Effects of resistance training on tendon mechanical properties and rapid force production in prepubertal children

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Submitted 9 April 2014; accepted in final form 3 June 2014

Achilles tendon properties (cross-sectional area, elongation, stress, strain, stiffness, and Young’s modulus), electromechanical delay, rate of electromyographic (EMG) increase (REI; slope of the EMG time curve), rate of force development (RFD; slope of the force-time curve), and rate of force development (RFD; slope of the EMG time curve) were measured before and after RT. Tendon stiffness and Young’s modulus increased significantly after RT in the experimental group only (−29% and −25%, respectively); all other tendon properties were not significantly altered, although there were mean decreases in both peak tendon strain and strain at a given force level (14% and 24%, respectively; not significant) which may have implications for tendon injury risk and muscle fiber mechanics. A decrease of −13% in EMD was found after RT for the experimental group, which paralleled the increase in tendon stiffness (r = −0.59); however, RFD and REI were unchanged. The present data show that the Achilles tendon adapts to RT in prepubertal children and is paralleled by a change in EMD, although the magnitude of this change did not appear to be sufficient to influence RFD. These findings are of importance within the context of the efficiency and execution of movement.

It is well accepted that resistance training (RT) is an effective and beneficial mode of exercise for children, adolescents, and young athletes if appropriately supervised (16). Positive outcomes of RT are reported to include improvements in cardiorespiratory fitness (7), bone mineral density (45), psychosocial health (17), and motor performance and coordination (3, 7). RT also promotes strength gains beyond those associated with normal growth (56) as a result of improved intermuscular coordination and an increased motor unit activation, rather than changes to muscle size or structure (48, 52).

As age- or training-related increases in muscular strength occur, a synchronous adaptation of the tendon’s mechanical properties must be achieved so that 1) these greater forces can be efficiently transmitted to the bone without damaging the tendon, and 2) the tendon elongation experienced under the greater loads is maintained, enabling skeletal muscle fibers to continue to work within their normal operating range (26, 64). In adults, tendons adapt to chronic increases in loading by increasing stiffness via tendon hypertrophy (11, 31) or an increase in the Young’s modulus, a dimensionless measure of material stiffness that provides information about the tendon’s underlying microstructure (33, 53). In children, age-related increases in body mass and muscular strength, which increase tendon loading, correlate well with age-related increases in Achilles tendon stiffness (62). This finding is consistent with the positive relationship observed between muscular strength and tendon stiffness in adults (42) and supports the notion that children’s tendons can also adapt to changes in chronic loading, as has been indicated in other young animals (e.g., (27)). Nonetheless, it is not known whether RT alters tendon mechanical properties beyond those associated with normal growth and development, or the mechanisms by which potential adaptations might occur. This is of particular importance because tendon stiffness is determined by both its physical dimensions [length and cross-sectional area (CSA)] and its material stiffness (Young’s modulus), both of which increase during childhood but at different rates, which leads to an assumption that certain aspects of tendon maturation are more important or receptive to adaptation than others (62). Furthermore, it has been shown that short-term RT does not necessarily result in muscle hypertrophy in children (48, 52). If RT improves muscular strength but does not result in adequate tendon adaptations in children, the change in ratio between these parameters could have important consequences for both the risk of strain-related injury and muscle fiber mechanics.

Understanding the relational change in muscle strength and tendon stiffness is important because tendons play an integral role in movement by transferring muscular forces across joints to the bones. Specifically, tendon stiffness influences explosive force production by affecting electromechanical delay (EMD), a measure describing the time lag between muscle activation and muscle force production, and the rate of force development (RFD), a measure of the rate of rise in force after the onset of a muscle contraction. A delay in transferring rapidly generated forces may have important consequences for balance and stability (21, 54) by slowing movement reaction times (25) and increasing the risk of falls (28, 50). Previous research indicates that EMD and RFD can be augmented with training in adults,

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and is typically attributed to an increased musculo-tendinous stiffness (22, 65), neural drive (1, 13, 61), or a combination of these mechanisms. Furthermore, we have previously shown that EMD and RFD are related to tendon stiffness and neural drive in children (63). However, it is not known whether or to what extent these parameters may be augmented with RT in children. Although EMD is influenced by the physiological mechanisms associated with the excitation-contraction coupling process (46), the tendon’s stiffness is believed to account for a large proportion of EMD by governing the time taken to stretch the tendon to a point at which muscular forces can be effectively transferred from the muscle to the bone (10). RFD on the other hand appears to be heavily dependent upon neuromuscular factors, evidenced by parallel increases in contractile RFD and rate of muscle activation (1, 13, 61). This is believed to occur because the rate of muscle activation, often estimated as the rate of electromyographic (EMG) amplitude increase (REI), has been previously related to muscle shortening velocity (44), a factor directly related to RFD (24). On the basis of this relationship, training-induced increases in RFD and REI may be expected to occur concurrently and be more strongly related to the change in REI than a change in tendon stiffness.

