

# Patellar ligament hypertrophy evaluated by magnetic resonance imaging in a group of professional weightlifters

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[Received 24 June 2012; Accepted 6 August 2012]

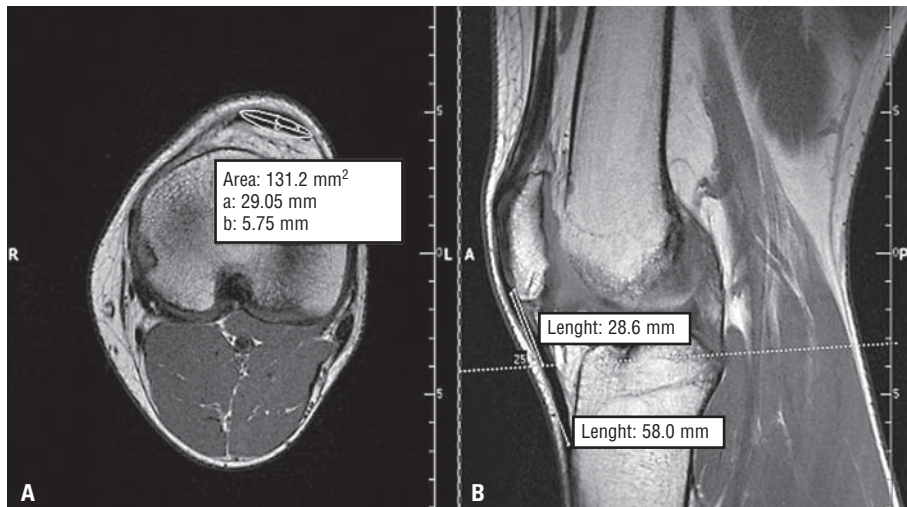
*The aim of this research was to investigate if perennial, professional weightlifting training, started at puberty, leads to hypertrophy of the patellar ligament (PL). The knee examinations were performed with a 1.5 T magnetic resonance imaging system. The area of the cross-sectional area (CSA) of the PL midsubstance was evaluated in T1-weighted images. A control group of 19 participants was also examined with the same protocol. A significant increase of the PL midregion CSA was observed in a group of weightlifters. The area of the PL midsubstance and the onset of training were very strongly, reversely correlated. This paper presents the first description of PL midregion hypertrophy due to professional weightlifting training initialised and continued from a pubertal spurt. The described overgrowth is more intensified than has been reported for other parts of the PL. Moreover, it has been observed in the region that is the least susceptible for injuries, which in another situation could also have led to increased volume of the PL. The described phenomenon should be considered by orthopaedic surgeons because it can influence the choice of the surgical technique for cruciate ligament reconstruction as the PL is one of the structures for harvesting autografts. (Folia Morphol 2012; 71, 4: 240–244)*

**Key words: patellar ligament, patellar tendon, hypertrophy, magnetic resonance imaging**

## INTRODUCTION

The patellar ligament (PL) is the most commonly injured anatomical structure in athletes who are engaged in sports that require jumping, such as volleyball, basketball, or running [12]. This ligament extends from an apex of the patella to the tibial tuberosity, conducting contraction of the quadriceps muscle. The PL in scientific literature in orthopedy is clinically known as the “patellar tendon” (PT). Dur-

ing knee flexion, the PL shifts laterally in the coronal plane and posteriorly in the sagittal plane. Moreover, its length sharply increases by about 6 mm as the knee flexes from full extension to 30°. Between 30° and 110°, the length remains relatively constant [3]. In the general population, the cross-sectional area (CSA) of the PT changes along its length from the proximal ( $84 \pm 8 \text{ mm}^2$ ), through the middle ( $75 \pm 8 \text{ mm}^2$ ), to the distal ( $124 \pm 7 \text{ mm}^2$ ) portion [1].



**Figure 1.** T1-weighted magnetic resonance images. The weightlifter; **A.** The patellar tendon area measured by a modifiable ellipsoid on the axial section; **B.** The measurement level (dashed line, in the half of the ligament length) on sagittal section.

