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1 **EARLY ANTERIOR KNEE PAIN IN MALE ADOLESCENT BASKETBALL**
2 **PLAYERS ARE RELATED WITH BODY HEIGHT AND ABNORMAL KNEE**
3 **MORPHOLOGY**

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1 **ABSTRACT**

2 *Objectives:* To compare knee torque, range of motion, quality of movement, and morphology
3 in dominant and nondominant legs of male adolescent basketball players with and without
4 anterior knee pain and untrained peers.

5 *Design:* Cross-sectional.

6 *Setting:* Sports performance laboratory.

7 *Participants:* Male basketball players aged 14–15 years with and without anterior knee pain
8 and healthy untrained subjects (n = 88).

9 *Main outcome measures:* Basketball players were allocated to a symptomatic or
10 asymptomatic group based on self-reported anterior knee pain. Associations between pain
11 and body mass, height, passive range of motion, muscle peak torque, coactivation,
12 neuromuscular control, proprioception, and ultrasound observations were investigated.

13 *Results:* The prevalence of pain did not differ significantly between sides. Of 176 knees
14 inspected, 44 were painful, and 26 of these exhibited abnormalities in ultrasonography.
15 Symptomatic players were 5.0 and 6.9 cm taller than asymptomatic players and controls,
16 respectively ($P < 0.05$). In athletes with knee pain, the odds ratios of morphological
17 abnormalities and greater height were increased by 8.6 and 5.0 times ($P < 0.001$).

18 *Conclusion:* Knee pain prevalence in adolescent basketball players was not related to
19 differences between sides but was higher in tall players. Knee pain was accompanied by
20 morphological abnormalities detected with ultrasound.

21

22 **Keywords** Diagnostic ultrasound, Proprioception/Balance, Electromyography, Isokinetic
23 dynamometry

INTRODUCTION

1
2 Regular physical activity improves skeletal muscle function and cardiovascular health,
3 and helps to prevent and reverse obesity, reduce anxiety and depression, and improve self-
4 confidence and cognitive function (Haskell et al., 2007). Because of these benefits, regular
5 sports participation is highly encouraged. However, with growing competitiveness in sport,
6 children, adolescents, and young adult participants are under increasing psychological
7 pressure to win. For this reason, physical training is often intense, frequent, and highly
8 specialized, which increases the risk of training becoming too intense and/or unbalanced, as
9 well as possible health problems and ~~acute~~ traumatic injuries (Frisch, Croisier, Urhausen,
10 Seil, & Theisen, 2009). Although healthy tendons and muscles can tolerate large loads,
11 constant overexertion and incorrectly performed exercises and loading while fatigued can
12 change the material properties of the joints, such as the knee joint, which can make them
13 more prone to overuse injuries and associated pain (Helland et al., 2013). Additional risk
14 factors for knee joint pain include muscle weakness, strength imbalance, motor control
15 deficit, knee joint laxity, and changes in the mechanical and morphological properties of
16 tendons and ligaments (Smith et al., 2012; Ladenhauf, Graziano, & Marx, 2013; Gagnier,
17 Morgenstern, & Chess, 2013). These factors can increase the stress on the lower limbs during
18 sports events.

19 Adolescents are particularly vulnerable to overuse injuries because their
20 musculotendinous system is undergoing significant structural, biomechanical, and,
21 consequently, functional alterations during this phase of rapid growth (Frisch et al., 2009;
22 Adirim & Cheng, 2003). Previous studies have shown that up to 54% of adolescent athletes

1 experience some form of knee pain annually (Foss, Myer, Magnussen, & Hewett, 2014).
2 Nontraumatic anterior knee pain is one of the most common complaints in young athletes
3 (Adirim & Cheng, 2003).

4 A high degree of sport specialization contributes to overuse injuries in young athletes
5 (Bell, Post, Trigsted, Hetzel, McGuine, & Brooks, 2016). The incidence of knee joint injuries
6 in children and adolescents is particularly high in organized team sports, including
7 basketball (Caine, Maffulli, & Caine, 2008; Frisch et al., 2009). This can be attributed to
8 repeated sprinting, stopping, changing direction, and jumping (Cumps, Verhagen, &
9 Meeusen, 2007). These movements place a particularly high amount of stress on the leading
10 knee and associated muscle–tendon unit. Adolescent basketball players therefore provide an
11 ideal population for evaluating the structural and functional changes associated with knee
12 pain and injury.

13 The purpose of the present study was to compare knee torque, range of motion (ROM),
14 quality of movement, and morphological indicators in the dominant and nondominant legs
15 of male adolescent basketball players with and without anterior knee pain and untrained male
16 adolescents. All basketball players who experienced knee pain started having minor
17 complaints that were not yet intense enough to warrant medical attention. This phase is thus
18 ideal for assessing the associations between pain and functional and morphological
19 adaptations. Understanding these relationships may help to prevent further development of
20 pain or even alleviate existing pain. We hypothesized that adolescent basketball players
21 would have 1) side-to-side imbalances in muscle strength, 2) impaired flexibility and
22 neuromuscular control and 3) show morphological abnormalities that can be detected

1 noninvasively with ultrasound. We expect that this would help identify the risk factors for,
2 and early indicators of, knee pain and injury.

