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Structural Achilles tendon properties in athletes subjected to different exercise modes and in Achilles tendon rupture patients

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Kongsgaard, M., P. Aagaard, M. Kjaer, and S. P. Magnusson. Structural Achilles tendon properties in athletes subjected to different exercise modes and in Achilles tendon rupture patients. *J Appl Physiol* 99: 1965–1971, 2005. First published August 4, 2005; doi:10.1152/japplphysiol.00384.2005.—The prevalence of Achilles tendon (AT) injury is high in various sports, and AT rupture patients have been reported to have a 200-fold risk of sustaining a contralateral rupture. Tendon adaptation to different exercise modes is not fully understood. The present study investigated the structural properties of the AT in male elite athletes that subject their AT to different exercise modes as well as in Achilles rupture patients. Magnetic resonance imaging of the foot and leg, anthropometric measurements, and maximal isometric plantar flexion force were obtained in 6 male AT rupture patients and 25 male elite athletes (kayak/control group $n = 9$, volleyball $n = 8$ and endurance running $n = 8$). AT cross-sectional area (CSA) was normalized to body mass. Runners had a larger normalized AT CSA along the entire length of the tendon compared with the control group ($P < 0.05$). The volleyball subjects had a larger normalized CSA compared with the control group ($P < 0.05$) in the area of thinnest tendon CSA. No structural differences of the AT were found in the rupture subjects compared with the control group. Rupture subjects did not subject their AT to greater force or stress during a maximal voluntary isometric plantar flexion than the other groups. The CSA of the triceps surae musculature was the strongest predictor of AT CSA ($r_s = 0.569$, $P < 0.001$). This study is the first to show larger CSA in tendons that are subjected to intermittent high loads. AT rupture patients did not display differences in structural or loading properties of the tendons.

tendon rupture risk; tendon cross-sectional area; tendon loading

ON THE BASIS OF IN VITRO MEASUREMENTS, most tendons, including the human Achilles tendon, are believed to have a breaking stress of about 100 MPa (6, 19, 48). However, whereas most tendons experience peak stresses below 30 MPa, the human Achilles tendon may experience peak stresses in excess of 70 MPa during maximal eccentric plantar flexions (19, 21). Therefore, the human Achilles tendon has a safety factor of ~1.5, whereas most tendons have a safety factor of 4 or greater (3, 19).

Tendons subjected to considerable stress, including the Achilles tendon, are believed to operate as springs by storing and releasing elastic strain energy during locomotion (3, 9, 19). From this standpoint it is advantageous to have a thin (and long) tendon (3, 19). On the other hand, a thicker tendon, which would yield less strain energy, would reduce the average stress (force/area) across the tendon and thereby provide a greater safety margin. However, it is possible that the relatively small cross-sectional area (CSA) and the large loads may explain why the human Achilles tendon is more exposed to overload injuries than most other human tendons. In fact, the human Achilles tendon is the most common tendon to rupture spontaneously (45% of all tendon ruptures), and the prevalence of Achilles tendinopathy is considerable in a variety of different sports, especially in runners and various ball sports (7, 8, 19, 37, 38). However, it remains largely unknown whether different forms of loading patterns are associated with tendon hypertrophy and/or qualitative changes of the human tendon tissue, which may be valuable in attempts to reduce the incidence rate of Achilles tendon injuries, including ruptures.

Studies using animal models have shown that the CSA and strength of tendons either increase (40, 46, 47), decrease (46), or remain unchanged (5, 41) in response to endurance training, and do thus not provide a coherent picture. It appears that the tendons of immature but not mature animals may hypertrophy in response to training (10, 39), and this may explain some of the inconsistencies between reports. Human cross-sectional studies have shown that runners have significantly larger Achilles tendon CSA than nonrunners (30, 36), which seems to corroborate recent reports of increased collagen synthesis after an acute bout of prolonged running (36 km) in trained runners (25). In contrast, 9 mo of running training did not increase Achilles CSA in untrained subjects (12). However, the tendon response to intermittent high loads (as those seen in jumping, sprinting and heavy strength training) is scarcely investigated. Studies using animal models have shown an increased tendon CSA after intermittent high-load training (4, 40), whereas studies on humans have not demonstrated any tendon hypertrophy with intermittent high-load training (22, 23, 35).

The etiology of Achilles tendon ruptures remains unresolved, despite its considerable incidence rate. Approximately 80% of Achilles tendon ruptures occur 3–6 cm above the calcaneal insertion (1, 11, 27). The specific site of rupture has traditionally been explained by a poor blood supply or by a local variation in the strain topography and focal stress concentration (2, 16, 20). However, it has been suggested that tendon ruptures are almost always preceded by pathological alterations related to overuse and tendinopathy (1, 16, 18). Curiously, it was recently shown that patients with a previous Achilles tendon rupture had a 200-fold risk of sustaining a contralateral rupture (1), although compensatory mechanisms associated with the initial rupture cannot be ruled out. Furthermore, because the incidence of tendon ruptures in western countries far exceeds that in Africa and East Asia it is possible that genetic factors play a role (14, 33). Thus it remains unknown whether Achilles tendon ruptures are related to predisposing factors or loading alone.

