ORIGINAL ARTICLE

Changes in tendon stiffness and running economy in highly trained distance runners

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Abstract The purpose of this study was to determine if changes in triceps-surae tendon stiffness (TST K) could affect running economy (RE) in highly trained distance runners. The intent was to induce increased TST K in a subgroup of runners by an added isometric training program. If TST K is a primary determinant of RE, then the energy cost of running (EC) should decrease in the trained subjects. EC was measured via open-circuit spirometry in 12 highly trained male distance runners, and TST K was measured using ultrasonography and dynamometry. Runners were randomly assigned to either a training or control group. The training group performed 4×20 s isometric contractions at 80% of maximum voluntary plantarflexion moment three times per week for 8 weeks. All subjects (mean $\dot{V}O_2\max = 67.4 \pm 4.6$ ml kg^{-1} min^{-1}) continued their usual training for running. TST K was measured every 2 weeks. EC was measured in both training and control groups before and after the 8 weeks at three submaximal velocities, corresponding to 75, 85 and 95% of the speed at lactate threshold (sLT). Isometric training did neither result in a mean increase in TST K (0.9 ± 25.8%) nor a mean improvement in RE (0.1 ± 3.6%); however, there was a significant relationship ($r^2 = 0.43$, $p = 0.02$) between the change in TST K and change in EC, regardless of the assigned group. It was concluded that TST K and EC are somewhat labile and change together.

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Introduction

Several key variables have been identified as important determinants of performance in endurance sports. These include, among others: maximal oxygen uptake ($\dot{V}O_2\max$), fractional utilization of $\dot{V}O_2\max$, the ability to withstand a disturbance in homeostasis, or tolerance, and running economy (RE) which is the energy cost that is required to perform the chosen task (Jones and Carter 2000). There is a strong association between RE and distance running performance, with RE being a better predictor of performance than $\dot{V}O_2\max$ in elite runners who have a similar $\dot{V}O_2\max$ (Saunders et al. 2004). Early research comparing elite distance runners ($\dot{V}O_2\max = 79$ ml/kg/min) with good distance runners ($\dot{V}O_2\max = 69$ ml/kg/min), indicated that the elite runners had a better RE than the good runners (Pollock 1977).

It is known that RE is likely influenced by a number of factors. These include training experience, altitude, biomechanics and anthropometry (Saunders et al. 2004). Therefore, training interventions aimed at improving these factors, may lead to improvements in RE and will conceivably lead to enhanced distance running performance. Little research, particularly with highly trained or elite runners, has been conducted to study the potential improvements in RE with the manipulation of one or more of these factors. In highly trained distance runners, where RE is already highly developed through years of endurance training, further improvements in RE are considered to be difficult to obtain (Saunders et al. 2004).

It has been shown that strength training improves RE in a group of well-trained runners (Storen et al. 2008), but the mechanism of these improvements was not resolved. Spurrs et al. (2003) previously demonstrated that 6 weeks of plyometric training led to improvements in RE in a group of trained distance runners. Possibly, the mechanism for improved RE was an increase in triceps-surae tendon stiffness (TST K) following training.

It has been shown that in a group of trained distance runners, the most economical runners displayed a higher TST K compared to the less economical runners (Arampatzis et al. 2006). Ultrasound was used to visualize the length change of the fascicles and tendon during contraction. The reason for this better economy in association with a stiffer tendon still remains largely speculative; however, it is suggested that the TST mechanics are altered in order to minimize the energetic cost of contraction (Arampatzis et al. 2006; Hof et al. 2002; Lichtwark and Wilson 2008).

Several training protocols have been shown to alter the mechanical properties of the TST of non-runners (Kubo et al. 2001a, b). Kubo et al. (2002) found that simultaneous resistance and stretching training programs resulted in a stiffer tendon. Kubo et al. (2001a, b) have demonstrated that 12 weeks of long-duration isometric training results in increased TST K in a group of active, but non-elite male subjects. It remains to be determined whether a similar training protocol can be as effective in trained distance runners where TST K is already high (Kubo et al. 2000b). Therefore, the approach taken in this study was to put a group of highly trained distance runners on a modified training program consisting of prolonged isometric contractions to see if the anticipated change in tendon stiffness resulted in altered RE. It was hypothesized that isometric training would result in an increased TST K, and that this change would correspond with a change in RE.

Methods

Subjects

Twelve highly trained male middle- and long-distance runners, all having regularly participated in regional, national or international competition in events ranging from 1,500 m to the marathon, took part in the study. At the time of the study, all subjects were in the pre-competition phase of their training for the 10-km cross-country distance, and all were following a similar periodized training plan. The runners gave their informed written consent to the experimental procedure which was approved by the University of Calgary Conjoint Health Research Ethics Board. All subjects performed distance running training between 6 and 12 times per week. The training volume of the subjects ranged from 70 to 170 km week^{-1}. None of the subjects had any neuromuscular or musculoskeletal injuries at the time of the study. Following pre-testing, subjects were randomly assigned to either a training (T) or control (C) group. Following pre-testing, one subject was removed from the study because it was deemed his regular training volume (<50 km week^{-1}) and experience with weight training would act as a confounding factor in data analysis. The remaining 12 subjects' characteristics (by group) are shown in Table 1.

