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Published in:
International Journal of Sports Medicine

DOI (link to publication from Publisher):
10.1055/a-2018-2781

Publication date:
2023

Document Version
Accepted author manuscript, peer reviewed version

Published: Int J Sports Med . 2023 Dec;44(13):995-1002. doi: 10.1055/a-2018-2781.

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Sebastian Klich[1], Kamil Michalik[2], Jolanta Rajca[3], Krzysztof Ficek[3,4], Cesar Fernández-de-las-Peñas[5,6], Adam Kawczyński[1], Pascal Madeleine[7]

[1] Department of Paralympic Sport, Wrocław University of Health and Sport Science, Wrocław, Poland; [2] Department of Human Motor Skills, Wrocław University of Health and Sport Science, Wrocław, Poland; [3] Galen Orthopedic Center, Bieruń, Poland; [4] Department of Physiotherapy, Academy of Physical Education in Katowice, Katowice, Poland; [5] Department of Physical Therapy, Occupational Therapy, Rehabilitation and Physical Medicine, Universidad Rey Juan Carlos, Alcorcón, Spain; [6] Cátedra Institucional en Docencia, Clínica e Investigación en Fisioterapia: Terapia Manual, Punción Seca y Ejercicio Terapéutico, Universidad Rey Juan Carlos, Alcorcón, Spain; [7] Aalborg University, Department of Health Science and Technology, Sport Sciences – Performance and Technology, Aalborg, Denmark

Published: Int J Sports Med . 2023 Dec;44(13):995-1002. doi: 10.1055/a-2018-2781.

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Abstract

The study aims to investigate morphological and mechanical properties and echogenicity of the patellar tendon (PT) after acute fatigue-induced alterations in sprint track cyclists. Fourteen elite track cyclists participated in this study. The exercise protocol consisted of three (on a distance of 62.5 m), one (125 m and 250 m) maximal start accelerations, and sprints from the standing start. Immediately after testing all measurements, PT stiffness and thickness were set at 5-10-15-20 mm distal from the apex of the patella and 5-10 mm proximal to the tibial tuberosity. CSA was set at proximal, middle, and distal, while echogenicity was at proximal and distal points. The results showed significant increases in PT stiffness at all reference points after start acceleration ($p<0.001$). PT thickness showed similar results as for stiffness, however except for location placed at TT-5 ($p<0.001$). CSA increased significantly in proximal, middle, and distal regions ($p<0.001$), while echogenicity of tendon increased in proximal and distal regions ($p<0.001$) after start acceleration. Regional-dependent alterations of PT thickness and stiffness may be related to anatomical and physiological mechanisms due to acute isometric contraction in the initial phase of standing start. Tendon echogenicity might be also useful in monitoring tendon mechanical properties and defining acute fatigue-induced changes.

Keywords: stiffness, thickness, overloading, ultrasonography, myotonometry

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Introduction

Track cycling standing start from a starting gate is characteristic of team sprint, 500m and 1000m time trial (TT) during sprint competition. Also, individual 3000m and 4000m TT or team pursuit required an "all-out" start. It is commonly known that peak power output (PPO) is one of the most important factors and a strong predictor of maximum velocity and tracks cycling performance [1, 2]. The PPO is related to body mass and correlated with the acceleration phase [1]. The training of standing start in sprint track cyclists focuses on developing PPO at maximal intensity during short-duration tasks <7 seconds [3]. On the other hand, this type of training is strenuous for lower limbs and may lead to overuse syndrome in lower limb muscles and/or tendons [4].

The patellar tendon (PT) overloading might lead to tendonitis and finally cause tendinopathy in cyclists [4], as a result of excessive angular traction during the pedal cycle, start acceleration with a bigger gear, and quadriceps isometric contraction during the deceleration phase [5]. Pain in PT is reported in approximately 25% of cases of injury in cyclists [6] and is felt directly under the inferior pole of the patella during knee extension [5]. Morphological alterations in PT are related to reparative overloading mechanisms due to tensile loads and compression mechanisms. Anatomically, the compression is caused by an impingement of the inferior patellar pole and the proximal posterior surface of PT [7].

