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Decreased Hip Flexion during Spike Jump-Landings after Fatigue is Predictive of Patellar Tendinopathy in Volleyball

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Context: Patellar tendinopathy (PT) is a highly prevalent overuse injury in volleyball. However, little is known if and how the risk for developing PT is increased through fatigue-induced alterations during repetitive jump-landing activities in volleyball.

Objective: The purpose of this study was to explore fatigue-induced risk factors for PT during a spike jump-landing task in volleyball.

Design: Prospective cohort study.

Setting: 3D biomechanical laboratory screening.

Patients or Other Participants: Seventy-nine adult, male volleyball players.

Main Outcome Measure(s): At baseline (pre-season), 3D full-body kinematics and kinetics were collected while performing a spike jump before and after a volleyball-specific fatigue protocol. Throughout the season, players were followed for the occurrence of PT and survival analysis with competing risks was performed to identify significant predictors for the development of PT ($p < 0.05$).

Results: During follow-up, 10 of the 79 players developed PT (13%). Players with significantly less hip flexion during the horizontal landing/push-off phase of the spike jump after fatigue were at higher risk for developing PT (HR = 0.898; 95% CI 0.826 to 0.977; $p = 0.023$) as well as players with a significantly more elongated rectus femoris muscle-tendon unit (HR = 3.258; 95% CI 1.136 to 9.343; $p = 0.032$).

Conclusions: Despite the low (injured) sample size of this study, preliminary research findings indicate less hip flexion and more elongated rectus femoris muscle-tendon units during landing after fatigue as potential risk factors for developing PT. Future prevention programs for PT may wish to focus on hip-specific exercises and technique modifications (e.g., more hip flexion during landing) under fatigued circumstances.

Keywords: Injury Prevention, Jumper's Knee, Stress, Strain, Exertion

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Key Points:

(1) The accuracy of risk factor screenings can be enhanced through screening fatigue-induced movement alterations during jump-landing.

(2) Less hip flexion during landing after fatigue increases the risk for developing patellar tendinopathy and may be associated with rectus femoris contractions from a more elongated configuration.

(3) Prevention programs for patellar tendinopathy may wish to focus on hip-specific exercises and technique modifications (e.g., more hip flexion during landing) under fatigued circumstances.

Patellar tendinopathy (PT) is a highly prevalent overuse injury in sports with repetitive bouts of jump-landing tasks. Volleyball players are mostly affected, with prevalence rates near 50% and incidence rates up to 30 injuries/100 players/season.[1,2] PT refers to persistent patellar tendon pain and loss of function related to mechanical loading.[3] This pathological condition often affects athletes' sports participation and even may lead to termination of their athletic career.[4] Identifying the risk factors for PT is necessary before developing effective prevention programs.[5,6] The aetiology of PT is multifactorial in nature and there is currently a lack of strong evidence concerning extrinsic (e.g., activity volume) and intrinsic risk factors (e.g., body weight, jump performance).[5,6]

Repetitive patellar tendon loading is considered to be a modifiable extrinsic risk factor for PT.[7,8] Accumulation of high eccentric (or even concentric) tendon loading is thought to produce microtraumas in the tendon, which can eventually lead to intra-tendinous histopathological changes.[7,8] The magnitude of the patellar tendon loads, and how athletes can accommodate to them, can be accurately quantified via biomechanics.[9] This illustrates the need to investigate biomechanical risk factors for PT during dynamic tasks such as jump-landings. Evaluation of the entire kinematic chain appears to be important as both local (knee) and non-local (proximal or distal to the knee) kinematic alterations may affect patellar tendon loading.[9] At the local level, more knee flexion during landing can increase both tensile and compressive loads onto the tendon.[10] Regarding the non-local factors, less ankle dorsiflexion may result in less load absorption, potentially resulting in more loads being transferred to the patellar tendon.[9] On the other hand, more trunk and/or hip flexion during landing can be associated with reduced patellar tendon loading due to a closer positioning of the ground reaction force vector with respect to the knee joint.[11] Besides that, kinematic changes during landing can alter contributions of the different muscle bellies of the quadriceps (i.e., rectus femoris and vastus intermedius/lateralis/medialis), of which the rectus femoris has already been associated with the development and perpetuation of PT symptoms.[12] Although the exact underlying mechanism for this remains speculative until now, less hip flexion during jump-landing can, for example, lead to rectus femoris contractions from a more elongated configuration, possibly altering tension and/or loading into the patellar tendon.

Previous prospective studies were unable to determine clear local and/or non-local jump-landing related biomechanical risk factors predictive for PT.[2,8,13] To enhance the accuracy of risk factor screenings, functional fatigue protocols have already been proven to be successful, increasing the ability to identify biomechanical risk factors during jump-landings in female physical education students.[14] The concept of fatigue is an evolving field in literature, with complex interactions between physical and psychological types of fatigue potentially affecting neuromuscular control, making risk factors in (pre-season) screenings more apparent.[14,15] Hence, it seems crucial to consider fatigue, especially since fatigue has also been shown to increase the risk for knee injuries by altering both local and non-local kinematics during jump-landing tasks.[11,15] Likewise, healthy basketball, soccer and volleyball players employ kinematic strategies during landing when fatigued (e.g., less knee flexion and more trunk flexion after functional fatigue protocols compared to baseline) that may lead to a reduction of patellar tendon loading.[16,17] The question then arises whether players are more prone to develop PT if they do not show local and/or non-local compensatory kinematic strategies to reduce patellar tendon loads during landing when fatigued.[17]

The primary purpose of this study was to explore whether changes in patellar tendon loading during volleyball's most challenging jump activity, i.e., the spike jump[17], induced by a volleyball-specific fatigue protocol, may increase the risk of developing PT. As a secondary purpose, we explored if sagittal plane trunk, hip, knee, and/or ankle kinematic changes after fatigue may contribute to an increased PT injury risk. It was hypothesized that increased patellar tendon loads when fatigued, accompanied by kinematic alterations like more knee flexion and/or less trunk-hip-ankle (dorsi-)flexion, may elevate the risk of PT. For further exploration, we also observed whether fatigue-induced changes in quadriceps muscle-tendon unit (MTU) lengths, particularly when contractions occur from a more elongated configuration, may contribute to PT injury risk.

Methods

1. Study Design

This prospective cohort study started with pre-season screenings (July-September 2021) including 3D full-body biomechanics when performing spike jumps before and after a volleyball-specific fatigue protocol. Thereafter, participants were followed for the occurrence of PT during one consecutive volleyball season (35 weeks). This study was registered at ClinicalTrials.gov (ID = X) and approved by the Ethical Committee of the Ghent University Hospital (ethical approval number = X).

2. Participants

For inclusion, participants had to meet the following criteria: (1) male competitive volleyball players (from all competition levels) since PT is higher prevalent in this cohort[1], (2) at least 18 years old, and (3) at least 6 months injury-free. One-hundred and fifty volleyball players (from 13 clubs) were screened for inclusion, of which 86 met the inclusion criteria. Since PT incidence is 30 injuries/100 players/season[2], 26 PT injuries are expected in this sample, assuming to be sufficient to perform an explorative Cox regression analysis. Written informed consent was obtained from each participant prior to inclusion.

3. Procedures

The pre-season screening started with a 10-minute warm-up consisting of familiarization with the fatigue protocol without inducing any noticeable fatigue. Thereafter, kinematics and kinetics were collected when performing spike jumps before and after the fatigue protocol.

Fatigue was induced by a five-circuit version of the high-intensity, intermittent exercise protocol (HIIP-5).[18] The circuits of the HIIP-5 include exercises mimicking volleyball activities (i.e., directional changes, jumps, sprints and side-steps) that are executed at the highest possible movement speed. These circuits are interspersed with passive rest periods of 30 seconds. The HIIP-5 induces acute and long-lasting volleyball-specific fatigue responses up to 30 minutes after HIIP-5, assuring a sufficiently large time window within which the post-fatigue biomechanical assessments were completed (average time frame to complete the assessments post-HIIP-5 was 4.0 minutes).[18]

Spike jump-landing biomechanics were collected before and after the HIIP-5. The spike jump incorporates an initial horizontal landing/push-off phase, which includes a stretch-shortening cycle and induces higher patellar tendon loads compared to jumps with a predominantly vertical landing component.[16,17] During the spike jump, participants ran from a self-selected distance towards a volleyball net. Then, they landed with both feet separately on two force plates prior to pushing-off vertically (referred to as the horizontal landing/push-off phase). The force plates were located in front of the net, which was attached at a standardized height of 2.43 m. Jump height effort was standardized by asking to swing with the dominant hand forward to an imaginary ball positioned just above the net. Participants were asked to perform five valid spike jumps both before and after the HIIP-5. Trials were discarded if (1) one foot did not fully touch the force plate, (2) both feet did not touch the separate force plates, or (3) participants showed an adaptation of their preferred stride lengths in an attempt to target the force plates.

