Residual force enhancement after lengthening is present during submaximal plantar flexion and dorsiflexion actions in humans

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Although the characteristics of RFE have been well documented, particularly in isolated animal muscle, the mechanisms underlying the sustained force enhancement after a stretch remain unresolved. It is well established that RFE is insensitive to stretch velocity (3, 7, 20, 35) but that it increases with stretch amplitude (3, 8, 13). Although it is widely accepted that the magnitude of RFE is greater when a stretch of given amplitude is applied at longer initial muscle lengths, RFE has also been observed at shorter muscle lengths on the ascending limb and plateau of the force-length relationship (10, 13, 15, 24, 31, 38).

One of the prevailing theories to account for RFE is the “sarcomere popping” hypothesis (22), which relies on the development of nonuniform sarcomere lengthening during stretch of an active muscle (1, 16, 22). This theory is based on the inherent instability that has been observed in skeletal muscle when stretched to long lengths (16), where further lengthening results in a decrease in overlap between thick and thin filaments and an increase in the strain of passive structural elements. This theory can account for the presence of RFE when stretches are applied at long muscle lengths (i.e., on the descending limb of the force-length curve). However, the same instabilities are not evident at shorter muscle lengths, and the sarcomere popping theory cannot explain the RFE that has been observed on the ascending limb and plateau of the force-length curve. Lee and Herzog (20) have provided evidence of a passive force enhancement (PFE) in the human adductor pollicis that was seen as a sustained increase in force once the stimulation has ceased. This led Herzog et al. (12), in a recent review of the topic, to propose that RFE is composed of both and active component, related to cross-bridge kinetics, and a passive component. It is conceivable, therefore, that the contribution of cross-bridge kinetics to RFE would decrease over time until it is completely abolished, with the remaining force enhancement attributed solely to the passive component.

Most of the experiments from which characteristics of RFE have been identified have been made on isolated muscle preparations in which it is possible to accurately measure and manipulate variables such as sarcomere length and stretch amplitude. However, it is also crucial to determine whether RFE is a characteristic of human skeletal muscle when it is working in situ as part of a functional musculotendinous complex. Previous studies investigating stretch-induced force enhancement in humans have been conducted on the forearm...
flexor muscles (11), the first dorsal interosseus (3), and the adductor pollicis (5, 20). These studies have shown that RFE can be observed after the stretch of small human muscles of the hand and forearm during maximal voluntary contractions (11, 20) and/or electrical stimulation (3, 5, 20). However, there have been no studies to date that have investigated the presence of RFE in larger human muscle under functionally relevant conditions. Therefore, the purpose of this study was to determine whether RFE is present following the stretch of large postural muscles from the anterior and posterior tibial compartments under submaximal voluntary activation. The low level of voluntary activation used in the present study enabled the assessment of RFE for a considerable time after the stretch (10 s) to account for the contribution of cross-bridge kinetics while minimizing the influence of fatigue. We hypothesize that RFE will decrease over time to reach a steady level indicative of a non-cross-bridge tension.

METHODS

Subjects. Six healthy male subjects with no history of ankle injury or neurological disorder participated in the study. Three subjects performed the experimental protocol on both left and right legs, giving a total number of nine experiments. The mean (±SE) age, height, and mass of the subjects were 35.4 ± 3.1 yr, 1.81 ± 0.21 m, and 86.4 ± 1.1 kg, respectively. Subjects provided informed consent before participating in the experiments, and the study was approved by the Human Ethics Committee of the local research institute.

Experimental set-up. The experimental set-up has been presented in detail previously (27). Briefly, submaximal constant-velocity plantar flexion and dorsiflexion efforts were performed using a custom-built ankle torque motor that accurately controlled ankle displacement, velocity, and acceleration. The ankle joint was aligned with the axis of the torque motor and securely attached with nonelastic strapping. To minimize the contribution of the gastrocnemius muscle to the resultant joint torque during plantar flexion efforts, the subjects assumed a kneeling position on a rubber surface with the knee flexed ~120° from full extension (60° internal knee angle). In this position, the length of the biarticular gastrocnemius muscle is reduced, resulting in a 60% decrease in its torque-producing capability (4, 27).