3

4

METHODS

Participants

6 The present study involved 88 male adolescents, who were divided into the following
7 three groups (Table 1): adolescent basketball players who had participated for ≥ 5 years in
8 basketball activities and who had experienced anterior knee joint pain during the previous
9 month (symptomatic group, $n = 29$) or had not experienced pain (asymptomatic group, $n =$
10 30). In both groups, players were excluded if they had experienced any acute or contact
11 trauma, including ligament or meniscus injury and/or associated pain in a proximal and/or
12 distal joint. Players who had prior or present pain that required medical attention were also
13 excluded from the study. The basketball players were recruited at the end of the playing
14 season (May–June) from a county basketball school. They played seven to eight times per
15 month for about 7 months per year in the national championships and attended 1.5-h training
16 sessions four to five times per week all year. The third group comprised healthy untrained,
17 non-basketball-playing adolescents without a history of knee pain (control group, $n = 29$).
18 Control subjects were recruited from a local high school and were matched for age and body
19 mass index (BMI) to the basketball players. The Kaunas Regional Biomedical Research
20 Ethics Committee approved the research protocol, which was consistent with the principles
21 outlined in the Declaration of Helsinki. Written informed consent was obtained from the
22 parent or guardian of each participant.

1

2 Pain

3 For all participants in the control, asymptomatic, and symptomatic groups, the same
4 investigator characterized the severity and frequency of knee pain for each knee according to
5 the Knee Injury and Osteoarthritis Outcome Score (KOOS; Lithuanian version LK1.0)
6 questionnaire using only the nine-item part related to pain. Each question is scored from 0
7 (no pain) to 4 (extreme pain) (Roos & Lohmander, 2003). Here, we report the average score
8 of the nine items in the test.

9

10 Testing procedures

11 Body mass was measured in kilograms (TBF-300 body composition scale; Tanita, UK
12 Ltd., West Drayton, UK) and height in meters (Anthropometry Martin, GPM SiberHegner,
13 Switzerland) in the laboratory. The ROM of the left and right knee joints was determined
14 with a goniometer. This was followed by a warm-up of 8 min of cycling at a power of 50–70
15 W at a cadence of 70 rpm on a cycle ergometer. Next, knee extensor and flexor muscle
16 proprioception, peak torque, and coactivation were measured in each leg on an isokinetic
17 dynamometer (System 3; Biodex Medical Systems, Shirley, NY, USA). The next day,
18 clinical observation of the left and right knee joints was performed, and patella tendon
19 dimensions were measured by ultrasound. This was followed by a neuromuscular control test
20 for visual inspection of movement stability. The leg used to kick a ball was considered the
21 dominant leg. The above methods are detailed in the following sections.

22

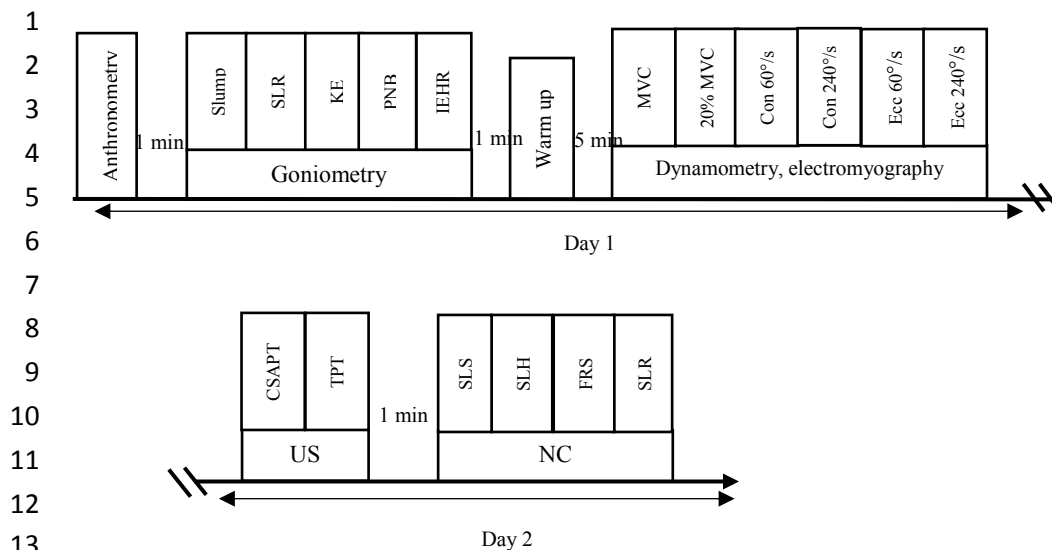


Figure 1. Graphical overview of the testing protocol.

Note. Slump – Slump Test; SLR – Straight Leg Rise Test; KE – Knee Extension Test (90/90); PNB – Prone Knee Bend Test; IEHR – Internal and External Hip Rotation; NC – Neuromuscular control; SLS – Single Leg Squat Test; SLH – Single Leg Hoop Test; FRS – Foam Roller Supine Test; SLR – Straight Leg Rise Test; MVC – maximal voluntary contraction force; Con – concentric muscle contraction; Ecc – eccentric muscle contraction; US – ultrasonography; CSAPT – cross-sectional area of the patellar tendon; TPT – thickness of the patellar tendon.

ROM

Passive ROM was determined in both legs with a standard goniometer and demarcated by the positions at which resistance to movement was felt (Sauer, Potter, Weisshaar, Ploeg, & Thelen, 2007). Participants did not report neurogenic pain in prone knee-bend, straight-leg raise, or seated slump tests. We first assessed resistance to movement in these neurodynamic tests (Boyd, Wanek, Gray, & Topp, 2009). To minimize subjective bias, all ROMs were determined by the same researcher. High intraclass correlation coefficients (ICCs) of 0.88–0.97 have been reported for the ROM tests used in the present study (Cibulka, Sinacore, Cromer, & Delitto, 1998; Gabbe, Bennell, Wajswelner, & Finch, 2004; Russell & Bandy,

1 2004; Santos, Ferreira, Malacco, Sabino, Moraes, & Felício, 2012; Neto, Jacobsohn, Carita,
2 & Oliveira, 2014).

3 The 90/90 test was performed by the participant in a supine position with the hip and knee
4 flexed to 90° and gently stretched until resistance was felt. The lateral epicondyle of the
5 femur was palpated, and the goniometer placed over the epicondyle. The lateral malleolus of
6 the tibia and the greater trochanter of the femur were marked. The arms of the goniometer
7 were aligned with these proximal and distal landmarks (Russell & Bandy, 2004).