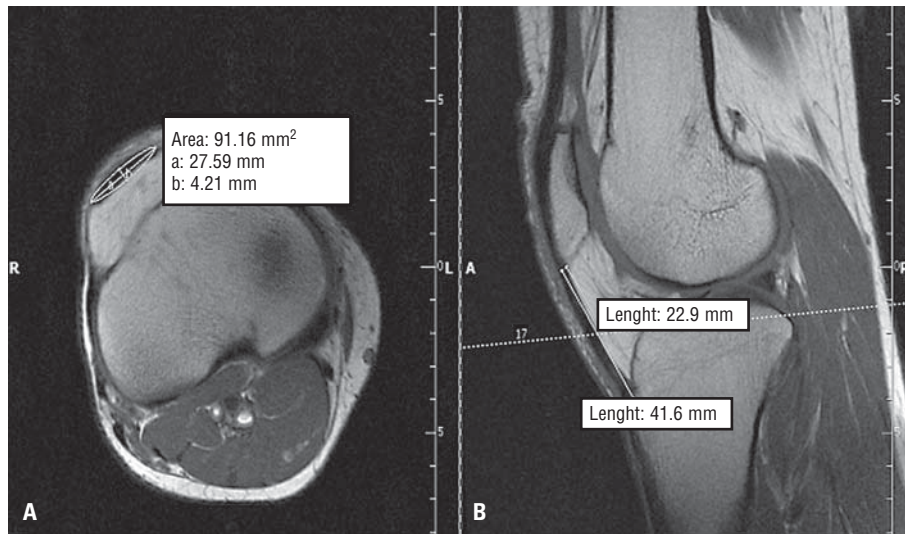
Although there is no consensus in the literature as far as PL hypertrophy due to exertion is concerned, most research suggests that this process is a protective adaptation to repeated injuries occurring during overload [7, 9, 13, 17]. On the contrary, this observation was made in a group of asymptomatic, high performance weightlifters who presented with neither history of PT trauma nor with any features of PT tendinopathy observed in magnetic resonance imaging (MRI) examinations. The aim of this report was to present and discuss probable causes of PL hypertrophy observed among high-performance weightlifters. To the authors' knowledge, such an intensified physiological hypertrophy of the PL mid-region has not been observed before.

## MATERIAL AND METHODS

Knee MRI examinations in high-performance weightlifters were carried out in the Department of Radiology in The Medical University of Lodz from October 2010 to November 2011. Assessment was a standard procedure carried out before the beginning of the training season. All athletes were representatives of the Polish National Weightlifting Team and participants of world, European, and Polish weightlifting championships. Nine weightlifters were submitted to the study. Due to pathological conditions in the joints of two sportsmen, only one knee was evaluated in each of those participants. The seven remaining subjects, who had both knees assessed, did not complain about PT region pain and they did not present any pain to palpation. More-

over, the morphology of the tendon assessed initially by ultrasonography showed no abnormalities.

The examinations were performed with an Avanto 1.5 T MRI system (Siemens, Germany), using a dedicated coil. All images were analysed retrospectively on a work station (Exhibeon, Pixel Technology, Poland) using software that allowed for the measurement of PL surface area by using a modifiable ellipsoid. The dimensions (cross-section) of the PL were evaluated on the basis of the T1-weighted images (parameters applied: TR 600 ms, TE 11 mm, FoV 160 mm, matrix  $320 \times 320$  mm, thickness 3 mm). The CSA was measured in the middle part of the PT (Figs. 1, 2). The level of measurement was determined on the basis of the sagittal section, mid distance between the patellar and the tibial attachment of the ligament. A control group of 19 males with no significant difference in age, height, and weight was also examined with the same protocol. None of the individuals from the control had participated in any systematic training or physical activity during the last year. The MRI examination was carried out due to pain in the knee joint. The primary ultrasonographic evaluation excluded the PL pathological condition. Data about the age, weight, and height was obtained for both groups. Additional information about the timespan of training and the age of training onset was collected from the sportsmen group. Collected data characterising the weightlifter group and the control is presented in a Table 1. The study complied with the Declaration of Helsinki and was approved by the local ethics committee.



**Figure 2.** T1-weighted magnetic resonance images. The control; **A.** The patellar tendon area measured by a modifiable ellipsoid on the axial section; **B.** The measurement level (dashed line, in the half of the ligament length) on sagittal section.

