Effect of androgenic-anabolic steroids and heavy strength training on patellar tendon morphological and mechanical properties

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A NUMBER OF CASE STUDIES DEPICTING tendon injury and rupture in bodybuilders (2, 3, 11) has often associated this problem with the widespread use of androgenic-anabolic steroids (AAS) in this population.

While the adverse effects of AAS on the reproductive, cardiovascular, and hepatic systems are well documented (7), the influence of this class of drugs on connective tissue remains understudied. In fact, existing knowledge mostly stems from often-discrepant reports from animal studies on short-term exposure to AAS and overloading (8, 17, 24).

These studies typically indicate that combined AAS and exercise induces an increase in tendon cross-sectional area and stiffness (8, 17). Failure strain seems reduced, without apparent change in ultimate strength (8, 15, 17). Some authors (16), yet not all (8), have also reported AAS-induced collagen dysplasia, inflammation, and fibrosis (14). Importantly, these effects have been associated with alterations in collagen fibril angle and the toe region of the tendon load-deformation curve, although the direction of these changes remains unclear (17, 24).

Ultrastructural changes in the crimp morphology are linked to the toe region length (also referred to as toe limit), during which the collagen fibrils are thought to be straightened out. Changes in this parameter may bear important mechanical consequences, inducing a lower failure strain or higher operating stresses when the toe limit is reduced or lengthened, respectively. However, to our knowledge, only one case study investigated these aspects in human subjects, failing to identify any sign of histological and ultrastructural alterations in the ruptured tendons of two bodybuilders (4). Yet the prevalence of tendon rupture is high in bodybuilders and essential evaluation of this structure before injury, including detailed assessment of tendon hypertrophy and mechanical properties, is missing. Indeed, tendon mechanical properties result from a complex tradeoff between effective transmission of force between muscle and bone and storage/release of elastic energy during complex movements such as locomotion or jumping [see Ref. 13 for review]. Several studies have shown that slow resistance training induces adaptive changes favoring tendon force transmission by increasing stiffness (1, 10, 21), via changes in material properties (21) and/or cross-sectional area (1, 10, 21). The potential influence of AAS upon these adaptations is unknown.

Hence the objective of this prospective study was to examine in vivo mechanical properties of patellar tendons subjected to long-term exposure to overloading and AAS abuse. By comparing highly trained individuals using AAS to trained and untrained subjects without any history of ASS, we aimed at identifying morphological and material features substantiating reports of altered collagen ultrastructure and metabolism. Specifically, we hypothesized that tendon rupture risk factors such as a higher operating stress and an increased toe limit strain would be seen in steroids users.

METHODS

Subjects and training history. Twenty-four volunteers were assigned to three groups: one group of resistance-trained individuals using steroids (RTS group), one group of resistance-trained individuals who had never used steroids (RT group), and one group without any history of regular heavy strength training or AAS abuse (CTRL group). Nine male athletes were initially recruited among bodybuilders for the RTS group. However, one of them dropped out of the study after suffering a patellar tendon rupture while performing an MVC during the testing session. The remaining eight subjects had been involved in heavy resistance training for several years (12.4 ± 5.4 yr), with one or two competitions per year. Training diaries were indica-
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of moderate- to high-intensity (60–80% of 1RM) and high-volume (4–5 sets × 8–15 repetitions, 3 to 5 times a week) regimens. Training programs targeted all major muscle groups through single- and multiple-joint exercises and were adjusted according to the competition calendar, the above regimens reflecting between-competition phases. Training of the thigh muscles typically included reclined leg presses, leg curls, leg extensions, squats, and hack squats. Pre-competition phases started 2–3 mo prior to competition and were generally arranged in weekly microcycles with one or two additional resting days. All subjects had been in the between-competition phase for at least 6 mo at the time of the present study. Eight subjects regularly involved in heavy resistance training were recruited from local students and powerlifting clubs for the RT group. All had been training for at least 5 years (6.3 ± 1.7 yrs on average). Typical training regimen for this group was 3 to 5 sessions per week, with 3 to 5 sets of 5 to 10 repetitions at 70 to 90% of 1RM, depending on periodization cycles. Exercises involving thigh muscles included leg extension, leg press, and various forms of squats, deadlifts, and lunges. Eight recreationally active males were recruited among the student population for the CTRL group. All subjects provided a written informed consent and the Ethics Committee of the Lithuanian University of Health Sciences approved the study.

