

Influence of static stretching on viscoelastic properties of human tendon structures in vivo

KEITARO KUBO, HIROAKI KANEHISA, YASUO KAWAKAMI, AND TETSUO FUKUNAGA
*Department of Life Science (Sports Sciences), University of Tokyo,
Komaba 3-8-1, Meguro, Tokyo 153-8902, Japan*

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Kubo, Keitaro, Hiroaki Kanehisa, Yasuo Kawakami, and Tetsuo Fukunaga. Influence of static stretching on viscoelastic properties of human tendon structures in vivo. *J Appl Physiol* 90: 520–527, 2001.—The purpose of this study was to investigate the influences of static stretching on the viscoelastic properties of human tendon structures in vivo. Seven male subjects performed static stretching in which the ankle was passively flexed to 35° of dorsiflexion and remained stationary for 10 min. Before and after the stretching, the elongation of the tendon and aponeurosis of medial gastrocnemius muscle (MG) was directly measured by ultrasonography while the subjects performed ramp isometric plantar flexion up to the maximum voluntary contraction (MVC), followed by a ramp relaxation. The relationship between the estimated muscle force (F_m) of MG and tendon elongation (L) during the ascending phase was fitted to a linear regression, the slope of which was defined as stiffness of the tendon structures. The percentage of the area within the F_m - L loop to the area beneath the curve during the ascending phase was calculated as an index representing hysteresis. Stretching produced no significant change in MVC but significantly decreased stiffness and hysteresis from 22.9 ± 5.8 to 20.6 ± 4.6 N/mm and from 20.6 ± 8.8 to $13.5 \pm 7.6\%$, respectively. The present results suggest that stretching decreased the viscosity of tendon structures but increased the elasticity.

medial gastrocnemius muscle; stiffness; hysteresis; flexibility

STRETCHING HAS BEEN RECOMMENDED to prevent injury and to improve performance by regaining joint range of motion, i.e., increasing flexibility (8, 25, 31). It has been documented that the potential mechanism for reduced risk of injury with increasing flexibility is the change in the viscoelastic properties of muscle-tendon units (31). Magnusson et al. (17), who determined the stiffness of muscle-tendon units as a result of observations on the ratio of the change in passive muscle moment to that in joint angle, suggested that repetitive stretches made hamstrings more compliant. In addition, Wilson et al. (32), who applied a damped oscillation technique to determine the stiffness of the upper limbs, showed that the enhancement in rebound bench press performance observed consequent to flexibility training was caused by a reduction in stiffness of muscle-tendon units,

increasing the utilization of elastic strain energy during the rebound bench press lift. It is well known that if an activated muscle is stretched before shortening, its performance is enhanced during the concentric phase. Many previous studies have indicated that this phenomenon is purported to be the result of strain energy stored in the tendon structures (e.g., Refs. 12, 28). Taken together, these previous results would indicate that the stretching training had made the viscoelastic properties of tendon structures compliant, and thus the stored elastic energy during stretch-shortening cycle increased. However, no attempt has been made to investigate the influences of stretching on the properties of human tendon structures in vivo.

Because most biological tissues act viscoelastically, if the muscle-tendon unit is stretched and then held at a constant length, the passive force at that length gradually declines, a phenomenon known as stress relaxation (17, 25). It has been demonstrated both in vitro (25) and in vivo (17) that repeated stretching of muscle-tendon units to a constant length significantly reduces peak passive tension. These findings suggest that stretching reduces the viscosity and/or stiffness of the muscle-tendon unit, which would be a factor to increase the joint range of motion. In addition, the viscoelastic materials of the muscle-tendon unit produce a variation in the load-deformation relationship that takes place between the loading and unloading curves during a cyclic tensile test (1). This is called hysteresis. For viscoelastic materials, greater energy is absorbed during loading than is dissipated during unloading (25). The hysteresis, i.e., the area within the loop, represents the energy loss as heat due to internal damping, and the area under the unload curve is the energy recovered in the elastic recoil (1). Wilson et al. (32) reported that flexibility training for the upper limbs induced a significant increase in work during the initial concentric portion of the rebound bench press lift. This led us to speculate that stretching may be an effective way to increase reused energy during exercise involving a stretch-shortening cycle, by reducing the hysteresis. However, only few studies have ever tried to quantify the hysteresis of the human muscle-tendon

Address for reprint requests and other correspondence: Keitaro Kubo, Dept. of Life Science (Sports Sciences), Univ. of Tokyo, Komaba 3-8-1, Meguro-ku, Tokyo 153-8902, Japan (E-mail: kubo@idaten.c.u-tokyo.ac.jp).