Joint torques about the ankle were measured using a torque transducer (Maywood Instruments, Hampshire, UK) located in the axle of the torque motor. The signal was amplified (Thermo Nobel, Stockholm, Sweden) and 50-Hz low-pass filtered (Neurolog, NL 125, Digitimer, Welwyn Garden City, Hertfordshire, UK) before undergoing analog-to-digital conversion at a sample rate of 250 Hz using a CED 1401plus and Spike2 data collection system (Cambridge Electronic Design, Cambridge, UK). Angular position and angular velocity signals were 50-Hz low-pass filtered and underwent analog-to-digital conversion in the same manner as the torque signal. Surface EMG recordings were made from soleus (Sol), medial gastrocnemius (MG), and tibialis anterior (TA) muscles using bipolar Ag-AgCl electrodes with a recording diameter of 2.5 mm and interelectrode distance of 15 mm (Sensor Medics, Yorba Linda, CA). Before electrode placement, the skin was shaved, lightly abraded with sandpaper, and cleansed with a 96% alcohol solution.

Experimental protocol. Subjects initially performed two maximal voluntary isometric plantar flexor efforts at a neutral ankle angle of 0° (defined as the sole of the foot perpendicular to the long axis of the leg). The mean joint torque was calculated over a 1-s period once the torque had plateaued, and the greater of the two trials was defined as the maximal voluntary isometric plantar flexion torque [maximal voluntary contraction (MVC)]. Submaximal plantar flexor efforts were then performed at ~25% MVC and the mean Sol EMG root mean square (RMS) was calculated for a 1-s period of constant activation using an analog RMS integrator (NL 705, Neurolog) with a time constant of 500 ms. The mean EMG RMS of these two trials was calculated over a 1-s period and set as the level of Sol activation to be maintained during each submaximal voluntary plantar flexion trial (PFv). This protocol was then repeated for maximal and submaximal voluntary dorsiflexion efforts, and the mean TA EMG RMS was calculated for the target level for submaximal voluntary dorsiflexion trials (DFv).

Lengthening PFv and DFv trials were performed through a range of motion of 0° to ~15° and 0° to ~15°, respectively, at an angular velocity of 15°/s while maintaining constant submaximal activation of Sol and TA, respectively (negative angles indicate dorsiflexion). The required level of Sol or TA RMS activation (25% of maximal effort) was visually displayed to the subject as a horizontal beam on a monitor, and the subject was instructed to match their EMG RMS, displayed as a second beam on the monitor, to the preset level. Once the required level of activation had been attained and was considered stable (~1 s), the ankle torque motor was set in motion on a verbal “go” command. During the movement, subjects were required to modulate their level of effort to keep the two horizontal beams matched, that is, maintain a constant submaximal level of Sol or TA EMG RMS. This constant level of activation was maintained throughout the duration of the movement and for at least 10 s after the end of the movement. Before and after each PFv and DFv trial, a passive recording was made in which the subject’s relaxed ankle (determined by visual inspection of the EMG records) was moved through the same 15° range of motion. After the subject’s ankle was passively moved to the final ankle angle, a submaximal isometric contraction was performed to the same required level of activation to enable the calculation of RFE.

To minimize torque variations due to fluctuations in the EMG level, trials with a deviation of more than ±5% of the required EMG RMS were excluded from further analysis. Each subject performed between 5 and 14 trials for PFv and DFv, from which the mean number of successful trials were 4.3 ± 0.6 for plantar flexion (range from 3 to 8) and 7.0 ± 0.7 (range from 4 to 9) in dorsiflexion. Despite this stringent criteria, it cannot by guaranteed that maintaining a constant level of EMG RMS reflects a constant level of muscle activation due to the potential for the movement of muscle fibers in relation to the muscle surface electrodes. However, in a previous study using this protocol (26), our laboratory has shown comparable results from both surface and intramuscular electrodes, which suggests that the EMG records from surface electrodes accurately reflects the constant level of muscle activation.

