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Force Per Active Area and Muscle Injury during Electrically Stimulated Contractions

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Abstract

Multiple mechanical factors have been implicated in the initiation of exercise-induced muscle injury. Although high absolute force levels are associated with greater injury, the importance of high force per active area independent of absolute force remains to be determined, especially in humans.

Purpose—This study sought to examine the role of specific force in muscle injury when peak eccentric force and total eccentric force were matched between two exercise bouts.

Methods—Ten subjects (six men, four women) performed 80 electrically stimulated (EMS) eccentric contractions of the right and left quadriceps femoris (QF) through a 90° arc at $\approx 45^\circ \cdot s^{-1}$. Specific force was manipulated by applying 25-Hz EMS to one thigh and 100-Hz EMS to the contralateral thigh, whereas force production was matched by lowering the EMS amplitude during the 100-Hz bout. T2 magnetic resonance images of the QF were collected before and 3 d after the eccentric exercise bouts. Injury was assessed via changes in isometric force and ratings of soreness in the QF over the course of 28 d after exercise and by determining changes in T2 relaxation time and muscle volume 3 d after exercise.

Results—The 100-Hz EMS induced a greater force loss ($P < 0.05$), soreness ($P < 0.05$), change in muscle volume ($P = 0.03$), and volume of muscle demonstrating an increase in T2 ($P = 0.005$) compared with 25-Hz EMS.

Conclusions—Our findings suggest that in humans, high specific force potentially plays an important role in the initiation of exercise-induced muscle injury.

Keywords

T2 MRI; SPECIFIC FORCE; SORENESS; ECCENTRIC CONTRACTIONS

Unaccustomed eccentric exercise is known to cause injury in skeletal muscle. This injury is often accompanied by a decrease in force production (21,28), delayed-onset muscle soreness (10,28), an increase in muscle volume (13,35), an increase in serum levels of creatine kinase (CK) (9,27), as well as sarcomere and ultrastructure disruption (15). Although the sequence of events that initiate contraction-induced injury remains unclear, several mechanical factors have been shown to play a role in the magnitude of the resultant injury (3,39). Initial muscle length (37), stretch of fibers beyond optimum length (strain) (6,23), peak contraction force (25,39), contraction velocity (6,39), work (20), and number of eccentric actions performed (25,37,40) have been shown to be determinants of muscle injury.

Much of the data regarding contraction-induced injury come from a variety of animal models. To date, less data exist regarding the role mechanical factors play in muscle injury in humans.

As in animal models, initial muscle length (8,31), peak force production (29), and number of eccentric actions (7) have been shown to play a role in the magnitude of injury. Force per active area of muscle (specific force or stress) is an additional factor suggested to be important in the initiation of contraction-induced muscle injury (18,25,39). Consistent with this idea is the finding that eccentric muscle actions result in greater injury than concentric and isometric actions because fewer motor units are recruited to generate a given absolute force (3).

It has been demonstrated that during electrically stimulated (EMS) contractions, stimulation frequency controls force per active area (5,16) and stimulation amplitude controls the area of muscle activated during EMS (1). In a previous investigation, we altered force per active area during two bouts of eccentric exercise by using EMS to match active muscle mass while manipulating force production by reducing stimulation frequency from 100 to 25 Hz (5). Using this protocol, high force per active area was found to result in greater muscle injury (5). Further insight into the role of specific force on muscle injury could be gained by matching peak eccentric force and total work generated between bouts of eccentric exercise imparting different specific forces. This could be accomplished by lowering EMS frequency while increasing the stimulation amplitude to activate a greater muscle mass.

The purpose of this study was to investigate the role of force per active area in contraction-induced muscle injury when peak eccentric force and total work were matched during an exercise bout. To accomplish this, two eccentric exercise protocols were used, and the magnitude and the time course of recovery from muscle injury were compared. In the first protocol, 25-Hz EMS was used to evoke a series of eccentric contractions of the quadriceps femoris (QF) muscle group. In the second protocol, 100-Hz EMS was used to evoke eccentric contractions of the contralateral QF at a higher force per active area. Peak eccentric force, work, range of motion, and contraction number were matched between the 100- and the 25-Hz protocols.