The main purposes of this study were to examine the effects of plantar flexor RT on the mechanical properties of the Achilles tendon in prepubertal children, and to determine the mechanisms underpinning potential adaptations. The Achilles tendon was chosen for examination because of its significant role in both daily and exercise-related tasks. It was hypothesized that children’s tendon stiffness would increase in response to RT. Because tendon hypertrophy is not commonly found following short-term RT in adults despite increases in tendon stiffness (33, 53), it was also hypothesized that RT-induced increases in tendon stiffness would be predominantly attributed to adaptations in material properties rather than to dimensional changes. To gauge the potential impact of increased tendon stiffness on muscle mechanics and physiological limits of the tendon, we also examined tendon stress and strain, and their individual components. In an effort to probe the mechanisms underpinning changes in explosive force production capacity in children after an intervention, a secondary purpose of the study was to examine the effects of plantar flexor RT on EMD and RFD and to determine whether any changes are related to changes in stiffness, REI, or both.

**METHODS**

**Ethical approval and participant information.** Ten boys and 10 girls (age 8.9 ± 0.3 yr) volunteered to participate in this study. Institutional ethical approval was granted by the Human Research Ethics Committee at Brunel University. The research was conducted in accordance with the guidelines set in the Declaration of Helsinki. Written assent and consent was provided by the children and their guardians, respectively. Physical activity readiness questionnaires were completed for each child by the guardians to ensure all children were physically capable of undergoing the RT intervention. All participants were free from known neuromuscular or musculoskeletal disorders, and were not involved in competitive sports or had previous RT experience. Each child’s peak height velocity offset (an indicator of biological maturity) was estimated to confirm their prepubertal status (40). Participants were divided into control (age 8.9 ± 0.3 yr; height 135.7 ± 6.6 cm; mass 33.0 ± 9.2 kg) and experimental (age 8.9 ± 0.2 years; height 136.3 ± 5.6 cm; mass 28.4 ± 4.7 kg) groups depending on school class, which were allocated at random 1 wk prior to study commencement. Each group contained 5 girls and 5 boys. Every child visited the laboratory on two separate occasions to allow data to be collected before and after 10 wk of plantar flexion RT.

**Resistance training program.** For the experimental group, plantar flexion RT was performed twice weekly as part of a class-based physical education lesson. The lesson was arranged as circuit training comprising nine stations each accommodating up to three children, with plantar flexion RT set up as one of the stations. For the class containing the control group, this station was replaced with a rest station. The remaining stations were varied throughout the program to help maintain participant motivation. They typically included unrelated activities such as basketball shooting, football dribble, hockey slalom, sit-ups, etc. Groups rotated between stations at the sound of the investigator’s whistle, which was blown when either all children at the plantar flexion RT station had completed one set of repetitions (experimental), or every 2 min (control). Rotations continued until all scheduled sets had been completed by the experimental participants, which correlated to ~50 min for the control group.

Children in the experimental group were familiarized with the recumbent (45° incline) calf-raise machine (GLPH1100; Body Solid, Forest Park, IL) on two separate occasions prior to the start of the 10-wk training period. During the first session, participants were taught the correct technique on an unloaded machine (foot plate mass of 30 kg). Once seated, participants placed the balls of their feet on the footplate of the machine and were instructed to slowly and fully extend the knees (Fig. 1). They then raised and lowered the footplate through a full but comfortable range of motion using slow concentric then eccentric ankle plantar flexion contractions (2–3 s per cycle) for both safety reasons and to achieve a long muscle contractile (and thus tendon loading) period. On the second occasion, participants completed a single set of 6–8 repetitions on an unloaded machine, and scored their perceived effort using a 7-level pictorial modification of the Borg scale of perceived exertion. Descriptors for each picture were very very easy, very easy, somewhat easy, somewhat hard, hard, very hard, and very very hard. The number of repetitions prescribed for the initial training session was then based on their perceived effort during this set (very hard to very very hard, 8 repetitions; somewhat hard to hard, 10 repetitions; very very easy to somewhat easy, 12 repetitions).

The first author of this study was always present at the resistance machine to ensure that the appropriate technique, applied load, and number of repetitions were adhered to and recorded. Because measurement of 1 RM in children is contraindicated, the frequency and intensity of the RT repetitions was based on progressive loading to generate strength gains where we could set loads on the basis of individual strength capacities without the need for continuous 1 RM monitoring. This was in accordance with the current recommendations for RT in primary school children (16). In the first 2 wk, two sets of
8–15 repetitions were performed with an emphasis on safety and technique rather than maximal loading. Therefore, participants trained without additional load being added to the RT machine (i.e., lifting the 30 kg footplate only). If 15 repetitions could be completed with this load by the fourth set, an additional load of 2.5 kg was added to generate a new 8–10 RM for the following session. This process was performed iteratively during the following 8 wk. In each session of the remaining 8 wk, three sets of 8–15 repetitions were performed. The mean (±SD) load (kg) lifted (i.e., load added to the weight stack) during the 10-wk RT schedule is shown in Table 1.

**Pre- and posttraining data acquisition.** Participant mass, height, tendon force at maximum voluntary contraction (MVC; peak tendon force), elongation, CSA, stress, strain, stiffness (k) and Young’s modulus (E), EMD, RDF, and REI were obtained pre- and post-RT. Comprehensive methodologies for calculating these variables have been detailed previously (62, 63). Therefore, only brief descriptions will be provided here.

