vicon Nexus (version 2.1) and processed in Visual 3D (C-motion version 6.0, Rochelle, USA). Kinematic data was filtered using a 6Hz low pass bidirectional Butterworth filter²⁶ and gaps were interpolated using a 3rd order least squares fit (maximum 10 frames)²⁷. Kinetic data was filtered separately at 50Hz²⁸.

Kinematics for variables based on previous research²⁵ (~~article submitted, Barker-Davies 2017~~): hip adduction, knee flexion, pelvic tilt, pelvic obliquity and trunk flexion occurring at the time of peak knee flexion upon each of the three analysed squats were averaged. MANOVA tests were conducted for each of the three squat variations with subsequent ANOVA and bonferroni corrected post-hoc comparisons where

applicable. Using G-Power²⁹ a medium effect size ($f^2(V)$ 0.25) combined with α -level (0.05) and 80% power required a total sample of 39 participants for a MANOVA global effect tests for 5 kinematic variables and 3 groups: controls, symptomatic and asymptomatic limbs of cases.

For curve analysis kinematic and kinetic data from the trunk, pelvis, hip and knee were normalised to 100% of the squat cycle between the points of minimum knee flexion. Means were plotted in pairwise comparisons between groups with bootstrapped t-tests and 95% confidence intervals to identify phases of the squat cycle where significant differences occurred.

Extensor knee moment was extracted at 30°, 45° and 60° during both concentric and eccentric phases. Force can be calculated from moment data if the length of the lever (moment arm) from where the centre of rotation occurs to where the force is applied is known. In this case this is the distance from knee joint centre to intersect the patella tendon. Smidt's³⁰ calculations provide an estimation of moment arm length which varies through knee flexion range. By dividing extensor knee moment by patella tendon moment arm as demonstrated by Frohm³¹ patella tendon force was calculated at 30°, 45° and 60°. Those values represent instantaneous data so to get an appreciation of mechanical loading throughout the squat cycle moment impulse was calculated. For paced squats extensor knee moment was integrated to calculate impulse (area under the curve) from minimum knee flexion angle to peak for the eccentric phase and peak to minimum for the concentric phase. Calculations were undertaken using SPSS (v 23, IBM, Armonk, NY, USA), Microsoft Excel (v 14.7.6, Microsoft, California, USA) and Matlab (v2014a, Mathworks, USA).

3. Results

Twenty-one cases and 22 healthy controls participated in the study. ~~In addition 2 control participants did not meet exclusion criteria having on-going lower limb injuries and were not enrolled.~~ 2 further healthy controls were screened and excluded having sustained lower limb injuries between invitation to participate and laboratory assessment (1 hamstring strain and 1 ankle sprain). A further 4 cases were screened and excluded: 2 cases declined consent, due to availability and 2 cases had concurrent intra-articular knee pathology. There were no significant demographic differences between groups (table 1). Cases had a median symptom duration of 18 months (range 10-120), median VISA-P of 45 (range 19-70), median neovascularity grade (Modified Ohberg Score³²) of 3.5 (range 2-4) and maximum tendon thickness of 7.9mm (SD=1.7). Asymptomatic limbs of cases had a maximum tendon thickness of 4.7mm (SD=1.9) and median neovascularity grade of 0 (range 0-4). The SKB, SLS and SLDS were completed by 20, 19 and 16 cases respectively as limited by pain. All controls completed the protocol without pain.

TABLE 1 – INSERT HERE

3.1 Kinematic and kinetic differences relevant to clinical examination

~~Cases squatted to significantly less depth than controls by 6.62° (95% CI 0.66-12.59, p<0.05) when performing the SLS (appendix 1) there were no other significant differences in a priori powered kinematics at peak knee flexion (table 2).~~ On their symptomatic side cases squatted to significantly less depth than healthy controls by 6.62° (95% CI 0.66-12.59, p<0.05) when performing the SLS (appendix 1). There were no other significant differences in hip adduction, knee flexion, pelvic obliquity, pelvic tilt or trunk flexion angles extracted at peak knee flexion between symptomatic limbs, asymptomatic limbs or healthy controls.