Running protocol

Subjects performed a graded exercise test to exhaustion for the determination of speed at lactate threshold (sLT) and maximal oxygen consumption ($\dot{V}O_2$max, ml kg^{-1} min^{-1}). Prior to arriving in the lab, subjects were told to wear cool, loose clothing and their own lightweight running shoes and were instructed not to consume any food or drink other than water for at least 90 min prior to the testing session. They were also asked to refrain from caffeine consumption and any vigorous physical activity for the previous 24 h. After a self-selected warm-up, the subjects began running on a motorized treadmill (Woodway Pro, Woodway USA, Waukesha, WI, USA) at a speed approximately 3 km h^{-1} below their most recent 10 km race performance. The treadmill belt speed was increased 0.8 km h^{-1} every 2 min. At the end of each 2-min stage, a fingertip blood sample was taken for blood lactate measurement (Lactate Pro). When blood lactate concentration rose more than 1 mM from the previous sample, the treadmill speed was decreased 0.8 km h^{-1}, and the grade was increased 2% every minute until volitional fatigue. This speed was assumed to be the sLT.

Expired gases were measured by a Parvomedics True-Max 2400 metabolic measurement cart (Salt Lake City, UT, USA). The metabolic cart was calibrated before and after each session by means of a two-point calibration using room air and a gas mixture of known composition (4% CO_2, 16% O_2, balance N_2). The flow sensor (Hans Rudolf 3813 heated pneumotachometer) was calibrated with a manual 3-l syringe. The accuracy values provided by the manufacturer were 0.03 and 0.1% for O_2 and CO_2 and $\pm2\%$ for volume. Maximal oxygen uptake ($\dot{V}O_2$max) was defined as the highest 30 s average $\dot{V}O_2$ during the test and was considered to have been reached if there was no increase (<100 ml min^{-1}) in $\dot{V}O_2$ with an increase in treadmill slope or two of the following occurred: (1) RER > 1.15; (2) blood lactate at the end of the test was greater than 8.0 mM; (3) subject reached their age-predicted maximal heart rate (220-age). All tests were terminated by volitional exhaustion and all subjects achieved $\dot{V}O_2$max by these criteria.

Table 1 Subject characteristics

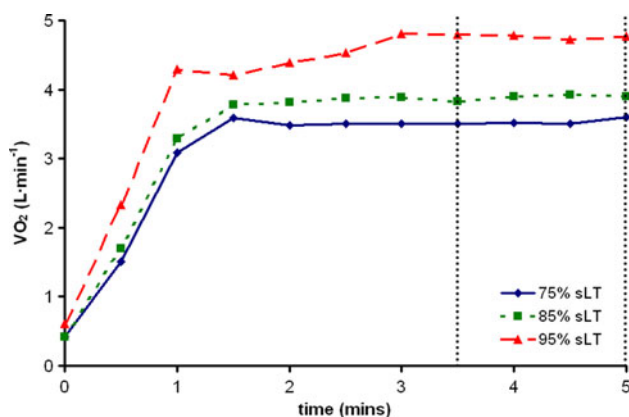| Group	N	Age (years)	Height (m)	Mass (kg)	Training volume[a] (km week^{-1})	$\dot{V}O_2$max (ml kg^{-1} min^{-1})	sLT	
							(m min^{-1})	($\dot{V}O_2$ as %$\dot{V}O_2$max)
Training	6	22.2 ± 3.1	1.80 ± 0.06	68.2 ± 8.3	108.5 ± 41.9	67.3 ± 3.1	273.5 ± 26.0	85.8 ± 3.8
Control	6	26.3 ± 6.0	1.76 ± 0.08	64.4 ± 5.5	107.9 ± 40.8	67.6 ± 6.4	262.7 ± 31.2	84.2 ± 4.7

Values are mean ± SD

[a] Training volume during the intervention period

Running economy

On a separate day, the subjects' RE was determined. The subjects were reminded of the same pre-testing instructions as prior to the first testing session. After a warm-up period of 10 min at a running speed of 160 m min^{-1}, the subjects ran on the treadmill at three different velocities in the same order (75, 85 and 95% sLT) for 5 min each. Velocities below the lactate threshold were chosen because at velocities above this, a physiological steady state for $\dot{V}O_2$ cannot be assured, and the measurement of oxygen uptake cannot account for all of the required energy (Svedahl and MacIntosh 2003). For each speed, the $\dot{V}O_2$ was measured and averaged every 30 s. The average $\dot{V}O_2$ over the final 2 min was taken as the steady-state $\dot{V}O_2$ for that speed. Steady-state was defined as a change in $\dot{V}O_2$ of less than 100 ml min^{-1} during the last 2 min of the run trials. All subjects reached a steady-state within 5 min based on this criterion. A typical RE test for one runner is shown in Fig. 1. Between stages, the subjects stood stationary on the treadmill for 5 min. Fingertip blood samples were taken for lactate analysis immediately before and after each 5 min running stage.