**Measurement of ankle joint moment.** The moment during the ankle joint was measured using an isokinetic dynamometer (Biodex Medical Systems, New York). Participants were seated with relative internal hip, knee, and ankle angles of 95°, 180°, and 90°, respectively. This joint configuration best matched that during RT and meant that the gastrocnemius muscles were at a mechanically advantageous length for maximal force production (12). Although an extended knee may have slightly less functional relevance than a flexed knee, it enabled the greatest tendon loading and provided maximal measures for the attainment of reliable force-elongation curves. The lateral malleolus of the right fibula was aligned with the dynamometer’s rotational center, and the foot, thigh, and torso were secured with stabilization straps. Plantar flexion contractions were completed with the participants’ arms crossed over their chest. They performed 5–8 submaximal isometric plantar flexion contractions to ensure that only the plantar flexor muscles were used in developing an ankle joint moment (contractions were performed under the instruction to “rotate the foot away from the body using the ball of the foot”). The maximum plantar flexor moment was then identified from three to five subsequent maximal contractions. This protocol provided both a task-specific warm-up and tendon preconditioning exercise (57). To minimize the likelihood of muscular fatigue, the familiarization and testing protocols were separated by a 5-min compulsory rest period.

Three plantar flexion MVCs were then performed with a maximum RFD. Participants were instructed not to flex the ankle prior to performing each plantar flexion and were given a 3-s count down before each contraction, during which they were instructed to push as hard and fast as possible. Verbal encouragement was provided during plantar flexion efforts for motivation (38). Consecutive contractions were separated by a 30-s rest period. Net plantar flexor moment and joint position data were sampled simultaneously at 1,000 Hz using a 12-bit A/D card (NI PCI-6071E; National Instruments, Austin, TX) and low-pass filtered using a fourth-order, zero-lag Butterworth filter with a 14-Hz cutoff frequency.

**Electromyographic measurement of muscle activity.** Muscle activity of the tibialis anterior (TA) was assumed to represent antagonist coactivation during plantar flexion, whereas gastrocnemius medialis (GM) muscle activity was captured for calculating both EMD and REI. The skin was rubbed with a sanitizing wipe before placing self-adhesive electrodes (Kendall H59P; Covidien, Ireland) approximately in parallel with the orientation of the underlying fascicles, using a bipolar setup with a center-to-center interelectrode distance of 20 mm. A reference electrode was positioned on the tibial plateau. Real-time EMG signals sent via telemetric transmission at 1,000 Hz to a remote receiver (Telemyo 2400R; Noraxon, Scottsdale, AZ) were synchronized with the dynamometer analog data. EMG signals were digitally filtered using a 10–500 Hz band-pass filter (Spike2 v5.12a software; Cambridge Electronic Design, Cambridge, UK). The EMG data were further processed to derive the various dependent variables as detailed below.

**Moment arm estimation.** Achilles tendon moment arm was estimated as the mathematical derivative of GM muscle-tendon junction (MTJ) excursion with respect to angular ankle displacement (19). The ankle joint was passively rotated throughout a 20° range of motion while displacement of the GM MTJ was imaged using real-time B-mode ultrasonography (45 mm linear array probe, Megas GPX, Esaote, Italy; 10 MHz transducer scanning). Probe movement relative to the skin was identified and adjusted by positioning a strip of echo-absorptive tape on the skin perpendicular to the approximate line of GM MTJ movement. Ultrasound images were captured at 25 Hz (ADV-C5; Grass Valley, France), and the GM MTJ was identified manually at each frame (Peak Motus v9; Vicon, Oxford, UK). Filtered angular displacement data were down-sampled to match the sampling frequency of the ultrasound data. A third-order polynomial was fitted through the GM MTJ excursion-ankle displacement curve over an angular displacement of 20°. The polynomial was then differentiated at the neutral (90°) ankle position to obtain the moment arm.

**Calculation of tendon properties.** TA EMG data were smoothed using a root-mean-square algorithm with a 100-ms averaging window. The TA EMG-moment relationship during a ramped dorsiflexion contraction was approximated by fitting a second-order polynomial to the data ($R^2 = 0.98 ± 0.01$ across participants) corresponding to at least the greatest level of TA EMG-moment data observed during the plantar flexion trials. Using these polynomials, the antagonist moment during plantar flexion was estimated and added to the net recorded moment to yield the gross plantar flexor moment. Achilles tendon force was then calculated as the ratio of the coactivation-adjusted plantar flexor moment to the Achilles tendon moment arm. Tendon elongation, defined as GM MTJ displacement from rest, was imaged with ultrasonography and processed in the same manner as described above. This measurement was corrected for distal tendon movement, estimated as the product of angular rotation and Achilles tendon moment arm length (20, 37) to reduce the possibility of underestimating tendon elongation and therefore overestimating tendon stiffness (37). Tendon stiffness (k) was estimated as the slope of the linear tendon force-elongation relationship between 10–90% of the maximum attained force for each participant (62). Post-RT, k was first calculated from the peak force attained post-RT (i.e., absolute force) to allow the assessment of this measure from a functional perspective, and second from the peak force attained pre-RT (i.e., relative force) to allow for the direct comparison of k from pre- and post-RT independent of any strength gains. These calculation methods are subsequently referred to as MVCpost and MVCpre, respectively. The mean intraindividual coefficient of variation for estimating $k$ from three MVCs was 6.9 ± 4.3%.