TABLE 2 – INSERT HERE

Exploratory curve analysis revealed the majority of significant differences were between ~~injured~~ symptomatic limbs and healthy controls (figure 2). There is greater abduction (valgus) positioning of the knee in ~~cases~~ symptomatic limbs with decreased abduction (valgus) moment compared to healthy controls throughout the movement. Symptomatic limbs ~~Cases~~ initiate and complete squatting with more trunk, hip and knee flexion which is associated with increased hip/knee extensor moment and hip/knee internal rotation moment at initiation and completion of the squat compared to healthy controls. There was only one instance of significant difference in curve analysis between symptomatic and asymptomatic limbs with symptomatic limbs exhibiting greater knee abduction angle between 10 and 14% of the squat cycle. All significant differences are summarised in table 3.

FIGURE 2 – INSERT HERE – Caption below:

Figure 2. Summary of kinematic and kinetic differences between symptomatic limbs of cases (blue) and healthy controls (red). Shaded areas represent bootstrapped 95% confidence intervals. The bar along the x-axis indicates time-points where significant difference occurred. There is greater abduction (valgus) positioning of the knee in cases (g) with decreased abduction (valgus) moment (h) compared to healthy controls throughout the movement. Symptomatic limbs initiate and complete squatting with more trunk (i), hip (a) and knee (d) flexion which is associated with increased hip (b)/ knee (e) extensor moment and hip (c)/ knee (f) internal rotation moment at initiation and completion of the squat.

TABLE 3 – INSERT HERE

3.2 Extensor knee moments and patella tendon force production relevant for exercise prescription

Further examination of knee moment and force production was analysed on the SLS and SLDS only as they are more standardised and more commonly used for exercise

prescription than the SKB. Extensor knee moments (table 4) extracted during the eccentric phase were generally slightly higher than the concentric phase (range ES 0.15-0.42 where $p < 0.05$ on SLS at 30° knee flexion within asymptomatic limbs and controls, all groups at 45° knee flexion and on SLDS within controls at 45° and 60° knee flexion (appendix 2)). Use of a decline board increased extensor knee moments and resulting patella tendon force by 9-18% compared to a flat surface (range ES 0.62-1.45, $p \leq 0.01$ (appendix 3)). There was a trend for lower extensor knee moments at all angles in the healthy control group compared to symptomatic and asymptomatic limbs but this was only significant at 30° knee flexion ($\chi^2 = 7.704$, $p < 0.05$) with asymptomatic limbs exhibiting greater extensor knee moment than healthy controls (mean ranks 25.85 v 39.74, $p < 0.05$, ES=0.39). Two-way mixed ANOVAs (group v 30°/45°/60° knee flexion) confirmed significant main effects on SLS and SLDS for greater extensor knee moment at increased knee flexion angle ($F \geq 658.3$, $p < 0.01$) but not group ($F \leq 1.380$, $p > 0.05$) with no interaction between group and knee flexion angle ($F \leq 0.598$, $p > 0.05$) (appendix 4).

TABLE 4 – INSERT HERE

Extensor knee moment impulse (table 4) was 30-43% greater during the eccentric phase than concentric phase of all squats with a larger effect size for symptomatic and asymptomatic limbs of cases (range ES 1.16-1.32, $p < 0.01$) compared to healthy controls (range ES 0.52-0.61). Despite this there was no interaction between group and phase ($F_{(2,60)} = 1.478$, $p > 0.05$) (appendix 5). In all groups eccentric, concentric and combined total extensor knee moment impulses were 36-51% greater (range ES 1.19-1.68, $p < 0.01$) (appendix 6) with the addition of the decline board. There were no differences between the groups in extensor knee moment impulses either on SLS or SLDS ($F \leq 1.615$, $p > 0.05$) (appendix 7). Participants-Cases found squatting on their symptomatic side more painful with the addition of the decline board (median 57 v