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unit *in vivo* and to investigate the effects of stretching on it (16).

Recent reports have shown that ultrasonography can be used to determine the stiffness and Young's modulus of human tendon structures *in vivo* (10, 12, 14, 15). Hence, we applied this technique to determine the magnitude of elongation in the tendon structure of human medial gastrocnemius (MG) muscle before and after static stretching and examine the changes in the stiffness and hysteresis. The purpose of the present study was to investigate the effects of static stretching on the viscoelastic properties of human tendon structures *in vivo*.

METHODS

Subjects

Seven healthy men (age 25.3 ± 1.4 yr, height 172.6 ± 4.9 cm, weight 70.8 ± 7.9 kg; means \pm SD) voluntarily participated in the present study as subjects. When the data were collected, the subjects were participating in recreational sports but had experienced neither strength training nor flexibility training programs. The subjects were fully informed of the procedures to be utilized as well as the purpose of this study. Written, informed consent was obtained from all subjects.

Measurement of Viscoelastic Properties of Tendon Structures

Before and after stretching, torque produced during isometric planar flexion and elongation in the tendon structures of MG was determined by a dynamometer (Myoret, Asics) and ultrasonography, respectively.

Measurement of torque. The experimental setup is schematically shown in Fig. 1A. The subject lay prone on a test bench, and the waist and shoulders were secured by adjustable lap belts and held in position. The right ankle joint was set at 0° (anatomic position) with the knee joint at full extension, and the foot was securely strapped to a foot plate connected to the lever arm of the dynamometer. Before the test, the subject performed a standardized warm-up and

submaximal contractions to become accustomed to the test procedure. The subject was instructed to develop a gradually increasing force from relax to maximal voluntary contraction (MVC) within 5 s, followed by a gradual relaxation within 5 s. The task was repeated two times per subject with at least 3 min between trials. Torque signals were analog-to-digital converted at a sampling rate of 1 kHz (MacLab/8, type ML780, AD Instrument) and analyzed by a personal computer (Performa 630, Macintosh). The measured values that are shown below are the means of two trials.

Measurement of elongation of tendon structures. A real-time ultrasonic apparatus (SSD-2000, Aloka) was used to obtain a longitudinal ultrasonic image of MG at the level of 30% of the lower leg length, i.e., from the popliteal crease to the center of the lateral malleolus. The ultrasonic images were recorded on videotape at 30 Hz, synchronized with recordings of a clock timer for subsequent analyses. The tester visually confirmed the echoes from the aponeurosis and MG fascicles. The point at which one fascicle was attached to the aponeurosis (P) was visualized on the ultrasonic image. P moved proximally during isometric torque development up to maximum (Fig. 2). A marker was placed between the skin and the ultrasonic probe as the landmark to confirm that the probe did not move during measurements. The cross-point between superficial aponeurosis and fascicles did not move. Therefore, the displacement of P (L) is considered to indicate the lengthening of the deep aponeurosis and the distal tendon (10, 12).

Calculations of strain, stiffness, and hysteresis. The L value at MVC was converted to the strain by the following equation

$$\text{Strain (\%)} = L \cdot TL^{-1} \cdot 100$$

where TL is the length of the tendon structure, i.e., the distance between the measurement site for L and the estimated insertion of the muscle.

To calculate the stiffness, first, the measured torque (TQ) during isometric plantar flexion was converted to muscle force (F_m) by the following equation

$$F_m = k \cdot TQ \cdot MA^{-1}$$

where k is the relative contribution of the physiological cross-sectional area of MG within plantar flexor muscles (6) and MA is the moment arm length of triceps surae muscles at 0° of ankle joint, which was estimated from the lower leg length of each subject as described by Visser et al. (27).