Peak tendon strain was calculated as the tendon elongation associated with peak tendon force, relative to its resting length, where resting tendon length was defined as the linear distance between the GM MTJ and the tendon insertion at the proximal calcaneus at a

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**Table 1. The 10-wk training schedule and load lifted per session**

<table>
<thead>
<tr>
<th>Week</th>
<th>1–2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Session 1</td>
<td>30.0 ± 0</td>
<td>32.5 ± 0</td>
<td>34.5 ± 1</td>
<td>41.7 ± 3.3</td>
<td>49.1 ± 2.7</td>
<td>51.0 ± 1.8</td>
<td>53.1 ± 1.7</td>
<td>58.9 ± 2.1</td>
<td>60.0 ± 2.1</td>
</tr>
<tr>
<td>Session 2</td>
<td>30.0 ± 0</td>
<td>32.5 ± 0</td>
<td>37.3 ± 0.7</td>
<td>44.7 ± 2.9</td>
<td>47.3 ± 1.6</td>
<td>50.8 ± 1.2</td>
<td>55.8 ± 3.4</td>
<td>59.5 ± 2.0</td>
<td>62.3 ± 2.2</td>
</tr>
</tbody>
</table>

Loads are presented as mean (±SD) in kilograms. Two sets of 8–15 RM were completed in each session during weeks 1 and 2, increasing to 3 sets during weeks 3–10. Pretraining and posttraining testing occurred during weeks 0 and 11, respectively.
neutral ankle angle. To derive tendon stress and Young’s modulus (E), the tendon’s CSA was transversely imaged at the narrowest part of the tendon, ~25 mm from the proximal calcaneus, using ultrasonography (8). Peak tendon stress was calculated as the ratio between the mean maximum attained force and smallest CSA measured from each participant. E was estimated as the slope of the tendon stress-strain relationship between 10–90% of peak tendon stress for each participant. As with k, each of these measures were also calculated using both MVCpre and MVCpost force levels.

Electromechanical delay. Filtered (unsmoothed) GM EMG data were corrected for digital conversion offsets and underwent full-wave rectification (Spoke 2 v5.14; Cambridge Electronic Design). Raw joint moment and GM EMG baseline activity were calculated over a 200-ms window. The signal onsets for both signals were defined as the times at which a value of the respective signal rose above two standard deviations from the baseline mean (36) for a minimum of 10 ms (14). Although these criteria were used in determining the signal onsets, onsets were visually verified to ensure accuracy because automated methods of signal onset detection are often less accurate (60). Trials displaying a moment decrease greater than 0.5 Nm from baseline were discarded because the moment onset could not be confidently identified. EMD of the GM muscle was calculated as the time lag between the onset of GM EMG activity and the onset of plantar flexor moment for each MVC (Fig. 3A). The mean intrindividual coefficient of variation for calculating EMD from three MVCs was 9.4 ± 4.8%.

Rate of force development. RFD was measured as the slope of the force-time curve obtained during isometric contractions. RFD measures provide both functional and mechanistic information about force development at different stages of a muscular contraction (2). In the present study, absolute RFD (RFDabs) was calculated from the onset of force to 25, 50, 100, and 200 ms. These intervals were chosen to provide insight into the physiological mechanisms that might underpin potential improvements in force production after RT and to match the intervals of previous studies [e.g., (1, 6)]. RFD was also calculated in relation to peak force (RFDnorm). By removing differences in strength between individuals or increases in strength following RT, RFDnorm can be directly compared between individuals and before and after RT (1, 6). Although improvements in RFD after RT are commonly reported in the earliest stages of force development in adults due to improvements in REI (1), the relationship between RFD and k has been shown to be strongest in later stages of force development in children (63). As such, it was deemed that improvements in RFD may have occurred at any stage of an MVC, warranting the calculation of RFDnorm to every 10% of MVC up to 90% MVC. RFDabs and RFDnorm are reported in kN/s and %MVC/s, respectively.

Rate of EMG amplitude increase. REI was measured as the slope of the EMG-time curve obtained from isometric contractions. Filtered GM EMG signals were smoothed using a root-mean-square over a 50-ms moving window (1, 6). After normalizing GM EMG data to peak EMG amplitude, absolute and normalized REI (REIabs and REInorm, respectively) were calculated from the onset of GM EMG activity to 25, 50, 75, and 100 ms intervals and to every 10% interval of peak EMG amplitude, up to 70%.

Statistical analysis. During the course of the RT intervention, one boy from the experimental group relocated and one girl from the control group was absent from the post-RT testing due to illness. Therefore, statistical analyses were performed for data on 9 experimental (5 girls, 4 boys) and 9 control (4 girls, 5 boys) participants. All data were analyzed using SPSS statistical software (SPSS v18.0, Chicago, IL). Pre-RT differences in descriptive body statistics (height, mass), tendon-related variables (CSA, elongation, stress, strain, k, and E), EMD, RFD, and REI time and force interval between groups were examined using independent t-tests to ensure that results would not be confounded by possible pre-RT group differences in musculoskeletal characteristics. Each variable was then examined with respect to time (pre-post) and group (control; experimental) using 2 × 2 factorial ANOVA. For these analyses, RFD and REI measured to time intervals were log-transformed to satisfy the assumptions of homogeneity of variance. In the case of significant time-by-group interactions, paired t-tests compared pre- to post-RT values in each group. The relationship between k and EMD was further investigated by calculating the Pearson product-moment correlation coefficients (r) and coefficients of determination (R2) of the group data illustrating the relative changes in EMD and k of each individual from pre- to post-RT. The results provided no case for examining the relationship between k and RFD (see Discussion). Statistical significance was accepted at P < 0.05.