43.5 $p < 0.01$, $ES = 0.69$). Correlation was not significant between pain on squatting and impulse (SLS $r = 0.264$, $p > 0.05$ and SLDS $r_s = 0.458$, $p > 0.05$). Correlation between pain on squatting and extensor knee moments was significant at 30° and 45° on SLS ($r = 0.458$, $p < 0.05$ and $r = 0.535$, $p < 0.05$ respectively) and at 60° on SLDS ($r_s = 0.641$, $p < 0.05$). VISA scores did not predict impulse generated on performing either squat (SLS $r_s = 0.090$ and SLDS $r_s = 0.053$, $p > 0.05$)(appendix 8).

4. Discussion

The main finding of this study is that cases with patellar tendinopathy on their symptomatic side only exhibit greater knee valgus on squatting compared to healthy controls.

Particular attention should be paid to examining the initial single leg stance posture as the majority of patients should be able to achieve this and several other significant differences were demonstrated at the start/finish of the squat cycle. These include increased trunk, hip and knee flexion associated with increased knee internal rotation moment and extensor knee moment in cases. Greater extensor knee moment at the start/finish of the squat cycle may be indicative of excessive patella tendon loading whilst the lack of differences at greater squat depth can be explained by the requirement in this model to move to a standardised position. Chronically increased extensor knee moment at minimal knee flexion angles typical for single leg stance and ambulation may represent a plausible risk factor for tendinopathy in this heterogenous population. Clinically some of these patients with degenerative pathology are of a level of symptomology that requires a reduction in step count before loading programmes can progress. Reduced knee abduction (valgus) and hip flexion moments in cases may represent failed attempts to resist valgus collapse. This alignment pattern has been shown to be induced by neuromuscular fatigue

which is another risk factor for musculoskeletal injury³⁶. Excessive knee valgus angle has been associated in similar SLS models with lumbar stress injury³³ and PFPS³⁴. In patellar tendinopathy reduced arch height in standing, which would likely occur with valgus alignment, has been demonstrated in a cross sectional study compared to controls³⁵. Conclusions relating to causation are limited by the cross-sectional nature of this study and further longitudinal research is planned.

The variations of squats in this study relate to practical and simple tests for clinical use. This study sought to standardise the depth and rate of squats so that peak patella tendon force was repeatable rather than an outcome measure in itself. Having achieved this its aim was to elucidate movement faults commonly looked for and identifiable without the use of specialist equipment. Had depth and rate not been standardised, as demonstrated by cases not quite managing to squat to the target of 60°, controls would likely have squatted much deeper than cases increasing the likelihood of false positive movement faults. This study is indicative of the low sensitivity of using solely peak kinematics as descriptors for such tests and has used curve analysis of the entire squat cycle to elucidate potentially important modifiable risk factors for pathology and targets for remedial physical therapy. What is considered abnormal by an experienced clinician can be difficult to quantify²⁵ (~~article submitted, Barker-Davies 2017~~) but may include several other parameters such as movement excursions, variability and changes in acceleration. Differences were almost exclusively seen between symptomatic limbs and healthy controls. The common practice of using the asymptomatic limb as a comparator is not supported by the results of this study as almost no differences were seen between sides of cases. This may be due to motor deficits occurring prior to development of pathology or cross-education from the symptomatic to the asymptomatic limb³⁷.

4.1 Using single leg squats to prescribe patella tendon loading

Modification of SLS to increase patella tendon loading as demonstrated here can be achieved by focusing on the eccentric phase, increasing the knee flexion angle and the addition of a decline board. Pain on squatting correlated with instantaneous moments, but not impulse, explaining more variance at angles where extensor knee moment is higher. This suggests pain is more an indicator of peak force rather than total dosage throughout movement. Pain may be related to force transmitted through the tendon above a threshold. Exercise into pain is a key feature of tendinopathy rehabilitation programmes proven to be effective^{38 39}. Mechanisms have been proposed for this including inducing tendon strain. In the Achilles high as opposed to low tendon strain exercise has been shown to induce superior mechanical adaptation in the form of increased stiffness⁴⁰. Pain on SLDS has been shown to correlate with reduced strain in patellar tendinopathy⁴¹. It may be that exercise into pain, which induces strain, is necessary to stimulate a healing response. This needs to be achieved without overloading the tendon⁴². ~~VISA score did not correlate with extensor knee moments indicating it has limited structural validity relevant to loading prescription. This supports the notion that~~ Loading dosage should be tailored to the individual within a pain monitoring framework⁴³.