4. Data Collection and Analysis

To monitor exertion induced by the HIIP-5, the following parameters were registered: (1) heart rate using a Polar system (Polar, Electro), (2) rate of perceived exertion for breathlessness (RPE-B) and legs (RPE-L) on a 20-point Borg scale, (3) HIIP-5 run-time using infrared timing gates (Microgate), and (4) spike jump height derived from the pelvic kinematic data.[17]

Kinematic data were collected with a 12 camera opto-electronic system (Oqus 3+, Qualysis, 300 Hz) and were synchronized with ground reaction force data gathered by two force plates embedded in the floor (AMTI, 1200 Hz). Retroreflective markers were placed on the skin according to the Liverpool John Moores University biomechanical model.[17] Kinematic and force data were processed in Qualisys (Qualisys Track Manager, Qualisys) and subsequently in Visual 3D software (Visual 3D v5, C-motion). Kinematic and force data were filtered using a fourth order Butterworth and critically damped low-pass filter at 20 Hz, respectively. Euler rotations (X-Y-Z) were used to calculate 3D full-body joint kinematics and kinetics. Since the spike jump is mainly a sagittal plane motion and patellar tendon loading is based on sagittal plane metrics, only sagittal plane data were utilized in this study. We also focused solely on the participants' leading leg to perform the spike jump due to higher patellar tendon loads in this leg.[17] The horizontal landing/push-off phase was defined as the period from initial contact to take-off, which was determined using the vertical component of the ground reaction force with a threshold set at 25 N.

Patellar tendon loading (peak force) was computed by dividing the net knee joint moment (normalized for body mass) by the patellar tendon moment arm, estimated as a function of the knee joint angle (Appendix 1).[19] Sagittal plane pelvis-trunk, hip, knee, and ankle kinematics were extracted as secondary parameters. For further exploration, the lengths of the different parts of the quadriceps MTU (rectus femoris and vastus intermedius/lateralis/medialis) were also computed since the amount of produced muscle-tendon force heavily depends on its length.[20] These lengths were estimated as a function of known hip and/or knee joint angles, which normalizes for thigh length (Appendix 1).[21] For the secondary and exploratory variables, discrete values were extracted at initial contact, peak joint angle/peak MTU length, peak knee flexion and take-off during horizontal landing/push-off. The averages of 5 trials were determined for every time point for pre-fatigue (baseline), post-fatigue (at least 30 seconds after HIIP-5), and their corresponding normalized Δ-value calculated (((Post – Pre) / Pre) x 100).

5. Injury Registration and Diagnostic Criteria

Injury data were collected during follow-up using a weekly and 3-montly retrospective questionnaire in the online platforms Panega Sports© and Research Electronic Data Capture (REDCap)©, respectively. When participants started to report patellar tendon complaints, they were contacted by phone to obtain more information concerning the nature of the injury. Moreover, these participants were asked to fill in the 'Victorian Institute of Sport Assessment Patellar tendinopathy questionnaire' (VISA-P), which evaluates symptom severity, knee function and ability to play.[22] To be included in the PT experimental group, participants had to meet the following criteria: (1) patellar tendon pain at the leading leg, and (2) loss of function, confirmed by a VISA-P score <80 or a sports stop ≥1 training/match due to patellar tendon pain. The total number of missed sessions due to these complaints was recorded to quantify the injury's impact on sports participation. Clinical differential diagnosis with other types of anterior knee pain was based on pain localization, with PT presenting as localized proximal patellar tendon pain[8], while other types of anterior knee pain such as patellofemoral pain syndrome (PFPS) are characterized by more diffuse pain.[23] Presence of ultrasonographic abnormalities (e.g., tendon thickening, swelling, hypoechogenicity, neovascularisation), taken from a physician in the clinical setting, was additionally documented if available.[3,7] PT complaints at the trailing leg were also registered and these participants were excluded from the analysis due to uncertainty concerning the effect of contralateral jump-landing patterns on PT injury risk. Other (self-reported) lower quadrant (i.e., low back and lower extremity) injuries were registered and included as competing risks, as these could either preclude the occurrence of the injury of interest (i.e., PT at the leading leg) or fundamentally alter its likelihood, for example by leading to sports discontinuation.[24] Throughout follow-up, participants were regularly contacted by phone to verify compliance with the injury registration method.

6. Exposure Time

The average amount of weekly volleyball participation (training and/or match) was registered during follow-up in Panega Sports© and REDCap©. Afterwards, time at risk (number of hours of volleyball participation) was calculated from the start of the study until the occurrence of the injury at interest (PT) or any other lower quadrant injury or until study ending/drop-out for participants who did not develop a lower quadrant injury.

7. Statistical Analysis

Statistical analysis was performed with IBM SPSS (version 28) and R (version 4.2.1) statistics. First, descriptive statistics were performed to check for potential confounders (demographics, history of patellar tendon complaints and indicators of exertion) (Table 1). Then, survival analysis with competing risks was applied to explore fatigue-induced biomechanical predictors for the development of PT. Survival analysis was used since it has the advantage of taking into account the individual amount of sports participation until injury or end of follow-up.[14] Lower quadrant injuries other than PT were included in the analysis as competing risks.[24] The assumptions of proportionality and linearity of the hazards were investigated by means of log-minus-log plots against time and the Schoenfeld residual global test. Only unadjusted univariate Cox regression analyses were performed due to the explorative nature of this study. For all biomechanical variables, *p*-values were determined for the normalized Δ-value to determine significant fatigue-induced predictors for PT (Table 2). In order to better interpret the effect of fatigue on these predictors, *p*-values were also determined for pre-fatigue and post-fatigue results (Appendix 2-3). Overall, the level of significance was set at $\alpha = 0.05$. Finally, thresholds for significant fatigue-induced predictors that may precipitate PT were defined with receiver operating characteristic (ROC) curve analysis using MedCalc software© (www.medcalc.org) (Table 3).[14]

Results

During follow-up, four players did not register any exposure time and three players developed PT complaints at the trailing leg, which excluded them from the statistical analysis. Therefore, a total number of 79 players were included in the analysis. Of them, 10 players developed PT at the leading leg during follow-up (13%). Four players discontinued training/match sessions (ranging from 1 to 7 sessions), while 6 players had no sports stop due to the injury. Reported VISA-P scores at symptom onset ranged from 57 to 79 points out of 100, with the time from pre-season screening to symptom onset varying between 3.9 and 30.3 weeks. Ultrasonography was conducted in two players with PT in which active signs of inflammation (e.g., increased swelling or neovascularization) were documented. The control group consisted of 35 injury-free players while 34 players developed competing risks (Figure 1). Body mass index was the only confounding variable that was significantly different between the PT and control group, with higher values for the PT group (Table 1). The PT injury rate was 0.7 events per 1000 hours of volleyball sports participation.

The Cox regression analysis revealed that patellar tendon loading was not a significant fatigue-induced predictor for PT. For the secondary/exploratory variables, hip flexion (at initial contact and at peak hip flexion) and rectus femoris MTU length (at peak knee flexion) were significant fatigue-induced predictors for PT. The hazard for developing PT increased approximately 1.1 times if hip flexion decreased by 1% post-fatigue compared to pre-fatigue. Moreover, the hazard for developing PT increased 3.3 times if rectus femoris MTU length increased by 1% post-fatigue compared to pre-fatigue (Table 2). To further explore the contraction dynamics of the rectus femoris MTU, the force-length profile of rectus femoris MTU was plotted during the entire horizontal landing/push-off phase

(Figure 2). Although the differences were small and there was considerable overlap in variability between the profiles, players with PT tended to show a greater increase in MTU length under fatigue compared to controls, which is reflected in a right shift of the profile with more elongation for 'similar' forces during almost entire horizontal landing/push-off. All other secondary/exploratory variables did not significantly alter the risk for developing PT (Table 2). Thresholds were only determined for the hip flexion angle, and revealed cut-off values of >5.9° and >3.5° decrease in hip flexion after fatigue at initial contact and peak angle, respectively (Table 3).