The aims of the present study were *1)* to investigate the structural properties of the Achilles tendon in male elite athletes regularly subjecting their Achilles tendons to different exercise modes and *2)* to compare that with the structural properties of subjects having sustained an Achilles tendon rupture to the contralateral leg.

MATERIALS AND METHODS

Subjects

The study conformed with the Helsinki II Declaration, and it was approved by the Copenhagen Ethics Committee. All subjects gave written informed consent to participate. Six male previous Achilles tendon rupture patients (mean ± SE age, height, and weight: 31.0 ± 0.7 yr, 181.5 ± 2.8 cm, 86.0 ± 6.3 kg) were recruited. All patients had sustained a spontaneous and complete unilateral rupture of the Achilles tendon and were included on average 1.2 yr postsurgery. Eight male elite volleyball players who had played in the highest division in Denmark for at least 3 yr were included to represent individuals subjected to intermittent Achilles tendon loads (mean ± SE age, height and weight: 25.3 ± 0.8 yr, 193.8 ± 2.8 cm, 89.5 ± 2.6 kg). Eight male elite long-distance runners (from the Danish national team, +80 km/wk for > 3 yr) were included to represent individuals subjected to repetitive Achilles tendon loads (mean ± SE age, height and weight: 28.6 ± 2.2 yr, 182.4 ± 2.7 cm, 69.5 ± 2.9 kg). Also, we included nine male elite kayak paddlers from the Danish national team to represent physically well-disposed young men, who had not subjected their Achilles tendons to exercise modes differently from normal young men (mean ± SE age, height and weight: 20.5 ± 1.1 yr, 181.9 ± 1.3 cm, 79.9 ± 1.9 kg). The kayak subjects were chosen because Achilles tendon stress and strain are minimal during kayaking. Thus the results for the kayak subjects will be regarded as reference/control values. The age and anthropometrical data of the subject groups are shown in Table 1. All elite athletes had a background of >7 h of specific training per week for at least 4 yr. All measurements of the rupture subjects were performed on their noninjured leg. For the athletes, all measurements were conducted on their preferred leg.

Measurements

Anthropometrical and force measurements. All subjects had their leg length (cm) measured. Leg length was defined as the distance from the palpated knee-joint line to the floor, and was measured with the subjects in a standing position. To measure maximal isometric plantar flexion force [MVC (N)] subjects were seated within a metal frame with the hip flexed to 90° and the knee fully extended as previously described (12, 36). In brief, the dominant, or noninjured, foot rested against a metal plate at 90° to the leg and with an axis of rotation corresponding to the lateral malleolus. Plantar flexion force (N) was measured by a strain-gauge load cell connected to the steel plate. The force signal was continuously recorded during 4 s and sampled at 50

Hz using an external analog-to-digital converter (DT 2801A, Data Translation, Malboro, MA). After five submaximal plantar flexions, which served as warm-ups, the subjects performed five maximal isometric plantar flexion efforts. Each maximal effort lasted for ~4 s separated by a 1-min rest period. Plantar flexion force was converted to moment (N·m) by multiplying the force measurement by the moment arm of the load cell. The highest obtained value of plantar flexion moment produced in any of the five efforts was used for analysis.

Magnetic resonance imaging (MRI). All subjects were instructed to refrain from any sports activities 24 h before the MR scans, to avoid any potential fluid accumulation. Sagittal plane MR T1-weighted spin echo (SE) images (GE Signa horizon LX 1.5 T) were obtained with the ankle in neutral position (90°), and with the following MR parameters: repetition time/echo time (TR/TE): 675/18 ms, field of view (FOV) 26 cm, matrix 384 × 256, slice thickness 3 mm and spacing 0 mm. To estimate the Achilles tendon moment arm a modified Reuleaux method was applied on the sagital images as previously described (28, 29, 42). The measurement of the moment arm from the MR images was performed three times for each subject, and the mean value was used for further analysis. Peak Achilles tendon force was calculated by dividing the recorded plantar flexion moment with the tendon moment arm. Although antagonist coactivation and joint rotation may influence the calculation of tendon force these were not measured, because previous studies have shown these to be marginal using the same force-measurement set-up as the present study (12, 29, 36). Free Achilles tendon length (dL) was obtained by measuring the distance from the most distal soleus insertion in the Achilles tendon to the tendon insertion in the calcaneus on the sagittal MR images (Fig. 1). Achilles tendon cross-sectional area was obtained from axial MR images (T1 weighted SE, TR/TE: 400/15 ms, FOV 12 cm, matrix 320 × 320, slice thickness 6 mm, spacing 2 mm). The cross-sectional area of the Achilles tendon was measured in the interval 3.2–12.0 cm above the inferior margin of the calcaneus. To avoid investigator bias an automated software routine was used to automatically outline and calculate the Achilles tendon CSA (mm²) using NIH Image software (Fig. 2) (http://rsb.info.nih.gov/nih-image). The mean value of three measurements of the same image was used for analysis. In each individual subject peak Achilles tendon stress (MPa) was calculated by dividing peak Achilles force (N) by the narrowest obtained Achilles tendon CSA (mm²). The area of the narrowest obtained Achilles tendon CSA (i.e., the narrowest, and presumably the weakest, portion of the tendon) is reported as narrowest Achilles tendon CSA. The anatomic CSA (ACSA) of the triceps surae was measured by axial MR imaging (T1 weighted SE, TR/TE: 400/16 ms, FOV 24 cm, matrix 256 × 256, slice thickness 10 mm, spacing 10 mm). The triceps surae ACSA was obtained 0–26 cm distally to the tibia plateau. Subsequently, the ACSA of soleus, gastrocnemius medialis, and gastrocnemius lateralis were visually outlined and measured using the scanner software program WEB 1000 (Agfa)(Fig. 3). The mean value of three measurements of the same image was used for analysis. The largest ACSA