Diet supplementation and drugs. Subjects of the RTS group had regularly supplemented their diet with vitamins and amino acids, via branched-chain amino acids and/or whey protein ingestion, and with an ergogenic supplement, creatine (continuously or in 2-mo cycles). Regular parenteral administration of AAS was reported by all the athletes. Typical dosages of AAS used by bodybuilders are reported elsewhere (5), but it was not possible to characterize drug abuse in the present study due to the exceptional duration (years) over which drug doses and types were modified and due to the reluctance of some subjects to disclose further details regarding the intake of these banned substances. Some of the RT and CTRL subjects reported occasional intake of vitamins or creatine but none of them had used AAS.

Maximal voluntary contraction. Maximal knee extension isometric torque was tested on an isokinetic dynamometer (System 3, Biodex Medical Systems, Shirley, NY and Cybex Norm, New York, NY). Each participant performed three maximal knee extension and one maximal knee flexion contractions at 90° of knee flexion, 0° corresponding to the knee being fully extended. To estimate the mechanical properties of the patellar tendon (see Tendon mechanical and material properties), two ramp contractions were performed at the same joint angle. The net torque was calculated offline as the net torque divided by the patella moment arm. The internal knee moment arm was estimated as a function of the femur bone length (23). The net torque was obtained by summing the extension torque and the estimated co-contraction torque produced by the knee flexor muscles. The latter was estimated from EMG recordings, by assuming linearity between EMG activity and isometric torque production. To this end, EMG activity of the biceps femoris muscle was recorded by means of two adhesive silver chloride electrodes (20 mm interelectrode distance, Ambu A/S, Ballerup, Denmark). Raw EMG signal was digitized (sampling frequency 2 kHz), stored, and analyzed with a commercially available software (Acqknowledge, Biopac System, CA, USA). EMG activity was quantified offline, by calculating the root mean square of the signal over a 0.5-s period around the peak torque.

Muscle architecture. Muscle thickness, pennation angle (Pa), and fascicle length (Lf) were measured with ultrasonography (10–15 MHz transducer, MyLab25, Esaote, Genoa, Italy) in the vastus lateralis. Ultrasound recordings were obtained on resting muscles, while subjects were lying supine. The ultrasound probe was positioned over the belly (~50% muscle length) of the vastus lateralis, so that fascicles were aligned with the probe direction. Subsequently, Pa, Lf, and thickness were measured with an image analysis software (ImageJ, Wayne Rasband, National Institutes of Health, Bethesda, MD, USA). The validity and test-retest reliability of ultrasound-based measurement of human muscle architecture have been reported previously (18, 22).

Tendon dimensions. The cross-sectional area of the patellar tendon was measured proximally, at midlength, and distally with ultrasonography (10–15 MHz transducer, MyLab25, Esaote, Genoa, Italy). These regions were selected based on previous reports (10, 21) indicating that hypertrophy was prominent near the insertions sites of the patellar tendon. The ultrasound probe was first positioned longitudinally over the proximal and distal insertions. An echo-absorptive marker at the interface with the skin allowed locating and marking precisely the insertion points. Tendon length was measured externally as the distance between insertions. Subsequently, the tendon CSA was scanned transversally at each insertion and at midlength. Measurements of tendon CSA were also normalized to knee extension maximal isometric torque, to account for the influence of actual daily stress experienced by the subjects.