As reported in previous studies using animal and human cadavers (e.g., Ref. 11), the F_m - L relation in the tendon structure was curvilinear consisting of an initial region (toe region) characterized by a large increase in L with increasing force and a linear region immediately after the toe region. The ratio of change in L to that in F_m at every 10% of MVC increased linearly with increasing force production levels from 10–50% of MVC and became almost constant in the range of 50–100% of MVC (12). In the present study, therefore, the F_m and L values above 50% of MVC were fitted to a linear regression equation, the slope of which was adopted as an index of stiffness (12).

The F_m - L curves during the ascending and descending phases of force development produced a loop. In the present study, the area of each of the curves under both the ascending and descending phases was calculated. Then the ratio of the area within the F_m - L loop (elastic energy dissipated) to the area beneath the curve during ascending phase (elastic energy input) was calculated as an index of hysteresis.

The repeatability for the stiffness and hysteresis measurements were investigated on two separate days in a prelimi-

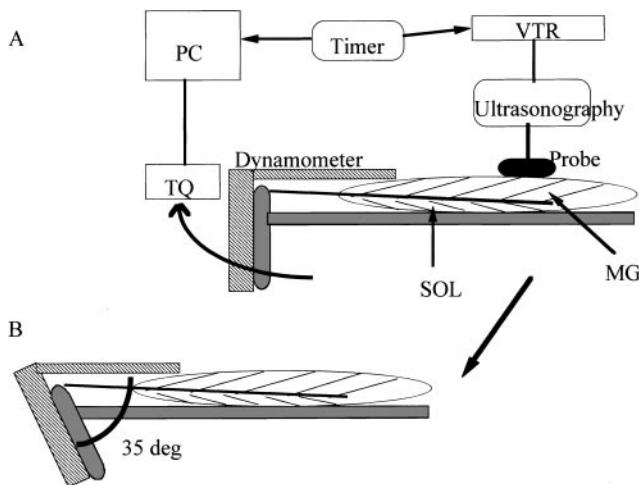


Fig. 1. A: schematic representation of experimental setup. PC, personal computer; VTR, videotape recorder; TQ, torque; MG, medial gastrocnemius muscle; Sol, soleus muscle. B: platform was moved to 35° of dorsiflexion and held for 10 min.