Table 1. *Characteristics of the groups*

	Kayak (*n* = 9)	Running (*n* = 8)	Volleyball (*n* = 8)	Rupture (*n* = 6)
Age, yr	20.5±1.1	28.6±2.2[b]	25.3±0.8[a]	32.0±0.7[be]
Body weight, kg	79.9±1.9[c]	69.5±2.9	89.5±2.6[ad]	86.0±6.3[d]
Years of elite training, yr	7.4±0.8	6.8±0.8	7.0±0.9	0
Height, cm	181.9±1.3	182.4±2.7	193.8±2.8[bcg]	181.5±2.8
Leg length, cm	51.7±0.4	52.9±0.9	57.8±1.0[bcf]	53.8±0.7[a]

Values are means ± SE; *n,* no. of subjects. Anthropometric data on the participating subject groups are given. Years of elite training was defined as number of years with >7 h of specific training per week. Leg length was measured as the distance from the palpated knee joint line to the floor. [a]Significantly greater value than kayak, $P < 0.05$. [b]Significantly greater value than kayak, $P < 0.01$. [c]Significantly greater value than running, $P < 0.05$. [d]Significantly greater value than running, $P < 0.01$. [e]Significantly greater value than volleyball, $P < 0.01$). [f]Significantly greater value than rupture, $P < 0.05$. [g]Significantly greater value than rupture, $P < 0.01$.

Fig. 1. Sagittal magnetic resonance image of the ankle. Free Achilles tendon length (dL) is defined as the distance from the most distal insertion of the soleus muscle in the Achilles tendon to the distal insertion of the tendon at the calcaneus.

for all three individual muscles were summed to give the maximal triceps surae ACSA.

Normalization of Data

The anthropometrical characteristics differed considerably between the different subject groups (see Table 1). The stresses imposed on the muscles and tendons are dependent on the applied loads, which largely depend on the body mass of the individual. Hence, to be able to compare the structural tendon properties and the functional performance between subject groups, tendon CSA, muscle ACSA, and

Fig. 2. Axial magnetic resonance image of the Achilles tendon. Cross-sectional area of the tendon recognized by the software and used for analysis is outlined.
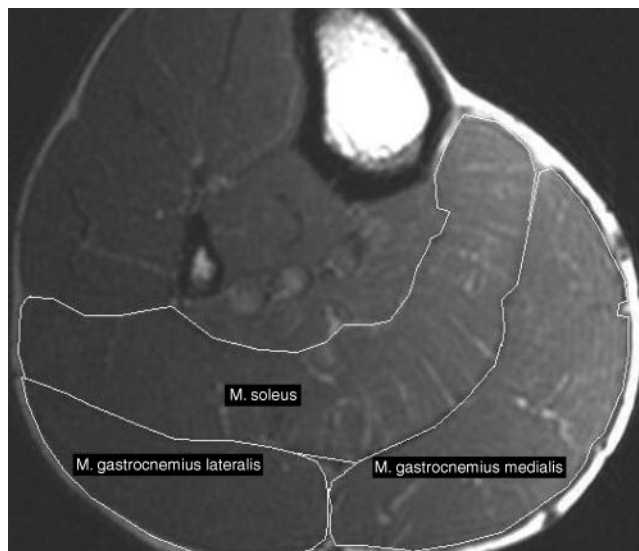

Fig. 3. Axial magnetic resonance image of the triceps surae muscle group. Cross-sectional areas of the muscles used for calculation are outlined.

strength parameters were normalized to body mass to the power of $3/4$ ($m^{3/4}$). The power of $3/4$ was chosen because allometric parameters that relate surfaces (e.g., tendon CSA) to body mass are closer to $3/4$ than to the 2/3 predicted by geometric similarity (32). The free Achilles tendon lengths were normalized to leg lengths (% of leg length).

Statistical Analysis

All data are presented as group means $\pm$ SE. The statistical analysis of the data was performed with SPSS standard version (10.0.5). The data were not normally distributed, and therefore nonparametric statistics were applied. Kruskal-Wallis one-way analysis of variance was used to detect overall significant differences between groups followed by Mann-Whitney test. Spearman's rho (r_s) was used to examine rank-order correlations between variables. All tests were two-tailed with an alpha level of $P < 0.05$.