8 Next, the prone knee-bend test for femoral nerve tension was performed on each leg. The
9 examiner passively flexed one knee slowly as far as possible until unilateral neurogenic pain
10 was felt along the lumbar area, buttock, and/or anterior thigh. Knee ROM was recorded as
11 the angle at which subjective symptoms were reported (Dutton, 2004).

12 For the internal and external hip rotation test, the participant lay in the supine position
13 with the hip and knee flexed 90°. The hip was then maximally rotated internally and
14 externally (Byrd, 2005).

15 The straight-leg raise test was performed in the supine position. The tested limb was raised
16 by the examiner with the knee fully extended and the foot in a relaxed position. The
17 contralateral limb was kept fully extended and in neutral rotation by a second examiner. The
18 goniometer was placed over the greater trochanter, with one arm aligned with the lateral
19 femoral condyle and the other aligned parallel to the table in the direction of the midaxillary
20 line (Neto et al., 2014).

21 For the seated slump test, the participant sat in a slouched position and actively flexed the
22 cervical spine as far as comfortably possible. The ankle was then passively dorsiflexed to
23 slight resistance, and the knee was slowly passively extended. The knee was extended until

1 full extension was achieved or until the participant reported the onset of neural-mediated
2 symptoms. The goniometer was aligned in a standard fashion with the axis over the lateral
3 femoral epicondyle, the stationary arm was aligned with the femoral trochanter, and the
4 moveable arm was aligned with the lateral malleolus (Davis, Anderson, Carson, Elkins, &
5 Stuckey, 2008).

6

7 Proprioception

8 A familiarization session for measurement of proprioception and peak torque was
9 performed 2 days before data collection. The participant sat in the isokinetic dynamometer
10 chair and performed a shorter version of the testing protocol described below. Proprioception
11 was assessed using an isokinetic dynamometer during both knee flexion and extension at a
12 knee angle of 90° (0° = leg fully extended). During the testing, repeated isometric knee
13 flexion and extension (10 trials at 1-s intervals) were performed in an attempt to match the
14 required 20% maximal voluntary isometric contraction (MVC). The participant could see the
15 force generated on the instrument's screen. To provide a measure of accuracy of isometric
16 contractions, we calculated the absolute error size (Magill, 2007) using the following
17 formula:

$$18 \quad \text{Absolute error} = \sum |x_i - T| / n$$

19 where x_i is the torque generated during trial i , T is the target torque, and n is the number of
20 trials. The vertical brackets $| |$ indicate that the difference was always given a positive value.

21 This gives an absolute error value in N·m. Not every individual produces the same maximal

1 torque, so to compare the data between different subjects, we also calculated the absolute
2 error (AE) size as a percentage of MVC as follows:

$$3 \quad \text{AE (\%)} = \text{AE} \times 100 / (20\% \text{ MVC}).$$

4 Moderate ICCs of 0.73–0.81 for the proprioception test have been reported (Zavieh et al.,
5 2016).

6

7 Peak torque

8 After completion of the proprioception evaluation, concentric and eccentric isokinetic and
9 isometric peak torque of the knee extensor and flexor muscles were measured using an
10 isokinetic dynamometer. First, an MVC was performed twice, each separated by a 2-min rest
11 interval with the knee joint at 90° (full extension = 0°). The participant then performed three
12 maximal contractions at angular velocities of 60°/s and 240°/s in both the concentric and
13 eccentric modes, and the best of three trials was used for further analysis. Each of the angular
14 velocities and the concentric and eccentric measurements were separated by ≥2-min rest to
15 prevent the development of fatigue. Two warm-up trials at submaximal intensity were
16 performed before measurement of maximal peak torque at each velocity. The ICC for peak
17 torque varies from 0.81 to 0.95 depending on exercise mode and velocity (Janusevicius et al.,
18 2017). The ratios of peak torque for concentric hamstring:concentric quadriceps and
19 eccentric hamstring:concentric quadriceps were calculated.

20

21 Electromyography

1 Electromyographic activity was assessed for the rectus femoris (RF) and the long head of
2 the biceps femoris (BF) muscles as described previously (Hermens, Freriks, Disselhorst-
3 Klug, & Rau, 2000). An MP150 instrument (Biopac Systems, Inc., CA, USA) was used to
4 record the electromyogram (EMG). Two self-adhesive disposable Ag–AgCl electrodes (10-
5 mm diameter, Ceracorta, Forli (FC), Italy) were placed on the hamstring and quadriceps
6 muscles with a 20-mm interelectrode distance, and the ground electrode was positioned on
7 the knee. A raw EMG was acquired with a sampling frequency of 1000 Hz and was filtered
8 using analogue high-pass (10 Hz) and low-pass (500 Hz) filters. The EMG signals were
9 analyzed using Acknowledge software (Biopac Systems). Coactivation of the RF and BF was
10 assessed using the root mean square (RMS) of the EMG signal. The analyzed EMG signal
11 was taken at 200 ms around the peak for isometric and 60°/s for concentric contractions, at
12 100 ms for 240°/s concentric and 60°/s eccentric contractions, and at 20 ms for eccentric
13 contractions at 240°/s. Coactivation of the BF was assessed as the EMG activity during knee
14 extension as a percentage of the EMG activity of the BF during maximal knee flexion either
15 at a constant length or during a constant-velocity contraction (Kellis & Baltzopoulos, 1996).
16 The ICCs for the RMS of the EMG signal are 0.80–0.88 (Larsson, Karlsson, Eriksson, &
17 Gerdle, 2003).