Electrical stimulation trials. The presence of RFE was investigated under conditions of electrical stimulation of the triceps surae (PFv) in four of the original subjects (total of 6 limbs tested). The same experimental set-up was maintained as indicated previously; however, because of the difficulty in obtaining suitable peripheral nerve stimulation in the flexed knee position, the subjects lay prone on the experimental bench with their knee fully extended (180°). Tibial nerve stimulation was applied via a Grass electrical stimulator (model S8800) and constant-current unit (model CCU1 A, Grass Instrument, Quincy, MA) using 1-ms, 50-Hz square-wave pulses. The cathode (Neuroline surface electrode, Medicotest, Oslo, Norway) was placed in the popliteal fossa, and the anode (100-mm × 50-mm rubber electrode) was placed proximal to the patella. The current level was gradually increased in consecutive trials, each lasting ~1 s, until the resultant isometric plantar flexor torque corresponded to ~15% of maximal voluntary isometric torque in the straight-knee position. During the dynamic trials, electrical stimulation was applied before the start of the movement. Once the torque level had stabilized (~1 s), the ankle torque motor was set in motion, and the electrical current was maintained throughout the duration of the movement. Five PFv trials were performed during lengthening conditions at the same velocity and over the same ankle displacement as indicated previously. Isometric trials were performed at an ankle angle of 0° and at

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the final ankle angle of \(-15^\circ\) dorsiflexion before and after each dynamic trial to enable the calculation of RFE.

**Data analysis.** A typical torque recording during a PF trial is shown in Fig. 1 along with the EMG RMS from Sol, MG, and TA and the angular displacement record. The plantar flexion torque increased above the isometric level during the stretch and reached a peak at the end of the stretch. The torque then decayed quickly after the stretch, reaching a steady value. The plantar flexion torque, as well as the EMG RMS from Sol, MG and TA were averaged over a 1-s window at the following periods (see Fig. 1): immediately before the stretch, 4–5 s after the stretch, 9–10 s after the stretch, and 4–5 s after the muscle was relaxed. During the stretch the EMG RMS was averaged over 1 s and the peak torque was recorded.

**Joint torque-angle profile.** In isolated muscle preparations, the magnitude of RFE is greater when stretches are applied at longer initial muscle lengths particularly on the descending limb of the force-length curve (8). To obtain an indication of the muscle lengths at which stretches were applied in the present study, the torque-angle profile of the triceps surae was determined from the torque response elicited by percutaneous stimulation of the tibial nerve with supramaximal doublets (interpulse interval = 10 ms) delivered from a constant-current stimulator (model D57A, Digitimer) as described earlier. Initial recordings were made at 0° ankle angle. The subject’s ankle was then passively moved to +15° plantar flexion, and the torque response to supramaximal doublet stimulation was recorded at this ankle angle and then at 5° increments between +15° plantar flexion and \(-15^\circ\) dorsiflexion (total range of motion was 30°). A small voluntary plantar flexion was performed before the stimulation at each incremental ankle angle to remove any thixotropic effects that may have developed as a result of the passive movement (28). The resting tension was noted at each increment and plotted against ankle angle to obtain the passive force-length profile.

The mean joint torque-angle profile is presented in Fig. 2. The passive joint torque was subtracted from the total joint torque to obtain an indication of the force-length profile of the contractile elements within the muscle (active torque). Data were normalized to the total joint torque at 0° ankle angle and presented as means ± SE for all subjects.

**Statistics.** The group means ± SE were calculated for all dependent variables. The normality of the data was tested using the Shapiro-Wilk’s W test, and subsequently the nonparametric Friedman test was used to examine the EMG RMS data at the different time points throughout the trial. The Wilcoxon matched-pairs test was used to identify significant differences between conditions. Statistical analysis was performed using SPSS for Windows (SPSS, Chicago, IL). Statistical significance was accepted at \(P \leq 0.05\), and, for multiple nonparametric comparisons, critical alpha levels were adjusted using a Bonferroni correction.