Tendon mechanical and material properties. Load-deformation properties of the patellar tendon were measured with ultrasound. Repeated measurements using this technique have previously shown a good interday reliability (20). The tendon was scanned longitudinally during a maximal isometric ramp contraction with the knee joint at 90° of flexion. Immediately prior to the ramp MVC, subjects performed a series of five submaximal isometric contractions (50% < torque < 80%) to ensure preconditioning of the tendon (12). The elongation of the patella tendon was measured in each video frame, as the distance between the patellar insertion and the tendon region anterior to the tibial plateau edge (6). By using combined ultrasound and electronic goniometer recordings, patellar displacement unrelated to muscle contraction was estimated posteriori during a passive knee flexion and subtracted from measured tendon elongation. This procedure enabled the correction of overestimated tendon elongation, due to change in knee joint angle during muscle contraction. The force-deformation data were plotted, and the best-fitting 2nd or 3rd degree polynomial function was applied to the data points. Stiffness was calculated as the slope of the obtained load-deformation curve in its linear portion, over 90% to 100% of the maximal force. This parameter was calculated at 1) the maximal common force level of all subjects (Stiffness) and 2) at the maximal common force level of each group (Stiffnessmax). This method ensured standardized between-groups comparisons of tendon mechanical properties at the same force level and at force levels typically exerted in each group. Young’s modulus was calculated as the product of stiffness and tendon length to CSA ratio. Stress and strain were obtained by normalizing tendon force to mean tendon CSA and tendon elongation to tendon length, respectively. Finally, toe limit strain was obtained as the zero-stress intercept of the linear portion of the stress-strain curve. This variable was calculated at stress levels corresponding to the maximal common force levels in each group. The intraday reliability of tendon mechanical properties was assessed in a subgroup (RT) on two stiffness measurements, yielding an intraclass correlation coefficient of 0.92 (0.60–0.99), with a typical error of 205 N·mm⁻¹. The same calculations were performed for the toe limit strain, indicating an intraclass correlation coefficient of 0.82 (0.26–0.97), with a typical error of 0.55 mm.

Statistics. Data were evaluated visually and statistically for normality of distribution. Relationships between variables of interest were tested by calculating the Pearson’s correlation coefficient. Between-group differences in muscle architecture parameters, MVC, tendon stiffness, Young’s modulus, and toe limit strain were analyzed with a one-way ANOVA. Differences in tendon CSA at proximal insertion, distal insertion, and midtendon length were assessed by using a factorial analysis of variance (tendon region × bodybuilding). A Fisher’s LSD test was used when interaction effects were significant. All data are reported as means ± standard deviations and significance level was set at P < 0.05.
**Table 1. Anthropometric measurements and maximal torque**

<table>
<thead>
<tr>
<th></th>
<th>RTS</th>
<th>RT</th>
<th>CTRL</th>
<th>F Ratio</th>
<th>P Value</th>
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<td>8</td>
<td>8</td>
<td>8</td>
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<td>Age, yr</td>
<td>29 ± 5</td>
<td>24 ± 3</td>
<td>29 ± 5</td>
<td>3.43</td>
<td>0.05</td>
</tr>
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<td>Body height, cm</td>
<td>178 ± 4</td>
<td>182 ± 6</td>
<td>175 ± 9</td>
<td>1.95</td>
<td>0.17</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>92 ± 13**</td>
<td>91 ± 4**</td>
<td>66 ± 9</td>
<td>18.15</td>
<td>&lt;0.01</td>
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<tr>
<td>Isometric torque, N·m</td>
<td>340 ± 87**</td>
<td>379 ± 38**</td>
<td>214 ± 55</td>
<td>14.80</td>
<td>&lt;0.01</td>
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<tr>
<td>VL muscle architecture</td>
<td></td>
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<tr>
<td>Pa, deg</td>
<td>21.2 ± 2.4**</td>
<td>19.1 ± 2.7*</td>
<td>15.0 ± 4.2</td>
<td>7.6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Lf, cm</td>
<td>8.6 ± 1.6</td>
<td>9.3 ± 1.1</td>
<td>8.6 ± 1.6</td>
<td>0.68</td>
<td>0.52</td>
</tr>
<tr>
<td>Thickness, mm</td>
<td>28 ± 3**</td>
<td>29 ± 3**</td>
<td>22 ± 3</td>
<td>14.56</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Tendon length, cm</td>
<td>4.9 ± 0.4</td>
<td>5.0 ± 0.7</td>
<td>4.6 ± 0.5</td>
<td>1.22</td>
<td>0.32</td>
</tr>
<tr>
<td>Tendon mean CSA, mm²</td>
<td>117 ± 13**§</td>
<td>131 ± 12**</td>
<td>98 ± 10</td>
<td>15.81</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Tendon mean CSA/torque, a.u.</td>
<td>36.1 ± 9.0**</td>
<td>35.4 ± 3.5**</td>
<td>48.0 ± 10.5</td>
<td>6.2</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Values are means ± SD. RTS, resistance-trained individuals using steroids; RT, resistance trained individuals who had never used steroids; VL, vastus lateralis; Pa, pennation angle; Lf, fascicle length; CSA, cross-sectional area. *P < 0.05, **P < 0.01 compared with CTRL. §P < 0.05.