RESULTS

Runners showed significantly larger normalized Achilles tendon CSA than kayak subjects from 4.8 to 9.6 cm and at 12.0 cm proximal to the inferior margin of the calcaneus (Fig. 4; $P < 0.05$). Furthermore, runners also had a greater tendon CSA than rupture subjects at 4.8 cm ($P < 0.05$) and between 7.2 and 10.4 cm ($P < 0.05$). Volleyball subjects had significantly greater normalized Achilles tendon CSA than kayak subjects at the 8.0-, 10.4-, 11.2-, and 12.0-cm levels ($P < 0.05$), as well as a greater tendon CSA than rupture subjects at the 9.6-, 10.4-, and 12.0-cm levels($P < 0.05$).

The normalized Achilles tendon CSA of rupture patients did not differ from that of kayak subjects at any level (Fig. 4). The narrowest normalized Achilles CSA was significantly higher in runners than in kayak ($+21\%$) and rupture subjects ($+24\%$; $P < 0.05$; Table 2). Also, the narrowest normalized Achilles CSA tended to be significantly greater in volleyball subjects than in kayak subjects ($+15\%$; $P = 0.075$; Table 2). All subjects had their narrowest Achilles tendon CSA at 7.2–10.4 cm, which corresponds to 3–6 cm above the Achilles insertion on the calcaneus. There were no significant differences in maximal Achilles tendon CSA between any of the groups (Table 2). There were no significant differences in either
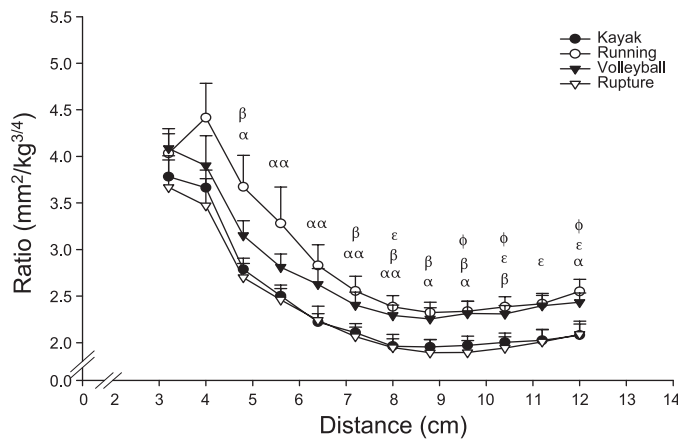
Fig. 4. Achilles tendon cross-sectional area normalized to bodyweight$^{3/4}$ at distances above the inferior margin of the calcaneus. Values are means ± SE. $^{\alpha}$Running significantly greater value than Kayak, $P < 0.05$. $^{\alpha}$Running significantly greater value than Kayak ($P < 0.01$). $^{\beta}$Running significantly greater value than Rupture ($P < 0.05$). $^{\epsilon}$Volleyball significantly greater value than kayak ($P < 0.05$). $^{\phi}$Volleyball significantly greater value than Rupture ($P < 0.05$).

absolute or normalized free Achilles tendon length between any of the groups, although individual range was very large (range: 13–26% of leg length) (Table 2).

Volleyball subjects had a significantly greater maximal triceps surae ACSA than the other groups (Table 2). Similarly, the volleyball subjects had a significantly greater normalized triceps surae ACSA than kayak subjects (+17%; $P < 0.01$). Runners also had a significantly greater normalized triceps surae ACSA than kayak subjects (+11%; $P < 0.05$; Table 2). The normalized triceps surae ACSA of the rupture subjects did not differ from that of the control group (Table 2).

Volleyball subjects had greater normalized plantar flexion strength than the runners (+27%; $P < 0.01$), kayak subjects (+23%; $P < 0.05$), and rupture subjects (+36%; $P < 0.05$) (Table 2). Also the volleyball subjects had a significantly greater absolute plantar flexion strength than the other groups (Table 2). There were no significant differences in normalized plantar flexion strength between the runners, kayak, and rupture groups (Table 2). When maximal plantar flexion force was converted to maximal Achilles tendon force, volleyball subjects had significantly greater Achilles tendon force than the

other three groups during the maximal plantar flexion effort (Table 2; $P < 0.05$). The volleyball subjects did not display greater stress than the kayak or the rupture group. However, the kayak and volleyball subjects displayed significantly greater peak Achilles tendon stress than the runners during maximal plantar flexion effort ($P < 0.05$; Table 2). The peak Achilles tendon stress of the rupture subjects was not significantly greater than any of the other groups (Table 2). Additionally, none of the rupture subjects (0/6) reported any symptoms before rupture.

The narrowest Achilles tendon CSA correlated to maximal tendon force ($r_s = 0.449$, $P < 0.05$) maximal triceps surae ACSA ($r_s = 0.569$, $P < 0.01$), maximal soleus ACSA ($r_s = 0.520$, $P < 0.05$) and maximal gastrocnemius ACSA ($r_s = 0.458$, $P < 0.01$). The predictability of the narrowest CSA of the Achilles tendon did not improve using multiple regression analysis. Thus maximal triceps surae ACSA remained the best predictor of narrowest Achilles tendon CSA.