The morphological properties of PT might be investigated using ultrasonographic imaging [4, 8]. A normal PT is characterized as an echoic fibrillar structure, however, a PT with tendinopathy may lead to disruption in fibrillar structure and hypoechogenicity [9]. Previous studies have evaluated PT by measuring thickness, cross-sectional area (CSA), and echogenicity. The thickness of PT has been assessed at a single location – 10

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mm inferior to the patellar pole [10-14]. However, multiple locations have also been analyzed [4, 14-16]. Holm et al. [14] and Toprak et al. [15] measured PT thickness at 6 mm and 10 mm proximal to the patellar pole and tibial tuberosity, while Klich et al. [4, 8] evaluated PT thickness at 5-10-15-20 mm inferior to the apex of the patella. Castro et al. [16] adjusted the measurement to the length of PT (at 25 %, 50 %, and 75 %). The CSA has mostly been examined at proximal, medial, and distal points from the apex of the patella [14, 17-19], and at a single point located at 10 mm inferior from the patella pole [12, 20]. Additionally, tendon echogenicity analysis depends on thickness assessment [21]. The PT thickness is important to monitor to reveal early-stage overloading and micro-injuries in conjunction with thickness assessments [4].

The current body of literature is limited to a few studies conducted in-field conditions during specialized training on the velodrome and/or competition [4, 22, 23]. Real-time analysis of changes in tendon morphology may influence the optimization of training and competition loads. Furthermore, this current study may also provide a better understanding of possible overloading mechanisms in PT. The main goal of this current study was to investigate the thickness and stiffness of PT after standing start accelerations. The second goal was to describe acute fatigue-induced regional-dependent alterations in morphological, mechanical properties, and echogenicity of PT in track cyclists specialized in sprint events.

Materials & Methods

This study was conducted to evaluate morphological (thickness and CSA) and mechanical (stiffness) alterations in PT properties before and after repeated standing start acceleration exercises. The morphological alterations were based on the measurement of thickness using ultrasonography imaging, stiffness assessed by myotonometry, and echogenicity

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using computer-assisted grey-scale analysis. Measurements were made in the same order starting with (1) myotonometry including stiffness of PT, (2) ultrasonography of PT including thickness and CSA measurement, and (3) PT echogenicity. The total time of preparing all measurements took approximately 5 min per subject. The study was conducted following STROBE guidelines [24]. All measurements were taken on the right (dominant side) lower extremity.

An eligible group of 14 elite track cyclists who specialized in sprint events participated in this study. All description of participant demographics, training experience, and duration is included in Table 1. The thigh length was defined as the distance from the anterior superior iliac spine to the medial joint line [4]. All subjects were tested to identify the dominant leg during pedaling [25] and identified as being right leg dominant. The track cyclists were competitors of the Polish national team and specialized in sprint events with a mean training experience of 9±3 years (mean±SD). The exclusion criteria included: (1) current or previous thigh and knee injury or symptoms; and (2) prior history of surgery in the lower extremity. Each subject read and signed an informed consent form approved by the Senate Research Ethics Committee (project identification code: XX/XXXX approval date: XX.XX.XXXX). The study was conducted according to the Declaration of Helsinki.

The G*Power software (version 3.1.9.2; Kiel University, Kiel, Germany) was used to estimate the required sample size. We calculated the power (1-β) for repeated measure ANOVA within factors, by defining the sample size as 13 (for all ultrasound and myotonometric measurements), set a minimum expected effect size (Cohen's *f*) of 0.5, an α level of 0.05, and a power of 0.95 and correlation for repeated measures of 0.6.

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The experimental procedure took two weeks because two groups of seven track cyclists participated in the data collection. These subjects had two days of rest before the main experiment and data collection. They were also asked to avoid strenuous physical activity two days before participating in the study.