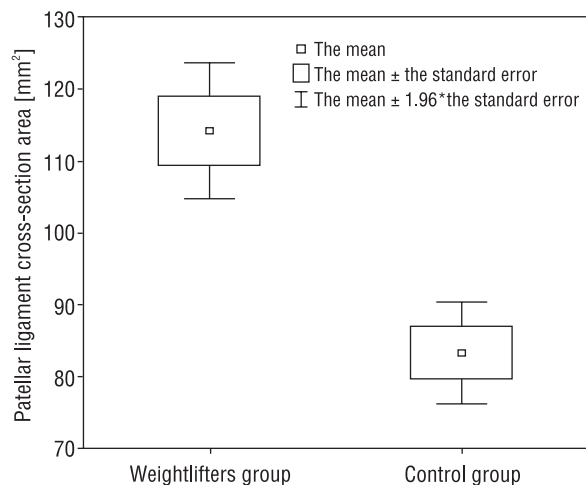
**Table 1.** Characterisation of the weightlifters group and the control

	Weightlifters (n = 16)				Control (n = 19)			
	Mean	Minimum	Maximum	Standard deviation	Mean	Minimum	Maximum	Standard deviation
Age [years]	26.1	21	34	4.2	26.6	19	36	5.3
Weight [kg]	92.6	78	120	14.8	86.6	72	106	8.8
Height [m]	1.78	1.68	1.88	0.06	1.8	1.69	1.9	0.05
Body mass index	29.1	27.1	34.7	2.7	26.8	23.8	31.7	2.2
Training participation [years]	15.5	10	25	4.7				
Age of training onset [years]	10.6	9	12	0.8				

For the statistical analysis the Mann-Whitney test, the t-test, and the Spearman's rank correlation test were employed.  $P < 0.05$  was rated as significant. Statistical analyses were performed using STATISTICA 9.1 (Statsoft, Tulsa, OK, USA).

### RESULTS

Mean CSA of the PL (PT) midregion was 37.1% greater in the weightlifter group than in the control group ( $114.29 \pm 19.3 \text{ mm}^2$  vs.  $83.38 \pm 15.82 \text{ mm}^2$ ). The significance of the difference was confirmed with the Mann-Whitney test (Fig. 3). Onset of the training was significantly and very strongly correlated with PT midregion CSA. The Spearman's coefficient was  $-0.74$ , which indicates that the younger a weightlifter was at the moment of the training onset the more increased was the observed PT hypertrophy. No influence of the training timespan on the PT CSA could be determined.



**Figure 3.** A box-and-whiskers diagram displaying difference in the cross-sectional area of the patellar tendon midregion between the weightlifters and the control group.

## DISCUSSION

A tendon tissue is not static and its matrix adapts in a process of remodelling mediated by tenocytes (tendon fibroblasts) according to the level, direction, and frequency of an applied load [15]. Collagen synthesis increases in the PT after vigorous endurance exercise and remains elevated for up to 72 hours post-exercise [9]. An additional reduction in metalloproteinase activity in the early recovery phase may support tendon overgrowth [15]. On the other hand, tendon hypertrophy seems to be secondary to changes in tendon composition and its material properties. Increased stiffness and elevated Young's modulus with no changes in PT CSA were found in subjects who had 8–14 weeks of resistance training [10, 14] or even in groups that had been training for years [11]. This is especially true for adult patients [13, 14] and may be linked to an age-related remodelling of the tendon, e.g. decrease in the number of tenocytes [2, 15]. This can result in insufficient production of new collagen fibres resulting in unobserved macroscopic hypertrophy. Similar inconsistencies concern potential Achilles tendon hypertrophy [8, 16]. Despite all the above, it is still uncertain what factors are responsible for the tendon hypertrophy process. It has been proven that a tendon's adaptation to stress is not constant along its length. Couppé et al. [1] analysed side-to-side differences in PT CSA in elite athletes. The lead extremity, which was on average 22% stronger than the non-lead extremity, had a greater distal and proximal PT CSA, which was not observed in the mid-tendon region. Seynnes et al. [17] and Kongsgaard et al. [9] drew similar conclusions. Hypertrophy of tendon attachments subjected to tendon-bone compressive forces may indicate that not only the intensity of loading but also the direction of the working force is a cause of uneven tendon overgrowth. On the other hand, most of the PL traumas occur at the insertion regions, which were defined as the most susceptible to overgrowth. Hypotheses explaining this phenomenon assume that heavy resistance training can induce micro-injuries of the tendon that remain undetected during examination and give no symptoms. Therefore, hypertrophy could result from the tendon repair and/or remodelling processes supporting protection for the weaker tendon regions [17]. Nevertheless, the hypertrophy observed in this research concerned the part of the tendon that is not susceptible to injury, which indicates that tendon overgrowth was probably caused by the process of physiological adaptation to repeated stress.