**RESULTS**

**Isometric plantar flexion and dorsiflexion joint torque.** The means (±SE) isometric joint torques from maximal and submaximal plantar flexion and dorsiflexion actions are presented in Table 1. Despite strict criteria for maintaining the required level of muscle activation, the average dorsiflexion torque (28.5% MVC) was slightly greater than the desired level (25% MVC). Also shown in Table 1 is the mean joint torque from isometric contractions at the final joint angle (Isom Long) and the passive torque recorded after moving the relaxed ankle to the final joint angle. Isom Long was significantly greater than the isometric torque produced at the initial joint angle for each activation condition. The increase in isometric torque at the longer muscle lengths can be attributed to the increase in passive torque arising from the rotation of the relaxed ankle to the final joint angle. As shown in Fig. 2, the passive joint torque is subtracted from the total torque to provide an indication of the joint torque produced by the contractile elements of the muscle (active joint torque). This torque-angle profile indicates that, when the joint total torque is corrected for the contribution of passive torque, the active joint torque continues to increase at each dorsiflexion increment (i.e., on the ascending limb of the force-length relationship).

It must be acknowledged that the joint torque-joint angle relationship is not determined by the muscle force alone, but it
is dependent on the moment arm of the muscle force, which varies with joint angle. According to Maganaris et al. (21), the estimated moment arm for the Achilles tendon is 5.6 cm when the ankle is at 30° plantar flexion and decreases to 4.3 cm at 15° dorsiflexion. If a similar change in moment arm occurred in the present study, the “active torque” shown in Fig. 2 would in fact underestimate the muscle force produced by the triceps surae muscle group, resulting in a leftward shift in the force-length relationship (i.e., to shorter muscle lengths). The length dependence of force development is also influenced by the level of muscle activation, with the force-length curve shifted to the right for twitch contractions and for submaximal tetanic contractions (32). Therefore, although the joint torque-joint angle relationship in this study may not precisely match the force-length relationship of a maximally active isolated muscle, it is more than likely that the stretches in this study were applied on the ascending limb of the force-length relationship.

Submaximal voluntary plantar flexion and dorsiflexion actions. Figure 3 illustrates the general features of the torque response during a DFv trial. On application of a stretch during an isometric contraction at the initial joint angle, the joint torque increased, reached a peak at the end of the stretch, and decayed afterward to a steady level at the final joint angle. The time course of the torque response to the imposed muscle lengthening was similar for all conditions and resembles the lengthening-induced force enhancement that has been observed in isolated mammalian muscle fibers (25). Also shown in Fig. 3 is the joint torque recorded during an isometric contraction at the final joint angle (Isom Long). It can be seen that the steady torque after the stretch remained higher than the isometric torque at the longer muscle length. As an index of this force enhancement the isometric torque at the longer muscle length was subtracted from the steady joint torque after the stretch (RFE).

RFE after stretch. Although the torque decayed after the stretch, eventually reaching a steady-state level, the torque record during voluntary activation was not sufficiently smooth to enable quantification of the tension decay by exponential curve fitting as has been commonly performed in isolated muscle preparations (2, 25). Therefore, to obtain an indication of the decay in joint torque after stretch, the mean RFE was measured over a 1-s period between 4 and 5 s after the stretch and again at 9–10 s after the stretch. The torque measured between 4 and 5 s after the stretch was significantly greater than that measured between 9 and 10 s poststretch in each of the three experimental conditions (see Table 2). In several trials in which activation was maintained for longer than 10 s after the end of the stretch, the isometric torque showed no evidence of further decay indicating, that a steady state had been reached.

Table 1. Joint torque measures for isometric plantar flexor and dorsiflexor efforts

<table>
<thead>
<tr>
<th></th>
<th>MVC, N·m</th>
<th>Submaximal, N·m</th>
<th>Submaximal %MVC</th>
<th>Isom Long, N·m</th>
<th>Pass Long, N·m</th>
</tr>
</thead>
<tbody>
<tr>
<td>PFv</td>
<td>100.81±5.38</td>
<td>24.72±4.65</td>
<td>24.35±4.87</td>
<td>34.43±3.20*</td>
<td>10.75±0.80</td>
</tr>
<tr>
<td>DFv</td>
<td>34.61±1.87</td>
<td>9.47±0.28</td>
<td>28.52±1.58</td>
<td>17.67±0.98*</td>
<td>4.83±0.43</td>
</tr>
<tr>
<td>PFs</td>
<td>103.76±5.10</td>
<td>15.24±1.92</td>
<td>14.44±3.29</td>
<td>27.64±1.76*</td>
<td>9.14±0.17</td>
</tr>
</tbody>
</table>

Values are means ± SE for maximal voluntary contractions (MVC) and submaximal voluntary contractions (Submaximal) for plantar flexion and dorsiflexion trials (PFv, and DFv, respectively; n = 9) and electrically stimulated plantar flexion trials (PFs; n = 6). Isom Long, mean joint torque from Isometric contractions at the final joint angle; Pass Long, passive torque recorded after moving the relaxed ankle to the final joint angle. *Significantly different from Submaximal, P < 0.05.