After baseline measurements, all subjects took part in a standard 15-minute track cycling warm-up consisting of 5 km with moderate intensity riding around the velodrome (heart rate range over 150 to 165 bpm) and gear ratio 48:16 (chain ring:cog). The track used for the experimental procedure was an indoor 250 m length velodrome with a wood surface, with bands inclination at 42^{o} and straight inclination at 12^{o}. Moreover, this velodrome is approved by the Union Cycliste Internationale (UCI) to organize international track cycling events. After this part of the warm-up, cyclists performed three start accelerations: one acceleration from the slow ride (on a distance of 62.5 m) followed by two standing starts from the gate (on a distance of 12.5 m) interspaced by 5-min of passive rest. The last part of the warm-up was dedicated to generating maximum sprint effort that lasted 6 s on the cycle ergometer (Wattbike Ltd, Nottingham, UK) [3]. After the warm-up, 15-min of rest was given to change the gear ratio (50/15). After the rest, a repeated sprint acceleration protocol was performed. The main goal of this cycling task was to provoke acute fatigue-induced alterations in PT by performing five repeated cycling exercise tasks at different distances on a track velodrome. The exercise protocol consisted of (1) three maximal start accelerations and sprints from the standing start at a distance of 62.5 m (1/4 lap), (2) one maximal start acceleration and sprints from the standing start at a distance of 125 m (1/2 lap), and (3) one maximal start accelerations and sprints from the standing start on distance 250 m (1 lap), with each repetition separated by 8-min of rest. Immediately after testing, all measurements were taken in the same order

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as baseline. During the data collection all track cyclists performed a cool-down exercise, i.e., a 15-min ride on the track at a cadence of ~90 rpm).

To determine the moment of inertia (I) for the bicycle and rider we used equation $I=mr^2$, where *m* is the combined mass of the rider (in full racing outfit) and bicycle mass of two additional tires and rims, and *r* is the outside radius of the bicycle tire. Inertial load (IL) was defined as $\frac{1}{2}IG^2$, where *I* is above described moment of inertia and *G* is the gear ratio front (50)/rear (15) for all riders [26].

A hand-held myotonometer device (MyotonPro, Myoton Ltd, Estonia) was used to measure the stiffness of PT on the dominant lower extremity (right) at six different locations set at 5-10-15-20 mm distal from the apex of the patella and 5-10 mm proximal to the tibial tuberosity (Figure 2). The probe was placed perpendicular to the tested area and generated three impulses exerted on the testing area. Participants were lying in a supine position with their knees flexed at 30° and their feet on the massage table [27]. A pillow was placed under the popliteal space during the assessments. Data collection was performed by two examinators, where the first marked reference points using a tape measure for anthropometric measurements, while the second examiner was responsible for measuring the stiffness of PT.

Ultrasonography was performed using an ultrasound scanner (HS-2200, Honda, Toyohashi, Japan) with a 7.5 (6.0 to 11.0) MHz and 40 mm linear array transducer (HLS-584 M, Honda, Toyohashi, Japan) in a greyscale B-mode. The settings of the ultrasound system were standardized for all participants and kept identical for all measures. The scan depth was set to 1.8 mm, according to [4, 8]. The axial resolution of ultrasound images was found to be 0.068 mm per pixel. Ultrasound imaging of PT thickness and cross-sectional area was performed according to recommendations of the European Society of

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Musculoskeletal Radiology [28]. Participants were placed in the same position for the measurement of PT stiffness. The dominant knee was positioned in this position to avoid possible anisotropy related to the concave profile as a result of posterior thigh muscles and PT extension [11]. The linear transducer was placed longitudinally, first distal from the patella and after proximal from the tibial tuberosity. The thickness of the PT was assessed in six locations, set at 5-10-15-20 mm distal from the apex of the patella, and 5-10 mm proximal from the tibial tuberosity (Figure 3A, B). Tendon borders were defined inferiorly as the first hyperechoic region between the subcutaneous tissue and the deep fascia layer. To evaluate CSA the linear transducer was placed perpendicular to the PT at three locations indicating anatomical points (proximal, middle, and distal), set at 10 mm (proximal) and 20 mm (middle) distal from the patella pole, as well as 10 mm (distal) proximal from the tibial tuberosity [14, 17].