METHODS

Subjects

Ten adults (six men, four women) aged 26 ± 3 yr (mean $\pm$ SD), with a height of 173 ± 11 cm and a mass of 65 ± 12 kg volunteered to participate in this study. Subjects were screened for medical or orthopedic conditions that would preclude strenuous exercise or magnetic resonance (MR) imaging of the knee extensors. All subjects were recreationally active by self-report, but none had performed resistance training with the lower body during the previous 12 months. All experimental procedures were approved by The Institutional Review Board of The University of Georgia, and subjects provided written informed consent before participation.

General protocol

The study was conducted over the course of 6 wk. Subjects were familiarized to the isometric strength test and the electrical stimulation protocol during several sessions over the initial 2 wk. Each subject performed eccentric exercise with both their right and left QF. The 100- and the 25-Hz protocols were randomly assigned to the dominant and nondominant legs. Baseline isometric strength was determined at the end of the familiarization period. Eighty electrically evoked eccentric contractions were performed at the beginning of the third week in both the right and the left QF muscle group. Stimulation amplitude was adjusted such that force production was matched between the 25- and the 100-Hz bouts across all 80 contractions. Isometric strength and muscle soreness were assessed immediately after exercise and 1, 2, 3, 4, 7, 10, 14, 21, and 28 d after exercise. T2 MR images were collected on the third day (approximately 72 h) after exercise. Both legs were injured simultaneously because previous

research (30) as well as data from a companion study demonstrated that injury in one limb does not affect assessment of soreness or strength in the contralateral limb.

Assessment of maximal voluntary isometric force

Isometric knee extension was performed as described previously (5) on a custom-built chair with the leg secured via an inelastic strap to a rigid lever arm so that the knee was fixed at $\sim 70^\circ$ below horizontal. The moment arm was established by positioning a load cell (model 2000A; Rice Lake Weighing Systems, Rice Lake, WI) parallel to the line of pull and perpendicular to the lever arm. Torque was recorded from the load cell using a MacLab analog-to-digital converter (model ML 400; ADInstruments, Milford, MA) with a sampling rate of 100 Hz. Values were transferred to a portable computer for storage and analysis (Apple Computer, Cupertino, CA). Subjects were familiarized to the strength testing during three to five sessions over a 2-wk period before the study with baseline strength established at the end of this 2-wk period. During each testing session, subjects performed a series of maximal voluntary isometric contractions (MVIC) with 2 min of rest between trials. Subjects were given verbal encouragement to maintain their effort for 3–5 s. For each effort, torque was measured from the plateau region of the torque tracing. Once two efforts differed by less than 5%, the larger of the two efforts was considered the subjects, MVIC and recorded for further analysis. Day-to-day reliability of the assessment of MVIC in this manner has been tested over five consecutive days previously (unpublished observations). An intraclass correlation coefficient of 0.99 was found across the five testing days with a mean within-subject coefficient of variation of 3.2% (SD = 2.2) that ranged from 0.8% to 8.5% across the testing days (unpublished observations).

EMS eccentric exercise

Subjects performed unilateral, eccentric actions with the left and the right QF on a seated knee extension machine (Magnum Fitness System; Badger Fitness; Milwaukee, WI). Stimulation electrodes (6.98×10.16 cm; Uni-Patch, Wabasha, MN) were placed on the skin over the distal vastus medialis and the proximal vastus lateralis of both the right and the left thighs. A commercial stimulator (TheraTouch model 4.7; Rich-Mar Corporation, Inola, OK) was used to evoke all contractions. Each bout consisted of eight sets of 10 repetitions with 3 min of rest provided between sets. To perform each eccentric repetition, subjects were seated in the knee extension apparatus with the thigh at 90° relative to the torso. Two researchers lifted the weight to the starting position (full knee extension). EMS was then applied to smoothly lower the weight through a 90° arc (from 0° to 90° below horizontal) to a 2-s count. During each set, when the weight could no longer be lowered in a smooth, controlled manner, the weight was lowered by $\sim 5\%$, and the set continued until all 10 contractions were complete. The next set was begun using this weight. Exercise proceeded in this manner until all eight sets were complete.