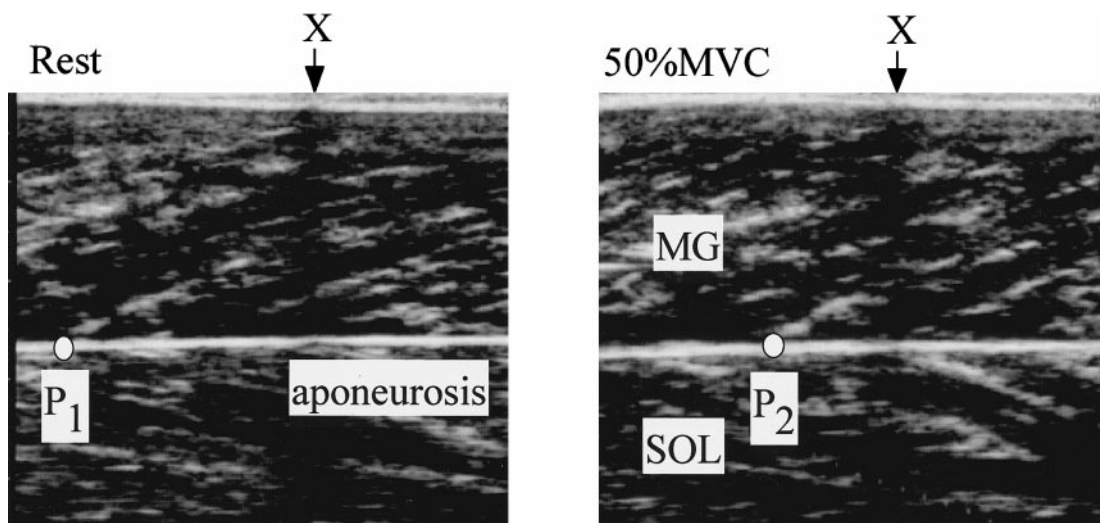


Fig. 2. Ultrasonic images of longitudinal sections of MG muscle during isometric contraction. A marker (X) was placed between the skin and the ultrasonic probe as the landmark to confirm that the probe did not move during measurements. The cross-point (P) between superficial aponeurosis and fascicles did not move. P was determined from the echoes of the deep aponeurosis and fascicles. P moved proximally during isometric torque development from rest (P_1) to 50% of maximal voluntary contraction (50%MVC; P_2). The distance traveled by P (L) was defined as the length change of tendon and aponeurosis during contraction.

nary study with 19 young men (22.6 ± 2.8 yr, 171.5 ± 6.1 cm, 69.2 ± 5.8 kg). The average values of stiffness and hysteresis obtained in the two tests were 23.2 ± 5.3 N/mm and $22.2 \pm 8.8\%$, respectively. Both the measured stiffness and the hysteresis had considerable interindividual variations: 13.8–34.3 N/mm in stiffness and 9.7–37.2% in hysteresis. However, there were no significant differences between test and retest values of stiffness and hysteresis. The test-retest correlation coefficient (r) was 0.90 for stiffness and 0.86 for hysteresis. The coefficient of variation was 5% for stiffness and 11% for hysteresis.

Static Stretching

Static stretching was administered to the right lower leg of the subject. The posture of the subject and setup were similar to those for the measurement of viscoelastic properties of tendon structures as mentioned above. The platform of the dynamometer, which was attached to the sole of the subject's foot, was moved to 35° of dorsiflexion with a constant velocity of $5^\circ/s$ (Fig. 1B) and held at this position for 10 min. The passive torque (Nm) during the stretching was detected by the dynamometer. Throughout the stretching, the subjects were requested to relax completely and not offer any voluntary resistance. To confirm the potential contribution of the contractile component during the stretching, we recorded electromyographic (EMG) activities from MG, lateral gastrocnemius, soleus, and tibial anterior muscles with Ag/AgCl surface electrodes (5 mm in diameter) placed on the belly of each muscle with a 25-mm interelectrode distance. The passive torque and EMG signals were transmitted to a computer (Performa 630, Macintosh) at a sampling rate of 1 kHz. The EMG was full-wave rectified and integrated for every 1 min of the stretching to give integrated EMG.

Statistics

Descriptive data included means \pm SD. The significance of difference between before and after the static stretching was analyzed by a paired Student's t -test. The level of significance was set at $P < 0.05$.

RESULTS

Before the stretching, the Fm- L relationship was nonlinear in form, as previously reported for animal and human tendons in vitro (Fig. 3A). The initial region of the ascending curve (toe region) was characterized by a large increase in L with increasing Fm. Moreover, the Fm- L curves during ascending and descending phases produced a loop (Fig. 3B). The strain, stiffness, and hysteresis ranged from 6.6 to 10.4% ($8.1 \pm 1.6\%$), from 15.8 to 34.3 N/mm (22.9 ± 5.8 N/mm) and from 9.7 to 33.7% ($20.6 \pm 8.8\%$), respectively.

The passive torque during stretching showed a peak at an initial phase (36.1 ± 7.0 Nm), after which torque decayed to a plateau with lengthening time (Fig. 4). The mean rate of decline in passive torque was $23.6 \pm 8.5\%$. During the stretching, EMG activities of all the muscles tested were very small and did not change significantly, whereas the passive torque declined (Table 1).

There was no significant difference in MVC values between those before and after stretching. However, the L values at near MVC became significantly greater after stretching (Fig. 5). The Fm- L loop, on the other hand, became significantly smaller after stretching (Fig. 6). The measured viscoelastic parameters are shown in Table 2. The stretching induced significant decreases in stiffness and hysteresis and increases in the areas under both loading and unloading curves.