The ultrasound data were then subjected to further analysis carried out with ImageJ, an open-source image enumeration software package (US National Institute of Health, Bethesda, MD, USA) [29]. The analysis of the area and echo intensity were carried out in two locations of the patellar tendon (measured from the apex of the patella and tibial tuberosity), in the right and left extremities, and in two-time points (before and after the sprint). Image data were converted to an 8-bit integer giving values ranging from 0 (black, no reflection) to 255 (white, substantial reflection). The measurements of area and echo intensity were performed in manually indicated regions of interest (ROI) in two locations of the patellar tendon. The vertical borders of ROI were indicated 5 and 20 mm from the apex of the patella (Fig. 3C), and 5 and 10 mm from the tibial tuberosity (Fig. 3D) (according to the methodology of tendon thickness measurements). Horizontal borders of ROI were manually drawn inferiorly as the first hyperechoic region between

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the subcutaneous tissue and the deep fascia layer (according to the methodology of tendon thickness measurements). The echo intensity (in grayscale 0-255) was automatically calculated in the defined ROI. Based on the ROI histogram the mean value was computed.

The SPSS statistical software (version 18., SPSS Inc., Chicago, Illinois, USA) was used for data analysis. Mean values ± standard deviation with confidence interval (CI 95%) were reported. The normality of the data distribution was applied through the Shapiro–Wilk tests, while homogeneity of variance was analyzed by Levene's test. The analyzed data were normally distributed for all parameters, while the variances for all parameters were equal. A two-way, repeated measure analysis of variance (RM-ANOVA) with *Time* (baseline and immediately after) and *Location* (for stiffness and thickness: PT-5, PT-10, PT-15, PT-20, TT-10, TT-5; CSA: proximal-medial-distal; and echogenicity: proximal-distal) was conducted. If a significant interaction between variables was found, the Bonferroni adjustment for multiple comparisons was used for post hoc tests ($p=0.01$). The effect size was estimated using partial eta square (η^2), classified as small ($.2<\eta^2<.49$), medium ($.5<\eta^2<.79$), or large ($\eta^2\leq.8$) [30]. Pearson's correlation test was applied to determine the relationships between mean power output per body weight and immediately post but also pre-post differences of stiffness, thickness, CSA, and echogenicity. The correlation coefficients were classified as trivial (0.0), small (0.1), moderate (0.3), strong (0.5), very strong (0.7), nearly perfect (0.9), and perfect (1.0) [31]. For all statistical tests, a p-value ≤ 0.05 was considered significant.

Results

Table 2 shows the mean±SD of PT stiffness at baseline and immediately after repeated start acceleration exercise. The two-way RM-ANOVA revealed a statistically significant

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main effect of *Time* ($F_{1,156}$=293.7, $p \leq 0.001$, η^2=0.65) and *Location* ($F_{3,156}$=8.9, $p \leq 0.001$, η^2=0.22). Moreover, the analysis showed an interaction effect between *Time* and *Location* ($F_{1,156}$=2.5, p =0.04, η^2=0.14) for PT stiffness. Post-hoc analysis showed significant increases in PT stiffness at all reference points (PT-5, PT-10, PT-15, PT-20, TT-10, TT-5) after start acceleration ($p \leq 0.001$).