DISCUSSION

The primary findings of this study were that persons who subjected the Achilles tendon to repetitive loads (runners) and intermittent loads (volleyball players) had a larger normalized Achilles tendon CSA compared with non-weight-bearing athletes (kayakers) and subjects with a previous Achilles tendon rupture. Furthermore, both the strength and the CSA of the triceps surae muscles were related to the Achilles tendon CSA. Additionally, the structural properties of the Achilles tendon in subjects who had previously sustained an Achilles tendon rupture did not differ from the control subjects (kayakers). During maximal isometric plantar flexion the load and resulting tendon stress of the tendon were similar in this patient group and the controls (kayakers). Thus these measures are unlikely to explain the reported 200-fold increase in risk of getting a contralateral rupture (1).

In the present study, the CSA of the Achilles tendon was measured continuously along the length of the free tendon to obtain a CSA value at the narrowest and therefore presumably the weakest (greatest stress) segment of the tendon (narrowest Achilles tendon CSA). The narrowest Achilles tendon CSA in the present study ranged from 50.8 mm^2 for the kayak group to 63.5 mm^2 for the volleyball group. Previous studies have reported narrowest Achilles tendon CSA in the range from 33.8

Table 2. *Absolute and normalized Achilles tendon properties*

	Kayak ($n = 9$)	Running ($n = 8$)	Volleyball ($n = 8$)	Rupture ($n = 6$)
Achilles tendon moment arm, mm	52.0±0.7	52.8±1.5	57.4±1.8^{ace}	53.3±0.6
Free Achilles tendon length, mm	49.3±6.1	70.5±7.9	52.1±4.9	54.0±5.4
Normalized free Achilles tendon length, %	9.6±1.2	13.3±1.4	9.0±0.7	10.1±1.1
Maximal Achilles tendon CSA, mm^2	101±5.6	106±8.6	119±5.9	101±5.4
Narrowest Achilles tendon CSA, mm^2	50.8±2.9	55.1±2.1	63.5±2.9^{ace}	51.5±1.2
Narrowest normalized Achilles tendon CSA, mm^2/kg$^{3/4}$	1.9±0.1	2.3±0.1^{ae}	2.2±0.1	1.9±0.1
Maximal triceps surae ACSA, mm^2	4,553±165	4,546±181	5,804±285^{bde}	5,000±196
Normalized maximal triceps surae ACSA, mm^2/kg$^{3/4}$	170±3.9	189±5.1^{a}	199±6.8^{b}	180±10.0
Plantar flexion strength, N·m	188±12.9	164±9.4	254±16.4^{adf}	178±12.7
Normalized plantar flexion strength, N·m/kg$^{3/4}$	7.06±0.46	6.85±0.36	8.70±0.43^{ade}	6.42±0.55
Maximal Achilles tendon force, N	3,619±235	3,127±174	4,421±228^{ade}	3,347±234
Peak Achilles tendon stress, MPa	72.4±5.2^{c}	57.4±4.2	70.3±4.12^{c}	65.0±4.2

Values are means ± SE; *n*, no. of subjects. Normalized free Achilles tendon length (%) was calculated as free Achilles tendon length divided by leg length. ^{a}Significantly greater value than kayak, ($P < 0.05$). ^{b}Significantly greater value than kayak, $P < 0.01$. ^{c}Significantly greater value than running, $P < 0.05$. ^{d}Significantly greater value than running, $P < 0.01$. ^{e}Significantly greater value than rupture, $P < 0.05$. ^{f}Significantly greater value than rupture, $P < 0.01$.

to 127 mm^2 in untrained subjects (12, 29, 30, 36). Thus the measured narrowest Achilles CSA in the present study falls well within previously reported values. However, the range of previously reported values is substantial, which may be related to different measurement and analysis methods.

On the basis of in vitro tests, the failure stress of tendons are generally considered to be close to 100 MPa (6, 19, 48), and the safety factor is around eight for most tendons (3, 19). In our study, peak Achilles tendon stress during maximal isometric plantar flexion was 74 MPa for the kayak subjects, 57 MPa for the runners, 70 MPa for the volleyball subjects and 65 MPa for the rupture subjects, which yields a safety factor of ~1.5. Therefore, even a small reduction in the tensile strength of the Achilles tendon might increase the risk for sustaining a tendon rupture or suffering an overload-associated injury. Hypothetically the safety margin can be augmented by an increase in tendon CSA and thereby the failure strength. In the present study it was shown that although volleyball players can generate greater forces during plantar flexion the resulting tendon stress was similar to that of the kayakers and rupture subjects as a function of the tendon CSA. Similarly, the relatively low tendon stress in runners can be ascribed to the greater tendon CSA (see Table 2). However, to what extent the tendon CSA is related to injury risk remains to be established.

Eighty percent of all Achilles tendon ruptures occur 3–6 cm above the calcaneal insertion (1, 11, 27), which corresponded to the narrowest Achilles tendon CSA of the present data. The narrowest section of the tendon will be subjected to the greatest stress for a given load. Therefore, we examined its relationship to maximal triceps surae ACSA, maximal soleus ACSA and/or maximal gastrocnemius ACSA. Approximately 30% of the variance in tendon CSA could be explained by the muscle characteristics, and maximal triceps surae ACSA was the best predictor of the narrowest Achilles tendon CSA. So if tendon CSA adapts to muscle size or strength, physical activity such as resistance training could theoretically be a potent stimulus for tendon hypertrophy.