INSERT TABLE 5 HERE

This data can be used to prescribe progressive loading as follows (table 5). During an isometric hold for example patella tendon force at 60° will be roughly twice that at 30° and 10-20% higher with the addition of a decline board. For an isotonic movement if patients cannot tolerate the concentric phase, for example due to fatigue, 30-43% more extensor knee moment can be generated focussing on the eccentric phase. For the full isotonic movement progression to a decline board will increase load by 36-51%. The additional loading a decline board provides is enhanced on isotonic movement compared to isometrics not only because the knee

joint centre is moved further away from the centre of mass¹⁸ but in practice greater squat depth is achieved even when instructed to reach a specific target. Low frequency exercise (3 second pacing) has been shown to induce superior mechanical adaptations than high frequency exercise (1 second pacing) with matched tendon strain⁴⁰. Progression from isometric to isotonic then heavier loading phases (eccentric loading or heavy slow resistance) prior to the introduction of plyometrics is a pragmatic approach which allows for patient feedback on pain response.

4.2 Limitations

Patella tendon moment arms should be subject specific but as no regression equations have been made based on readily measurable anthropometrics⁴⁴ it is reasonable to seek a match in terms of the whole study population. Smidt's participants³⁰ are similar to the military population in age, height and weight and therefore this technique to derive patella tendon force was used. Some cases were unable to complete the full protocol and on failing to squat to the full depth stipulated sample size was reduced for the more demanding tests and may have selected those with less severe pathology. This was mitigated by the test order starting with the least demanding task to maximise data capture.

Finally the nature of the pain experienced by cases in this controlled laboratory setting is likely to differ to that encountered in the real world. Cases often described pain being triggered by more plyometric activity such as jumping which generates increased extensor knee moment compared to walking, stair climbing and kicking and at a faster rate³. It was decided not to include a jumping test in this study due to the anticipated drop out rate as was seen with the more demanding decline squat. Further, causing a flare in symptoms in a cohort undergoing clinical treatment would have been undesirable. The use of controlled pacing and range of motion in

squatting was designed to limit the chance of adverse effects, of which there were none.

5. Conclusion

Knee valgus alignment is a plausible risk factor for patellar tendinopathy. Conclusions relating to causation are limited by the cross-sectional study design. Increasing squat depth, use of a declined surface and isolating the eccentric phase enable progression of loading prescription guided by pain.

Highlights

- Patellar tendinopathy cases exhibit excessive valgus collapse on single leg squat
- Peak patella tendon force on single leg squat is similar in cases and controls
- Pain on activity correlates with tendon force and can be used to titrate loading
- Forces can be increased by eccentric phase loading and addition of a decline board

Conflict of Interest Statement

All authors declare no conflicts of interest.

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Table 1 Demographics of participants