RFE values from the voluntary activation conditions are presented in Table 2. When expressed as a percentage of the joint torque recorded from the isometric contraction at the final joint angle (Isom Long), the RFE values measured 9–10 s after the stretch were 7 and 12% for PFv and DFv, respectively.

EMG. Figure 4 displays the mean (±SE) EMG RMS for Sol, MG, and TA for the four measurement periods of each trial. Data are normalized to the EMG recorded during an MVC when that muscle was acting as an agonist (i.e., Sol EMG normalized to Sol activation during an MVC plantar flexion). As expected, there was no significant difference in Sol RMS or TA RMS throughout the lengthening PFv and DFv trials, respectively. Furthermore, there was no significant difference in MG and TA RMS throughout PFv trials. During DFv, however, there were significant differences in both Sol and MG activation. Subsequent analysis revealed that Sol RMS during the stretch was significantly greater than that immediately before the stretch, whereas the mean MG RMS for 9–10 s after the stretch was significantly greater than that immediately before the stretch. These findings indicate a significant increase in antagonist activation during and after the dorsiflexion stretch, which may contribute to a decrease in the resultant dorsiflexion joint torque and, subsequently, an underestimate of RFE in this condition.
significant for DFv (Table 2). In this case, PFE accounted for a small PFE in each of the three conditions, this was only estimated by visual inspection of the raw EMG records. Although there was little change in the ankle muscles during the stretch recorded at the end of the stretch, the peak torque data were normalized to the isometric torque recorded at the long muscle length to obtain a measure of the force enhancement during the stretch. When expressed as a percentage of Isom Long, the peak torque reached during the stretch was 134\% for PFv and 135\% for DFv.

**Force enhancement during stretch.** Experiments on isolated animal muscle suggest that RFE is the remnant of a noncontractile force that makes a sizeable contribution to the peak force enhancement during the stretch (9, 25). Because the peak incremental force occurred at the end of the stretch, the peak torque data were normalized to the isometric torque recorded at the long muscle length to obtain a measure of the force enhancement during the stretch. When expressed as a percentage of Isom Long, the peak torque reached during the stretch was 130\% ± 3\% for PFv, and 135\% ± 4\% for DFv.

**Electrical stimulation (PFv).** To enable further comparisons between experiments on animal and human muscles, RFE was also investigated under electrical stimulation of the plantar flexors (Fig. 5). The results from PFv trials were similar to those for PFv trials; RFE values were 17 and 13\% of Isom Long when measured 4–5 and 9–10 s after the end of the stretch, respectively (Table 2), whereas the force enhancement during the stretch was 134\% ± 4\%.

PFE. PFE after the stretch was measured as the difference between the resting torque after the stretch and the resting torque recorded after an isometric contraction at the final joint angle. Complete relaxation of the ankle muscles was confirmed by visual inspection of the raw EMG records. Although there was a small PFE in each of the three conditions, this was only significant for DFv (Table 2). In this case, PFE accounted for ~25\% of the total force enhancement (RFE).

**DISCUSSION**

This is the first study to show that RFE is present during submaximal voluntary activation of human calf (triceps surae and TA) muscles. Furthermore, these experiments were carried out within the working range of the muscle-tendon complex on the ascending limb of the force-length curve, indicating that RFE is a functionally relevant characteristic of human skeletal muscle.