DISCUSSION

The average stiffness before static stretching was 23 N/mm. To make a comparison with prior findings on the stiffness of tendon structures without the influences of dimensional differences, we tried to convert

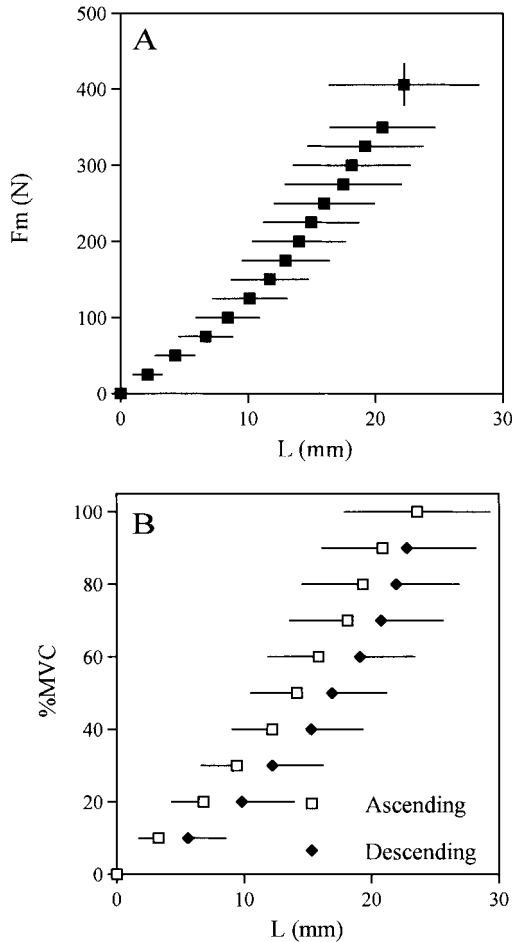


Fig. 3. Data from 7 subjects before stretching. *A*: the initial region of the curve was characterized by a large increase in *L* with increasing muscle force (*F_m*). Mean stiffness was 22.9 ± 5.8 N/mm. *B*: the *F_m*-*L* curves during ascending and descending phase produced a loop. The hysteresis value was $20.6 \pm 8.8\%$.

the *F_m*-*L* curve to a stress-strain curve and then calculate the Young's modulus, i.e., the slope of the stress-strain curve, using the procedure described by Ito et al. (10) and Kubo et al. (12). For this purpose, cross-

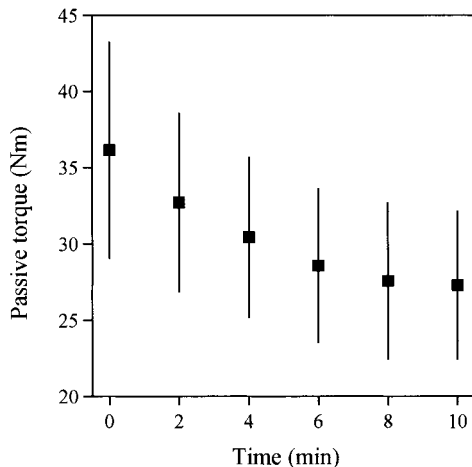


Fig. 4. Passive torque during stretching showed an initial peak, after which torque decayed to a plateau.

Table 1. Initial and final 1-min *iEMG* responses during 10-min stretching

	MG	LG	Sol	TA
Initial 1-min <i>iEMG</i> , mV	0.56 ± 0.14	0.37 ± 0.11	0.53 ± 0.20	0.40 ± 0.24
Final 1-min <i>iEMG</i> , mV	0.52 ± 0.13	0.39 ± 0.11	0.56 ± 0.15	0.39 ± 0.25

Values are means \pm SD. *iEMG*, integrated electromyogram; MG, medial gastrocnemius; LG, lateral gastrocnemius; Sol, soleus; TA, tibial anterior. There were no significant differences.