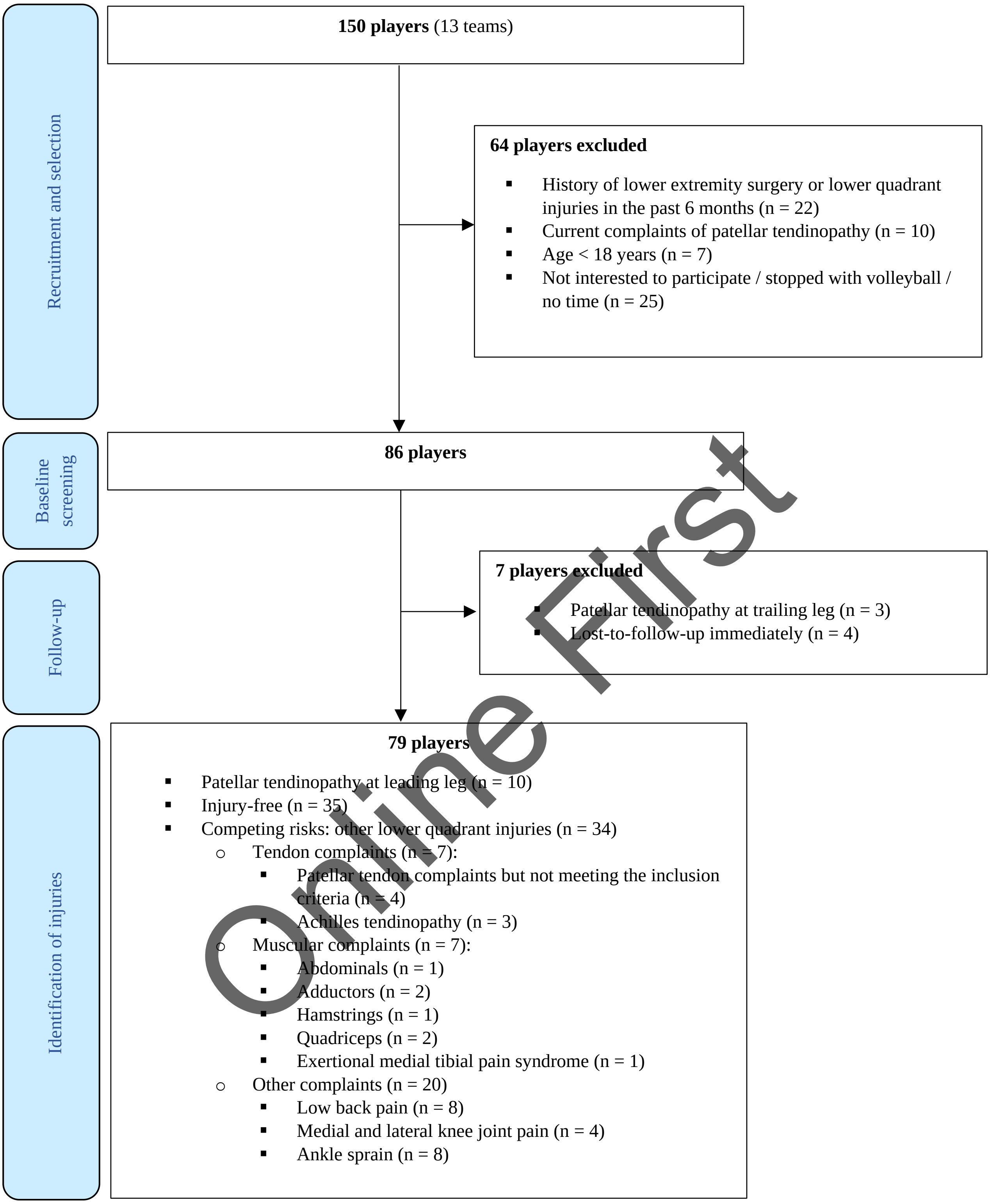


Figure 1. Flow Diagram of Participant Selection Process in the Study.

Table 1. Potential Confounders for Patellar Tendinopathy Injury Risk.

Variables	Control (n = 69)	PT (n = 10)	*p*-value*	Hedge's *g* effect size
Demographics				
Age (yrs.)	23.35 ± 5.05	23.00 ± 4.37	0.837	0.069
Weight (kg)	79.74 ± 11.16	84.88 ± 14.21	0.192	0.441
Height (m)	1.86 ± 0.07	1.83 ± 0.05	0.236	0.400
Body mass index (kg/m²)	**23.10 ± 2.86**	**25.30 ± 3.56**	**0.031**	**0.739**
Volleyball experience (yrs.)	12.96 ± 5.67	11.40 ± 7.07	0.435	0.263
Volleyball participation per week (h)	7.03 ± 2.82	6.50 ± 1.58	0.565	0.194
Elite competition level (%)	5.8	0.0	0.435	-
Setter, middle, outside hitter, libero (%)	15.9, 26.1, 43.5, 15.5	0.0, 30.0, 60.0, 10.0	0.514	-
History of previous patellar tendon complaints (>6 months ago)				
Prevalence (%)	23.9	20.0	0.787	-
Duration (yrs.)	3.68 ± 4.95	1.00 ± 0.00	0.468	0.555
Indicators of exertion				
Heart rate at HIIP-5 ending (% of theoretical maximum)	96.65 ± 3.73	95.47 ± 5.17	0.381	0.295
RPE-B at HIIP-5 ending (6-20)	18.55 ± 1.57	18.10 ± 1.73	0.404	0.281
RPE-L at HIIP-5 ending (6-20)	15.75 ± 2.88	15.30 ± 3.68	0.660	0.149
Run-time during HIIP-5 (min)	5.68 ± 0.26	5.85 ± 0.42	0.289	0.572
Spike jump height, Δ post- vs pre-fatigue (cm)	-3.07 ± 3.54	-4.42 ± 2.77	0.254	0.385

The control group consisted of 35 injury-free players and 34 players with competing risks. Values are expressed as mean ± SD (if possible). Significant variables are highlighted in bold. * Student *t* tests or one-way ANOVAs were used for continuous variables, Chi-Square tests for binary variables.

Table 2. Fatigue-Induced Biomechanical Predictors for Patellar Tendinopathy.

Variables	Control (n = 69)	PT (n = 10)	*p*-value*	Hazard ratio with 95% CI	Hedge's *g* effect size
<u>Primary outcome variable</u>					
Patellar tendon loading, peak (%), ↘ (-) / ↗ (+)	-2.98 ± 7.86	0.74 ± 5.81	0.115	1.080 (0.984-1.186)	0.482