**Comparison with previous human muscle studies.** Stretch-induced force enhancement has been examined previously in human experiments on muscles of the forearm (11) and the hand (3, 5, 11, 20). The level of RFE in the present study (7–13\%) is comparable with that reported for these previous studies (10\%, Ref. 5; and 12–17\%, Ref. 20). De Ruiter et al. (5) reported a steady-state force enhancement of 6 N (~10\% of isometric force) after stretch of the electrically stimulated adductor pollicis muscle. Interestingly, the amount of force enhancement was largely independent of the level of activation, staying relatively constant across initial isometric force levels ranging from 30 to 55 N (5). Gülch et al. (11) have also reported a small level of force enhancement following stretch of the voluntarily activated forearm flexor muscles that increased following a training regime that included an eccentric component. However, the authors also noted that the isometric force recorded immediately before the stretch, and the level of integrated EMG, were significantly lower than that for a pure isometric contraction at the same muscle length. Similar observations were made by Lee and Herzog (20) in experiments investigating RFE during maximal voluntary activation of the adductor pollicis. These findings raise the possibility that the level of RFE determined by comparing the steady-state force after a stretch with that of a purely isometric contraction at the corresponding muscle length may be influenced by variations in the level of muscle activation. In an attempt to avoid the confounding influence of variations in muscle activation, we provided direct visual feedback of the RMS EMG signal and instructed the subjects to maintain a constant level of muscle activation throughout each trial. Although this protocol resulted in very stringent control of the activation in the agonist muscle, no feedback was provided to the subject concerning the level of synergist or antagonist muscle activation. This lack of feedback may have contributed to the slight increase in activation of the antagonist muscles (Sol and MG) during DFv trials. The slight increase in antagonist activation may have led to an underestimate of the RFE in this condition, but it would not alter the main findings of this study. It is unclear why the level of antagonist activation increased for stretches applied to

**Table 2. RFE and PFE measures for plantar flexor and dorsiflexor efforts**

<table>
<thead>
<tr>
<th></th>
<th>RFE 4–5 s, N·m</th>
<th>RFE 9–10 s, N·m</th>
<th>RFE 4–5 s, %</th>
<th>RFE 9–10 s, %</th>
<th>PFE, N·m</th>
</tr>
</thead>
<tbody>
<tr>
<td>PFv</td>
<td>3.33±0.50</td>
<td>2.38±0.46*</td>
<td>9.17±1.05</td>
<td>6.52±0.98</td>
<td>0.21±0.16</td>
</tr>
<tr>
<td>DFv</td>
<td>2.76±0.31</td>
<td>2.42±0.31</td>
<td>13.56±1.37</td>
<td>11.72±1.37</td>
<td>0.63±0.14*</td>
</tr>
<tr>
<td>PFs</td>
<td>4.78±0.59</td>
<td>3.59±0.37*</td>
<td>17.34±3.82</td>
<td>13.26±4.49</td>
<td>0.25±0.17</td>
</tr>
</tbody>
</table>

Values are means ± SE joint torque for residual force enhancement (RFE) and passive force enhancement (PFE). RFE data are also presented as \% of the isometric joint torque at the final joint angle. *Significantly different from RFE 4–5 s (P < 0.05). †Significantly different from resting torque after Isom Long, P < 0.05.

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![Fig. 4. Normalized EMG root mean square. Mean (±SE) EMG root mean square values for Sol (triangles), MG (squares), and TA (circles) during submaximal lengthening plantar flexion (A) and dorsiflexion (B) trials. Pre, prestretch. **Significantly greater than Pre for Sol, P < 0.05. **Significantly greater than Pre for MG, P < 0.05.](http://jap.physiology.org/Downloadedfrom/10.1152/jappl.00283.2006)
the dorsiflexion but not plantar flexion muscles, although the coactivation of these muscle groups may have increased the stability of the ankle joint during eccentric muscle actions. An increase in joint stiffness may be more desirable when the stretches are applied to the smaller TA muscle, which is less accustomed to eccentric contractions than the larger triceps surae muscle group, which routinely experience lengthening actions during normal gait.

In addition to maintaining a constant level of muscle activation, the accurate quantification of RFE after a stretch requires that muscle activation be sustained long enough for the poststretch force to decay to a steady level. As shown in this study and in other experiments (5) on human muscle, this time period can be in the order of 10 s. Therefore, experiments that investigate RFE during maximal voluntary contractions (e.g., Refs. 11, 20) may be susceptible to the influences of fatigue. The use of submaximal voluntary activation in the present study has eliminated the possibility that the measurement of RFE during voluntary activation were influenced by muscle fatigue.