	Controls n=22	Cases n=21	Difference between groups/sides p value
Age, years, mean (SD)	34.0 (6.5)	30.5 (5.9)	0.075
Height, m, mean (SD)	1.79 (0.06)	1.78 (0.08)	0.991
Weight, Kg, mean (SD)	85.3 (9.2)	88.4 (10.8)	0.312
BMI, Kg/m ² (SD)	26.8 (2.72)	27.7 (3.2)	0.306
Pre-test VAS pain, 0-100mm, mean (SD)	0 (0)	25.9 (15.3)	-
VAS pain on SKB, 0-100mm, mean (SD)	0 (0)	*Symptomatic side: 35.1 (22.3) *Asymptomatic side: 8.4 (14.3)	*<0.0005
VAS pain on SLS, 0-100mm, mean (SD)	0 (0)	*Symptomatic side: 41.5 (22.9) *Asymptomatic side: 7.1 (13.1)	*<0.0005
VAS pain on SLDS, 0-100mm, mean (SD)	0 (0)	*Symptomatic side: 43.8 (26.5) *Asymptomatic side: 10.5 (14.0)	*<0.0005
Roles, frequency	Aircraft technician=4, Administrative Officer=5, Military Nurse=6, 5=Physical Training instructors, 2=Doctors	3=Electric/engineer technician, 1= Seaman, 2=Driver, 2= Mechanic, 3=IT/Comms engineer, 5=Infantry, 3=Admin. Officers, 1=Radars operator, 1=Tank loader,	-
Leg dominance	Right=20 Left=2	Right=21 Left=1	-
Injured leg	-	Right=12 Left=9	-

P values relate to independent t-test unless otherwise stated. *Value relates to comparison between symptomatic and asymptomatic sides with significant difference paired t-tests and all residuals normally distributed. BMI=Body Mass Index, VAS=Visual Analogue Scale, SKB=Small knee bend, SLS=Single leg squat, SLDS=Single leg decline squat.

Table 2 Mean kinematic variables at peak knee flexion (SD) powered for *a priori* MANOVA.

Variable	Group	Small knee bend (°)	Single leg squat (°)*	Single leg decline squat (°)	ANOVA (Single variable within group)
Hip adduction	Symp	^a 6.1 (7.2)	^{ab} 9.6 (7.5)	^b 5.4 (6.5)	p=0.002
	Asymp	4.8 (5.8)	8.6 (6.8)	5.1 (6.1)	p=0.072
	Control	^a 7.4 (5.3)	^{ab} 13.9 (7.6)	^b 9.8 (6.2)	p<0.0005
Knee flexion	Symp	^{ac} 49.9 (6.7)	^{a*} 57.9 (6.3)	^c 65.2 (9.0)	p<0.0005
	Asymp	^{ac} 49.5 (5.2)	^{ab} 61.2 (6.9)	^{bc} 66.4 (10.4)	*p<0.0005
	Control	^{ac} 50.2 (9.7)	^{ab*} 64.6 (8.3)	^{bc} 72.2 (6.7)	*p<0.0005
Pelvic Obliquity	Symp	1.0 (5.2)	0.9 (6.6)	1.1 (4.8)	p=0.028
	Asymp	^c 3.0 (5.5)	1.9 (6.1)	^c 2.4 (6.0)	p=0.036
	Control	^{ac} 0.3 (4.0)	^a 5.6 (6.9)	^c 5.6 (5.8)	p<0.0005
Pelvic tilt	Symp	^a 14.8 (9.8)	^a 19.7 (10.0)	19.7 (10.2)	*p=0.026
	Asymp	15.0 (9.6)	19.6 (9.4)	20.0 (11.0)	p=0.030
	Control	^{ac} 12.8 (7.7)	^a 24.1 (10.9)	^c 22.5 (10.2)	p<0.0005
Trunk flexion	Symp	^c 8.5 (4.8)	[#] 11.9 (4.6)	^c 17.1 (9.1)	&p=0.020
	Asymp	^{ac} 8.4 (5.1)	^{a#} 14.4 (6.9)	^c 16.3 (7.5)	p=0.003
	Control	^{ac} 5.8 (5.3)	^{a#} 16.4 (11.1)	^c 18.1 (9.4)	p<0.0005
MANOVA (All variables between groups)		F _(10,98) =0.78 p=0.652	F _(4,52) =2.63 p=0.045	F _(10,90) =1.34 p=0.220	

Symp=Symptomatic limb, Asymp=Asymptomatic limb. MANOVA: *Bonferroni corrected post-hoc significant difference between groups. #Trunk flexion on single leg squats excluded from MANOVA due to heterogeneous variance. ANOVA: Bonferroni corrected post-hoc significant differences: between ^asmall knee bend and single leg squat, ^bsingle leg squat and single leg decline squat and ^csmall knee bend and single leg decline squat. *Mauchly's test of sphericity positive Greenhouse-Geisser correction applied. &Non-parametric Friedman test used with follow-up Wilcoxon matched pairs.