18

19 Ultrasonographic assessment

20 A radiologist with nearly 10 years clinical work experience assessed both knees in all
21 participants in the control, asymptomatic, and symptomatic groups using an ultrasound
22 device (Esaote MyLab 50 XVision, Italy) with a 7–12-MHz linear probe. The radiologist

1 was blinded to the group. The participant was diagnosed as having morphological
2 abnormalities if ultrasound inspection exposed pathological signs such as oedema,
3 increased connective tissue deposition, or fragmentation of the tibial tuberosity ossification
4 centre, which is linked to Osgood–Schlatter syndrome. Other characteristic signs were
5 recorded as morphological abnormalities; these included semimembranosus bursitis,
6 observed as an enlarged semimembranosus bursa, bipartite patella, as a patella with an
7 unfused accessory ossification centre, or thickening of the patella, which indicates patellar
8 tendonitis. If the participant had noticed any of these abnormalities, he was advised to
9 consult an orthopaedic surgeon, but we did not request the examination results.

10 The morphology of the knee joint is represented here as the patellar tendon (PT) cross-
11 sectional area (CSA) and thickness. The CSA and thickness in both the longitudinal and
12 transverse planes were measured in both knees while the participant was in a supine position
13 with his knees supported and flexed (~30°). The PT was assessed in two areas: 1 cm from
14 the apex of the patella, and 1 cm above the tibia attachment. The data from these two PT
15 measurement areas was averaged for further analysis. High ICCs of 0.82–0.93 for PT
16 dimension measurements have been reported (Gellhorn & Carlson, 2013).

17

18 Neuromuscular control

19 The following day, neuromuscular control was assessed by a physical therapist with >10
20 years' experience. High to moderate ICCs of 0.77–0.92 for the tests used in the present study
21 have been reported (Mens, Vleeming, Snijders, Koes, & Stam, 2001; Stensrud, Myklebust,
22 Kristianslund, Bahr, & Krosshaug, 2011).

1 During the single-leg squat, the participant squatted five times on each leg and then
2 performed five single-leg hops on each leg (Bailey, Selfe, & Richards, 2011). In these two
3 tests, position and movement were assessed and graded as follows: 1, the ankle, knee and hip
4 were maintained in line, the pelvis was horizontal, and the trunk was vertical in the sagittal
5 plane; 2, the participant displayed minor disturbances in the lower extremity and trunk
6 alignment in the sagittal plane and/or a slight pelvic drop; 3, the participant displayed
7 moderate disturbances in the lower extremity and trunk alignment in the sagittal plane and/or
8 moderate pelvic drop; and 4, the participant displayed severe disturbances in the lower
9 extremity and trunk alignment in the sagittal plane and/or significant pelvic drop.

10 For the foam roller supine test, the participant lifted each leg five times from the supine
11 position lying on a foam roller with the hands not touching the floor.

12 Finally, for the active straight-leg raise test, the participant was asked to lift each leg 20
13 cm straight five times while lying supine on the floor (Mens et al., 2001). The last two tests
14 were assessed as follows: 1, stable pelvis; 2, minor sway of pelvis; 3, moderate sway of
15 pelvis; 4, the participant falls off the roller or strong rotation or sway of the pelvis from the
16 horizontal position. Raising one leg with a little sway and the other leg with a larger sway
17 was considered a minor disturbance, and moderate pelvic sway during the active straight-leg
18 raise test was considered a moderate disturbance.

19

20 Statistical analysis

21 Data are presented as mean and standard deviation (SD). The Kolmogorov–Smirnov test
22 was used to check whether the data were normally distributed. If not normally distributed,

1 the data were log-transformed or the inverse was taken to ensure a normal distribution, which
2 was then used for statistical analysis. In some cases, no normal distribution developed even
3 after transformation of the data. In these cases, a nonparametric exact Wilcoxon test was used
4 to evaluate the differences, such as for the neuromuscular control tests. For normally
5 distributed data, a repeated-measures ANOVA was used with a within-factor (side: dominant
6 vs. nondominant) and a between-factor (group: symptomatic vs. nonsymptomatic vs. control)
7 analysis. When a significant group effect but no side \times group interaction was found, Tukey's
8 post hoc test was performed to locate the differences. A significant interaction between side
9 and group indicated that the side difference differed between the three groups.

10 To identify any associations between knee pain and parametric variables (peak torque,
11 ROM, AE, RMS), these variables were divided into two percentiles using the median value
12 to calculate the odds ratios for a 2×2 contingency table and 95% confidence intervals (CIs).
13 The participants were allocated to a low (3 or 4 points) or high (1 or 2 points) group for each
14 neuromuscular control test or for the number of morphological abnormalities in positive and
15 negative groups, and the odds ratios and CIs were calculated for nonparametric variables.
16 The sample size required to detect a change of 15% in peak torque, which is considered to
17 be clinically relevant, was 25 at a one-tailed $\alpha = 0.05$ and statistical power $\beta = 0.80$. For all
18 statistical tests, differences were regarded as significant at $P < 0.05$.

19

20

RESULTS

21 Participant characteristics

1 Thirteen players had bilateral pain, while 16 players had unilateral (8 in the dominant, 8
2 in the non-dominant leg) pain. The pain intensity and frequency in the symptomatic athletes
3 were 1.5 ± 0.7 points and 10.7 ± 5.7 days per month. The pain intensity and frequency did
4 not differ between legs (1.4 points and 10.9 days in the dominant leg; 1.5 points and 10.4
5 days in the nondominant leg, $P > 0.05$). The symptomatic players were taller than the
6 asymptomatic players and controls ($P < 0.05$), but body mass and BMI did not differ
7 significantly between groups (Table 1). The odds ratio of having knee pain was increased 5.0
8 times in tall players compared with asymptomatic players and controls (95% CI 2.2–11.2; P
9 < 0.001).

10 **Table 1.** Participants' characteristics.