RESULTS

Intervention compliance in the control and experimental groups was 92.6% and 96.7%, respectively. The mean training load for the experimental group increased from 30.0 kg in the first training session to 62.3 kg in the last training session (increase of 208%; Table 1). There were no significant differences between the control and experimental groups for any variable measured pre-RT (P = 0.118–0.992). For each variable measured, F-statistics and levels of significance for repeated measures ANOVA interaction effects can be found in Table 2, whereas mean (± SD) values and percentage changes in tendon variables (both for MVCpre and MVCpost) are presented in Table 3.

Table 2. ANOVA interaction effect statistics for descriptive data, tendon-related variables, EMD, RFD, and REI

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Time × Group</th>
<th>F(1, 16)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass</td>
<td></td>
<td>0.888</td>
<td>0.360</td>
</tr>
<tr>
<td>Height</td>
<td></td>
<td>0.250</td>
<td>0.624</td>
</tr>
<tr>
<td>Tendon CSA</td>
<td></td>
<td>0.417</td>
<td>0.528</td>
</tr>
<tr>
<td>Isometric force</td>
<td></td>
<td>0.587</td>
<td>0.456</td>
</tr>
<tr>
<td>Stress, MVCpost</td>
<td></td>
<td>0.001</td>
<td>0.976</td>
</tr>
<tr>
<td>Stress, MVCpre</td>
<td></td>
<td>0.334</td>
<td>0.572</td>
</tr>
<tr>
<td>Strain, MVCpost</td>
<td></td>
<td>1.889</td>
<td>0.188</td>
</tr>
<tr>
<td>Strain, MVCpre</td>
<td></td>
<td>1.542</td>
<td>0.232</td>
</tr>
<tr>
<td>Elongation, MVCpost</td>
<td></td>
<td>2.253</td>
<td>0.153</td>
</tr>
<tr>
<td>Elongation, MVCpre</td>
<td></td>
<td>1.904</td>
<td>0.187</td>
</tr>
<tr>
<td>Stiffness, MVCpost</td>
<td></td>
<td>10.368</td>
<td>0.005*</td>
</tr>
<tr>
<td>Stiffness, MVCpre</td>
<td></td>
<td>6.574</td>
<td>0.021*</td>
</tr>
<tr>
<td>Young’s modulus, MVCpost</td>
<td></td>
<td>9.245</td>
<td>0.008*</td>
</tr>
<tr>
<td>Young’s modulus, MVCpre</td>
<td></td>
<td>6.538</td>
<td>0.021*</td>
</tr>
<tr>
<td>EMD</td>
<td></td>
<td>3.176</td>
<td>0.096</td>
</tr>
<tr>
<td>RFDabs, ms</td>
<td></td>
<td>1.944–0.006</td>
<td>0.185–0.807</td>
</tr>
<tr>
<td>RFD, %</td>
<td></td>
<td>1.338–0.009</td>
<td>0.264–0.924</td>
</tr>
<tr>
<td>REIabs, ms</td>
<td></td>
<td>0.205–0.016</td>
<td>0.658–0.902</td>
</tr>
<tr>
<td>REI, %</td>
<td></td>
<td>2.942–0.009</td>
<td>0.106–0.926</td>
</tr>
</tbody>
</table>

CSA, cross-sectional area; EMD, electromechanical delay; MVCpost, maximum voluntary contraction after resistance training; MVCpre, maximum voluntary contraction before resistance training; REI, rate of electromyographic increase; RFD, rate of force development. The MVCpre variable was normalized to pretraining MVC force. *P < 0.05.
significant for the control group ($P = 0.378$ and $P = 0.659$, respectively). Despite this, no time-by-group interactions were found for stress, strain, or elongation, calculated using either MVC$_{pre}$ ($P = 0.174–0.976$) or MVC$_{post}$ ($P = 0.222–0.572$). Additionally, no time-by-group interactions were found for tendon CSA ($P = 0.517$) or peak force ($P = 0.456$). Group mean and individual changes in tendon variables (on the basis of MVC$_{pre}$) are presented in Fig. 2.

EMD decreased on average by 9.8 ms ($-13\%$) in the experimental group (pre-RT 76.3 ± 11.0 ms; post-RT 66.5 ± 10.2 ms) and remained unchanged in the control group (pre-RT 74.5 ± 10.8 ms; post-RT 74.2 ± 12.3 ms), however, the time-by-group interaction for EMD did not reach statistical significance ($P = 0.096$). Changes in EMD were moderately correlated with changes in $k$ in the experimental group ($r = -0.59$, $P = 0.066$) and poorly correlated in the control group ($r = -0.24$, $P = 0.278$), as shown in Fig. 3. No time-by-group interaction effects were found for RFD ($P = 0.185–0.924$) or REI ($P = 0.106–0.926$) calculated to any interval examined after RT (Figs. 4 and 5). The lack of change in RFD precluded an examination of the relationship between the change in $k$ and the change in RFD. Group means and percentage changes for each tendon-related variable (both MVC$_{pre}$ and MVC$_{post}$) are reported in Table 3.