The RE at each speed was expressed as an energy cost (EC, kJ kg^{-1} km^{-1}), using the average respiratory exchange ratio (RER) over the same period and the caloric equivalent (kcal L O_2^{-1}) of the oxygen uptake. This is the recommended method of expressing RE (Fletcher et al. 2009). EC was calculated as:
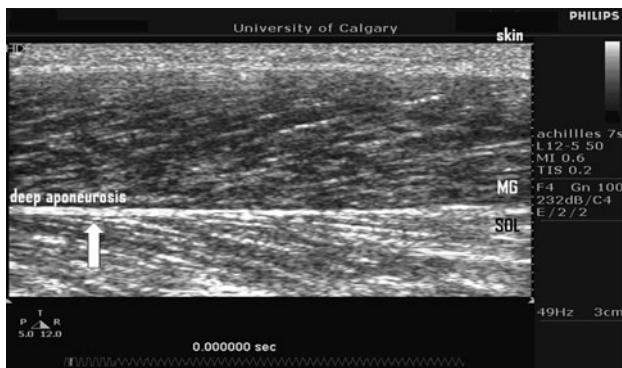
$$EC(kJ\,kg^{-1}\,km^{-1}) = \dot{V}O_2 \times \text{Caloric Eq.} \\ \times 4.1839\,kJ\,kcal^{-1}\,S^{-1}\,BM^{-1} \\ \times 1{,}000 \quad (1)$$

where $\dot{V}O_2$ is measured in L min^{-1}, caloric equivalent is in kcal L^{-1} of O_2, speed (S) is measured in m min^{-1}, body mass (BM) is measured in kg and 1,000 is m km^{-1}.

Tendon mechanical properties

In order to determine TST K, each subject performed maximal voluntary isometric ankle plantarflexion contractions (MVC) of their right leg immediately prior to the RE test. A familiarization trial was conducted on a previous day. The subjects laid prone with their knee at 180° and their ankle at 90°. Before each MVC, the axis of rotation of the dynamometer (Biodex Medical Systems Inc., Shirley, NY, USA) was carefully aligned with the axis of rotation of the ankle joint. The shank and unshod foot were affixed to the dynamometer using a series of Velcro straps. To further familiarize the subjects with the protocol and to ensure at least one visually distinctive fascicle–aponeurosis cross-point was obtained, a warm-up consisting of 3–5 min of submaximal isometric plantarflexions was performed. Afterward, the subjects performed three isometric ramp MVC plantarflexions. Moment during the MVC was sampled at 100 Hz. The trial eliciting the highest moment was used for analysis.

During each MVC, a 12.5-MHz linear array ultrasound probe (Philips Envisor, Philips Healthcare, Eindhoven, The Netherlands) was used to visualize the deep aponeurosis of the medial gastrocnemius (MG) (Fig. 2). Ultrasound scans were captured at 49 Hz. The ultrasound probe was placed on the MG muscle belly and secured using a custom-built apparatus (Fig. 3). To ensure the probe did not move during the contraction, a point on the ultrasound images, where a muscle fascicle attaches to the deep aponeurosis, was identified both before and after a test contraction for


Fig. 1 Oxygen uptake ($\dot{V}O_2$, L min^{-1}) of one runner at all three measured velocities. *Vertical dashed lines* encompass the four values used to calculate steady-state $\dot{V}O_2$

Fig. 2 Muscle ultrasound image of the right medial gastrocnemius (*MG*) and soleus (*SOL*) at rest. In the ultrasound scan, the *white stripes* represent echoes generated by collagen-rich tissue surrounding the echo-absorptive fascicles. The *oblique stripes* are interfascicular tissue echoes, and the *horizontal white stripes* are aponeurotic echoes. The *white arrow* indicates an identifiable landmark on the aponeurosis which was seen in multiple images as it moves more proximally (to the right in this image) and was used to calculate MG tendon stiffness

each subject. This point did not shift following the test contraction. An external function generator (B-K Precision 3010, Dynascan Corp., Chicago, IL, USA) was manually started at the initiation of the contraction and acted as a time-stamp for synchronization between image and moment data collection. Ultrasound images were recorded, and a clear echo point, where a fascicle inserts into the deep aponeurosis, was identified throughout the contraction, and its displacement was measured using publicly available image analysis software (*ImageJ*, NIH, Baltimore, MD, USA). This displacement of a fascicle–aponeurosis junction was interpreted as tendon elongation.