EMS stimulation consisted of 450- μ s biphasic square wave pulses at a constant current. The current used in each eccentric exercise bout was determined in the following manner. EMS at 25 Hz was then applied to one QF, and the current was increased until it was sufficient to isometrically evoke 25% of MVIC. EMS was then applied at 100 Hz to the contralateral QF, and the current was increased until isometric force was similar to that evoked at 25 Hz. These currents were then recorded and used during the eccentric exercise protocols.

Assessment of muscle injury

MVIC and ratings of muscle soreness were obtained before, immediately after, and on days 1, 2, 3, 4, 7, 10, 14, 21, and 28 after eccentric exercise. MVIC was assessed as described above. Muscle soreness was assessed using a 100-mm visual analog scale. The scale was anchored with “0” being a complete lack of soreness and with “100” being the worst soreness imaginable.

Evidence suggests that the VAS scale is both valid and reliable for determining pain responses to noxious stimuli (34). Subjects rated their soreness during a dynamic contraction of the QF. Subjects lifted a light weight (10% of concentric one repetition maximum) through a 90° concentric arc (from 90° to 0° below horizontal) and a 90° eccentric arc (from 0° to 90° below horizontal) at 45°·s⁻¹. T2 MR images were collected before and on day 3 (approximately 72 h) after the exercise protocols. Imaging was performed 72 h postexercise because this time point provided a balance between allowing sufficient time for elevation of the T2 signal, which has been shown to peak 5–10 d after exercise (13,36), while still falling within the reported time course of near peak changes in other markers of muscle injury such as swelling, muscle soreness, and isometric force loss (13,35).

MRI and analysis

Subjects were positioned supine in a 3.0-T whole body imager (Signa; General Electric, Milwaukee, WI). A 20-cm-diameter extremity coil (General Electric) was centered on an indelible ink mark placed approximately 40% of the distance between the top of the patella and the greater trochanter. A series of 8 to 12 T2-weighted, spin echo, transaxial images (TR/TE = 1600/30,60) 10 mm thick and 5 mm apart were collected in a 20-cm field of view and a 256 × 256 matrix along a 20-cm region of the thigh. T2 images were calculated from the two echo times assuming a monoexponential decay (12).

MRI has been used as a noninvasive method to study muscle injury (13,33,35). A delayed increase in the signal intensity of T2 (transverse) relaxation of skeletal muscle water has been observed after eccentric exercise and has been shown to peak 2–6 d after exercise (13). This delayed increase correlates with markers of ultrastructure disruptions in muscle biopsies from both humans (32) and animals (24) and follows a similar time course to traditional markers of injury such as soreness, serum CK levels, swelling, and force loss (13,35).

Images were analyzed using WinVessel 2.01 (Meyer, Michigan State University, East Lansing, MI), essentially as described previously (5). After spatial calibration, a region of interest (ROI) was defined in each image by manually tracing the outline of the anatomical cross-section area of the QF. The mean T2 values were determined for the QF in each slice, and all slices were averaged to obtain a mean T2 of the QF muscle group over the imaged region. QF volume (cm³) in the imaged region was determined by summing the pixels count from each ROI and multiplying by the area of a single pixel (FOV/matrix; 20 cm/256 pixels = 0.078 cm² per pixel) then multiplying by slice thickness (1 cm) and spacing (0.5 cm). In this context, “volume” is used to refer to the volume of muscle that fell within the region that was imaged. This region contains a large portion of the muscle belly but did not cover the entire length of the QF muscle group. Mean T2 values and volumes were also determined for the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), and vastus intermedius (VI) in the same manner by tracing the outline of each individual muscle. Mean T2 values were determined from the preexercise (resting) images and the 3-d postexercise images. For the QF muscle group and each individual muscle, the change in T2 signal intensity ($\Delta T2$) was calculated by subtracting the pre-T2 from the post-T2. Change in muscle volume or swelling was calculated for the entire QF and each of the four individual muscles using the following equation: [(postvolume — prevolume) / prevolume] × 100 for the QF and for each individual muscle.