**DISCUSSION**

The first purpose of the present study was to test the hypothesis that planar flexor RT would change the mechanical properties of the Achilles tendon in prepubertal children. The main finding was that the mechanical properties of the Achilles tendon were significantly altered by 10 wk of twice-weekly RT in previously untrained prepubertal children. In the experimental group, tendon stiffness ($k$) increased by 29% when calculated using absolute tendon force and elongation (i.e., MVC$_{post}$). These values are similar to those reported previously for adults (15–64%) performing moderate duration (8–14 wk) RT programs (31–33, 53, 58). The results suggest that immature human Achilles tendons respond to chronic loading in a similar way to mature tendons, thereby adding to previous findings by confirming, for the first time, that strength training can increase tendon stiffness in children.

Tendon stiffness is believed to have a substantial effect on movement performance (35), therefore an increase in tendon stiffness in pediatric populations may be considered beneficial for several reasons. First, the time taken to stretch a stiff tendon to a point at which forces are transmitted to the bone is shorter than that for a more compliant tendon, and should translate into faster force development and movement performance (see below). Second, tendon stiffness influences the proportion of a muscle-tendon unit’s overall length change completed by the tendon and muscle individually (51), which may have implications for movement efficiency by influencing both muscle fascicle shortening velocity and the tendon’s elastic energy storage capacity (5, 35). Finally, tension-sensitive mechanoreceptors located in the muscle (e.g., golgi tendon organs and muscle spindles), which provide important proprioceptive feedback (21), would be influenced by the tendon’s stiffness and the subsequent length change of the muscle fibers during contraction. This feedback may be greater when tendons are stiffer, which could influence spatial awareness of the limbs (51) and affect the regulation of balance and motor control. Further research is required to determine whether the magnitude of change in tendon stiffness elicited by strength training in children is sufficient to affect these aspects of movement performance and efficiency.

Importantly, no change in tendon CSA was detected after RT, leading to the conclusion that tendon stiffness increased as a result of a significant (~25%) increase in the Young’s modulus ($E$). Increases in the Young’s modulus, which are indicative of changes in the tendon’s underlying microstructure, may include changes within the tendon’s extracellular matrix (29) such as an increase in collagen content (34) or mean fibril diameter (39), changes in intermolecular cross-linking type (23) and density (9), and closer alignment of collagen fibers with the direction of force transmission (15) to facilitate an increase in fibril density. The lack of change in tendon CSA is consistent with previous studies in adults after a similar duration of RT (33, 53) and suggests that a higher loading intensity or a greater duration of training may be required to elicit significant tendon hypertrophy (11, 55). Together, these results suggest that the developing tendon may be more susceptible to qualitative adaptations than quantitative changes, which is likely to have implications for other tendon

**Table 3. Pretraining and posttraining tendon mechanical properties (mean ± SD)**

<table>
<thead>
<tr>
<th>Tendon Characteristic</th>
<th>Group</th>
<th>Pretraining</th>
<th>Posttraining, MVC$_{post}$</th>
<th>%Δ</th>
<th>Posttraining, MVC$_{pre}$</th>
<th>%Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tendon CSA, mm$^2$</td>
<td>Con</td>
<td>40.7 ± 7.2</td>
<td>41.8 ± 7.9</td>
<td>4.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Exp</td>
<td>35.8 ± 6.3</td>
<td>36.7 ± 5.9</td>
<td>2.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tendon length, mm</td>
<td>Con</td>
<td>151.6 ± 32.9</td>
<td>153.8 ± 29.4</td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Exp</td>
<td>160.3 ± 21.3</td>
<td>164.5 ± 24.3</td>
<td>2.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Force, N</td>
<td>Con</td>
<td>1872.4 ± 478.3</td>
<td>2104.5 ± 430.5</td>
<td>12.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Exp</td>
<td>1595.3 ± 300.0</td>
<td>1925.2 ± 370.8</td>
<td>20.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak elongation, mm</td>
<td>Con</td>
<td>11.5 ± 4.0</td>
<td>12.6 ± 2.5</td>
<td>7.4</td>
<td>11.5 ± 2.1</td>
<td>-2.0</td>
</tr>
<tr>
<td></td>
<td>Exp</td>
<td>11.6 ± 2.7</td>
<td>10.3 ± 2.4</td>
<td>-10.5</td>
<td>9.2 ± 2.1</td>
<td>-20.3</td>
</tr>
<tr>
<td>Peak strain, %</td>
<td>Con</td>
<td>8.1 ± 3.2</td>
<td>8.5 ± 2.8</td>
<td>5.4</td>
<td>7.8 ± 2.9</td>
<td>-3.2</td>
</tr>
<tr>
<td></td>
<td>Exp</td>
<td>7.3 ± 1.6</td>
<td>6.2 ± 1.4</td>
<td>-13.7</td>
<td>5.5 ± 0.7</td>
<td>-23.9</td>
</tr>
<tr>
<td>Peak stress, N/mm</td>
<td>Con</td>
<td>47.1 ± 14.1</td>
<td>51.7 ± 14.9</td>
<td>9.5</td>
<td>46.9 ± 12.4</td>
<td>-0.6</td>
</tr>
<tr>
<td></td>
<td>Exp</td>
<td>44.6 ± 9.1</td>
<td>50.8 ± 7.0</td>
<td>13.3</td>
<td>44.4 ± 8.4</td>
<td>-0.6</td>
</tr>
<tr>
<td>Stiffness, N/mm</td>
<td>Con</td>
<td>162.5 ± 41.8</td>
<td>167.4 ± 36.0</td>
<td>2.9</td>
<td>164.8 ± 31.4</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td>Exp</td>
<td>138.4 ± 36.7</td>
<td>177.8 ± 31.9*</td>
<td>28.5</td>
<td>170.9 ± 32.1*</td>
<td>23.5</td>
</tr>
<tr>
<td>Young’s modulus, MPa</td>
<td>Con</td>
<td>629.4 ± 235.3</td>
<td>663.1 ± 261.1</td>
<td>5.4</td>
<td>639.0 ± 249.4</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>Exp</td>
<td>642.0 ± 171.2</td>
<td>799.1 ± 149.8*</td>
<td>24.5</td>
<td>798.2 ± 173.3*</td>
<td>24.4</td>
</tr>
</tbody>
</table>