Group	Number	Age (years)	Height (cm)	Body mass (kg)	BMI	Training experience (years)
Symptomatic	29	14.5 ± 0.6	179 ^{a,b} ± 8	65.4 ± 10.6	20.3 ± 2.1	6.3 ± 1.6
Asymptomatic	30	14.0 ± 0.6	174 ± 11	61.4 ± 13.2	20.0 ± 2.5	6.3 ± 1.4
Control	29	14.2 ± 0.7	172 ± 9	63.4 ± 15.1	21.2 ± 4.1	–

11 ^a: significantly different from control at $P < 0.05$.

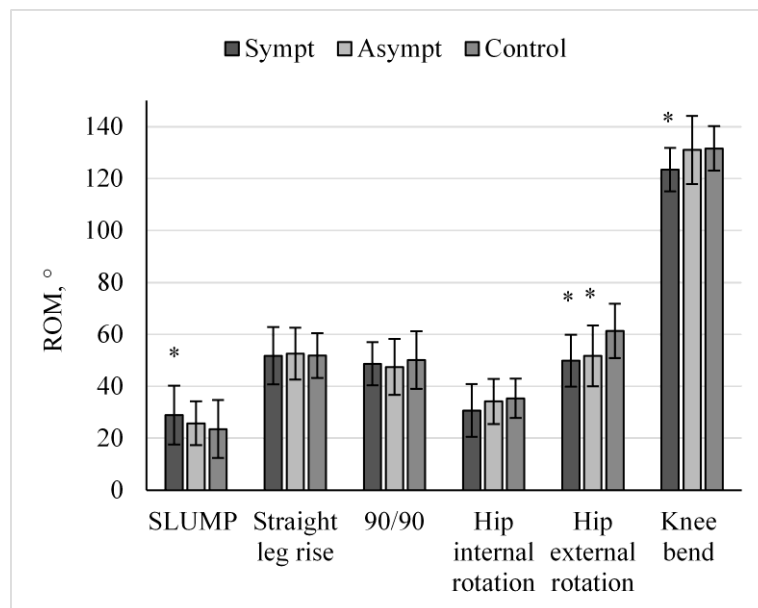
12 ^b: significantly different from asymptomatic at $P < 0.05$. Data are expressed as mean \pm standard
13 deviation.

14

15 **ROM**

16 There were no significant side differences, side \times group interactions, or significant
17 associations with pain for ROM in any joint. Figure 2 shows that symptomatic players had a

1 larger ROM than controls in knee extension during sitting (slump test, $P = 0.020$), and a
 2 smaller ROM in hip external rotation ($P < 0.001$) and knee bend in the prone position ($P <$
 3 0.001). These variables did not differ significantly between the symptomatic and
 4 asymptomatic groups.



5
 6 **Figure 2.** Comparison of ROM in controls and symptomatic and
 7 asymptomatic basketball players.

8 *: significantly different from control at $P < 0.05$. The data are presented as mean \pm standard
 9 deviation.

10

11 Peak torque

12 There were no significant group \times side interactions or associations between torque and
 13 knee pain. The isometric torque, concentric torque at $60^\circ/s$, and eccentric torque at $60^\circ/s$ were
 14 higher in the dominant than in the nondominant leg (Table 2; $P < 0.05$). Both symptomatic
 15 and asymptomatic players developed higher torques than controls during concentric $60^\circ/s$
 16 extension and flexion and $60^\circ/s$ eccentric flexion (Table 2; $P < 0.05$). In addition,
 17 symptomatic players developed higher torque than controls for isometric flexion, concentric

- 1 240°/s extension and flexion, and eccentric 60°/s and 240°/s flexion and extension (Table 2;
- 2 $P < 0.05$). The symptomatic players developed higher torques than asymptomatic players for
- 3 isometric extension and 60°/s eccentric flexion (Table 2; $P = 0.044$).

1 **Table 2.** Comparison of peak torque, ratio, and coactivation (CoAc) for the dominant and nondominant leg of controls (C), and symptomatic (S), and asymptomatic (A) basketball players.

	Symptomatic		Asymptomatic		Control		Side effect	Group effect
	Dominant	Nondominant	Dominant	Nondominant	Dominant	Nondominant		
Isometric								
MVC extension	175±29	162±34	159±48	144±38	160±42	149±35	D>ND; P<0.05	S>A; P=0.044
CoAc	(11.7)	(15.7)	(15.4)	(14.9)	(17.5)	(18.0)	NS	S<C; P=0.012
MVC flexion	88±21	78±18	79±26	71±23	71±17	64±21	D>ND; P<0.05	S>C; P<0.001
CoAc	(8.0)	(7.3)	(8.1)	(7.8)	(7.9)	(7.7)	NS	NS
Concentric								
60°/s extension	154±29	147±35	145±42	135±33	130±34	128±34	D>ND; P<0.05	S,A>C; P=0.041
CoAc	(14.0)	(14.9)	(17.0)	(16.6)	(16.9)	(18.3)	NS	NS
60°/s flexion	108±26	102±23	103±29	93±25	91±26	83±19	D>ND; P<0.05	S,A>C; P=0.048
CoAc	(7.9)	(8.2)	(6.6)	(6.2)	(9.9)	(8.9)	NS	A<C; P<0.001
240°/s extension	96±16	92±20	87±23	86±21	84±23	83±21	NS	S>C; P=0.025
CoAc	(18.3)	(17.0)	(17.4)	(19.3)	(16.2)	(18.6)	NS	NS
240°/s flexion	79±19	76±15	75±21	71±18	71±22	67±17	NS	S>C; P<0.001
CoAc	(10.0)	(10.3)	(9.0)	(8.0)	(11.4)	(9.7)	NS	A<C; P=0.043
Eccentric								
60°/s extension	199±39	198±49	190±60	179±50	173±43	172±43	NS	S>C; P=0.017
CoAc	(14.7)	(18.0)	(17.8)	(22.0)	(18.9)	(22.3)	D<ND; P<0.05	S<A,C; P<0.05
60°/s flexion	136±34	129±35	124±42	109±32	115±34	105±29	D>ND; P<0.05	S>A,C; P=0.041
CoAc	(8.0)	(9.5)	(10.1)	(7.5)	(10.3)	(9.2)	NS	NS
240°/s extension	143±44	159±46	132±59	126±54	133±57	122±56	NS	S>C; P=0.048
CoAc	(17.1)	(15.6)	(21.2)	(17.3)	(16.5)	(21.0)	NS	NS
240°/s flexion	130±37	122±42	118±40	106±37	112±26	104±29	NS	S>C; P=0.046
CoAc	(13.3)	(12.6)	(17.8)	(16.5)	(15.4)	(14.6)	NS	NS