The volume of muscle demonstrating an increase in T2 signal intensity (VDI T2) was determined on a pixel-by-pixel basis, essentially as done previously (5,33). Pixels with a T2 between 20 and 35 ms were assumed to represent muscle in preexercise images, whereas pixels with a T2 greater than 35 ms were considered to represent noncontractile tissue such as fat and connective tissue (33). The mean and SD of the T2 values from pixels in the 20- to 35-ms range were calculated. Pixels falling within this range were summed across all images and a volume was calculated as described above. Additionally, pixels with a T2 greater than 35 ms were

summed across all images. Pixels in matching postexercise images with a T2 greater than the mean ± 1 SD of muscle from the preexercise images (determined from the pixels falling in the 20- to the 35-ms range in the preexercise images) were considered elevated. These pixels were summed across all postexercise images, and subsequently pixels from preexercise images with a T2 greater than 35 ms were subtracted. The remaining pixels were considered to represent muscle tissue that had an elevated T2 (i.e., VDI T2). This was done to correct for pixels containing noncontractile tissue such as fat, which have elevated T2 values, compared to muscle, which would be found in both sets of images. VDI T2 was calculated as described above and expressed relative to muscle volume from the preexercise images.

Statistical analysis

Results are expressed as mean $\pm$ SD. All statistical tests were performed using the Statistical Package for the Social Sciences version 14.0 (SPSS Inc, Chicago, IL). Dependent sample *t*-tests were performed to compare differences in mean T2 signal intensity of the QF muscle group, and each of the individual quad muscles preexercise and postexercise MR images in both the 100- and the 25-Hz thighs. Differences in VDI T2, change in mean T2, and change in volume (swelling) between the 100- and the 25-Hz thighs were examined using dependent *t*-tests. A two-factor stimulation frequency (100 and 25 Hz) $\times$ muscle (RF, VM, VI, and VL) within-subjects ANOVA was performed to examine differences in the change in mean T2 and change in volume (swelling) between thighs and muscles of the QF. A two-factor stimulation frequency (100 and 25 Hz) $\times$ time (days posteccentric exercise) within-subjects ANOVA was performed to examine differences in MVIC and ratings of soreness. When a significant interaction was found, a one-way repeated-measures ANOVA was performed to test for simple effects and followed, when appropriate, by dependent measures *t*-tests. Main effects were only interpreted in the absence of a significant interaction. A Bonferroni correction for multiple comparisons was used when pairwise comparisons evaluating differences in means for soreness, MVIC, and individual muscles of the QF were made. Significance was set experiment-wise *a priori* at $\alpha \leq 0.05$.

RESULTS

Eccentric exercise

Peak eccentric weight lifted 59 ± 31 kg and weight lifted across the 80 contractions (work) did not differ between the 100- and the 25-Hz exercise protocols (Fig. 1). On average, the weight lifted during each protocol declined 54% from 59 (31) to 27 (26) kg ($P < 0.001$) from the first contraction to the eightieth. Stimulation amplitude (mA) was 30% greater ($P = 0.002$) during the 25-Hz protocol (Table 1).

MRI

Representative resting (preexercise) and 72 h postexercise T2 images are presented in Figure 2. Mean absolute T2 signal intensity for the QF muscle group and each individual quadriceps muscle (RF, VM, VI, and VL) are shown in Table 2. After the 100-Hz exercise protocol, an 18% increase ($P < 0.001$) in mean absolute T2 intensity was observed. The 25-Hz protocol induced a much smaller, 2%, but nonetheless significant ($P = 0.04$) increase in T2 signal intensity. When examined on a muscle-by-muscle basis, a significant muscle by time interaction ($P = 0.003$) was found with the 100-Hz protocol producing an 18% ($P = 0.006$), 26% ($P < 0.001$), 13% ($P = 0.002$), and 11% ($P = 0.008$) increase in absolute T2 intensity in the RF, VM, VI, and VL muscles, respectively (Table 2). Although a significant interaction ($P = 0.02$) was found preexercise to postexercise after the 25-Hz protocol, none of the individual differences between muscles reached statistical significance.