Secondary/exploratory outcome variables					
Pelvis-trunk flexion (%), ↘ (-) / ↗ (+)					
Initial contact	-19.33 ± 113.40	16.48 ± 49.64	0.089	1.015 (1.000-1.030)	0.329
Peak pelvis-trunk flexion	11.68 ± 72.09	23.52 ± 48.66	0.483	1.003 (0.996-1.009)	0.168
Peak knee flexion	-64.76 ± 806.29	274.99 ± 793.12	0.185	1.001 (1.000-1.001)	0.418
Take-off	33.78 ± 161.10	9.83 ± 35.80	0.823	0.999 (0.992-1.007)	0.156
Hip flexion (%), ↘ (-) / ↗ (+)					
Initial contact	**-7.65 ± 6.80**	**-16.43 ± 13.21**	**0.006**	**0.922 (0.874-0.972)**	**1.110**
Peak hip flexion	**-4.14 ± 4.99**	**-7.85 ± 7.65**	**0.023**	**0.898 (0.826-0.977)**	**0.685**
Peak knee flexion	-4.71 ± 7.86	-6.91 ± 11.33	0.218	0.957 (0.892-1.027)	0.261
Take-off	-59.43 ± 265.66	-127.75 ± 304.33	0.731	1.000 (0.998-1.001)	0.250
Knee flexion (%), ↘ (-) / ↗ (+)					
Initial contact	-16.18 ± 12.78	-20.90 ± 8.63	0.295	0.976 (0.933-1.021)	0.377
Peak / Peak knee flexion	-3.63 ± 3.35	-2.38 ± 2.52	0.337	1.130 (0.877-1.455)	0.378
Take-off	11.25 ± 64.96	1.23 ± 23.27	0.860	1.002 (0.985-1.018)	0.161
Ankle dorsiflexion (%), ↘ (-) / ↗ (+)					
Initial contact	-1.16 ± 3.12	-0.37 ± 2.61	0.386	1.090 (0.899-1.323)	0.254
Peak ankle dorsiflexion	-2.02 ± 2.48	-0.56 ± 2.68	0.223	1.173 (0.910-1.513)	0.578
Peak knee flexion	-2.14 ± 2.47	-0.63 ± 2.52	0.197	1.168 (0.928-1.471)	0.603
Take-off	-2.06 ± 5.88	-1.99 ± 7.69	0.720	1.018 (0.922-1.125)	0.012
Rectus femoris MTU length (%), ↘ (-) / ↗ (+)					
Initial contact	-0.18 ± 0.55	0.05 ± 0.88	0.333	1.557 (0.660-3.672)	0.376
Peak length	-0.09 ± 0.53	0.24 ± 0.66	0.110	2.443 (0.881-6.778)	0.599
Peak knee flexion	**-0.18 ± 0.57**	**0.21 ± 0.67**	**0.032**	**3.258 (1.136-9.343)**	**0.657**
Take-off	0.09 ± 0.54	0.34 ± 0.64	0.104	2.683 (0.903-7.968)	0.454
Vastus intermedius MTU length (%), ↘ (-) / ↗ (+)					
Initial contact	-2.21 ± 1.76	-2.98 ± 1.42	0.158	0.771 (0.535-1.110)	0.446
Peak length	-0.53 ± 0.51	-0.33 ± 0.34	0.255	2.766 (0.454-16.868)	0.403
Peak knee flexion	-0.53 ± 0.51	-0.33 ± 0.34	0.253	2.787 (0.454-17.108)	0.406
Take-off	-0.11 ± 1.55	-0.25 ± 1.41	0.105	1.554 (0.917-2.633)	0.233
Vastus lateralis MTU length (%), ↘ (-) / ↗ (+)					
Initial contact	-1.94 ± 1.55	-2.61 ± 1.24	0.168	0.750 (0.497-1.131)	0.441
Peak length	-0.28 ± 0.31	-0.15 ± 0.17	0.168	10.177 (0.287-360.742)	0.422
Peak knee flexion	-0.28 ± 0.31	-0.15 ± 0.17	0.157	11.170 (0.294-423.941)	0.424
Take-off	-0.10 ± 1.39	0.22 ± 1.26	0.108	1.624 (0.904-2.916)	0.227
Vastus medialis MTU length (%), ↘ (-) / ↗ (+)					
Initial contact	-1.75 ± 1.39	-2.36 ± 1.12	0.164	0.724 (0.458-1.144)	0.443
Peak length	-0.28 ± 0.30	-0.15 ± 0.17	0.179	9.809 (0.277-347.103)	0.421
Peak knee flexion	-0.28 ± 0.30	-0.15 ± 0.17	0.170	10.529 (0.283-391.700)	0.424
Take-off	-0.09 ± 1.24	0.20 ± 1.12	0.107	1.730 (0.894-3.347)	0.230

The control group consisted of 35 injury-free players and 34 players with competing risks. Normalized Δ-values (%) are expressed as mean ± SD. Significant predictors are highlighted in bold. * Unadjusted univariate Cox regression with competing risks.

Table 3. Cut-Off Values for Fatigue-Induced, Significant Predictors for Patellar Tendinopathy.

Fatigue-induced predictors	Cut-off (°)	Sensitivity with 95% CI (%)	Specificity with 95% CI (%)	LR +	LR -
Less hip flexion at initial contact	>5.9	80.0 (44.4-97.5)	69.6 (57.3-80.1)	2.6	0.3
Less peak hip flexion	>3.5	80.0 (44.4-97.5)	62.3 (49.8-73.7)	2.1	0.3

LR + = positive likelihood ratio, LR - = negative likelihood ratio.

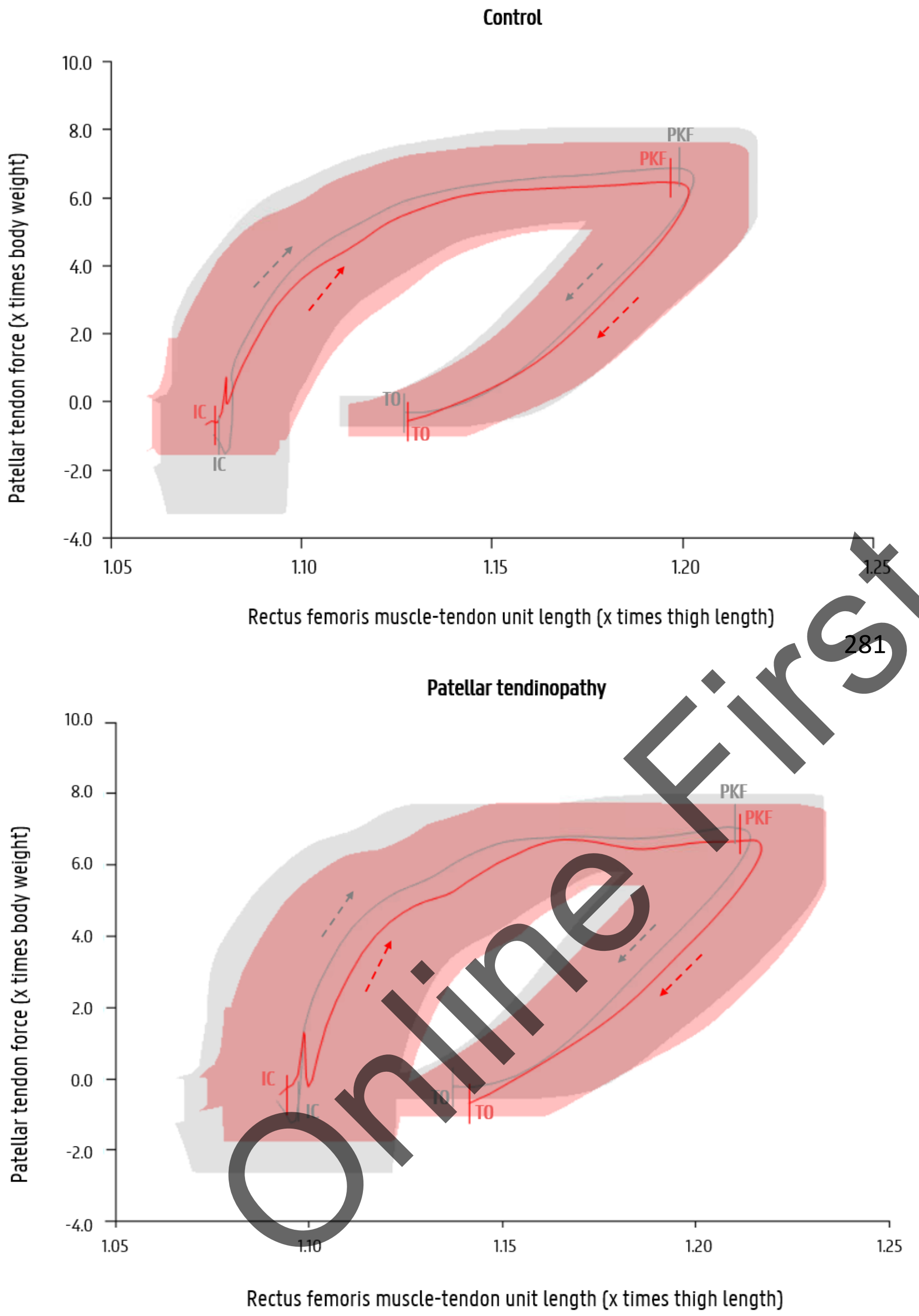


Figure 2. Force-Length Relationship Before and After Fatigue.

Trajectories are presented as mean and standard deviation clouds. Initial contact, peak knee flexion and take-off are indicated as IC, PKF and TO, respectively. Pre-fatigue and post-fatigue are indicated in grey and red, respectively. The arrows indicate the direction of time.

Discussion

1. Synthesis of the Results

This is the first study that explored fatigue-induced biomechanical risk factors for PT when performing a spike jump-landing task in a small number of male volleyball players. Contrary to what we expected, changes to patellar tendon loading with fatigue did not increase the risk for PT. Nevertheless, we found that players with less hip flexion after fatigue were at higher risk for developing PT and consequently found that an elongated rectus femoris MTU also increased the risk.