2 Note: peak torque is presented in N m, coactivation and force ratios are presented as percentages. MVC: maximal isometric voluntary contraction torque;

3 NS: not significant. Values are expressed as mean ± standard deviation.

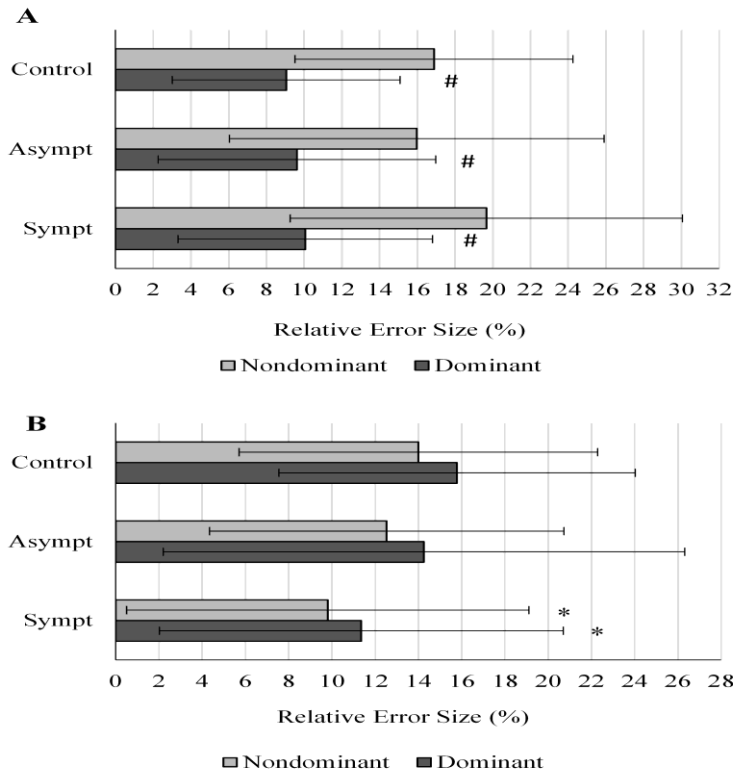
1 Coactivation

2 In all groups, coactivation was lower during knee flexion than during knee extension ($P < 0.05$).
3 Coactivation was lower in the dominant than in the nondominant leg only during eccentric $60^\circ/s$
4 flexion ($P < 0.05$; Table 2). Coactivation was lower in symptomatic players than in controls during
5 isometric and eccentric $60^\circ/s$ extensions and was lower in asymptomatic players than in controls
6 during concentric $60^\circ/s$ and $240^\circ/s$ flexion and eccentric $60^\circ/s^1$ extension (Table 2; $P < 0.05$).
7 Coactivation did not differ significantly between the symptomatic and asymptomatic players and
8 was not significantly associated with knee pain.

9 Proprioception

10 There were no significant differences in matching the actual extension torque to a required
11 torque between groups and no significant associations between the ability to match torque and
12 knee pain (Fig. 3). Matching was better in the dominant than in the nondominant leg only in
13 extension ($P < 0.001$). Matching was better during flexion in symptomatic players than in controls
14 ($P < 0.05$, Fig. 3).

15



1 **Figure 3.** Comparison of the error magnitude during repeated contractions at 20% maximal
 2 voluntary isometric contraction (MVC) between groups for the dominant and nondominant legs
 3 in controls, symptomatic, and asymptomatic basketball players.
 4 A, leg extension; B, leg flexion; *: different from controls at $P < 0.05$; #: difference between dominant
 5 and nondominant legs at $P < 0.05$. The data are presented as mean \pm standard deviation.
 6
 7

8 Neuromuscular control

9 There were no significant side differences or side \times group interactions in the neuromuscular
 10 control tests. Controls scored higher than the basketball players for the single-leg squat and jump
 11 and active straight-leg raise tests ($P < 0.05$, Fig. 4). When the data were combined for athletes and
 12 nonathletes, the poorer scores in the active straight-leg raise test were strongly associated with
 13 having a knee disorder, as shown by a more than twofold elevation in the odds ratio (95% CI 1.1–
 14 4.2; $P < 0.05$).

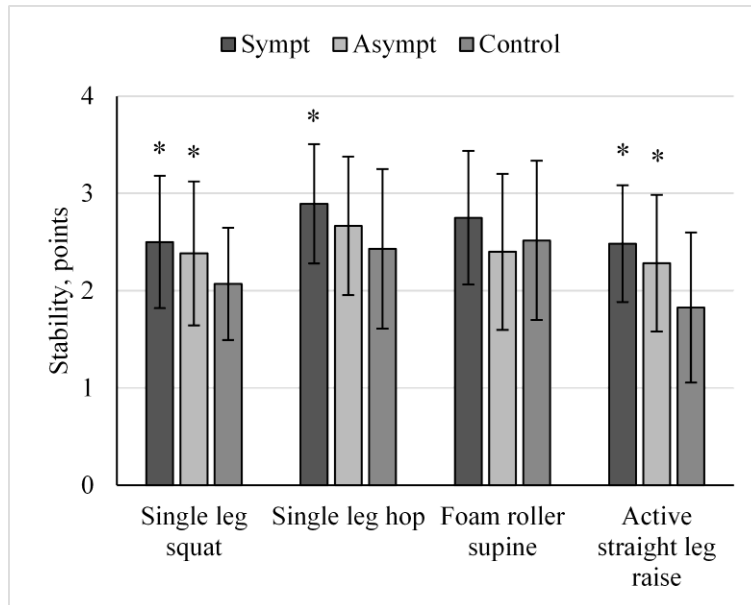


Figure 4. Comparison of visual inspection of stability during movement in controls, symptomatic (S), and asymptomatic (A) basketball players.