Correction for ankle joint rotation

It has been documented that it is extremely difficult to completely prevent any joint rotation during an attempted isometric contraction despite using external fixations (An et al. 1984; Maganaris and Paul 1999). This joint rotation would contribute to the apparent tendon elongation measured during the contraction if it is not accounted for (Muramatsu et al. 2001; Spoor et al. 1990). This inevitable joint rotation would also result in a lower measured ankle joint moment since the application of force to the dynamometer foot plate is no longer perpendicular to the dynamometer lever arm thus not an indication of moments generated about the ankle joint. To determine the amount of joint rotation, the ankle joint motion was imaged at 24 Hz using a portable video camera (Sony Digital 8 Handycam, Sony Canada, Toronto, ON, Canada) mounted on a tripod. Two to four small dots were drawn on the medial aspect of the unshod right foot and ankle in order to
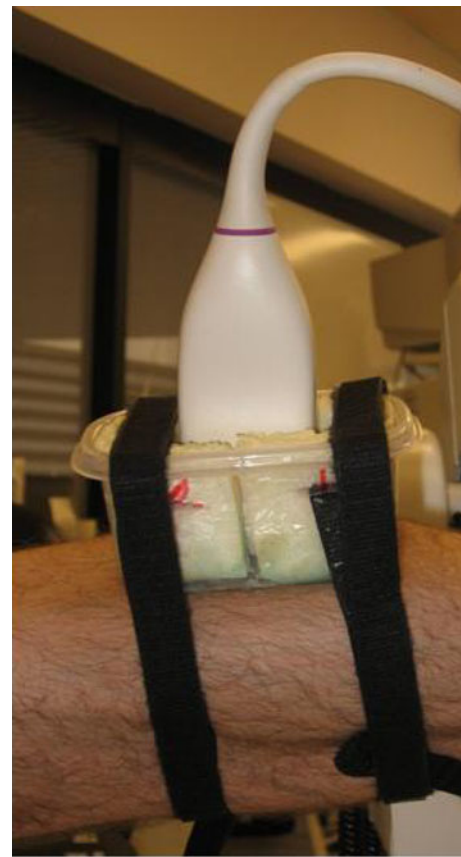
Fig. 3 Ultrasound probe secured to the MG using a custom-built apparatus. A series of Velcro straps was affixed to a Styrofoam template, ensuring the ultrasound probe remained secure during contractions

monitor foot position during the contraction. These markers were used to determine joint angle change during the contraction using *Image J*. In contrast with Arampatzis et al. (2005) who assumes the resultant force is perpendicular to the biodex lever, we assumed the moment about the ankle resulted in a force perpendicular to the foot. Any change in angle of the foot relative to the biodex lever will result in an underestimation of the ankle joint moment. To estimate this error, we measured the change in angle of the foot relative to the biodex lever, and the corrected moments were calculated as:

$$M_C = M_M \times \cos(\Phi)^{-1}$$

where M_C and M_M are the corrected and measured moments, respectively, and Φ is the angle of the foot at peak moment. The corrected moments were on average 2.9% higher (range 0.7–7.4%) than the moments measured by the dynamometer. The corrected moments were used for further calculations of force and stiffness. To determine the elongation of the tendon not attributable to stretch, the ankle was passively rotated from maximal dorsiflexion to maximal plantarflexion at $5° \, s^{-1}$ using the dynamometer

while the subject lay passive with the leg relaxed. Three repetitions served as conditioning trials. On the fourth trial, ultrasound images were collected, and the translation of a distal fascicle–aponeurosis cross-point was quantified. This permitted the tendon displacement attributed to joint rotation to be subtracted from the apparent elongation measured during the MVC. The moment arm of the TST was estimated using the tendon travel method (An et al. 1984) under in vivo conditions (Ito et al. 2000; Maganaris 2000). The displacement of the fascicle–aponeurosis cross-point (dL, mm) caused by rotating the ankle from 5° of dorsiflexion to 5° of plantarflexion (dθ, rad) was calculated from the passive rotation trials described above. The TST moment arm was calculated as the ratio $dL/d\theta$ (mm rad^{-1}). Triceps surae force was calculated by dividing the ankle joint moment by the TST moment arm.

The measured force–elongation data were fitted to a quadratic equation:

$$\text{Force} = A(dL)^2 + B(dL) \tag{2}$$

TST K, defined as the force–elongation slope from 25 to 45%, 30 to 70% and 50 to 100% of maximal isometric plantarflexion force, respectively, was calculated from the quadratic force–elongation relationship. We used these ranges of force because it was felt that the usual approach of using 50–100% (Kubo et al. 2000a, b, 2001b) might emphasize a range of force that was not relevant to running (Lichtwark and Wilson 2006).

Isometric training

The training protocol was adapted from that of Kubo et al. (2001a, b) since these previous studies have demonstrated that similar training protocols caused increased TST K in physically active men of similar age. The training protocol consisted of 4 × 20 s isometric plantarflexions at 80% MVC with 1-min rest between repetitions, three times per week for 8 weeks. Training was performed on both right and left legs, independently. All training was performed on the same dynamometer in the same position as the previous test sessions, and all training was monitored by an investigator. TST K and MVC were measured every 2 weeks in the training group. Training load was adjusted based on the most recently measured MVC. EC was measured before and after 8 weeks in both the training and control groups. All subjects continued their usual run training through-out the study.

Reliability

Reliability of image analysis for tendon elongation and K was assessed on the same images of ten subjects on two different days by the same individual. Mean elongation

difference was −0.02 mm (<0.01%), and mean K difference was −9.61 N mm^{-1} (0.03%). Intraclass correlation for elongation and K were $r = 0.980$ and 0.965, respectively. Day-to-day variability in elongation and K was assessed at 45, 70 and 100% MVC for K measures at 0 and 2 weeks in the six subjects undergoing the training protocol. There was no significant change in ankle MVC or TST K over this period. Variability was assessed using Bland–Altman plots. The apparent technical error of measurement, calculated according to Hopkins (available at http://sportsci.org/resource/stats/xrely.xls) was 6.1% (4.2–11.4%, 95% CI) for elongation and 7.3% (4.1–12.6%, 95% CI) for K. It is difficult to know how much this apparent error is due to a real week-to-week change experienced by individual subjects.