Fatigue-induced alterations to patellar tendon loading were not predictive for PT in this study. It is currently unclear whether this is a true observation, or whether this may have been influenced by other factors (e.g., calculation method, low sample size, competing risks, compensations in the kinematic chain). As such, patellar tendon loading was calculated based on sagittal plane knee kinematics and kinetics solely, potentially neglecting additional rotational forces or muscular co-contractions.[25,26] Moreover, a closer look at the actual patellar tendon loading values showed that fatigue decreased patellar tendon loads in the control groups, while these loads did not reduce in the PT experimental group (Appendix 2-4). These observations are in line with our study hypothesis but may not have proved significant due to the low sample size or due the large number of competing risks in this study. Indeed, higher patellar tendon loads were observed in the competing risk group compared to the injury-free group, which may have increased the averaged patellar tendon loading for the total control group (Appendix 4). Future studies should again consider examining fatigue-induced patellar tendon loading alterations, possibly calculated with advanced biomechanical models, in larger sample sizes.

Fatigue-induced less hip flexion during landing increased the risk for developing PT. Previous research also found that less hip flexion is associated with current symptoms of PT and can even predict its presence and severity.[12,27] Landing with less hip flexion is suggested to increase tensile loads acting on the patellar tendon due to a posterior location of the body's centre of mass.[27] However, patellar tendon loading was not predictive for PT in this study. This might be explained by the fact that more pelvis-trunk flexion was also found both before and after fatigue in the PT group (Appendix 2-3), which could negate the posterior location of the centre of mass.[11] In fact, more pelvis-trunk flexion during landing has been shown to re-shift the body's centre of mass and resultant ground reaction force vector more anteriorly relative to the knee joint, potentially reducing external knee joint moments and subsequent patellar tendon loading.[11] This proximal compensation strategy appeared to not fully eliminate the risk of developing PT, as less hip flexion (with similar knee flexion angles) during landing may have proximally elongated the only bi-articular MTU of the quadriceps, that is the rectus femoris, and this was also found a predictor for PT in this study.

Contractions of the rectus femoris MTU from a more elongated configuration after fatigue increased the risk for developing PT. This is a clinically relevant finding as it has been stated that the majority of the patellar tendon fibres originate from the central fibres of the quadriceps tendon, that is, from the rectus femoris, which extend over the anterior surface of the patella.[12] Up to this day, the impact of such suboptimal contraction dynamics on PT injury risk remains very much hypothetical. Two hypotheses are explored here, which are based upon the assumption that the test conditions in this study were representative of match and training conditions.[18] Hypothesis 1 assumes that the rectus femoris contractile (muscle fibres) and elastic elements (tendon and aponeurosis) act as one rigid entity.[12] This implies that length changes to the entire rectus femoris MTU may also increase elongation (strain) within the patellar tendon up to values near its peak length. High levels of tendon strain are associated with histopathological deterioration of the collagenous network due to the accumulation of micro-trauma and these changes typically occur at the proximal patellar tendon region.[7] Confirming this hypothesis, rectus femoris MTU length at peak knee flexion was very close to its peak length (Appendix 2-3), suggesting that this could also be the case for the patellar tendon. As an argument against this hypothesis, increased knee flexion after fatigue was not found to be predictive for PT, given that the amount of knee flexion has previously been associated with the amount of patellar tendon strain.[10] Moreover, relatively small differences in MTU length changes were observed between the injured and control group in the present study (Table 2). Hypothesis 2 assumes that, to optimally store and return elastic strain energy, elastic elements should not act too stiff, nor too compliant relative to the force capacity of the contractile element, also called as MTU tuning.[20] Such imbalance between muscle force capacity and tendon stiffness has already been suggested to increase tendon strain in the proximal patellar tendon region.[7] It is then hypothesized that the observed rectus femoris MTU lengthening may impede optimal MTU tuning. To give more insight into this matter, we exploratively plotted the joint work contribution relative to the overall joint work before vs. after fatigue for the injured and control group (Appendix 5). During landing, no meaningful differences in joint work distribution with fatigue were observed between both groups. However, during push-off, players with PT demonstrated a 5% relatively greater decrease in hip joint work and a 4% relatively greater increase in knee joint work after fatigue compared to controls. This disproportionate shift in energy distribution between the hip and knee joint during push-off may reflect changes in elastic energy storage and release of the rectus femoris MTU. More hip flexion during the initial landing phase might, therefore, bring the gluteal musculature in a more optimal configuration that would consequently allow for better energy release at the level of the hip. This may substantially decrease the demands of the rectus femoris MTU at the level of the knee during push-off. Future studies should explore such hypotheses to provide more insight into how fatigue-induced suboptimal contraction dynamics of the rectus femoris MTU may increase PT injury risk.

Although knee flexion did not increase the risk for PT in this study, both hip and knee motion after fatigue still seem important to consider when determining fatigue-induced PT injury risk due to the bi-articular function of the rectus femoris. We assumed that decreased hip flexion and (to a lesser extent) increased knee flexion after fatigue may increase rectus femoris MTU lengthening and subsequent PT injury risk. In an attempt to develop PT injury risk profiles, we divided players into four quadrants with colour codes (green → yellow → orange → red) corresponding to increased injury risk based on their changes in both hip and knee flexion after fatigue (Figure 3). Decreases in hip flexion were considered more decisive for developing PT than increases in knee flexion as these better predicted rectus femoris MTU length increases (hip: r = -0.48, knee: r = 0.39). We also indicated the threshold for peak hip flexion decreases of >3.5° that may precipitate PT with a red line. To confirm the utility of this risk profiling, we indicated those players that developed PT throughout study follow-up (red dots). As expected, the majority of players with PT were situated in the orange and red quadrants, and above the threshold of >3.5° peak hip flexion decrease. Nine players with PT decreased hip flexion, and two of them even increased knee flexion after fatigue, which may elongate the rectus femoris MTU both proximally and distally. One player that developed PT, however, was labelled as biomechanically 'safe' as he was situated in the green quadrant. This player may have been more at risk due to other, non-biomechanical factors (i.e., high body mass, history of patellar tendon complaints).[6,28] Due to the multifactorial nature of PT, future prospective studies are needed to confirm the predictive value of biomechanical markers in interaction with other markers of injury for PT on multiple large cohorts.[29]

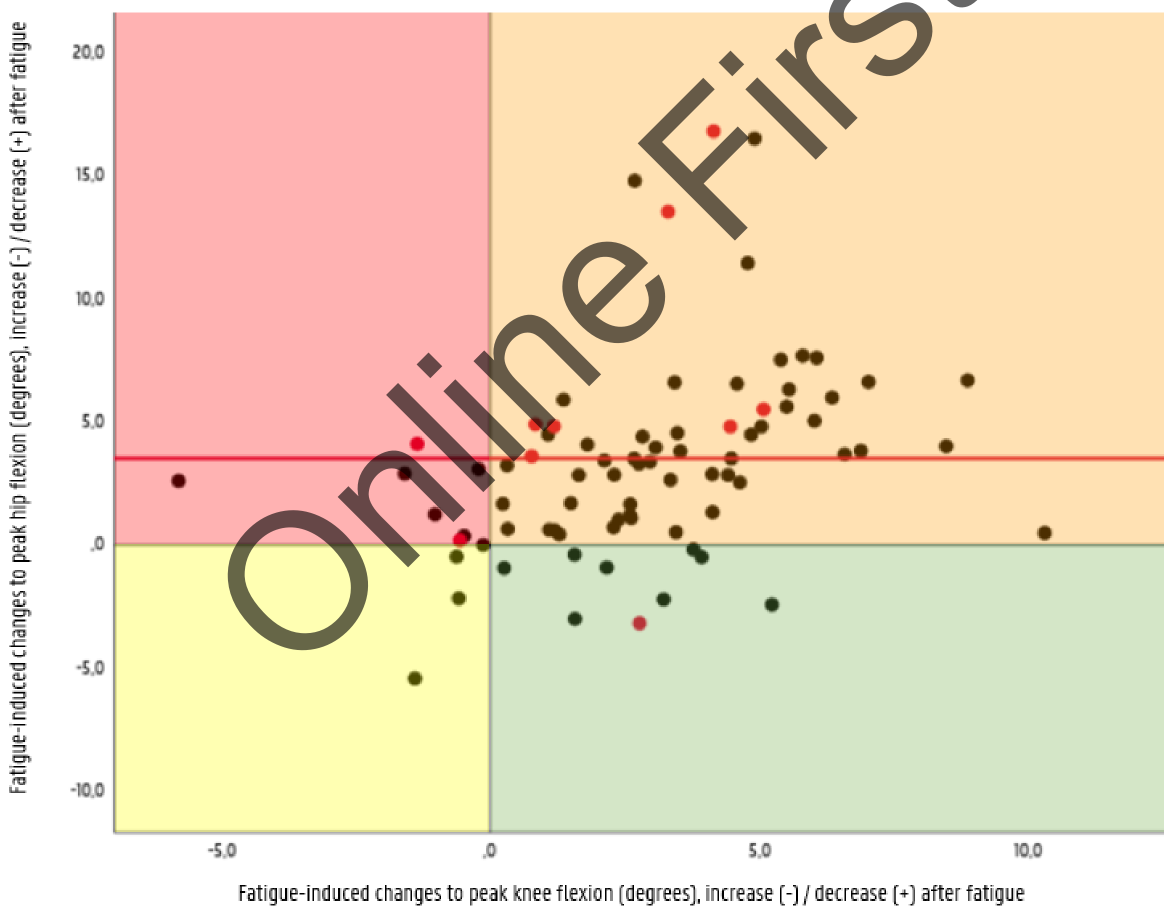
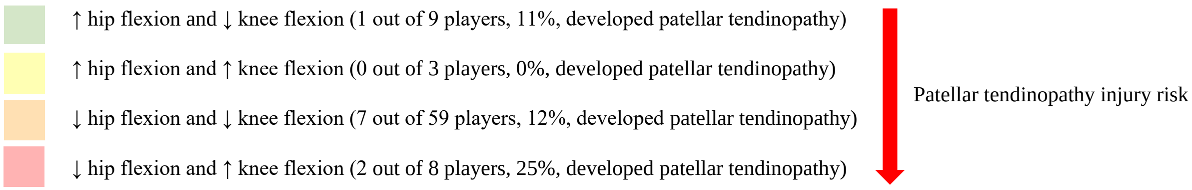