*: significantly different from controls at $P < 0.05$. The data are presented as mean \pm standard deviation.

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Knee joint morphology

7 There were no significant side differences or side \times group interactions for patella tendon
 8 morphology. **Of the 176 knees inspected with ultrasound, 26 showed morphological abnormalities.**
 9 **Out of these 26, 22 were painful, but 4 were without pain (2 from non-symptomatic basketball**
 10 **players and 2 from non-athletes). There were also 20 painful knees without morphological**
 11 **abnormalities.** The most frequent changes in the 26 knees with morphological abnormalities were
 12 characteristic of Osgood–Schlatter syndrome (50%), semimembranosus bursitis (19%), bipartite
 13 patella (15%), and patellar tendonitis (15%). The odds ratio of morphological abnormalities in
 14 participants with knee pain was elevated by 8.6-fold (95% CI 3.7–19.5; $P < 0.001$). Patella tendon
 15 CSA and thickness did not differ significantly between groups (Fig. 5).

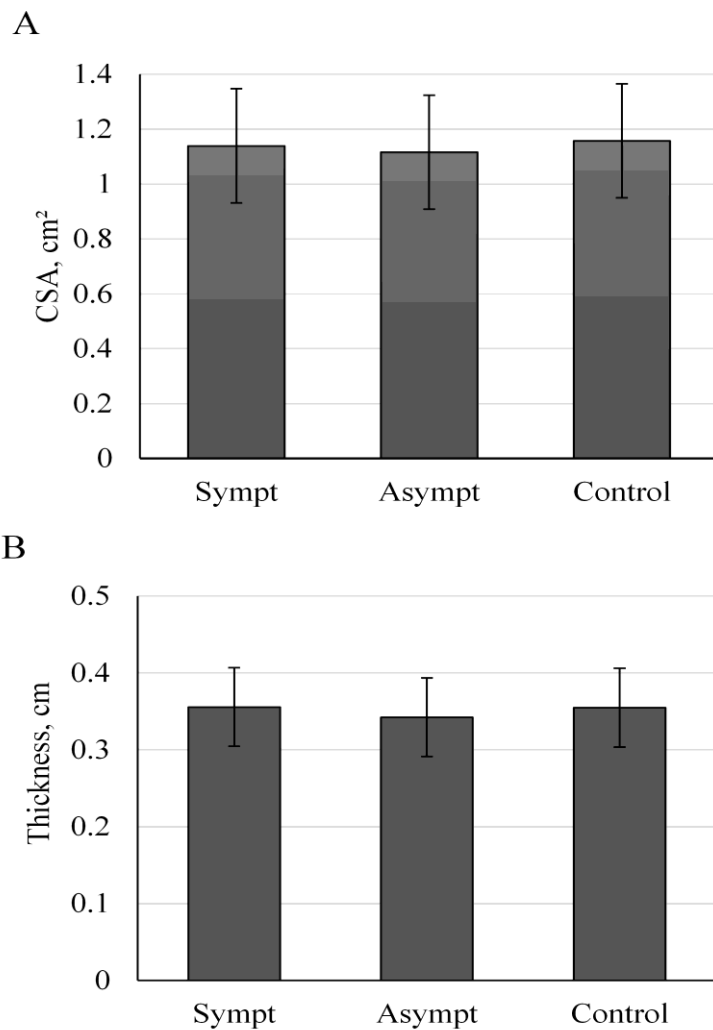


Figure 5. Comparison of patellar tendon cross-sectional area (A) and thickness (B) in controls, symptomatic, and asymptomatic basketball players.

DISCUSSION

We found that adolescent basketball players with knee pain were taller and more frequently showed morphological abnormalities of the knee joint. Although these abnormalities may be related to greater stress on their knees compared with the other groups, there were no significant differences between asymptomatic and symptomatic players in the ability of their knee extensors and flexors to develop torque. There was also no evidence that the side-to-side differences in muscle strength contributed to the development of anterior knee pain in these adolescent basketball

1 players. Because a taller player has a longer femur and tibia, it is possible that the longer bones
2 but similar muscle strength as in asymptomatic players placed greater stress on the knee joint
3 because of the longer lever arm of the bones.

4 Consistent with previous investigations (Daneshjoo, Mokhtar, Rahnama, & Yusof, 2012;
5 Śliwowski, Jadczyk, Hejna, & Wieczorek, 2015), our study found that the dominant leg was
6 stronger in each group. Bilateral strength differences were similar between groups, and there was
7 no association between leg dominance and pain symptoms. These results are also consistent with
8 data from upper body measurements showing that side-to-side differences between arms are
9 unlikely to contribute to throwing-related pain in children (Mickevičius et al., 2016).

10 There is evidence that greater muscle strength is associated with a higher risk of injury. For
11 instance, in adolescent female basketball players, greater hip abduction strength is associated with
12 an increased risk for the development of patellofemoral pain (Herbst, 2015). Baseball players with
13 greater throwing power are more prone to injury of the shoulder (Adirim & Cheng, 2003). At first
14 glance, our data seem to support this notion because the basketball players had stronger knee
15 extensors and flexors than controls. However, the knee extensors and flexors had a similar torque-
16 generating capacity in asymptomatic and symptomatic players. Therefore, it seems unlikely that
17 the knee pain in the symptomatic players was caused by the stronger knee extensors and flexors
18 *per se*.