**RESULTS**

Anthropometric measurements of the subjects are presented in Table 1.

*Muscle strength and architecture.* Mean MVC isometric torque was 59% and 77% higher in RTS and RT groups, respectively, than in the CTRL group (Table 1), without any significant difference between trained groups.

Consistent with this higher strength, muscle pennation angle and thickness were higher in RTS (41% and 27%, respectively) and RT (27% and 33%, respectively) groups than in the CTRL group (Fig. 1, Table 1). These variables were not statistically different between trained groups. By contrast, muscle fascicle length was similar between groups (Table 1), indicating that VL hypertrophy in trained groups resulted mainly from addition of in parallel sarcomeres.

*Tendon dimensions.* Tendon length did not differ significantly between groups (Table 1). However, tendon mean CSA was 19% and 34% larger in RTS and RT groups, respectively, than in the CTRL group, with a significantly larger tendon in the RT group compared with the RTS group (Table 1, Fig. 2). ANOVA analyses indicated significant main effects for measurement location (F = 25.26, P < 0.01) and for group (F = 39.02, P < 0.01) but no interaction (Fig. 3). Tendon cross section area was largest in trained subjects not using steroids (P < 0.05). However, correlation analyses indicated a significant relationship between tendon CSA and MVC torque (r = 0.81, P < 0.01). When normalized to quadriceps maximal torque, differences in tendon CSA between trained steroids users and nonusers disappeared, while this ratio was ~35% higher in CTRL (Table 1, Fig. 3).

*Tendon mechanical properties.* In the RTS group, Stiffnessₚ and Stiffness_max were 40% (P < 0.01) and 70% (P < 0.01) higher, respectively, than in the CTRL group (see Table 2 and Fig. 4). When comparing the RT to the CTRL group, the higher Stiffness did not quite reach significance (+21%, P = 0.06) but Stiffness_max was 34% higher (P < 0.05). The difference in Stiffness between RTS and RT groups was not significant either (15%, P = 0.09), but Stiffness_max was 26% higher in the RTS group (P < 0.05).

Young’s modulus at common force level was not significantly different between groups, despite a 18% higher value for the RTS group, compared with RT and CTRL groups (P = 0.08 in both cases) (Table 2). However, Young’s modulus at group maximal force level was higher in the RTS group than in both the RT (30%, P < 0.01) and CTRL (44%, P < 0.01) groups. There was no significant difference in Young’s modulus between RT and CTRL groups (Table 2).

Tendon maximal strain and toe limit strain did not significantly differ between groups (Table 2 and Fig. 4). Since scatter analyses indicated that these variables were linearly related with a similar slope in all groups, an analysis of variance was also performed on toe limit strain to maximal strain ratios. However, differences in normalized toe limit strain did not reach statistical significance either (P = 0.18).

**DISCUSSION**

This is the first report on in vivo measurements of patellar tendon in humans subjected to long-term exposure to overloading and steroids. Comparisons between trained (RTS and RT) and untrained (CTRL) subjects who do not use steroids indicate that
the patellar tendon is stiffest and presents a higher tensile modulus in trained individuals using steroids. Tendon cross section area normalized to quadriceps maximal torque was significantly lower in trained subjects but was not different between trained steroids users and nonusers. Contrary to our hypothesis, maximal (operating) strain was similar in both groups and toe limit strain was not significantly higher than in controls.

The observed differences in muscle isometric torque and architecture between trained and untrained subjects are consistent with the hypertrophic response typically observed in response to heavy strength training. Considering the elliptic shape of the VL CSA, one can predict an exponential relation between muscle thickness (assimilated to an ellipse minor axis) and CSA (II × half-minor axis × half-major axis) with hypertrophy. This suggests that the difference in muscle CSA was even higher than the observed ~30% differences in thickness. The lack of statistical differences in knee extension torque, VL muscle thickness, and pennation angle between RTS and RT groups confirms that the patellar tendon was exposed to comparable levels of force in these subjects.