The two-way RM-ANOVA revealed a statistically significant main effect of *Time* ($F_{1,84}$=152.1, $p \leq 0.001$, η^2=0.64) and *Location* ($F_{5,84}$=28.1, $p \leq 0.001$, η^2=0.63) for PT thickness. Moreover, a statistically significant interaction effect between *Time* and *Location* ($F_{5,84}$=6.2, $p \leq 0.001$, η^2=0.27) was found. Post-hoc analysis showed significant increases in PT thickness at all locations after start acceleration except TT-5 ($p<0.001$). Greater thicknesses were observed at PT-5, PT-10, and PT-20 after sprint acceleration, compared with PT-15, TT-10, and TT-5 ($p<0.01$ for all points). A significantly lower thickness was found at PT-15 after sprint acceleration, compared with PT-20 ($p<0.001$) (**Table 2**).

The two-way RM-ANOVA revealed a statistically significant main effect of *Time* ($F_{1,39}$=1051.1, $p \leq .0001$, η^2=0.96) and *Location* ($F_{2,39}$=152.9, $p \leq 0.001$, η^2=0.89) for PT CSA. Moreover, a statistically significant interaction effect between *Time* and *Location* ($F_{2,39}$=375.8, $p \leq 0.001$, η^2=0.95) was found. Post-hoc analysis showed significant increases in proximal, middle, and distal PT CSA after start acceleration ($p<0.001$). Greater CSA was observed at the location proximal to the apex patella after start acceleration, compared with the location medial to the apex patella after start acceleration ($p<0.001$). Lower CSA was also found distal to the apex patella after sprint acceleration, compared with the location placed proximal ($p=0.005$) and medial ($p<0.001$) to the apex patella after sprint acceleration (**Table 2**).

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The two-way RM-ANOVA revealed a statistically significant main effect of *Time* ($F_{1,26}$=86.9, p≤.0001, η^2=0.70) and *Location* ($F_{1,26}$=317.9, p≤0.001, η^2=0.92) for PT echogenicity. Moreover, a statistically significant interaction effect between *Time* and *Location* ($F_{1,26}$=12.7, p=0.001, η^2=0.33) was found. Post-hoc analysis showed significant increases in PT echogenicity placed proximal and distal to the apex patella (p<0.001). Moreover, higher echogenicity was found distal to the apex patella after start acceleration, compared with the location placed proximal to the apex patella after start acceleration (p=0.005) (**Table 2**).

Pearson's correlation test showed very strong negative correlations between mean power output and stiffness TT-10 (r=-0.61; p<0.05), stiffness TT-5 (r=-0.75; p<0.01), thickness TT-10 (r=-0.58; p<0.05), CSA proximal (r=-0.59; p<0.05), CSA middle (r=-0.55; p<0.05), positive for echogenicity distal (r=0.66, p<0.01). Also, the post-thickness PT-15 and inertial load were strongly correlated (r=0.60; p<0.05). Additionally, very strong relationships were found between the baseline to immediate-post differences of stiffness PT-5 (r=0.61; p<0.05), stiffness TT-10 (r=-0.62; p<0.05), stiffness TT-5 (r=-0.77; p<0.01), thickness TT (r =-0.56 p<0.05) in relation to mean power output.

Discussion

This study may provide novel findings and explanations regarding acute fatigue-induced mechanisms in PT, including both ultrasonographic and myotonometric evaluation of the tendon's properties. Moreover, this current study showed for the first time an increase in PT thickness and stiffness at the same locations placed 5-10-15-20 mm distal from the apex of the patella and 5-10 mm proximal to the tibial tuberosity after standing start acceleration training in elite sprint track cyclists. Our results demonstrated also an acute increase in tendon's CSA at three different locations (proximal, medial, and distal), as

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well as echogenicity including a 20 mm interval distal from the apex of the patella and a 10 mm interval proximal to the tibial tuberosity. The reported increase in PT thickness, stiffness, and CSA may indicate fiber disruption due to alterations in the collagen content, collagen type, as well as extracellular matrix density [32, 33]. Moreover, the increase in echogenicity may also be an indicator of fatigue-induced alteration in PT [34, 35]. The results of our study showed acute spatial changes in the morphological and mechanical properties of PT after repeated standing start acceleration exercises. Alterations in morphological properties were evaluated using ultrasonography imaging, while the mechanical property was assessed using myotonometry at the same six locations on PT. Our previous studies have demonstrated good to excellent intra- and inter-reliability for this measurement procedure using ultrasound [8] and similar changes in PT thickness after 200 m flying start competition in elite sprint track cyclists [4].