Figure 3. Fatigue-Induced Biomechanical Patellar Tendinopathy Injury Risk Profiles.

Players who did not develop patellar tendinopathy during follow-up are presented with black dots (n = 69), those who developed patellar tendinopathy are indicated with red dots (n = 10). Players were divided into four quadrants with colour codes based on their change (decrease or increase) in hip and knee flexion after fatigue. Rectus femoris MTU lengthening and subsequent patellar tendinopathy injury risk increased according to the colour of the quadrant (green → yellow → orange → red). The threshold for peak hip flexion decreases of >3.5° that may precipitate patellar tendinopathy is indicated with a red line.



2. Limitations and Research Implications

First, this study did not conduct pre-season VISA-P questionnaires, nor clinical and/or ultrasonographic examinations, implying that asymptomatic pathological tendons may have been included and structural/functional changes from baseline could not be sufficiently monitored during follow-up. Second, multivariate Cox regression analysis was not appropriate due to an insufficient number of players who developed PT during study follow-up (n = 10), which was far below the expected 26 injuries as estimated from previous prospective injury surveillance.[2] A closer examination on this revealed that the prevalence of previous patellar tendon complaints was 3.3 times lower in our study population compared to the population on which the above estimation was based (i.e., 22.8% vs. 75.0%, respectively),[2] which may have resulted in a lower initial risk for our athletes.[28] Next to the lack of multivariate analyses in our study, a high number of biomechanical risk factors were measured without correcting the level of α. Consequently, the results of this study remain largely explorative and need to be confirmed in future studies with larger numbers of players. Moreover, we only included male participants which is why the study results cannot simply be extrapolated to the female population. Third, patellar tendon loading, as calculated in this study, may potentially underestimate true tendon loading.[25,26] In line with this, MTU length changes, as calculated in this study, may include intra- and/or inter-subject anatomical/anthropometric variations, and may even not accurately reflect in vivo 3D athletic muscle function.[21] In this context, patellar tendon strain could not be calculated since patellar motion is unmeasurable using skin markers. Future studies could simultaneously determine muscle fibre length through ultrasonography to derive tendon length. Strain gauges (e.g., shear wave tensiometers) may also have the capacity to measure tendon strain in a more direct way.[30] Finally, the high variability in reported duration from pre-season screening to PT symptom onset implies that cumulative fatigue and/or biomechanical adaptations may have influenced symptom development throughout the season. Supplemental biomechanical assessments at critical time points during the season seem crucial, for example after mid-season breaks or in the later stages of the season when levels of physical fitness may well be different and/or accumulated fatigue/loading occurs.[31,32]

3. Clinical Implications

Considering the explorative nature of this study, we can only give some preliminary clinical recommendations. The observation of fatigue-induced jump-landing biomechanics made it possible to more accurately identify those volleyball players at risk for PT (Appendix 2-3). All risk factors that were significant pre-fatigue became stronger predictors post-fatigue (i.e., rectus femoris MTU length at initial contact and pelvis-trunk flexion at initial contact/peak pelvis-trunk flexion/peak knee flexion/take-off). Moreover, the fatigue protocol revealed additional risk factors post-fatigue that were not significant pre-fatigue (i.e., rectus femoris MTU length at peak length/peak knee flexion/take-off and hip flexion at initial contact/peak hip flexion). This adds evidence that volleyball players should additionally be screened under fatigued conditions when investigating PT injury risk, aligning with previous screening recommendations for other lower extremity overuse injuries like exertional medial tibial pain (EMTP), where fatigue has also been shown to make differences more apparent by decreasing neuromuscular function.[14] Screenings for PT injury risk should predominantly focus on detecting an adverse decline in hip movement strategies (i.e., less hip flexion during horizontal landing/push-off) after fatigue as it may impede optimal rectus femoris MTU function. Especially those players that demonstrate fatigue-induced hip flexion decreases of >5.9° and >3.5° at initial contact and at peak, respectively, might be closely monitored throughout the entire season and may benefit from participating in customized injury prevention programs. Such injury prevention programs may incorporate hip-specific exercises (e.g., improving strength/fatigue resistance of the gluteal muscles) and/or technique modifications (e.g., more hip flexion during landing) under fatigued circumstances.[12,33] Future studies should explore the effectiveness of such interventions in populations at risk for PT.

Conclusion

This is the first explorative prospective study to investigate fatigue-induced biomechanical risk factors for PT during a spike jump-landing in volleyball. Despite the low (injured) sample size of this study, inclusion of a fatigue protocol enhanced the identification of risk factors, with less hip flexion during landing and more elongated rectus femoris MTUs after fatigue emerging as preliminary contributors to PT development. Assessment and training of these risk factors are thought to be essential for reducing PT injury incidence in the future.

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Appendix 1. Calculation of Patellar Tendon Loading (Force) and Quadriceps Muscle-Tendon Unit Lengths.

Patellar tendon loading (force) was computed by dividing the net sagittal plane knee joint moment (normalized for body mass) by the patellar tendon moment arm length, which was predicted based on Herzog & Read as follows:

Patellar tendon moment arm length (cm) $= 4.71 + (0.042 \times \beta) + ((-0.896 \times 10^{-3}) \times \beta^2) + ((0.447 \times 10^{-5}) \times \beta^3)$.

In this equation, β represents the knee joint angle in the sagittal plane, measured in degrees.[19]

Quadriceps muscle-tendon unit (MTU) lengths were calculated and normalized for thigh length according to the following regression equations of Hawkins & Hull:

- *Rectus femoris MTU length (x times thigh length)* $= 1.107 + ((-1.50 \times 10^{-3}) \times \alpha) + ((1.99 \times 10^{-3}) \times \beta)$
- *Vastus intermedius MTU length (x times thigh length)* $= 0.496 + ((3.88 \times 10^{-3}) \times \beta) + ((-1.63 \times 10^{-5}) \times \beta^2)$
- *Vastus lateralis MTU length (x times thigh length)* $= 0.569 + ((4.06 \times 10^{-3}) \times \beta) + ((-2.07 \times 10^{-5}) \times \beta^2)$
- *Vastus medialis MTU length (x times thigh length)* $= 0.489 + ((3.07 \times 10^{-3}) \times \beta) + ((-1.53 \times 10^{-5}) \times \beta^2)$

In these equations, α and β represent the hip and knee joint angles in the sagittal plane, measured in degrees, respectively.[21]

Appendix 2. Pre-Fatigue Biomechanical Predictors for Patellar Tendinopathy.