To our knowledge, the present study is the first to demonstrate a larger tendon CSA in human tendons subjected to high tensile tendon loading. Numerous animal studies have examined the effect of endurance training on tendon tissue properties, while the effect of intermittent high loading is scarce. Birch et al. investigated the tendon CSA in horses subjected to 18 mo of high- or low-intensity training (4). There appeared to be an intensity-specific tendon hypertrophy with high-intensity training, but not with low-intensity training (4). Similarly, the Achilles tendon CSA in rats subjected to 4 mo of high-intensity training increased (40). However, no previous human studies have demonstrated an increased tendon CSA in response to training involving intermittent high-tension tendon loading. It has recently been shown that 14 wk of strength training in elderly subjects did not result in increased patella tendon CSA, but a remarkable increase in stiffness and Young's modulus (35). Others have shown that 8–12 wk of isometric strength training in young men did not increase patella or Achilles tendon CSA (22, 23). However, it is possible that 14 wk or less of resistance training is insufficient time to detect measurable changes in tendon CSA.

The larger Achilles tendon CSA in runners confirms previous findings by Rosager et al. (36), who showed that runners had markedly greater Achilles tendon CSA (+22%) than age-matched controls. Additionally, runners had a 36% greater Achilles tendon CSA in the most distal part of the tendon compared with nonrunners (30). Recent human studies have demonstrated that collagen I synthesis is increased in response to acute endurance running (13, 24, 25). Taken together these data suggest that endurance training may induce tendon hypertrophy. On the other hand it was recently shown that 9 mo of endurance running in untrained subjects did not result in Achilles tendon hypertrophy (12). Similarly, Buchanan and Marsh (5) did not find an increase in tendon CSA in guinea fowls subjected to 12 wk of endurance running. Interestingly, 12 mo of endurance training in pigs increased tendon CSA by 21%, while a reduction of CSA was noted after 3 mo (45, 47). Such a biphasic response may explain the lack of tendon hypertrophy in the study by Hansen et al. (12), i.e., that tendon hypertrophy is preceded by atrophy and that a significant tendon hypertrophy might have occurred if the intervention period had been prolonged. Thus it is possible that in humans it requires long-term (years) training for tendon hypertrophy to take place. Another possible factor for the incoherent picture of with respect to tendon hypertrophy is maturation. Studies investigating the effect of endurance training have often used immature animals (40, 46, 47), and data suggest that exercise is more likely to result in tendon hypertrophy in immature than mature animals (10, 39). Thus it cannot be discounted that the greater Achilles tendon CSA seen in runners and volleyball subjects may be a result of loading during the growing years, or of genetic factors.

It was recently shown that a tendon segment with a length beyond 80 mm displayed inferior fatigue quality (increased creep and decreased time to rupture), which suggests that tendons are composed of longitudinal 'structural units' below 80 mm (43). If these structural units do not span the entire length of the tendon there must be a pathway for force transduction which may also involve shear forces. Albeit purely speculative, a long tendon would experience a greater amount of shear force during loading, making it more prone to fatigue rupture. The notion that tendon length may affect fatigue quality and the fact that Achilles tendon rupture patients have a 200-fold increased risk of sustaining a contralateral rupture prompted us to investigate tendon length as a possible predisposing factor. However, the subjects of the present study with a prior Achilles tendon rupture had similar structural dimensions of the tendon and produced similar stress on the tendon during maximal isometric plantar flexion compared with the other subject groups. Others have also been unable to show a relationship between tendon length and injury (26). Therefore, features predisposing subjects to Achilles tendon ruptures are most likely intrinsic to the tendon. It has been shown that the number of medium to large-size fibrils (diameter of 60–150 nm) in the distal core of the Achilles tendon is reduced in ruptured Achilles tendons compared with healthy tendons (31). Similarly, Józsa et al. (17) have shown that the average size of collagen fibers in ruptured tendons are decreased. Whether this reduced number of larger fibrils is a genetic factor or whether it is developed from overloading remains unknown.

In the present study all the rupture subjects were asymptomatic before the rupture, which is in accordance with previous studies (1, 16, 31) and supports the notion that painful tendinopathy does not precede Achilles tendon rupture. However,

despite the absence of pain before ruptures it has been shown that all tendon ruptures (including those of the Achilles tendon) display histopathological alterations indicative of degenerative changes (15, 18). It has also shown that achillodynia-related tendon swelling and decreased echotexture were strongly related to the risk of sustaining an Achilles tendon rupture, whereas no ruptures occurred in tendons sonographically characterized as normal (34). Additionally, data on horse tendons have shown substantial exercise-induced hyperthermia (43–45°C) during sustained gallop, which may cause fibroblast death (44). This may potentially reduce the ability to adapt to habitual loading, although this remains to be established in a human model. Thus the etiology of Achilles tendon ruptures remains poorly understood, and additional studies should address cellular and molecular factors potentially associated with increased risk of Achilles tendon injury. Furthermore, long-term studies that examine the effect of different training regimes on tendon properties and injury risk are warranted.