Marked differences in tendon mechanical and material properties were observed between groups, although significance level was not always reached when these variables were measured at a force level common to all subjects. These small discrepancies, P values ranging from 0.06 to 0.09, could be ascribed to the higher forces exerted by trained subjects and the nonlinear pattern of the tendon force-elongation relation at submaximal force levels. Nevertheless, tendon stiffness was greater in the trained groups than in the CTRL group. This result is consistent with previous observations of increased stiffness in the patellar tendon following resistance training lasting 9 to 12 wk in healthy young subjects (10, 21). However, in contrast to the RT group, the high magnitude of tendon stiffness in RTS subjects (+40% and 70% compared with the CTRL group) largely differs from the 19-to-24% increases found after resistance training (10, 21). Furthermore, maximal tendon stiffness was greater in the RTS group than in trained subjects not using steroids. This difference is at odds with the similar muscle force and size of these groups. Apart from the potential role played by steroids per se (discussed below), this difference may be explained by tendon loading modalities: precise comparison of the training programs used by the subjects over several years is difficult but steroids are ergogenic drugs enabling athletes to train with larger volumes. In line with this hypothesis, typical numbers of training sets and repetitions summarized in the Methods section suggest that tendon mean time under tension may have been longer for the RTS group. As suggested by positive correlations between increases in patellar tendon stiffness and muscle hypertrophy, but not muscle strength, after short-term training (21), a larger time under tension may be more important than maximal force generating capacity in the training-induced changes in tendon mechanical properties. Nevertheless, an increase in tendon stiffness has been observed in rats in response to combined exercise and AAS exposure and with AAS alone (15). Combined with the large volumes of heavy resistance training, such an increase could be linked with a higher occurrence of microinjury. Longitudinal studies controlling loading parameters are required to ascertain the link between AAS abuse and tendon higher stiffness observed in the present study.

Consistently with stiffness measurements, tensile modulus was larger in trained subjects using steroids, denoting differences in tendon material properties in this group. As for tendon stiffness, differences in Young’s modulus between RTS and the other groups (30–44%) exceeded the increase typically observed in this parameter (20%) after short-term training (21). Interestingly, differences in tensile modulus between trained and untrained subjects were not significant, despite the latter presenting lower tendon stiffness. This ostensible disagreement is in fact solved when considering tendon CSA, larger in the RT group than in the CTRL and RTS groups. Hence the present data indicate that years of heavy resistive loading resulted in an expectable increase in patellar tendon stiffness in RT and RTS subjects, but this adaptation was achieved in different ways in steroids users and nonusers. In the RT group, the higher tendon stiffness seems to be underpinned by a larger tendon CSA, whereas the higher stiffness
values observed in RTS subjects are largely attributable to differences in tendon material properties.

The larger tendon CSAs measured in both trained groups, in particular near the insertion sites, are in line with the pattern of patellar tendon hypertrophy observed after a resistance training program (10, 21). Yet, the magnitude of the differences in mean tendon CSA between trained groups and the CTRL group was on average four- to sixfold the increase typically reported after a few months of resistance training (10, 21). To investigate tendon hypertrophy relative to the actual stress experienced in daily life, tendon CSA was normalized to muscle torque. This variable was chosen as a surrogate of quadriceps cross-sectional area, which could not be inferred satisfactorily from the present measurements. This approach is supported by the theoretical link between tendon and muscle scaling in mammals (9) and by the correlation between the present measurements of tendon mean CSA and isometric torque. Normalized tendon CSA was substantially reduced in trained groups compared with CTRL, without any difference between steroids users and nonusers. At first glance, these results suggest that, despite an adaptive hypertrophy, tendon