Statistics

Values are presented as mean ± standard deviation (SD). Three-factor ANOVAs were performed for K (group × force range × time) and EC (group × speed × time) where time and speed were within subjects factors. Where there was no three-way interaction, two and one-factor ANOVAs were used to examine the two-way interactions and main effects (Maxwell and Delaney 2004). Where a main effect was found, Tukey's post-hoc tests were utilized to test for differences between means. Pearson product–moment correlations identified significant relationships. Statistical significance was set at $p \leq 0.05$.

Results

Average MVC for both the training and control groups are presented in Table 2. Two-factor ANOVA revealed no significant group by time interaction and no main effect of group. Average MVC for all subjects increased significantly over the course of the 8 weeks from 146.3 ± 37.9 to 172.3 ± 50.7 N m ($p = 0.02$). There was a significant increase in MVC from 0 to 4 and 0 to 6 weeks of training in the training group ($p = 0.014$ and $p = 0.016$, respectively). MVC was also significantly increased from 2 to 4

Table 2 Mean isometric moment (N m), pre- versus post-isometric training

	N	Pre-training	Post-training
Training	6	157.2 ± 28.3	191.2 ± 32.6
Control	6	135.3 ± 45.5	153.4 ± 61.1
Total	12	146.3 ± 37.9	172.3 ± 50.7

Data are expressed as mean ± SD. There was no significant interaction of group × time; however, there was a significant main effect of time on isometric moment ($p = 0.018$)

Table 3 Mean energy cost ($kJ\ kg^{-1}\ km^{-1}$), pre- versus post-isometric intervention

|	Pre-training			Post-training		
	75% sLT	85% sLT	95% sLT	75% sLT	85% sLT	95% sLT
Training	4.17 ± 0.33	4.27 ± 0.18	4.46 ± 0.16	4.21 ± 0.27	4.26 ± 0.28	4.44 ± 0.21
Control	4.40 ± 0.30	4.49 ± 0.22	4.64 ± 0.16	4.40 ± 0.20	4.50 ± 0.21	4.63 ± 0.16

Data are expressed as mean ± SD. There was no significant group × speed × time interaction nor any group × speed, group × time or speed × time interactions; however, there was a significant main effect of speed ($p < 0.001$)

and from 2 to 6 weeks ($p = 0.003$ and $p = 0.018$, respectively). The training program did result in a small increase in strength of the training group, but oddly, the control group experienced a similar increase in strength.

Values for EC at the start and end of the study are shown in Table 3 for the control and training groups. There was no significant time × speed × group interaction for EC. There was, however, a significant main effect of speed ($p < 0.001$), indicating that EC increased when speed increased from 75 to 95% of sLT. Blood lactate concentrations during the runs for all subjects were $1.5 ± 0.4$, $2.0 ± 0.5$ and $3.5 ± 1.0$ mM prior to training and $1.4 ± 0.4$, $2.0 ± 0.6$ and $3.5 ± 1.0$ mM following the training protocol. There was also no group × time interaction for blood lactate.

A force–tendon elongation curve from which TST K was calculated for one subject is presented in Fig. 4. TST K prior to and after the 8 weeks of isometric training for both the control and training groups is shown in Table 4. There was no significant interaction of time × force level × group. There was also no group × time interaction. There was, however, a significant main effect of force
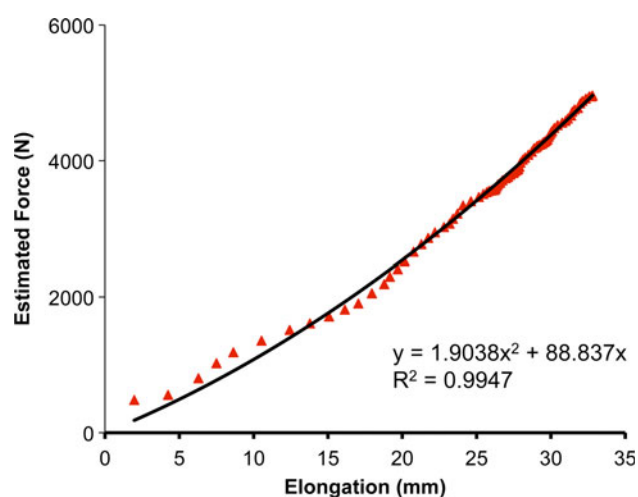
Fig. 4 Force–elongation curve during a typical MVC. *Filled triangles* represent measured force–elongation data for a typical subject. The *solid line* represents the fitted quadratic equation from which stiffness was calculated

level ($p < 0.001$), indicating that TST K was greater when measured at a higher force range. This higher TST K at higher force ranges is consistent with an increasing slope associated with the quadratic relationship of the line that was fit to the data; see Fig. 4.