Table 3 Timepoints in squat cycle of significant differences in kinematic/kinetic data

Test	Movement	Angle/ moment	% of squat cycle with sig. diff.	Direction of effect
Hip Joint				
SKB ^a	Flexion	Angle	1-27	Sympt>CON
SLS	Flexion	Angle	1-13	Sympt>CON
SLDS	Internal rotation	Moment	31-83	CON>Sympt
SKB ^c	Internal rotation	Moment	1-22, 97-98	Sympt>CON
SLS	Internal rotation	Moment	2, 4-6	Sympt>CON
SLS	Internal rotation	Moment	38-44, 47-57, 59-62	CON>Sympt
SLDS ^b	Flexion	Moment	1-15, 17, 93-94, 96-101	CON>Sympt
SKB	Flexion	Moment	2-37, 86-101	CON>Sympt
SLS	Flexion	Moment	2-21, 94-101	CON>Sympt
Knee joint				
SLDS	Abduction	Angle	1-10	Sympt>CON
SLS	Abduction	Angle	1-20, 33-87, 89-101	Sympt>CON
SKB ^g	Abduction	Angle	1-34, 40-101	Sympt>CON
SLDS	Flexion	Angle	38-72	CON>Sympt
SKB ^d	Flexion	Angle	1-26, 89-101	Sympt>CON
SLS	Flexion	Angle	1-10	Sympt>CON
SLS	Flexion	Angle	49-54	CON>Sympt
SLDS ^h	Abduction	Moment	1-81, 86-101	CON>Sympt
SKB	Abduction	Moment	1-101	CON>Sympt
SLS	Abduction	Moment	2-101	CON>Sympt
SLDS	Internal rotation	Moment	1-17, 24-25, 30, 84-101	Sympt>CON
SKB ^f	Internal rotation	Moment	1-26, 91-101	Sympt>CON
SLS	Internal rotation	Moment	1-6, 9-14, 92-101	Sympt>CON
SKB ^e	Extensor	Moment	1-25, 89-98, 101	Sympt>CON
SLS*	Extensor	Moment	6-8	Sympt>CON
SLS	Abduction	Angle	10-14	Sympt>Asympt
Trunk				
SLDS ⁱ	Flexion	Angle	1-16, 96-101	Sympt>CON
SLS	Flexion	Angle	2-13	Sympt>CON

SKB=small knee bend, SLS=single leg squat, SLDS=single leg decline squat, Sympt=symptomatic limbs of cases, Asympt=asymptomatic limbs of cases, CON=control. ^{a-i}Represented in figure 2 with letter corresponding to graph.

Table 4 Instantaneous extensor knee moments and moment impulse at varying degrees of squat depth and phase of the squat cycle