<table>
<thead>
<tr>
<th>Force level, N</th>
<th>RTS</th>
<th>RT</th>
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<th>F Ratio</th>
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<th>F Ratio</th>
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<td>6,000</td>
<td>6,000</td>
<td>4,000</td>
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<td>4,000</td>
<td>4,000</td>
<td>4,000</td>
<td>4,000</td>
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<tr>
<td>Stiffness, N/mm</td>
<td>3860 ± 964**§</td>
<td>3179 ± 722**</td>
<td>3052 ± 604*</td>
<td>2754 ± 377</td>
<td>2277 ± 145</td>
<td>11.43 &lt; 0.01</td>
<td>7.13 &lt; 0.01</td>
</tr>
<tr>
<td>Young’s modulus, GPa</td>
<td>1.56 ± 0.25**§§</td>
<td>1.28 ± 0.18</td>
<td>1.20 ± 0.35</td>
<td>1.08 ± 0.33</td>
<td>1.08 ± 0.07</td>
<td>7.74 &lt; 0.01</td>
<td>2.19 0.14</td>
</tr>
<tr>
<td>Strain, %</td>
<td>7.1 ± 1.2</td>
<td>5.9 ± 1.1</td>
<td>7.4 ± 1.0</td>
<td>6.0 ± 1.1</td>
<td>6.8 ± 1.6</td>
<td>0.47 0.63</td>
<td>1.01 0.38</td>
</tr>
<tr>
<td>Stress, MPa</td>
<td>51.9 ± 4.9**§</td>
<td>34.6 ± 3.3**§</td>
<td>46.1 ± 4.3*</td>
<td>30.8 ± 2.9**</td>
<td>41.2 ± 4.5</td>
<td>11.02 &lt; 0.01</td>
<td>17.01 &lt; 0.01</td>
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Values are means ± SD. Force level (N) columns denote variables measured at the highest force level common to all subjects (4,000 N) or at the highest force level in each group (4,000 N for CTRL and 6,000 N for RTS and RT). *P < 0.05, **P < 0.01 compared with CTRL. §P < 0.05, §§P < 0.01 compared with RT.

Fig. 4. Patellar tendon force-elongation (A) and stress-strain relations (B). The force-elongation curve was drawn from mean values ± SD from the untrained group (CTRL), the trained group not using AAS (RT), and the trained, AAS users group (RTS). Stress-strain curves were drawn from mean values ± SD. The intercept between the dashed line and the X axis denotes the toe limit strain.
safety factor (failure stress to peak daily stress ratio) may be reduced with long-term resistance training, regardless of steroids abuse. Consistent with this hypothesis, analyses of mechanical properties confirmed that the stress experienced by the patellar tendon during MVC was lower in CTRL subjects. However, further analyses revealed that maximal tendon stress was 13% higher in the RTS group than in the RT group. Taken together, these data suggest that tendon hypertrophy occurred with long-term training but was insufficient in subjects using steroids to limit tendon stress at the same levels observed in nonusers. These observations, combined with the similar strain measured in all groups may have some clinical implications: despite a higher maximal stress, the patellar tendon of RTS subjects operates in the same strain range than RT and CTRL subjects. Considering that in rats, tendon ultimate strength seems unaffected by combined AAS and loading (17), the above findings suggest that patellar tendon may experience more damage at higher strain values in AAS users. In vitro studies measuring the ultimate tensile strength of tendinous tissue in this population are required to test this hypothesis.

Contrary to our expectation, toe limit strain and normalized toe limit strain (to maximal strain) were not statistically different between groups. These findings contrast with reports from animal studies (17, 24) but are in line with ultrastructural analyses of ruptured tendon fibrils from two bodybuilders (4). They suggest that long-term exposure to AAS and/or overloading did not appreciably affect collagen ultrastructure or that such alteration did not have substantial impact on the tendon toe region measured in vivo. Nevertheless, combined resistance training and AAS intake affects collagen metabolism measured in serum, notably by increasing net synthesis of type I collagen (19). Direct evidence of such an increase is currently lacking in tendon tissue, but in rats, combined AAS and training have been shown to inhibit matrix metalloproteinases activity, whereas this parameter was upregulated with exercise alone (14). Hence the higher tendon stiffness and the different material and morphological properties observed in the RTS group, compared with the RT group, could be explained by differences in collagen remodelling. Future studies should investigate this possibility in a longitudinal design controlling for training modality, AAS dosage, and diet.

CONCLUSIONS

Following long-term heavy resistance training and AAS abuse, the patellar tendon is characterized by higher stiffness and tensile modulus than in force-matched individuals subjects to training only. Comparisons of tendon CSA normalized to maximal muscle torque indicate that tendon hypertrophy is similar in trained individual using steroids or not. Yet the higher maximal stress measured in steroid users may limit the tendon safety factor in this population. Despite the lack of evidence for collagen ultrastructural damage, the present measurements of tendon mechanical and material properties may reflect difference in collagen remodelling.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


REFERENCES