Variables	Control (n = 69)	PT (n = 10)	*p*-value*	Hazard ratio with 95% CI	Hedge's *g* effect size
Primary outcome variable					
Patellar tendon loading (x times body weight)	7.24 ± 1.08	7.55 ± 0.69	0.538	1.262 (0.597-2.669)	0.292
Secondary/exploratory outcome variables					
Pelvis-trunk flexion (degrees), extension (-) / flexion (+)					
Initial contact	**21.88 ± 11.90**	**33.36 ± 15.75**	**0.025**	**1.058 (1.007-1.112)**	**0.916**

Peak pelvis-trunk flexion	**24.47 ± 11.17**	**35.58 ± 16.08**	**0.023**	**1.063 (1.008-1.122)**	**0.929**
Peak knee flexion	**16.25 ± 11.38**	**26.77 ± 17.91**	**0.049**	**1.054 (0.997-1.115)**	**0.846**
Take-off	**-9.64 ± 11.46**	**1.58 ± 15.06**	**0.017**	**1.073 (1.006-1.145)**	**0.930**
Hip flexion (degrees), flexion (-) / extension (+)					
Initial contact	-54.09 ± 10.80	-46.22 ± 15.19	0.091	1.045 (0.990-1.104)	0.684
Peak hip flexion	-70.22 ± 11.28	-62.27 ± 15.09	0.108	1.041 (0.989-1.097)	0.667
Peak knee flexion	-48.12 ± 11.01	-44.21 ± 15.11	0.631	1.013 (0.961-1.067)	0.335
Take-off	-0.35 ± 9.13	5.63 ± 14.19	0.103	1.054 (0.986-1.126)	0.601
Knee flexion (degrees), flexion (-) / extension (+)					
Initial contact	-25.57 ± 6.45	-26.92 ± 5.42	0.328	0.951 (0.862-1.050)	0.211
Peak / Peak knee flexion	-82.44 ± 7.63	-85.06 ± 6.76	0.322	0.960 (0.888-1.038)	0.345
Take-off	-10.21 ± 5.24	-10.85 ± 5.20	0.896	0.992 (0.884-1.114)	0.122
Ankle dorsiflexion (degrees), dorsiflexion (-) / plantarflexion (+)					
Initial contact	-77.43 ± 6.55	-75.62 ± 6.60	0.469	1.034 (0.950-1.126)	0.273
Peak ankle dorsiflexion	-86.78 ± 5.88	-88.33 ± 6.04	0.285	0.928 (0.804-1.071)	0.261
Peak knee flexion	-85.72 ± 5.95	-87.24 ± 6.11	0.345	0.938 (0.818-1.077)	0.253
Take-off	-31.97 ± 5.49	-32.36 ± 3.73	0.638	0.969 (0.851-1.104)	0.071
Rectus femoris MTU length (% thigh length), ↘ (-) / ↗ (+)					
Initial contact	**107.68 ± 1.47**	**109.12 ± 2.67**	**0.012**	**1.590 (1.094-2.312)**	**0.865**
Peak length	120.37 ± 1.58	121.47 ± 1.99	0.081	1.536 (0.934-2.526)	0.670
Peak knee flexion	119.89 ± 1.55	121.00 ± 2.02	0.072	1.558 (0.958-2.533)	0.681
Take-off	112.68 ± 1.56	113.70 ± 2.00	0.088	1.425 (0.944-2.152)	0.628
Vastus intermedius MTU length (% thigh length), ↘ (-) / ↗ (+)					
Initial contact	0.58 ± 0.02	0.59 ± 0.02	0.313	1.189 (0.851-1.660)	0.224
Peak length	0.70 ± 0.01	0.71 ± 0.01	0.269	1.553 (0.718-3.359)	0.364
Peak knee flexion	0.70 ± 0.01	0.71 ± 0.01	0.266	1.556 (0.721-3.361)	0.367
Take-off	0.53 ± 0.02	0.54 ± 0.02	0.892	1.023 (0.736-1.423)	0.123
Vastus lateralis MTU length (% thigh length), ↘ (-) / ↗ (+)					
Initial contact	0.66 ± 0.02	0.66 ± 0.02	0.309	1.195 (0.849-1.682)	0.227
Peak length	0.76 ± 0.00	0.76 ± 0.00	0.211	3.012 (0.501-18.107)	0.378
Peak knee flexion	0.76 ± 0.00	0.76 ± 0.00	0.199	3.133 (0.509-19.271)	0.383
Take-off	0.61 ± 0.02	0.61 ± 0.02	0.890	1.023 (0.741-1.412)	0.123
Vastus medialis MTU length (% thigh length), ↘ (-) / ↗ (+)					
Initial contact	0.56 ± 0.01	0.56 ± 0.01	0.309	1.263 (0.807-1.977)	0.227
Peak length	0.64 ± 0.00	0.64 ± 0.00	0.218	3.467 (0.455-26.383)	0.377
Peak knee flexion	0.64 ± 0.00	0.64 ± 0.00	0.212	3.520 (0.461-26.870)	0.380
Take-off	0.52 ± 0.01	0.52 ± 0.01	0.890	1.030 (0.673-1.577)	0.123

The control group consisted of 35 injury-free players and 34 players with competing risks. Values are expressed as mean ± SD. Significant predictors are highlighted in bold. * Unadjusted univariate Cox regression with competing risks.

Appendix 3. Post-Fatigue Biomechanical Predictors for Patellar Tendinopathy.

Variables	Control (n = 69)	PT (n = 10)	*p*-value*	Hazard ratio with 95% CI	Hedge's *g* effect size
Primary outcome variable					
Patellar tendon loading (x times body weight)	6.99 ± 0.95	7.58 ± 0.56	0.138	1.741 (0.838-3.621)	0.639
Secondary/exploratory outcome variables					
Pelvis-trunk flexion (degrees), extension (-) / flexion (+)					
Initial contact	**20.76 ± 11.51**	**33.88 ± 14.73**	**0.006**	**1.075 (1.020-1.133)**	**1.089**
Peak pelvis-trunk flexion	**24.56 ± 10.51**	**37.87 ± 13.54**	**0.003**	**1.092 (1.029-1.159)**	**1.208**
Peak knee flexion	**17.86 ± 10.93**	**30.56 ± 14.64**	**0.007**	**1.085 (1.016-1.159)**	**1.100**
Take-off	**-8.56 ± 11.05**	**3.40 ± 11.34**	**0.005**	**1.092 (1.020-1.168)**	**1.045**
Hip flexion (degrees), flexion (-) / extension (+)					
Initial contact	**-49.87 ± 10.30**	**-38.32 ± 12.49**	**0.004**	**1.093 (1.021-1.171)**	**1.081**
Peak hip flexion	**-67.19 ± 10.58**	**-56.77 ± 11.20**	**0.008**	**1.085 (1.014-1.160)**	**0.969**
Peak knee flexion	-45.57 ± 9.96	-39.85 ± 10.15	0.205	1.041 (0.975-1.112)	0.568
Take-off	0.53 ± 8.80	7.55 ± 11.33	0.054	1.071 (0.993-1.155)	0.762
Knee flexion (degrees), flexion (-) / extension (+)					
Initial contact	-21.40 ± 6.26	-21.14 ± 3.88	0.893	0.993 (0.897-1.100)	0.042
Peak / Peak knee flexion	-79.42 ± 7.58	-82.99 ± 6.37	0.233	0.954 (0.887-1.026)	0.475
Take-off	-10.02 ± 5.13	-11.33 ± 6.46	0.522	0.966 (0.872-1.071)	0.244
Ankle dorsiflexion (degrees), dorsiflexion (-) / plantarflexion (+)					
Initial contact	-76.48 ± 6.41	-75.31 ± 6.45	0.686	1.021 (0.926-1.126)	0.182
Peak ankle dorsiflexion	-85.01 ± 5.99	-87.71 ± 4.26	0.115	0.890 (0.763-1.039)	0.460
Peak knee flexion	-83.87 ± 6.01	-86.59 ± 4.62	0.159	0.911 (0.794-1.045)	0.458
Take-off	-31.32 ± 5.74	-31.68 ± 4.20	0.572	0.965 (0.853-1.092)	0.063
Rectus femoris MTU length (% thigh length), ↘ (-) / ↗ (+)					
Initial contact	**107.48 ± 1.50**	**109.16 ± 2.12**	**0.002**	**1.897 (1.235-2.916)**	**1.048**
Peak length	**120.26 ± 1.47**	**121.75 ± 1.65**	**0.008**	**2.010 (1.185-3.411)**	**0.994**
Peak knee flexion	**119.67 ± 1.42**	**121.24 ± 1.70**	**0.003**	**2.100 (1.294-3.407)**	**1.069**
Take-off	**112.77 ± 1.47**	**114.09 ± 1.93**	**0.018**	**1.669 (1.082-2.575)**	**0.851**
Vastus intermedius MTU length (% thigh length), ↘ (-) / ↗ (+)					
Initial contact	0.57 ± 0.02	0.57 ± 0.01	0.847	1.033 (0.746-1.430)	0.022
Peak length	0.70 ± 0.01	0.71 ± 0.01	0.167	1.668 (0.821-3.388)	0.492
Peak knee flexion	0.70 ± 0.01	0.71 ± 0.01	0.165	1.673 (0.823-3.397)	0.495
Take-off	0.53 ± 0.02	0.54 ± 0.02	0.531	1.101 (0.816-1.486)	0.234
Vastus lateralis MTU length (% thigh length), ↘ (-) / ↗ (+)					
Initial contact	0.65 ± 0.02	0.65 ± 0.01	0.834	1.036 (0.746-1.437)	0.017
Peak length	0.76 ± 0.01	0.76 ± 0.00	0.113	3.396 (0.698-16.535)	0.496
Peak knee flexion	0.76 ± 0.01	0.76 ± 0.00	0.102	3.634 (0.710-18.594)	0.500
Take-off	0.61 ± 0.02	0.61 ± 0.02	0.533	1.099 (0.819-1.474)	0.231
Vastus medialis MTU length (% thigh length), ↘ (-) / ↗ (+)					
Initial contact	0.55 ± 0.01	0.55 ± 0.01	0.836	1.047 (0.681-1.610)	0.017
Peak length	0.64 ± 0.00	0.64 ± 0.00	0.119	4.061 (0.659-25.025)	0.498
Peak knee flexion	0.64 ± 0.00	0.64 ± 0.00	0.112	4.223 (0.668-26.715)	0.501
Take-off	0.52 ± 0.01	0.52 ± 0.02	0.533	1.132 (0.769-1.669)	0.231