sectional area and length of the tendon were obtained from a previous report (50 mm² for the Achilles tendon; Ref. 33) and from the distance between the measurement site and estimated insertion of the muscle. The obtained Young's modulus with an average of 280 MPa agreed with our recent observations on that of tendon structures in knee extensors (250 MPa; Ref. 12), but it was considerably lower than those previously reported for animal and human cadaver tendons, 0.6–1.8 GPa (1, 11). This discrepancy can be attributed to the fact that the stiffness value determined in the present study represents the elasticities of both outer tendon and aponeurosis, whereas the above-quoted research on Young's modulus investigated the outer tendon only (1, 11). Few studies have ever tried to investigate the elastic properties of aponeurosis of animals (3, 13, 23). Scott and Loeb (23) stated that the elastic properties of aponeurosis were similar to those of tendon. On the other hand, Ettema and Huijing (3) and Lieber (13) showed that the aponeurosis strain was greater than that of tendon. We are unable to offer reasons for the discrepancy. Anyway, the strain of tendon structures obtained in the present study, 8.1%, lies within the range of aponeurosis strain values, 8–10%, which were directly determined using animal materials (3, 13). This supports that the lower Young's modulus obtained in the present study is attributable largely to the elasticity of the aponeurosis. Recently, Maganaris and

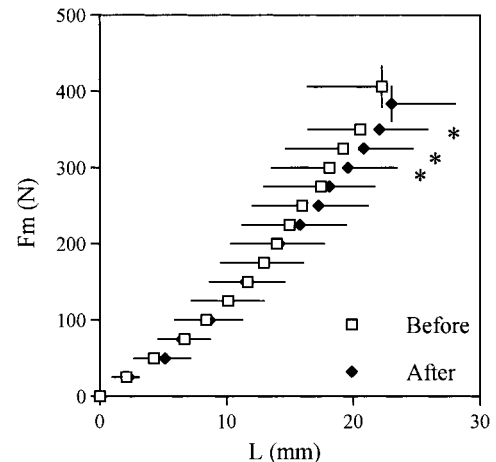


Fig. 5. Data from 7 subjects. *L* tended to be greater after stretching than before. *L* above 300 N was significantly greater after the stretching than before. *Significant difference between before and after stretching.

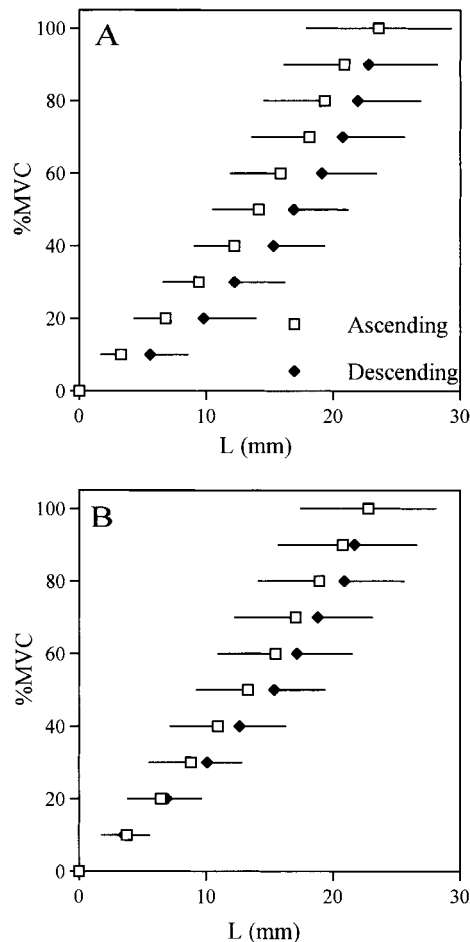


Fig. 6. Data from 7 subjects. Fm-L curves during ascending and descending phase produced a loop (hysteresis). The hysteresis was significantly smaller after (A; $13.5 \pm 7.6\%$) than before stretching (B; $20.6 \pm 8.8\%$).

Paul (15) demonstrated that the aponeurosis strain values were significantly greater than those of tendon in tibial anterior muscle. Furthermore, Roberts et al. (21) stated that most of energy storage in running turkeys must have occurred in the aponeurosis, thus suggesting that the large extensibility of the aponeurosis has been shown to make muscle contractions more efficient during movement. Namely, the present results on stiffness suggest that the human tendon structures in vivo are quite compliant, and therefore care must be taken when estimating the dynamics of the muscle-tendon unit during human movements using data on tendon elasticity that have been obtained from experiments using human cadavers and/or animals.