Con, control group; Exp, experimental group. MVC$_{pre}$ and MVC$_{post}$ variables were normalized to pretraining or posttraining MVC force, respectively. *Significant change relative to control, $P < 0.05$.
properties such as tensile strength and tendon extension under an applied load.

Significant increases in isometric MVC (and thus peak tendon force) were found in the experimental group (+21%), although this increase was not statistically different from the control group (+12%) and was less than the change in dynamic strength (mean load lifted per set increased from 30 to 62 kg over the training period). Because changes in absolute strength affect the calculation of the tendon’s mechanical properties, tendon elongation, strain, stress, stiffness, and the Young’s modulus were also calculated using MVC

\[\text{pre}\] data. Independently of how they were calculated, the mean decreases in peak tendon elongation (−11% and −20% for MVC

\[\text{post}\] and MVC

\[\text{pre}\], respectively) and strain (−14% and −24%, respectively) in the experimental group were not statistically significant. Although this result may in part reflect the relatively low number of study participants, we also observed that responses were variable between subjects, with some subjects showing marked changes in elongation (see Fig. 2). In individuals in whom these decreases were quite apparent, the risk of tendon strain injury following RT might be decreased, which is important because it has been previously suggested that children’s load-bearing tendons may operate with little safety margin with respect to

![Fig. 2](image-url)

**Fig. 2.** Mean (black) and individual (gray) changes in tendon mechanical and material properties [calculated using maximum voluntary contraction after resistance training (RT) (MVC

\[\text{post}\]) for control (CON; \[n=9\]) and experimental (EXP; \[n=9\]) participants before (PRE) and after (POST) the training intervention. \[*P < 0.05\].

![Fig. 3](image-url)

**Fig. 3.** A: representative trace of dynamometer moment and gastrocnemius muscle (GM) electromyographic (EMG) signal used to obtain electromechanical delay (EMD) measurement. B: relationship between changes in tendon stiffness and changes in EMD. Filled circles and open diamonds represent the experimental and control groups, respectively. Trend lines for the experimental (solid line) and control groups (dashed line) are shown. The relationship between the changes in tendon stiffness and EMD for the experimental group, examined using a regression analysis, was nonsignificant \([P = 0.077]\).
tendon injury (47) due to having a lower ultimate tensile strength (15). Reductions in elongation may also have negative consequences for movement efficiency by reducing the tendon’s capacity for energy storage (35). For individuals in whom changes in elongation and strain were less apparent, muscle force production and efficiency may have been maintained by allowing fibers within the muscle group to continue to work within their normal operating range after the training (26, 64). Predictably, peak stress remained unchanged after RT when calculated using either force level due to a combination of lack of tendon hypertrophy and a nonsignificant change in peak tendon force. Further studies are needed to determine whether greater changes in tendon properties might meaningfully affect tendon mechanics, injury risk, and elastic energy reutilization after more extensive or intensive training periods, although the present data suggest there may be a minimal, albeit variable, influence.

The second aim of the present study was to examine the effects of plantar flexor RT on EMD and RFD for the purpose of determining whether any changes were related to changes in tendon stiffness, REI (i.e., rate of muscle activation), or both. The decrease in EMD found in the experimental group was not statistically different from that of the control group (−13% and 0% for the experimental and control groups, respectively; \( P = 0.096 \)), although the lack of statistical significance may have resulted from the inclusion of an experimental participant whose EMD increased appreciably, but was not found to be an outlier. This result, however, was indicative of a heterogeneity in the change in EMD (see Fig. 3) where responders and nonresponders were evident (i.e., +12% to −38% change). Also, the mean decrease in EMD of −10 ms, which was found post-RT in the experimental group, may be considered sufficient to provide a meaningful improvement in the magnitude of muscular force attainable within a short time frame, and thus be of functional importance (41, 50). Given these findings, and according to wave propagation theory (\( v = \sqrt{\frac{k}{\mu}} \), where \( v \) is the wave velocity, \( \mu \) is the mass per unit length of the material, \( x \) is the elongation of the material from its resting length, and \( k \) is the material’s stiffness), which states that the velocity of force transmission through a viscoelastic material is partly governed by its stiffness, the relationship between the change in EMD and change in tendon stiffness was explored and a moderate correlation was found (\( r = -0.59 \)).