In summary, the present study show that persons who subject their Achilles tendon to repetitive loads (runners) and to intermittent loads (volleyball players) had a larger normalized Achilles tendon CSA than control subjects. Furthermore, both strength and the CSA of the triceps surae muscles were positively related to the Achilles tendon CSA. Additionally, we did not find any differences in structural or loading capacity of the Achilles tendon in subjects who had previously ruptured their contralateral Achilles tendon compared with control subjects.

GRANTS

REFERENCES

1. **Aroen A, Helgo D, Granlund O, and Bahr R.** Contralateral tendon rupture risk is increased in individuals with a previous Achilles tendon rupture. *Scand J Med Sci Sports* 14: 30–33, 2004.
2. **Astrom M and Westlin N.** Blood flow in chronic Achilles tendinopathy. *Clin Orthop* 166–172, 1994.
3. **Biewener AA and Roberts TJ.** Muscle and tendon contributions to force, work, and elastic energy savings: a comparative perspective. *Exerc Sport Sci Rev* 28: 99–107, 2000.
4. **Birch HL, McLaughlin L, Smith RK, and Goodship AE.** Treadmill exercise-induced tendon hypertrophy: assessment of tendons with different mechanical functions. *Equine Vet J Suppl* 30: 222–226, 1999.
5. **Buchanan CI and Marsh RL.** Effects of long-term exercise on the biomechanical properties of the Achilles tendon of guinea fowl. *J Appl Physiol* 90: 164–171, 2001.
6. **Butler DL, Grood ES, Noyes FR, Zernicke RF, and Brackett K.** Effects of structure and strain measurement technique on the material properties of young human tendons and fascia. *J Biomech* 17: 579–596, 1984.
7. **Clement DB, Taunton JE, and Smart GW.** Achilles tendinitis and peritendinitis: etiology and treatment. *Am J Sports Med* 12: 179–184, 1984.
8. **Cook JL, Khan KM, and Purdam C.** Achilles tendinopathy. *Man Ther* 7: 121–130, 2002.
9. **Cutts A, Alexander RM, and Ker RF.** Ratios of cross-sectional areas of muscles and their tendons in a healthy human forearm. *J Anat* 176: 133–137, 1991.
10. **Elliott DH.** Structure and function of mammalian tendon. *Biol Rev Camb Philos Soc* 40: 392–421, 1965.
11. **Galloway MT, Jokl P, and Dayton OW.** Achilles tendon overuse injuries. *Clin Sports Med* 11: 771–782, 1992.
12. **Hansen P, Aagaard P, Kjaer M, Larsson B, and Magnusson SP.** Effect of habitual running on human Achilles tendon load-deformation properties and cross-sectional area. *J Appl Physiol* 95: 9375–9380, 2003.
13. **Heinemeier K, Langberg H, Olesen JL, and Kjaer M.** Role of TGF-β_1 in relation to exercise-induced type I collagen synthesis in human tendinous tissue. *J Appl Physiol* 95: 2390–2397, 2003.
14. **Jozsa L and Kannus P.** Histopathological findings in spontaneous tendon ruptures. *Scand J Med Sci Sports* 7: 113–118, 1997.
15. **Jozsa L and Kannus P.** Spontaneous rupture of tendons. In: *Human Tendons.* Champaign, IL: Human Kinetics, 1997, p. 254–325.
16. **Jozsa L, Kvist M, Balint BJ, Reffy A, Jarvinen M, Lehto M, and Barzo M.** The role of recreational sport activity in Achilles tendon rupture. A clinical, pathoanatomical, and sociological study of 292 cases. *Am J Sports Med* 17: 338–343, 1989.
17. **Jozsa L, Lehto M, Kvist M, Balint JB, and Reffy A.** Alterations in dry mass content of collagen fibers in degenerative tendinopathy and tendon-rupture. *Matrix* 9: 140–146, 1989.
18. **Kannus P and Jozsa L.** Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. *J Bone Joint Surg Am* 73: 1507–1525, 1991.
19. **Ker RF, Alexander McN R, and Bennett MB.** Why are mammalian tendons so thick? *J Zool Lond* 216: 309–324, 1988.
20. **Kjaer M.** Role of extracellular matrix in adaptation of tendon and skeletal muscle to mechanical loading. *Physiol Rev* 84: 649–698, 2004.
21. **Komi PV, Fukashiro S, and Jarvinen M.** Biomechanical loading of Achilles tendon during normal locomotion. *Clin Sports Med* 11: 521–531, 1992.
22. **Kubo K, Kanehisa H, and Fukunaga T.** Effects of different duration isometric contractions on tendon elasticity in human quadriceps muscles. *J Physiol* 536: 649–655, 2001.
23. **Kubo K, Kanehisa H, and Fukunaga T.** Effects of resistance and stretching training programmes on the viscoelastic properties of human tendon structures in vivo. *J Physiol* 538: 219–226, 2002.
24. **Langberg H, Skovgaard D, Asp S, and Kjaer M.** Time pattern of exercise-induced changes in type I collagen turnover after prolonged endurance exercise in humans. *Calcif Tissue Int* 67: 41–44, 2000.
25. **Langberg H, Skovgaard D, Petersen LJ, Bulow J, and Kjaer M.** Type I collagen synthesis and degradation in peritendinous tissue after exercise determined by microdialysis in humans. *J Physiol* 521: 299–306, 1999.
26. **Lian O, Holen KJ, Engebretsen L, and Bahr R.** Relationship between symptoms of jumper's knee and the ultrasound characteristics of the patellar tendon among high level male volleyball players. *Scand J Med Sci Sports* 6: 291–296, 1996.
27. **Maffulli N and Wong J.** Rupture of the Achilles and patellar tendons. *Clin Sports Med* 22: 761–776, 2003.
28. **Maganaris CN, Baltzopoulos V, and Sargeant AJ.** In vivo measurement-based estimations of the human Achilles tendon moment arm. *Eur J Appl Physiol* 83: 363–369, 2000.
29. **Magnusson SP, Aagaard P, Dyhre-Poulsen P, and Kjaer M.** Load-displacement properties of the human triceps surae aponeurosis in vivo. *J Physiol* 531: 277–288, 2001.
30. **Magnusson SP and Kjaer M.** Region-specific differences in Achilles tendon cross-sectional area in runners and non-runners. *Eur J Appl Physiol* 2003.
31. **Magnusson SP, Qvortrup K, Larsen JO, Rosager S, Hanson P, Aagaard P, Krogsgaard M, and Kjaer M.** Collagen fibril size and crimp morphology in ruptured and intact Achilles tendons. *Matrix Biol* 21: 369–377, 2002.
32. **Markovic G and Jaric S.** Movement performance and body size: the relationship for different groups of tests. *Eur J Appl Physiol* 92: 139–149, 2004.
33. **Moller A, Astron M, and Westlin N.** Increasing incidence of Achilles tendon rupture. *Acta Orthop Scand* 67: 479–481, 1996.
34. **Nehrer S, Breitenseher M, Brodner W, Kainberger F, Fellinger EJ, Engel A, and Imhof F.** Clinical and sonographic evaluation of the risk of rupture in the Achilles tendon. *Arch Orthop Trauma Surg* 116: 14–18, 1997.
35. **Reeves ND, Maganaris CN, and Narici MV.** Effect of strength training on human patella tendon mechanical properties of older individuals. *J Physiol* 548: 971–981, 2003.
36. **Rosager S, Aagaard P, Dyhre-Poulsen P, Neergaard K, Kjaer M, and Magnusson SP.** Load-displacement properties of the human triceps surae aponeurosis and tendon in runners and non-runners. *Scand J Med Sci Sports* 12: 90–98, 2002.
37. **Sandmeier R and Renstrom PA.** Diagnosis and treatment of chronic tendon disorders in sports. *Scand J Med Sci Sports* 7: 96–106, 1997.
38. **Smart GW, Taunton JE, and Clement DB.** Achilles tendon disorders in runners—a review. *Med Sci Sports Exerc* 12: 231–243, 1980.
39. **Smith RK, Birch HL, Goodman S, Heinegard D, and Goodship AE.** The influence of ageing and exercise on tendon growth and degeneration-