In the present study, the loading and unloading curves during a cyclic force development test produced a loop (i.e., hysteresis) as observed in vitro (e.g., Ref. 25). In addition to evidence that passive torque during stretching gradually decreased without any changes in EMG, the appearance of hysteresis suggests that the human tendon structures in vivo have viscoelastic profiles. The area surrounded by the loop represents the

Table 2. Measured parameters before and after stretching

	MVC, Nm	Estimated max Fm, N	Estimated MA, mm	Strain, %	Elastic Energy, J	Stiffness, N/mm	Hysteresis, %
Before	112.8 ± 7.4 (100-123)	406 ± 26 (362-444)	50.5 ± 1.8 (49.7-53.7)	8.1 ± 1.6 (6.6-10.4)	3.6 ± 1.2 (2.0-5.3)	22.9 ± 5.8 (15.8-34.3)	20.6 ± 8.8 (9.7-33.7)
After	110.7 ± 11.3 (102-128)	398 ± 40 (367-461)		8.6 ± 1.7 (6.9-11.0)*	4.0 ± 1.2 (2.2-6.1)*	20.6 ± 4.6 (13.8-28.1)*	13.5 ± 7.6 (6.5-25.9)*

Values are means \pm SD (ranges in parentheses). MVC, maximal voluntary contraction; max Fm, maximum muscle force; MA, moment arm length. * Significantly different from before.

energy loss as heat due to internal damping, whereas the area under the unloading curve is the energy recovered in elastic recoil (1). Hence, the hysteresis of tendon structures should be taken into account when estimating the dynamics of the muscle-tendon unit during human movements (28). However, no study has ever tried to quantify the hysteresis of human tendon structures *in vivo*. The hysteresis determined in the present study averaged 21%. This ranks high within the range of the values reported for 18 species of adult quadrupedal mammals, 3–20% (19). However, the hysteresis value obtained in the present study is comparable to that of human tendons at various strain rates *in vitro*, ~25% (9).

To calculate the F_m , we estimated moment arm length and relative contribution of MG to the triceps surae muscles in terms of physiological cross-sectional areas. The variation in moment arm length and relative contribution of MG among subjects might have caused the large variability in the measured parameters. The moment arm length and physiological cross-sectional areas of respective muscles of each subject would be necessary for an accurate absolute F_m determination. In the present study, we aimed to study whether the tendon properties changed after static stretching. In addition, there were no significant differences in the activation levels (integrated EMG) of each triceps surae muscles before and after stretching (data not shown). Therefore, we considered that this F_m calculation based on these assumptions would be valid to study the changes of the tendon properties after static stretching.

The stretching technique used in this study made the tendon structures more compliant by decreasing stiffness from 22.9 ± 5.8 to 20.6 ± 4.6 N/mm. Furthermore, the areas under both the loading and unloading curves increased and hysteresis decreased significantly after stretching. The hysteresis is considered an indication of the viscosity of the tissue (1). Therefore, the observed lower hysteresis after stretching can be interpreted as a decline of viscosity within tendon structures. The *in vivo* data of the present study are consistent with previous findings regarding the influences of stretching on the viscoelasticity of tendons, obtained through observations *in vitro*. For example, Viidik (26) reported that stretching the tail tendon of rat increased its compliance. Similarly, Wang et al. (29) observed an elongation in wallaby tail tendon on cyclic stretching. In addition to these findings on the elasticity, Frisen et al. (5) and Viidik (26) showed that repeated cyclic stretches of rat tendons decreased hysteresis, suggesting a reduction of energy dissipation in the tissues after stretching.