The measurement procedure for PT thickness and stiffness consisted of six reference points located along the tendon, including four points located distal to the apex of the patella, and two located proximal to the tibial tuberosity. This study was also the first to evaluate spatial changes in thickness and stiffness at the same locations in PT after standing start acceleration training in elite sprint track cyclists.

We observed a significant increase in PT thickness in all measured points immediately after repeated standing start acceleration. The largest difference between immediately post and baseline, as well as a percentage increase in thickness, was observed at a point located 20 mm distal from the apex of the patella (main difference: 0.88 mm; +28%), while the lowest difference was found at 5 mm proximal to the tibial tuberosity (main difference: 0.18 mm; +6%). Klich et al. [4] observed the largest increase in thickness at a point located 15 mm and 20 mm from the apex of the patella after 200 m

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flying start in sprint track cycling (+8% for both locations) and after 4000 m pursuit race (+7% for both locations). The present study showed a strong and statistically significant correlation between the post-thickness of PT-15 and inertial load. The increase in thickness and relationship with inertial load might be related to the specifics of a standing start, because, in contrast to a flying start, a standing start requires maximum isometric strength of the quadriceps to generate maximal power output during the gate release phase [36, 37]. Immediately after gate release, the drive phase follows where sprinters must generate maximum power as quickly as possible and maintain it as long as possible [2]. The second difference might be related to different gear ratios during standing start and flying start. Moreover, endurance track cyclists performed standing start during a 4000 m pursuit race, however, endurance events require a lower gear ratio than in sprint competition.

Previous studies used myotonometric evaluation of PT to measure stiffness at the middle location of the tendon [38, 39]. Cristi-Sánchez et al. [39] reported greater PT stiffness in soccer players compared with non-athletes as a result of higher force transmission during muscle contraction. Those studies are case cross-sectional studies; however, we have not found any investigation reporting an acute influence of different muscle actions, especially isometric contraction on tendon stiffness. In this current study, we found a significant increase in PT stiffness after standing start accelerations, however, the greatest and lowest increase was not similar in thickness. The largest difference between immediately post and baseline, as well as a percentage increase in stiffness, was observed at a point located 5 mm proximal to the tibia tuberosity (main difference: 316 N/m; +124%), while the lowest was seen at 5 mm distal from the apex of the patella (main difference: 90 N/m; +35%). Our study revealed region-dependent acute changes in PT

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characteristics after standing start acceleration training in elite sprint track cyclists. Changes in PT stiffness after standing start accelerations should be considered as the effect of isometric contraction during the gate release phase and anisometric generation of maximal power during the drive phase. Burgess et al. [40] and Kubo et al. [41] observed an increase in PT stiffness after isometric training, as an effect of the quadriceps muscle hypertrophy in a frame of training-induced adaptation. Moreover, increased stiffness in athletes might be related to changes in the interaction of the muscle-tendon unit and protection mechanism against damage [42]. The greatest increase in PT stiffness at 5 mm proximal to the tibial tuberosity may be a result of tendon elongation. O'Brien et al. [43] have observed that increased stiffness may be caused by greater CSA. In this current study, distal CSA (close by tibial tuberosity) had the lowest increase among proximal and middle CSA, while, the distal echogenicity had greater echo intensity compared with proximal. Yu and Boseck [44] have indicated that alterations in signal echogenicity are related to mechanical properties and may be dependent on the magnitude of the tendon's tension. Suydam and Buchanan [45] have found that echogenicity may indicate non-functional alterations in the Achilles tendon since factors like torsion of the tendon and multiaxial loading influence mechanical properties. It should be noted that PT has a different morphological structure than the Achilles tendon, thus additional research is needed to explain changes in PT echo intensity.