The 100-Hz protocol produced a contrast shift in a much larger (45% vs 3%; $P < 0.001$) volume of the QF compared with the 25-Hz protocol (Fig. 3A). Additionally, a significantly greater change in muscle volume and ΔT_2 occurred after exercise at 100 Hz compared with 25 Hz (Fig. 3B and Fig. 3C). A significant muscle by stimulation frequency interaction was observed for ΔT_2 ($P = 0.004$) and swelling ($P = 0.004$). Each of the four muscles of the QF demonstrated greater ΔT_2 ($P \leq 0.03$) and swelling ($P \leq 0.008$) after exercise at 100 Hz (Fig. 4A and Fig. 4B). Although no differences were observed among the RF, VM, VI, and VL in the 25-Hz thigh, the VM of the 100-Hz thigh demonstrated a greater ΔT_2 than the VI and VL ($P \leq 0.03$; Fig. 4A) whereas the RF exhibited significantly more swelling than the VI and VL ($P \leq 0.03$; Fig. 4B).

Isometric torque

A significant stimulation frequency by time interaction was found ($P = 0.006$) with regards to changes in MVIC. One-way ANOVA ($P < 0.001$) indicated that MVIC values were reduced compared with baseline immediately postexercise and on days 1, 2, 3, 4, 7, 10, and 14 ($P \leq 0.05$ for each day; Fig. 5) after the 100-Hz exercise. One-way ANOVA ($P < 0.001$) indicated that MVIC was reduced immediately postexercise and on days 1, 2, and 3 ($P \leq 0.05$ for each day; Fig. 5) after the 25-Hz protocol. When compared between thighs, torque loss was statistically similar immediately after each exercise protocol, but strength was impaired to a greater extent on days 1–7 ($P < 0.05$ for each day; Fig. 5) postexercise in the 100-Hz thigh.

Ratings of muscle soreness

A significant stimulation frequency by time (days postexercise) interaction ($P = 0.001$) was found for ratings of muscle soreness. One-way ANOVA ($P < 0.001$) indicated that ratings of soreness differed from baseline immediately postexercise and over the next 4 d ($P < 0.05$ for each; Fig. 6) after the 100-Hz protocol. Soreness differed from baseline (one-way ANOVA; $P = 0.002$) immediately postexercise and over the next 3 days ($P < 0.05$ for each day; Fig. 6) after the 25-Hz protocol. When compared between protocols, rating of soreness were greater in the 100-Hz thigh immediately postexercise and on days 1–3 ($P < 0.05$; Fig. 6) after exercise.

DISCUSSION

The major finding of the present study was that when peak eccentric force, contraction speed, range of motion, and total eccentric work were matched, eccentric exercise performed using 100-Hz EMS induced greater muscle injury than exercise performed at 25 Hz. This finding suggests it was not peak eccentric force or total eccentric work but rather the higher force per active area during the 100-Hz protocol that was the critical mechanical component in the initiation of muscle injury. Conflicting results have been reported in previous studies regarding the relationship between certain mechanical components of eccentric contractions and the magnitude of muscle injury. Peak eccentric force (25,39), work (20), strain (6,23,37), contraction velocity (6,39), initial muscle length (37), and force per active area (18) have been shown to be determinants of the magnitude of muscle injury in various animal models. Faulkner et al. (11) have suggested that the disagreement could be attributed to the fact that a variety of animals (mice, rats, rabbits, toads) as well as several *in vitro*, *in situ*, and *in vivo* experimental models have been used to study contraction-induced injury. In human models, initial muscle length (8,31), peak force (29), and force per active area (5) have been associated with muscle injury. The results of the present study, indicating the importance of specific force in the initiating of muscle injury, agree with the animal results of Hentzen et al. (18) and extend our previous findings (5) by demonstrating that force per active area determines the magnitude of muscle injury independent of peak eccentric force and total eccentric work. The results of the present study would appear to be at odds with the findings of McCully and Faulkner (25) and Warren et al. (39), which indicated that peak eccentric force was best determinant of the

magnitude of contraction-induced injury. However, because activation of the exercised muscle is likely complete in the animal models used in these studies, contraction force is essentially a surrogate for specific force. Thus, these studies potentially provided further evidence of the role of by force per active area in the initiation of contraction-induced injury.