Figure 5a shows the relationship between TST K and EC at 75% sLT for all subjects prior to the training program. At 75% sLT, the relationship was significant at the 30–70% MVC ($p = 0.03$) and 50–100% MVC ($p = 0.02$). The relationships were similar at 85% sLT at these same force ranges ($p = 0.05$ and $p = 0.04$, respectively). The relationship between TST K and EC at 95% sLT was significant only at 95% sLT ($p = 0.05$). The relationship was not significant at any of the other measured force levels, but a trend was evident ($p = 0.07$ and $p = 0.12$). The relationship between TST K and EC measured at 75% sLT following training is shown in Fig. 5b. The relationship was significant at all three measured force levels at 75% sLT ($p = 0.01$, $p = 0.03$ $p = 0.02$) and 85% sLT ($p = 0.02$, $p = 0.03$ and $p = 0.04$). The relationship was significant only at the lower measured force ranges (25–45% MVC and 30–70% MVC) at 95% sLT ($p = 0.04$ and $p = 0.05$), and a trend was evident at the highest force level ($p = 0.057$).

The relationship between MVC and TST K in the training and control groups was significant at all three force ranges prior to the training program ($r^2 = 0.33$, 0.36, 0.35; $p < 0.05$ across force ranges). Following the training program, the strength of the relationship was increased compared to before training ($r^2 = 0.57$, 0.60, 0.60; $p < 0.005$ across force ranges).

There was a significant relationship between the change in TST K and the change in MVC following the training protocol, in both the control and training groups when stiffness was measured from 30 to 70% MVC and 50 to 100% MVC ($p = 0.04$ and 0.02, respectively). This result was not significant when stiffness was measured from 25 to 50% MVC ($p = 0.10$).

Because there was no group × time interaction for the results of TST K and EC, we combined groups for consideration. Figure 6 shows the relationship between the change in TST K and the change in EC, measured as the

Table 4 Tendon stiffness (N mm^{-1}), pre- versus post-isometric intervention

	Pre-study			Post-study		
	25–45% MVC	30–70% MVC	50–100% MVC	25–45% MVC	30–70% MVC	50–100% MVC
Training	300 ± 71	322 ± 59	366 ± 56	322 ± 76	366 ± 93	434 ± 122
Control	248 ± 90	285 ± 106	340 ± 130	218 ± 93	249 ± 113	295 ± 145

Data are expressed as mean ± SD. There was no significant group × force range × time interaction nor any group × force range, group × time or force range × time interactions; however, there was a significant main effect of force range ($p < 0.001$)
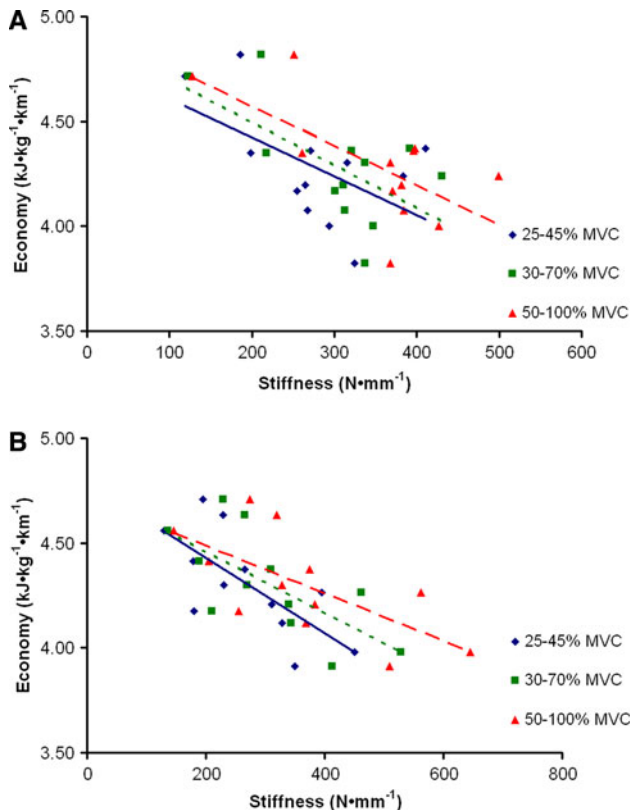
Fig. 5 The relationship between economy (75% sLT) and tendon stiffness at the three measured force levels prior to (**a**) and following training (**b**). *Filled diamonds* represent stiffness measured from 25 to 45% MVC, *filled squares* represent stiffness measured from 30 to 70% MVC and *filled triangles* represent stiffness measured from 50 to 100% MVC. *Solid lines* represent the economy–stiffness relationship from 25 to 45% MVC; *dashed lines* represent the relationship from 30 to 70% MVC, and *long-dashed lines* represent the relationship from 50 to 100% MVC. The relationship was significant at the 30–70% MVC ($p = 0.03$) and 50–100% MVC ($p = 0.02$). The relationships were similar at 85% sLT at these same force ranges ($p = 0.05$ and $p = 0.04$, respectively). The relationship at 95% sLT was not significant at any of the measured force levels, but a trend was evident ($p = 0.051$, $p = 0.07$ and $p = 0.12$). Following training, the relationship at 75% sLT following was significant at all three measured force levels at 75% sLT ($p = 0.02$, $p = 0.02$ and $p = 0.03$) and 85% sLT ($p = 0.02$, $p = 0.03$ and $p = 0.04$). The relationship was significant only at the lowest measured force range at 95% sLT
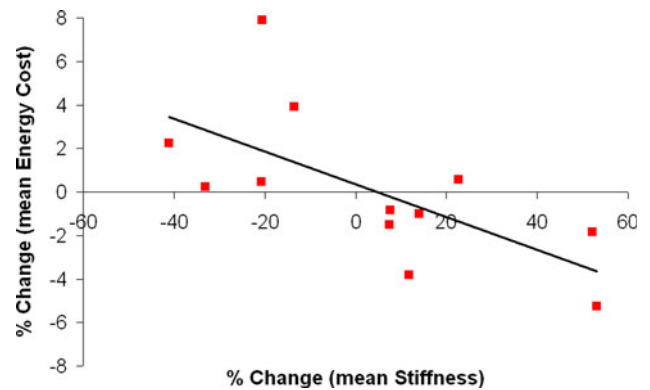
Fig. 6 Relationship between relative change in stiffness and change in economy. Data are expressed as an average % change from baseline across all measured velocities (75, 85 and 95% sLT) and force levels (25–45, 30–70 and 50–100% MVC) following the 8-week training protocol for all subjects. The relationship is significant ($r = -0.723$; $p = 0.005$)