19 The lower coactivation during muscle contraction in players compared with controls is
20 interesting because lower coactivation may be advantageous for the development of maximal
21 voluntary contraction force. The cause of the lower coactivation in the basketball players is not
22 known, but inappropriate coactivation of antagonist muscles may predispose the knee joint to a
23 higher risk of injuries (Aagaard, Simonsen, Magnusson, Larsson, & Dyhre-Poulsen, 1998). More

1 important perhaps is that inappropriate coactivation may have contributed to the impaired stability
2 of trunk/lower-limb movements observed in the basketball players. Scattone and Serrão (2014)
3 reported that impaired trunk/lower-limb kinematics are associated with an increased incidence of
4 overuse knee injury in adolescent females. Consistent with this, we found that lumbopelvic balance
5 during the active straight-leg raise test was associated with a more than twofold higher prevalence
6 of knee pain in our sample. These observations suggest that particular emphasis should be placed
7 on maintenance of core stability during athletic conditioning.

8 Both impaired stability of movement and limited ROM or hypermobility (extended ROM) have
9 been reported to contribute to lower-extremity joint disorders (Tyler, Nicholas, Mullaney, &
10 McHugh, 2006). In the symptomatic players in our study, limited ROM was greatest during the
11 prone knee-bend test in the quadriceps. There is evidence that increased nerve mechanosensitivity
12 plays a protective role in the prevention of muscle damage (Boyd et al., 2009). The smaller ROM
13 was not only limited to the quadriceps, but also found in hip external rotation. Although a ROM
14 deficit or hypermobility may contribute to knee problems in symptomatic players, these may not
15 be directly related because ROM did not differ significantly between the symptomatic and
16 asymptomatic players. Therefore, whether alterations in ROM are a prelude to knee problems
17 remains unclear.

18 Similar to previous observations in bilateral sports (Zhang, Ng, Lee, & Fu, 2014), our study
19 found no significant differences in PT CSA and thickness between groups. However, others have
20 observed thickening and larger CSA in athletes in unilateral sports (Couppé et al., 2008) and
21 female volleyball (bilateral) players (Toprak et al., 2012). The discrepancy between studies may
22 be related to the type of sport and/or the sex of the athletes because sex and sport specificity are
23 risk factors for patellar tendinopathy (van der Worp, van Ark, Roerink, Pepping, van den Akker-

1 Scheek, & Zwerver, 2011). Regardless of the reason for the discrepancies between studies, the
2 lack of adaptation to the large stresses and strains on the tendon in the basketball players in the
3 present study may have increased the risk for developing the clinical changes in the knee observed
4 by ultrasound imaging in symptomatic players.

5 Ultrasound imaging revealed that 57% of the symptomatic group exhibited pathological
6 changes in the knee joint. The odds ratios indicated that the prevalence of knee pain was 8.6-fold
7 higher in athletes with morphological modifications in the knee joint. Interestingly, the ultrasound
8 observations characteristic of Osgood–Schlatter syndrome were not accompanied by a lower
9 torque-generating ability (data not shown). This lack of an association suggests that, despite these
10 abnormalities, the tendon mechanical and material properties required to transmit force remain
11 unchanged. Nevertheless, overuse or repetitive microinjuries can damage the muscle–tendon
12 binding sites and, in turn, affect movement stability (Wild, Steele, & Munro, 2013). The
13 observations that the symptomatic patients were taller and that 13 of the 26 detected abnormalities
14 with ultrasound were characteristic of Osgood–Schlatter syndrome may indicate that the taller
15 players were in their growth spurt. This syndrome is associated with more anterior knee pain
16 because of inflammation of the PT at the tibial tuberosity beginning in late childhood.

17 None of the functional parameters differed significantly between symptomatic and
18 asymptomatic players, and they are therefore unlikely to explain the development of knee pain in
19 adolescent male basketball players. However, the symptomatic players were taller than the
20 asymptomatic players, and it is possible that their greater height reflected a higher growth rate.
21 This is significant because the incidence in the number of injuries during puberty (Brenner, 2007;
22 Frank, Jarit, Bravman, & Rosen, 2007) is positively related to the rate of increase in height
23 (Faulkner, Davison, Bailey, Mirwald, & Baxter-Jones, 2006).

1 One limitation of this study is that we did not assess the reproducibility of our **ultrasound**
2 **assessments and other tests**. However, the literature (cited in the description of the different tests)
3 shows good reliability and given that all procedures were performed by experienced researchers,
4 we believe that the reliability of the different tests would have been as high as that reported. A
5 second limitation is that anterior knee pain is multifactorial in nature, which makes assessment
6 complicated. It is possible that recall of pain in the previous month introduced some bias, but this
7 bias was minimized by asking the parents and coaches for confirmation of knee problems. **In**
8 **addition, it would be interesting if symptomatic players and asymptomatic players with**
9 **morphological abnormalities were followed to assess whether the symptoms were transient and**
10 **the morphological abnormalities preceded the development of symptoms**. Finally, we did not
11 assess the pubertal status of the participants, which would have helped us to better ascertain to
12 what extent the pain was associated with accelerated growth.

13

14

CONCLUSIONS

15 Side-to-side differences **of muscle strength, flexibility and neuromuscular control** in 14–15-
16 year-old male basketball players were not related to the early detection of anterior knee pain.
17 However, the risk of knee pain was higher in tall players and was often accompanied by
18 morphological abnormalities detected by ultrasound. The risk of pain was slightly related to a
19 lower lumbopelvic balance, which highlights the need to maintain core stability during athletic
20 conditioning.

21

Conflict of interest

23

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4 None.

5

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