This study has some practical implications for scientists and sprint track cycling coaches. It is noteworthy that the regular training process not only develops physical performance (PPO, optimal torque, and cadence) but also causes morphological adaptation of skeletal muscles and tendons. Therefore, it is important to monitor acute and longitudinal changes in sports such as sprinting in track cycling. Appropriate

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preparation for the competition phase may lead to the reduced possible occurrence of overuse syndrome in muscles and/or tendons. Thus, training should include a strength exercises phase like traditional gym-based training for increasing the total number of collagen fibrils, their diameter, and fibril packing density.

Some of the potential limitations should be noticed to consider in future studies. First, we have recruited under 23 age track cyclists and more experienced track cyclists who specialized in sprint events could provide more specific information about acute fatigue-induced alterations in PT. Second, we only assessed the acute effect after standing start acceleration training. Future studies should also report post-exercise assessments to monitor recovery processes. Additionally, future studies could implement simultaneous investigation of PT thickness and stiffness by using shear-wave elastography.

Conclusions

Standing start accelerations, as one of the principal training exercises, may lead to gathering post-exercise alterations in morphological (tendon thickness, CSA), mechanical properties (stiffness), and echogenicity of PT. Regional-dependent alterations of PT thickness and stiffness may be related to anatomical and physiological mechanisms due to acute isometric contraction in the initial phase of standing start. However, the anatomical characteristics of PT might be associated with an adaptation to different loadings. Tendon echogenicity might be also useful in monitoring tendon mechanical properties and defining acute fatigue-induced changes. In general, those measurement procedures could be used in the early evaluation of overuse syndrome.

Acknowledgments

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The experiments complied with the current laws of the country in which they were performed. The datasets generated and analyzed during the current study are not publicly available, but are available from the corresponding author who was an organizer of the study. The authors have no conflicts of interest to declare.

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Table and figures list

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Table 1. Mean±SD of the participant demographics, training experience, and duration.

Variables	Elite sprint track cyclists n=14
Age (year)	21±1.7
Body height (cm)	179±3.9
Body mass (kg)	85.6 ±7.5
Body Mass Index (kg/m^2)	26.6±1.7
Training experience (year)	9±3
Isometric force [kgf]	143±32
Peak power output [W]	1969±256
Peak power output [W/kg]	23±2
Mean power output [W]	1819±225
Mean power output [W/kg]	21±2

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Table 2. Patellar tendon stiffness [N/m], thickness [mm], cross-sectional area [mm^2] and echogenicity at baseline and immediate after repeated sprint acceleration protocol. Mean ± SD values.

	Baseline	Immediate after	Immediate-Post to Baseline Mean Difference (95% CI)	*P* value*
Stiffness [N/m]				
PT-5	257±38.2	358±32.2	90 (87,93)	*p*≤.001
PT-10	266±8.0	387±57.5	103 (96,110)	*p*≤.001
PT-15	241±29.6	387±48.2	140 (134,146)	*p*≤.001
PT-20	254±27.0	356±33.8	105 (101,109)	*p*≤.001
TT-10	247±20.2	404±34.9	195 (170,220)	*p*≤.001
TT-5	255±31.2	468±64.6	316 (275,357)	*p*≤.001
Thickness [mm]				
PT-5	3.56±0.25	4.24±0.38	0.64 (0.60,0.67)	*p*≤.001
PT-10	3.52±0.21	4.16±0.40	0.58 (0.49,0.66)	*p*≤.001
PT-15	3.18±0.35	3.59±0.11	0.44 (0.40,0.48)	*p*≤.001
PT-20	3.17±0.40	4,05±0.50	0.86 (0.75,0.96)	*p*≤.001
TT-10	2.96±0.33	3.28±0.25	0.56 (0.42,0.70)	*p*≤.001
TT-5	3.05±0.21	3.23±0.23	0.13 (0.11,0.15)	*P>0*.001
CSA [mm^2]				
PT-5	162±12.7	180±12.7	15 (14,17)	*p*≤.001
PT-20	120±7.12	134±7.13	14 (13,15)	*p*≤.001
TT-5	114±1.80	123±1.81	6 (5,7)	*p*≤.001
Echogenicity				
Proximal	75.78±5.63	91.55±7.06	12 (9,14)	*p*≤.001
Distal	83.16±8.10	101.46±9.97	17 (15,19)	*p*≤.001