Instantaneous extensor knee moment and patella tendon force extracted during the eccentric phase of squat movement					Extensor knee moment impulse throughout squat movement				
Knee flexion angle (°)	Extensor knee moment (Nm/Kg) (SD)	Patella tendon force (N/Kg)	ANOVA between groups (F)	Pairwise Differences SLDS v SLS (Nm/Kg)	Phase of Squat	Moment Impulse (Nm.s/Kg)(SD)	ANOVA between groups (F)	Pairwise SLDS v SLS (Nm.s/Kg)	Pairwise Eccentric v Concentric (Nm.s/Kg)
Single leg squat (SLS)									
30	Symp 0.75 (0.12)	15.31	# $\chi^2=7.704$ p=0.021 #Asymp>Con p=0.035 ES=0.39	-	Eccentric phase	Symp 2.00 (0.41)	1.615 p=0.207	-	*0.60 (43%)
	Asymp 0.78 (0.12)	15.92				Asymp 1.96 (0.35)			*0.50 (34%)
	Control 0.67(0.10)	13.67				Control 1.78 (0.58)			*0.42 (31%)
45	Symp1.15 (0.16)	23.47	2.981 p=0.059	-	Concentric phase	Symp 1.40 (0.28)	0.942 p=0.396	-	-
	Asymp 1.16 (0.15)	23.67				Asymp 1.46 (0.27)			
	Control 1.05 (0.14)	21.43				Control 1.36 (0.37)			
60	Symp 1.46 (0.22)	31.06	0.924 p=0.405	-	Full ROM	Symp 3.40 (0.59)	1.156 p=0.322	-	-
	Asymp 1.51 (0.13)	32.13				Asymp 3.43 (0.57)			
	Control 1.40 (0.18)	29.79				Control 3.14 (0.83)			
Single leg decline squat (SLDS)									
30	Symp 0.83 (0.12)	16.94	2.227 p=0.118	*0.09 (12%)	Eccentric phase	Symp 2.71 (0.41)	0.421 p=0.659	*0.71 (36%)	*0.69 (34%)
	Asymp 0.85 (0.14)	17.35		*0.08 (10%)		Asymp 2.80 (0.41)		*0.84 (43%)	*0.67 (32%)
	Control 0.77 (0.12)	15.71		*0.11 (16%)		Control 2.67 (0.48)		*0.89 (50%)	*a0.66 (30%)
45	Symp 1.32 (0.15)	26.99	1.847 p=0.168	*0.17 (15%)	Concentric phase	Symp 2.02 (0.37)	# $\chi^2=0.666$ p=0.717	*0.62 (44%)	-
	Asymp 1.30 (0.16)	26.53		*0.14 (12%)		Asymp 2.13 (0.36)		*0.67 (46%)	
	Control 1.24 (0.13)	25.31		*0.19 (18%)		Control 2.05 (0.40)		*0.69 (51%)	
60	Symp 1.67 (0.19)	35.53	0.902 p=0.403	*0.23 (16%)	Full ROM	Symp 4.73 (0.67)	0.544 p=0.584	*1.33 (39%)	-
	Asymp 1.68 (0.14)	35.74		*0.16 (11%)		Asymp 4.93 (0.69)		*1.50 (44%)	
	Control 1.63 (0.13)	34.68		*0.23 (16%)		Control 4.72 (0.60)		*1.58 (51%)	

*Significant difference p<0.0005 #Parametric assumptions violated Kruskal-Wallis H and Bonferroni corrected Mann Whitney-U used.

^aResiduals not normally distributed Wilcoxon matched pairs used. Greater extensor knee moment and therefore patella tendon force is generated at deeper knee flexion angles and on using a decline board. Symp=Symptomatic limbs, Asymp=Asymptomatic limb. The eccentric phase of squatting results in greater impulse than the concentric phase with use of a decline board resulting in greater impulse.

Table 5. Recommendations for Progressive Loading Rehabilitation for patellar tendinopathy

Suggested progressive loading exercises for the patellar tendon
<i>Maximum pain on activity $\leq 3/10$ without flare-up in symptoms the following day</i>
Start with 30° knee flexion isometric hold
Progress to 60° knee flexion isometric hold to double patella tendon force (PTF)
Do not progress knee flexion angles above 60°. There is an exponential increase in patellofemoral force
Add decline board to isometric hold to increase PTF 10-20% further
Introduce isotonic exercises
Progress to a decline board to increase moment impulse (extensor torque throughout the whole movement) up to 50%
Isolate the eccentric phase to increase moment impulse by a further 30%
Once able to tolerate isotonic exercise on decline board or isolating eccentric phase progress to weighted squats prior to plyometric activities

Figure 1

a. Hip flexion angle

b. Hip flexion moment

c. Hip internal rotation moment

d. Knee flexion angle

e. Knee flexion moment

f. Knee internal rotation moment

g. Knee adduction angle

h. Knee adduction moment

i. Trunk flexion angle

Figure 2