Another probable conclusion is that the compensatory reaction of tendon hypertrophy that is pathological in adult individuals is a natural process for youths during their pubertal growth spurt. Tissues prone to growth in this period can be induced into extensive hypertrophy in response to heavy resistance training. Moreover, youths demonstrate higher potential for regeneration processes that may be also manifested by intensive cell proliferation due to strain. In males, synergy of androgens and oestrogens is well defined in the process of the growth spurt, epiphyseal maturation, and bone mineral accretion [5]. Furthermore, tenocytes are susceptible to hormonal influence; hence, hormonal imbalances during puberty may stimulate musculoskeletal system metamorphosis [11]. The authors evaluated the CSA only at the level of the midregion where the plane was approximately perpendicular to a long tendon axis, ensuring the accuracy and precision of the measurements. In spite of the fact that this region was described as the least predisposed to hypertrophy and all of the weightlifters were free of any injuries in this part, 37% of tendon overgrowth was observed. This result is, to the authors' knowledge, the highest described in the literature. In previously published material, enlargement of PT CSA has been only 4–27% [2, 9, 17]. Such extensive hypertrophy is probably caused by more factors than those discussed above. All the athletes in this study began training before or during their pubertal spurt and continued until the end of maturation of the skeletal system. Other factors that may contribute to the described process are the characteristics of weightlifter training. Firstly, it comprises exercise in which sportsmen crouch with maximally bended knees, and secondly athletes usually increase their knee load by lifting additional weight. In this position the PT is supremely taut and high longitudinal strain is exerted. Also the period of training, although not significantly correlated with the intensity of hypertrophy, probably plays an additive role. Morphological changes in the tendon due to stress accumulate more slowly than in muscle [17], which may explain the absence of observed changes in other reports where the training protocol comprises only a few weeks of training [10, 14] with no overloads that are typical for professional weightlifting training. All the aforementioned causes of PT overgrowth seem to be confirmed by observation of cruciate ligament hypertrophy in professional weightlifters [6]. Statistical analyses indicate that the age of training onset is more important to the process of PL hyper-



trophy than the duration of the training. Clinical implications of PT hypertrophy may be injury risk reduction resulting from an increase in tendon tissue strength of about 50 to 100 N per every additional mm<sup>2</sup> of CSA. Moreover, it has been demonstrated that patients with wider PT who had bone-patella-bone autograft ACL reconstruction recovered faster after the surgery and did better 3 months postoperatively than those with smaller tendons [4].

There are some limitations in this study. Firstly, the PT attachment regions were not assessed. The analysis concerned only the PL midregion in order to evaluate the physiological adaptation and to eliminate the influence of the enthesopathies. Secondly, due to the observant character of this research, no histological samples could be obtained, so the morphology and the mechanical properties of hypertrophied tissue remain unknown. Although it disabled detailed analysis of physiological hypertrophy of the PT, the authors of this research believe that data obtained from the medical history and the physical examination enabled reliable analysis of the observed phenomenon.

## CONCLUSIONS

In conclusion, the process of tendon hypertrophy is probably provoked by many factors like the level, direction, and frequency of an applied force as well as the loading history. Nevertheless, the most likely factor responsible for the described physiological overgrowth is a training onset overlapping the pubertal growth spurt. It seems to confirm the hypothesis that the immature musculoskeletal system is highly prone to stress stimuli, and that muscle tendons adapt to increased loading primarily through the process of overgrowth. Additionally, the presented report is another example confirming the well-accepted statement that some of the anatomical dimensions that were evaluated in the general population find no reflection in data obtained from sportsmen involved in heavy resistance training.

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