**Voluntary and electrical stimulation.** For lengthening plantar flexion actions, the RFE in trials in which the muscle was activated by electrical stimulation (PFv = 13%) was twice as large as that for voluntary activation trials (PFv = 6.5%). However, because of the limitations of the experimental design, it is not possible to directly compare the results from the two different activation conditions. To achieve constant voluntary activation of Sol in PFv trials, it was necessary to perform the lengthening trials in a flexed knee position, thereby minimizing the contribution from the gastrocnemius muscle group (see METHODS), whereas for PFv trials, a fully extended knee was required to achieve optimal stimulation of the tibial nerve. Therefore, the differences between the two conditions may arise from the method of stimulation (voluntary activation vs. electrical stimulation) or from the different contributions from gastrocnemius in the straight-knee and flexed-knee positions. It is worth noting, however, that similar differences in the amount of RFE for voluntary and stimulation conditions were reported by Lee and Herzog (Ref. 20; 12 and 17% for voluntary and stimulated trials, respectively), which may reflect a specific effect of the type of muscle activation. Furthermore, for PFv trials, the stimulation intensity was adjusted so that the level of muscle activation produced a plantar flexion torque that was ~15% of that produced during a MVC. Although the level of force generation by the muscle was submaximal, it cannot be excluded that this force was produced by the maximal activation of a small number of motor units. Therefore, it is possible that the RFE measurements were influenced by fatigue; however, this would have led to an underestimate of RFE due to the different stimulus durations in the PFv trial (~12 s) compared with the Isom Long trials (~2 s).

It is interesting to note that, in the present study, there was little difference in the peak torque during the stretch between the voluntary and stimulation trials (130% for PFv and 134% for PFv). Again, this finding is consistent with that of Lee and Herzog (20), which suggests that the differences in knee angle were not a confounding factor in the present study. These results, however, may appear to conflict with previous studies examining the angle-specific joint torque during lengthening contractions that have reported a significant increase in joint torque during electrical stimulation compared with voluntary activation (e.g., Ref. 27). The primary difference in these studies may arise from the joint angles (and hence muscle lengths) at which the torque was recorded; e.g., previous studies recorded the joint torque at a neutral ankle angle (0°) and the present study examined the lengthening torque at a longer muscle length (15° dorsiflexion), which will recruit a greater torque contribution from noncontractile elements (as shown in Fig. 2).

Although the normalized peak torque during stretch for DFv trials (135%) was similar to both PFv and PFv trials, the amount of RFE was significantly greater during DFv trials (11.7%) than for PFv trials (6.5%; P < 0.05). Unfortunately it was not possible to accurately quantify the joint angle-joint torque relationship for the dorsiflexion muscle group using either voluntary activation or electrical stimulation; the level of

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**Fig. 5.** Representative traces from a submaximal electrical simulation trial. Traces are shown for plantar flexion torque (middle traces) and joint angle (bottom) during a lengthening trial. The duration of electrical stimulation (Stim) is also shown (top). Plantar flexion torque increased during the stretch and decayed afterward to a level that was greater than the torque produced from an isometric trial at the final joint angle. After stimulation has ceased, the plantar flexion torque decreased to a level that is indistinguishable from the passive torque at the final joint angle.
 voluntary activation is known to change depending on the joint angle (4), and stimulation of the common peroneal nerve innervates not only TA but also the plantar flexion muscles peroneus longus and brevis. Therefore, it cannot be excluded that differences in RFE between dorsiflexion and plantar flexion are influenced by variations in the working lengths of the two muscle groups (i.e., TA working on the descending limb of the force-length relationship). An alternative explanation is that the amount of RFE differed according to the fiber-type populations of the two muscle groups. As discussed in the following section, we consider that the noncontractile elements of the sarcomere are primarily responsible for the RFE after stretch. It is well established that the structural proteins such as titin are different between fast and slow muscle fibers. In slow muscle fibers, the titin isoforms are larger and their stress-strain relation is shifted to longer sarcomere lengths than fast fibers (39). If, as we believe, extension of the titin filament plays an important role in the development of RFE, then slow fibers would need to be stretched to greater lengths to produce an equivalent amount of RFE to fast fibers. It is possible that fiber-type differences in the properties of structural proteins could contribute to the difference in RFE between the fast TA and slow SOL muscles.