difference between the final value and initial value for all subjects and averaged across all three force levels and running speeds, respectively. There was a significant relationship between the % change in TST K and % change in EC when all velocities and force levels were combined ($r^2 = 0.44$, $p = 0.02$). Note that the range of values of change in TST K is relatively much greater than the range of values of change in EC.

Discussion

The main finding in this study was that there was a significant relationship between the relative change in average EC across all three measured running velocities and the relative change in TST K when all subjects were considered. Furthermore, our results confirmed that a negative relationship exists between RE, measured as EC, and TST K, confirming the results of Arampatzis et al. (2006) despite the current study having a more homogeneous group of runners. This suggests that a higher TST K is

associated with a lower energy cost to run a given distance. This observation must be tempered with previous suggestions that there appears to be an optimal tendon stiffness, beyond which the energy cost of running must increase (Lichtwark and Wilson 2007, 2008).

It is not well understood how specific mechanical alterations of tendon can affect the energy cost of muscle contractions; however, it is suggested that the energy cost of contraction is related to both the amount of shortening and the shortening velocity (Stainsby and Lambert 1979). Lichtwark and Wilson (2008) have suggested that muscle fascicles and their tendon interact in order to minimize the contractile energy cost of a working muscle during movement. These authors speculate that the behavior of tendons and fascicles are each adjusted in order to maximize the power–velocity relationship. In doing so, high forces are permitted without requiring higher motor-unit activation. In particular, with submaximal activation, such as is seen during running, the power–velocity relationship is maximized at low shortening velocities (Chow and Darling 1999). It is speculated that the findings of the current study are the result of specific mechanical alterations of the triceps surae tendon in order to optimize the muscle fascicle shortening velocity to maximize power output at levels of muscle activation during the run trials. In doing so, the energetic cost of contraction can be minimized.

There has been ample research regarding muscle and tendon behavior during human movement with particular attention paid to the mechanics of the muscle's tendon which need not be elaborated here (Arampatzis et al. 2006; Hof et al. 2002; Lichtwark and Wilson 2008; Lichtwark and Barclay 2010; Stafilidis and Arampatzis 2007). There are apparent advantages of stiff tendons in some cases and compliant tendons in other cases. Each of these advantages will be described briefly below.

The lengthening of a tendon for energy storage is relevant in stretch-shortening cycles where a substantial pre-stretch of the tendon occurs early in a contraction. A compliant tendon allows more energy storage during lengthening. The energy absorbed in this case was kinetic or gravitational. This energy can subsequently be released upon shortening. A compliant tendon may also help by allowing the tendon to lengthen upon stretch, thereby keeping fascicle shortening velocity low and optimizing the power–velocity relationship.

Conversely, a highly compliant tendon may interfere with the direct transmission of muscle shortening to joint movement (Roberts 2002), and thus, a stiffer tendon may be best suited to provide power for certain movements, where a substantial pre-stretch of the tendon does not occur (Alexander 1974; Biewener and Roberts 2000).

In the case of the TST, it does not appear to undergo a substantial pre-stretch during running (Lichtwark et al. 2007); thus, energy storage can only come from active contraction of the muscles connected to the tendon. Reducing the compliance would reduce the energy transfer to the tendon and therefore reduce the energy cost of the associated muscle contractions. Where a substantial pre-stretch does not occur, a more compliant tendon requires greater muscle fiber shortening and/or velocity of shortening for a given joint movement. As a result, in the case of the TST, force transmission to the joint may be favoured over elastic energy storage and release since for a given amount and rate of muscle tendon unit shortening, less muscle fiber shortening is needed with a stiff tendon compared to a compliant one where additional fiber shortening is needed to accommodate tendon stretch.