This result is consistent with that of Kubo et al. (33) who found a significant decrease in EMD in conjunction with a significant increase in tendon stiffness after 12 wk of isometric training in adults, and with that of Grosset et al. (22) who found paired changes in EMD and musculo-tendinous stiffness after 10 wk of plyometric training in adults. Because we have recently shown that tendon stiffness is moderately correlated with EMD in children \( [r = -0.66; (63)] \) and that tendon stiffness was targeted by the RT intervention, this new result might indicate a causal relationship between stiffness and EMD in this population. However, it is likely that the decrease in EMD found currently at least partly results from other mechanisms, including changes in muscle contractile properties or intramuscular and intermuscular force transmission, given that 65% of the variability in the change in EMD was not explained by the change in tendon stiffness. Further research is therefore required to fully determine the causal link between tendon stiffness and EMD and the importance of other factors.

The effects of changes in tendon stiffness on contractile RFD were also examined as part of the second aim of the present study. On the basis of the positive relationship found between RFD and REI in children (18, 63) and simultaneous increases in these variables after a period of RT in adults (1, 25, 61), which suggest parallelism between these variables (30, 44), it was theorized that both RFD and REI could increase

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**Fig. 4.** Rate of EMG increase (REI) measured before (open circles) and after (filled circles) training, calculated as % peak amplitude EMG per second. A: REI measured to 25, 50, 75, and 100 ms after EMG onset. B: time (ms) to reach relative (to peak) EMG levels.

**Fig. 5.** Rate of force development (RFD) measured before (open circles) and after (filled circles) training, calculated as %MVC per second. A: RFD measured to 50, 100, 200, and 400 ms after EMG onset. B: RFD measured to each 10% interval of peak MVC up to 90% MVC.
concurrently in children as a result of RT. However, in accordance with performing the plantar flexion exercise at a slow velocity, neither REI nor RFD was found to change significantly as a result of RT in the present study; previous studies have also found a lack of neural change after RT performed with a slow movement velocity (6, 43). This lack of change in REI may represent a lack of improvement in the ability to recruit large motor units for force production. It should be recognized that motor unit recruitment has been shown to remain unchanged after short-term RT in children (52), although other neurological adaptations have been demonstrated (48). Children also do not have the same capacity for motor unit recruitment as adults (4). Together with this evidence, our findings suggest that short-term RT using slow movement velocities or RFDs does not significantly improve the rate of activation in children and thus may reduce the likelihood of improvement in the rate of muscular force development.

In the context of the present study, the lack of change in the rate of muscle activation allowed for a clearer examination of the effect of changes in tendon stiffness on RFD, which has been observed to change in parallel with aging to adulthood (63). Nonetheless, RFD also remained unchanged, indicating that changes in tendon stiffness of the magnitude elicited by the current training (~29%) were not sufficient to affect RFD, which left little rationale for further exploring the relationship between changes in RFD and changes in tendon stiffness. The fact that RFD remained unchanged also negated the need to adjust tendon stiffness estimates for changes in force application, which may have increased with loading rate due to the tendon’s rate-dependent properties (49, 59). It should be noted, however, that the relationship reported previously (63) was demonstrated over a broad range of tendon stiffness values (~500% difference in tendon stiffness between the least and most stiff children). In fact, the regression equations given in that study can be used to demonstrate that RFD calculated to 50, 200, and 400 ms would be predicted to increase by only 118, 386, and 512 N/s for an increase in tendon stiffness of 29%. These values align well with the results obtained in the present study (mean increases of 146, 472, and 684 N/s for 50, 200, and 400 ms, respectively; Fig. 5), suggesting that statistically significant changes would not have been detected with the magnitude and variation of tendon stiffness change elicited by the training intervention. Potentially, longer or more intensive training periods might yield more favorable results with respect to RFD adaptations resulting directly from increases in tendon stiffness if greater increases in tendon stiffness could be obtained. Regardless, considerable changes in tendon stiffness (i.e., greater than that found presently) appear to be necessary to meaningfully affect RFD. The present findings add to the literature by indicating that increases in tendon stiffness of the magnitude found currently have a limited influence on the rate of muscular force production in children (30).

The present results demonstrate for the first time that tendons adapt to heavy RT in prepubertal children. The increase in tendon stiffness was accompanied by an increase in the Young’s modulus rather than changes in tendon CSA, indicating that microstructural changes in the tendon most likely underpinned the increase in tendon stiffness. Changes to the mechanical properties of the Achilles tendon in the present study included nonsignificant but variable decreases in both peak tendon strain and elongation at a given force level (i.e., MVCpre), which may have implications for injury minimization and the operating length range of the muscle fibers during contraction in those children who showed notable responses. Regarding the changes in rapid force development, the changes in EMD were correlated with the changes in tendon stiffness (r = 0.59), although the overall mean change in EMD was not statistically different from the control group. This is consistent with the hypothesis that a stiffer tendon will transfer force faster to the skeleton and suggests that the changes in tendon stiffness might also be associated with certain aspects of movement performance; such a relationship should be explicitly examined in future research. However, no change in RFD was found after the training, indicating that even greater changes in tendon stiffness would have been necessary to have a meaningful effect on contractile RFD and that training that elicits an increase in the rate of muscle activation might more readily induce an increase in RFD in children. Given the potential implications of these findings, further research is warranted to gain a better understanding of the effects of RT in children with respect to both neural and mechanical adaptations.

REFERENCES


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