Mechanisms underlying RFE in muscle. Despite extensive investigations of stretch-induced force enhancement, the mechanisms underlying RFE are unresolved. Evidence for the development of nonuniform sarcomere lengthening during stretch (16) led Morgan (22) to propose a specific sarcomere popping hypothesis to account for RFE. However, as discussed in a recent review by Herzog et al. (12), this hypothesis fails to account for the RFE that has been observed when stretches were applied on the ascending limb and plateau of the force-length relationship. The proposal put forward by Herzog et al. is that RFE is composed of both an active component, determined by cross-bridge kinetics, and a passive component arising from the strain of structural proteins such as titin. Evidence for cross-bridge involvement in the development of RFE is taken from experiments on single frog fibers in which the magnitude of RFE increased in the presence of the myosin inhibitor, 2,3-butanedione monoxide (BDM) (29). On the basis that BDM causes a marked slowing of cross-bridge kinetics, the authors suggested that RFE may arise from slowly detaching cross bridges. However, these data must be interpreted with caution, because RFE should be assessed when the tension has reached a steady state and decreasing the cross-bridge detachment rate with BDM may lead to the overestimation of RFE.

The tension decay after stretch, as analyzed using a biexponential function (2), is composed of a fast (100–150 s⁻¹) component and a slow (2–10 s⁻¹) component, the amplitude of the fast component being approximately twice that of the slow component (25). Both the rate and the amplitude of the fast component are significantly reduced in the presence of the specific myosin inhibitor, N-benzyl-p-toluene sulfonamide (BTS), whereas the slow component was insensitive to BTS (25). It was therefore, suggested that the fast component represents the force contribution arising from cross bridges and the slow component represents a non-cross-bridge tension that was directly related to RFE. These observations indicated that the accurate measurement of RFE can only be made after sufficient time has elapsed for the fast, cross-bridge tension to decay to a steady state.

Although the extent to which nonuniform sarcomere lengthening contributes to RFE is still being debated, there is more consensus regarding the contribution of noncontractile elements to the long-lasting force enhancement. Edman and Tsuchiya (9) suggest the recruitment of a damped elastic element in parallel with the contractile components can account for RFE. According to Lee and Herzog (20), RFE contains a passive component (PFE) that can be seen as a sustained increase in force once the stimulation has ceased. In their experiments on whole frog sartorius muscle, however, van Atteveldt and Crowe (38) found no evidence of PFE. Although some PFE was observed following dorsiflexion stretches in the present study, the maximum contribution to the total force enhancement (i.e., RFE) was only ~25%. On this basis, we consider that PFE is insufficient to fully account for the RFE observed in these experiments.

Although the identity of the noncontractile element is unresolved, the presence of RFE in single myofibrils (30) indicates that it resides within the myofibril. The most widely cited candidate is the giant titin filament. Titin molecules possess the characteristics necessary to account for the force increase during stretch of a resting muscle (18), although the force arising from the extension of titin in single-molecule experiments may seem insufficient to account for the magnitude of passive forces recorded in vivo. However, as recently discussed by Tskhovrebova et al. (37), the complex arrangement of titin molecules in situ can have considerable effects on the persistence length and bending rigidity, thus producing values for titin stiffness that may be an order of magnitude or more greater than those recorded in vitro. The presence of RFE in an active muscle may be explained by an increase in titin stiffness on activation, through calcium-dependent titin-actin interactions (17, 19). If the stiffness of titin increases in the presence of calcium, it would presumably decrease after stimulation has ceased in the absence of calcium. It is conceivable, therefore, that the PFE observed in some conditions after the muscle activation has ceased is a remnant of the RFE that develops when an active muscle is stretched and calcium is readily available.

RFE has been observed in an extensive array of muscle preparations and is now considered a fundamental property of skeletal muscle. As shown in this study, the presence of RFE in human skeletal muscle under physiologically relevant conditions (i.e., submaximal voluntary activation of large postural muscles) suggests that this characteristic may be exploited in functional tasks. Interestingly, the magnitude of this force enhancement appears to differ in various muscle groups, which may reflect the composition of structural and contractile proteins. It is clear that the underlying mechanisms of RFE remain controversial, and further investigation of these issues and the functional benefits of this characteristic of skeletal muscle is warranted.

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