Significant differences *- within-group differences between baseline and IA.

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Figure 1. The experimental procedure includes measurements taken at baseline and immediately after sprint accelerations including PT stiffness, thickness, and CSA. After each sprint acceleration assessments of myotonometry and ultrasonography were taken.

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Figure 2. Reference location for PT stiffness set at 5-10-15-20 mm distal from the apex of the patella and 5-10 mm proximal to the tibial tuberosity.

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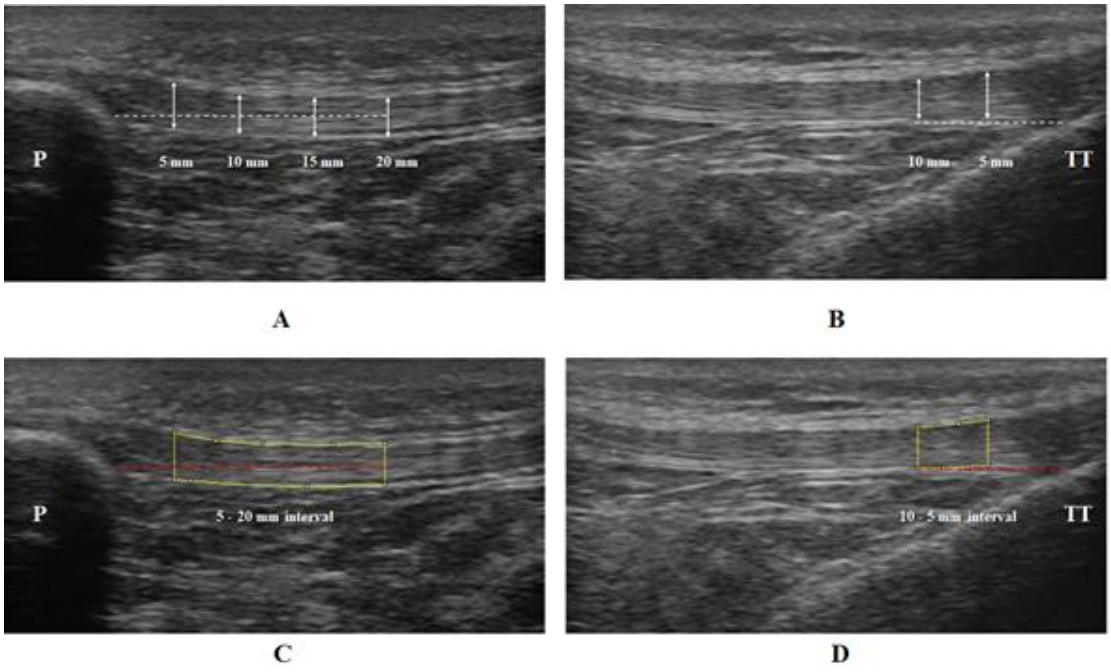

Figure 3. Ultrasound assessment of PT thickness (A and B), as well as area and echo intensity (C and D) measured from 5-10-15-20 mm distal from the apex of the patella (A), 5-10 mm proximal to the tibial tuberosity (B), the apex of the patella: red dotted line indicates the length of 20 mm (C), the tibial tuberosity: red dotted line indicates the length of 10 mm (D). In the yellow ROI area and echo intensity was computed.