Based on the results of this study, it appears that this mechanism may be the one that is operating for the triceps-surae muscles in this group of athletes. This speculation is consistent with previous findings which have shown that during running, very little pre-stretch of the medial gastrocnemius fascicles occur, and that these fascicles begin to shorten even prior to ground contact (Ishikawa et al. 2007; Ishikawa and Komi 2008), thus favouring force transmission over elastic energy storage in the triceps surae tendon. The optimal stiffness (Lichtwark and Wilson 2007) probably keeps muscle fiber length change to a minimum; so, it could be imagined that a tendon could be too stiff, thereby requiring muscle fiber shortening during each step in running. This would increase the energy cost of running.

It is somewhat disappointing that the current training protocol did not induce the same kinds of tendon adaptations as those previously demonstrated (Kubo et al. 2001a, b). The lack of a group effect in the EC–TST K relationship suggests that factors in addition to strength training were affecting strength and TST K. This could be a result of the current subject pool's higher TST K to begin with or that the substantial, long-term tendon mechanical adaptations from chronic run training require an adaptation period longer than the 8 weeks of the current study. It is also known that aerobic training interferes with adaptations to a strength training program (Nader 2006). This interference may have prevented us from seeing the anticipated substantial change in strength. However, it seems reasonable to surmise that TST K can change acutely, and this change can bring about a change in EC. The data support the theory that increased TST K does associate with improved EC (Arampatzis et al. 2006; Storen et al. 2008). Since the EC measurements were made immediately following the measurement of TST K, these measurements represent a condition that occurs at the same time. The strong relationship observed indicates that both measures are somewhat labile, and that they co-vary.

It cannot be ignored though that four of the 12 subjects experienced a decrease in tendon stiffness following the

training protocol. This was associated with an increase in energy cost of running. It is speculated that the short-term fatiguing effects of recent run training performed by these athletes may have caused these changes to occur.

Fatigue acutely affects RE, and this effect can persist for several days following exhaustive exercise (Hunter and Smith 2007; Nicol et al. 1991; Xu and Montgomery 1995). Furthermore, although not universally reported (Arampatzis et al. 2007; Mademli et al. 2006; Mademli and Arampatzis 2008), repetitive contractions have been shown to cause decreased tendon stiffness (Kay and Blazevich 2009; Kubo et al. 2001c, d; Wang et al. 1995). These inconsistent findings may relate to the absolute strain experienced by the tendon during the contractions in that high tendon strain (4–5%) is associated with decreased TST K, whereas low strain (<3%) is not (Arampatzis et al. 2007). High tendon stress associated with forceful muscular contractions occurs during running, and these repetitive contractions may actually result in subsequent, but transient decreased TST K. To date, it remains unclear how long this decrease in TST K persists following fatiguing contractions similar to those regularly performed by this subject pool, but there would have to be a subsequent positive consequence of these changes to bring about the training adaptations referred to above. On this basis, it is suggested that the concomitant decrease in TST K and increase in EC seen in four subjects (3 control, 1 training group subject in Fig. 6) may be a result of training performed in the days immediately preceding the final measurement of EC and TST K and may be a result of persistent fatigue-related decreases in TST K associated with recent run training. Taken together, the higher TST K as well as the shorter training protocol apparently prevented the anticipated significant increase in TST K within the training protocol.

The regular run training of the athletes in both the training and control groups and how TST K may be affected acutely as a result of this training cannot be overlooked. TST K is obviously a very labile feature, and can be affected in either a positive or negative way. Studies have shown what appear to be long-term changes in TST K that can be affected by various forms of strength training (Kubo et al. 2002, 2006), and short-term changes due to fatiguing repetitive contractions (Kay and Blazevich 2009; Kubo et al. 2001c, d). It is hypothesized that the TST strain experienced during running (approximately 5.5%; Lichtwark et al. 2007) is sufficient to induce short-term decreases in TST K. While the current study did not demonstrate a significant increase in TST K after performing an isometric training protocol, the fact that our subjects had a higher TST K than that previously reported (Arampatzis et al. 2006; Rosager et al. 2002) suggests that chronic run training, the type that is regularly performed by these athletes, probably contributes to both an increase in

TST K and a decrease in EC. It is quite possible that the change in TST K is one of the major factors that contributes to the change in the energy cost of running.

Finally, this study was the first to measure TST K at lower force ranges. In general, the relationship between TST K and EC both before and after the training program was strongest when K was measured at lower force ranges. This suggests that measuring TST K at lower force ranges may be more relevant in activities where submaximal muscle activation is required, like in running.

Conclusion

The current study found that both TST K and RE can change acutely, and that both variables appear to change together. It is speculated that this is a result of acute response to high-intensity training and/or a long-term training adaptation. The labile nature of the TST K and its resultant effects on EC suggests that this variable is an important one to consider in performances where EC is of importance. A short period of 8 weeks of strength training is not sufficient to alter the strength or stiffness of the TST tendon in a consistent manner in trained runners. Further studies are required to demonstrate the time-course of change for TST K following a bout of exercise. The labile nature of both TST K and EC has important implications for performance, such as during a taper in preparation for an important competition.

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