With regard to the acute influences of stretching on the viscoelasticity of human *in vivo*, however, the only information available from previous studies is data on the length changes in the muscle-tendon unit, estimated from the relation between passive torque and joint angle. Magnusson et al. (17, 18) observed a reduction of passive torque at a constant joint angle after repetitive stretches, suggesting that

stretching makes the muscle-tendon unit more compliant. Despite similarities in the muscle group subjected to stretching and the test protocol used for determination of stiffness, however, others failed to find any changes in the joint angle-passive torque relation after stretching (7, 30). One reason for the discrepancy might involve the difference between the stretch maneuvers used in each study. Taylor et al. (25) have documented *in vitro* that repeated stretching of muscle-tendon units to a constant length significantly reduces peak passive tension. In their results, after four stretches there was little alteration of the muscle-tendon unit, implying that a minimum number of stretches is required for most of the elongation in repetitive stretching (25). For human muscle-tendon units *in vivo*, however, treatment programs that consisted of 3–10 sets of 15- to 30-s stretching for hamstrings followed by a 20- to 30-s period of relaxation did not induce a significant change in the joint angle-passive torque relation after the stretching (7, 30). On the other hand, Magnusson et al. (17, 18), who administered five repetitions of 90-s static stretching with an interval of 30 s for hamstrings *in vivo*, observed that passive resistance in both the initial and final phases during stretch to a constant joint angle diminished with subsequent stretch. In the results of Magnusson et al. (17, 18), however, no significant decline in passive resistance was found after 40–45 s of the 90-s stretch for hamstrings. In addition, five repetitions of the 90-s stretch decreased the relative difference between passive resistances in the initial and final phases in every trial with each subsequent stretch (17, 18). These findings suggest that the existence of changes in the viscoelasticity of muscle-tendon units will depend on the duration rather than the number of stretches, and, if they occur, they should not be rapidly reversible. Although the stretch maneuver used in the present study was not repetitive, it induced a reduction of passive torque by 24% over 10 min, characterized by less change in the latter phase of the stretching (Fig. 4). Taking the above-mentioned point into account, the prescribed duration of stretch in this study may be assumed to be enough to change the viscoelasticity of the stressed tendon structures and to prevent them from returning rapidly.

The mechanisms that resulted in the decreases of stiffness and hysteresis after stretching are unknown. At least for the lowered stiffness observed in the present study, however, an acute change in the structure of the tendons might be involved. In a prior study using dogs (4), it has been documented that continual tensile stress applied during tibial lengthening apparently leads to an alteration in the biomechanical properties of the tendons that can result in a marked decrease of the elastic modulus, which is probably caused by strain-induced damage of the tendons and their repair processes. Stretch maneuvers for the human muscle-tendon unit *in vivo* can place stress on both the parallel and series elastic

components (18). As a result of observations on human in vivo, Magnusson et al. (18) suggested that the observed decline in the stiffness of muscle-tendon units after stretching would be an immediate adaptation of the parallel elastic component to a lower imposed load. However, the present result that the stiffness of the tendon structures decreased after stretch provides for the possibility that stretching induces change in the material function of the series elastic component, too. From the findings of Stromberg and Wiederhielm (24), the collagen fibers follow a wavelike course in the unstressed tendons, but they become aligned or parallel with increasing stress. If a similar phenomenon occurs in the tendon structures on completion of the stretch maneuver used in the present study, the observed reduction in the stiffness might be attributed to an acute change in the arrangement of collagen fibers.

The present results provide a physiological background to increases in the joint range of motion after stretching. Furthermore, the observed changes in the viscoelasticity of the tendon structures may be a reason for the delay in intrinsic muscle contraction after stretching that has been reported in previous studies (2, 22). The chief function of tendon structures is to transfer force produced by the contractile component to the joint and/or bone connected in series. A stiff tendon will be advantageous for performing brisk, accurate movements because it affects rapid tension changes (20). Inversely, if stretching has the effect of changing tendon structures to be more compliant, it will lead to a lower rate of force production and/or a delay of muscle activation. In fact, Rosenbaum and Henning (22) observed reductions of the rate of force development and EMG amplitudes and an increase in EMG latencies after static stretching of the triceps surae. From the standpoint of preventing athletic injuries, however, we can say that stretching and the subsequent decrease in stiffness diminish the imposed load across the muscle-tendon junction during rapid movements.

In conclusion, the present study showed that, using ultrasonography, it was possible to quantify the viscoelastic properties (stiffness and hysteresis) of human tendon structures in vivo. Furthermore, static stretching decreases the viscosity of tendon structures as well as increases the elasticity. This provides a physiological background for reducing passive resistance and improving joint range of motion after stretching.

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