hypotheses for the initiation and prevention of strain-induced tendinopathies. *Comp Biochem Physiol A* 133: 1039–1050, 2002.

40. **Sommer HM.** The biomechanical and metabolic effects of a running regime on the Achilles tendon in the rat. *Int Orthop* 11: 71–75, 1987.

41. **Viidik A.** Tensile strength properties of Achilles tendon systems in trained and untrained rabbits. *Acta Orthop Scand* 40: 261–272, 1969.

42. **Voigt M.** *Muscle-Tendon Elasticity in Vivo and Its Significance for Human Performance* (PhD thesis). Copenhagen, Denmark: University of Copenhagen, 1994.

43. **Wang XT and Ker RF.** Creep rupture of wallaby tail tendons. *J Exp Biol* 198: 831–845, 1995.

44. **Wilson AM and Goodship AE.** Exercise-induced hyperthermia as a possible mechanism for tendon degeneration. *J Biomech* 27: 899–905, 1994.

45. **Woo SL, Gomez MA, Amiel D, Ritter MA, Gelberman RH, and Akeson WH.** The effects of exercise on the biomechanical and biochemical properties of swine digital flexor tendons. *J Biomech Eng* 103: 51–56, 1981.

46. **Woo SL, Gomez MA, Woo YK, and Akeson WH.** Mechanical properties of tendons and ligaments. II. The relationships of immobilization and exercise on tissue remodeling. *Biorheology* 19: 397–408, 1982.

47. **Woo SL, Ritter MA, Amiel D, Sanders TM, Gomez MA, Kuei SC, Garfin SR, and Akeson WH.** The biomechanical and biochemical properties of swine tendons—long term effects of exercise on the digital extensors. *Connect Tissue Res* 7: 177–183, 1980.

48. **Wren TA, Yerby SA, Beaupre GS, and Carter DR.** Mechanical properties of the human achilles tendon. *Clin Biomech (Bristol, Avon)* 16: 245–251, 2001.