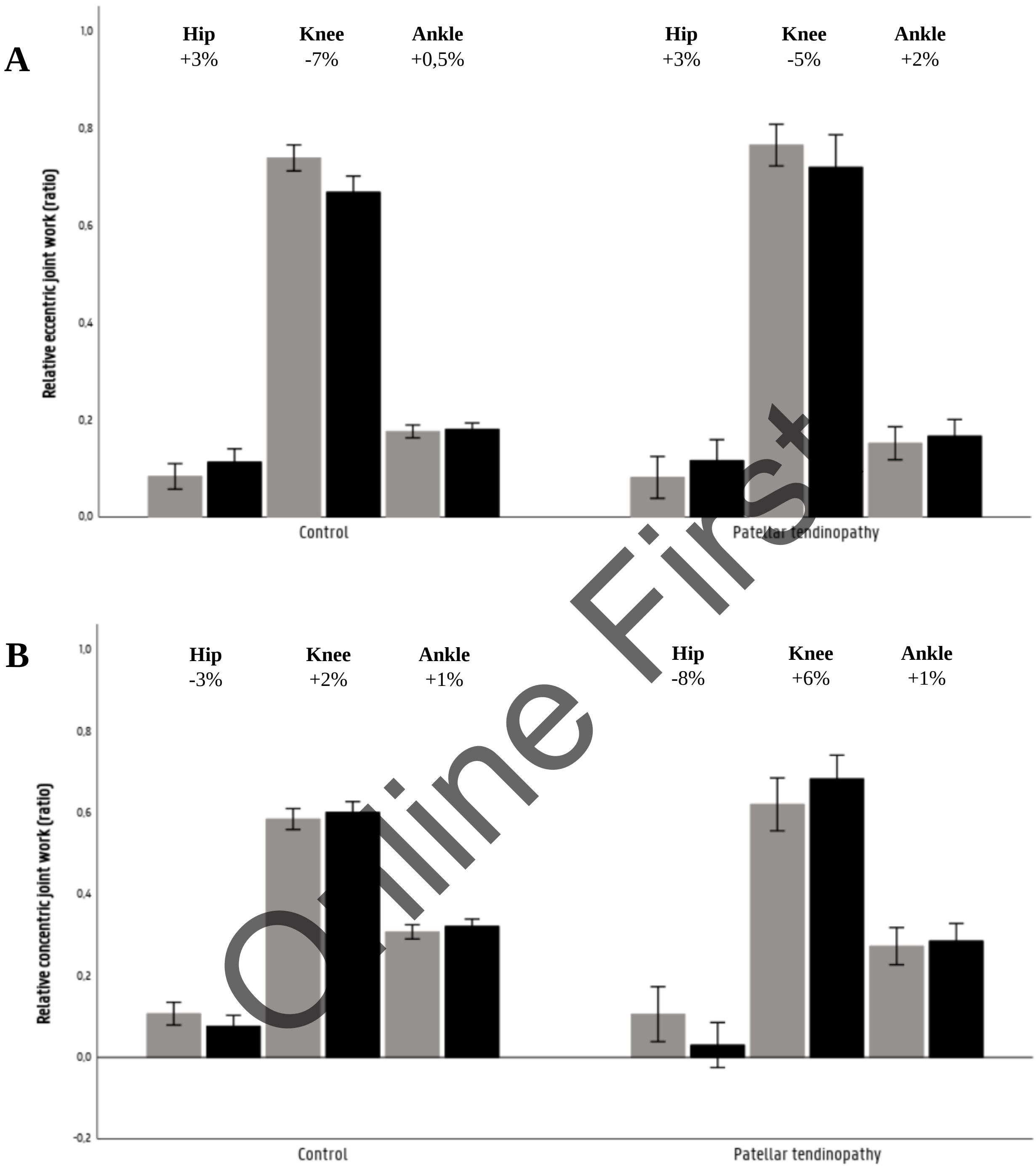
The control group consisted of 35 injury-free players and 34 players with competing risks. Values are expressed as mean ± SD. Significant predictors are highlighted in bold. * Unadjusted univariate Cox regression with competing risks.

Appendix 4. Additional Explorative Analysis on Patellar Tendon Loading.

Peak patellar tendon loading	Injury-free (n = 35)	Competing risks (n = 34)	Competing risks * (n = 24)	Patellar tendinopathy
Pre-fatigue (x times body weight)	7.12 ± 1.15	7.37 ± 0.99	7.45 ± 1.06	7.55 ± 0.69
Post-fatigue (x times body weight)	6.75 ± 0.99	7.24 ± 0.86	7.45 ± 0.86	7.58 ± 0.56
Average pre- and post-fatigue (x times body weight)	6.93 ± 1.05	7.31 ± 0.88	7.45 ± 0.92	7.57 ± 0.59
Delta Post – Pre (x times body weight), ↘ (-) / ↗ (+) after fatigue	-0.37 ± 0.45	-0.13 ± 0.60	-0.00 ± 0.58	+0.03 ± 0.44

* Other knee extensor mechanism issues (i.e., patellar tendon complaints that did not meet the inclusion criteria, quadriceps muscular complaints and medial/lateral knee joint pain) were excluded from the competing risk group to explore if these issues show injury mechanisms similar to the patellar tendinopathy experimental group. As excluding them increased (rather than decreased) patellar tendon loads in the competing risk group, we assume that these issues have different injury mechanisms than those observed in players who were assigned to the patellar tendinopathy experimental group.

Appendix 5. Relative Joint Work Before and After the HIIP-5 (mean and 95% CI).



Eccentric (negative, from initial contact to peak knee flexion) and concentric (positive, from peak knee flexion to take-off) joint work was extracted by integrating the joint power curve. Overall joint work was calculated by the sum of the hip, knee and ankle joint work, and for each joint, the relative contribution to the overall joint work (ratio) was calculated.

A: relative eccentric joint work (landing phase), B: relative concentric joint work (push-off phase). Pre-HIIP-5 = grey bar; post-HIIP-5 = black bar. The percentage change post- vs. pre-fatigue is reported for each joint in both groups.