Another potential explanation for the difference in injury between the two protocols could also be related to differences in the susceptibility of fast and slow muscle fiber types to exercise-induced injury. Previous studies have suggested that fast fibers are more susceptible to injury than slow fibers (14,38) potentially due to reduced structural proteins (14), lower contractile workload (38), and ability to generate higher forces (2). Because the force frequency curve is shifted to the right in fast muscle compared with slow muscle, increasing EMS frequency from 25 to 100 Hz will result in a marked increase in both absolute force and specific force in the fast fibers whereas little change in either would be expected in the slow fibers (22). This would suggest that the difference in injury between the protocols was due to increased injury to fast muscle fibers both because fast muscle is more susceptible to injury and because the difference in specific force applied to the individual fibers between the 25- and the 100-Hz protocols would likely be much greater in fast compared with slow fibers. This fits with the general hypothesis that specific force is more important in determining muscle injury than absolute or nonspecific force (more muscle recruited at a lower specific force), and that this may be especially true in the more susceptible fast fibers. Because muscle fiber-type distribution as well as evidence of fiber-type—specific damage was not measured in this study, confirmation of this explanation is not possible. An interesting aspect of the fiber-type—specific injury hypothesis would be to determine whether smaller (individuals with predominantly slow fiber types) or larger (individuals with predominately fast fiber types) differences in the magnitude of injury would result between the two stimulation protocols used in the present study. Our findings also further highlight the potential for differences in fiber-type distribution to account for the large between individual variation in injury that has been previously reported (19).

The primary mechanism that is thought to be responsible for causing exercise-induced muscle injury is the high stress placed on individual fibers (17,39). Failure of muscle structures in this manner would follow the normal stress theory (39), which suggests that the weakest contractile or series component of a muscle would fail when the applied tensile stress is greater than its yield strength. Specific force likely represents the best *in vivo* measure of tensile stress, and as such, it seems plausible that a specific force threshold could exist in skeletal muscle. Once the force per active area exceeds this threshold, the contractile or series elements of the muscle may fail resulting in muscle injury. Because the amount of muscle activated during each eccentric exercise protocols was not measured, we were unable to directly quantify the eccentric force per active area imparted upon the muscle during 100- and 25-Hz EMS in the present study. Others have speculated that contraction forces approaching or exceeding 115–120% of those achieved during MVIC could provide sufficient tensile stress to surpass the yield strength of individual muscle fibers (17,39). A recent study by Hentzen et al. (18) supports this idea by demonstrating that both isometric and eccentric contractions resulted in a similar amount of injury when a similar high stress was placed upon the contracting fibers.

In the present study, all four muscles of QF demonstrated an increase in mean T2 signal intensity as well as an increase in volume after exercise at 100 Hz. Suggesting that each muscle was activated and subsequently injured by EMS. Although injury appeared to have occurred in all four muscles, it was not uniform across the QF. The VM demonstrated nearly double the $\Delta T2$ of the VI and VL—suggesting greater injury occurred in the vastus medialis. Data from a previous study (35) also demonstrated differential injury among the four quadriceps muscles after voluntary eccentric exercise with the RF demonstrating greater injury than the three vasti muscles. The researchers concluded that performing seated knee extensions resulted in greater lengthening distances and peak forces in the RF relative to normal daily activities. In the present

study, the most likely explanation for the finding of a greater ΔT_2 in the VM was not that the VM experienced greater stress or was lengthened over a greater distance compared with the other muscles of the QF but rather due to electrode placement, a larger percentage VM was activated during exercise. It was surprising, that the greatest change in volume was observed in the RF (20%) not in the VM (15%), as might have been expected given the VM's greater ΔT_2 and the relationship between the increase in T_2 and an increase in muscle volume (35). Muscle size (for the RF) represents the most plausible explanation as to the observed difference in swelling. In the imaged area, the RF comprised only about 8% of the total QF volume (data not shown). Fluid appeared to accumulate in the more distal images in the intermuscular space between the RF and the VM, VI, and VL potentially accounting for its change in volume. Due to its small relative size, small absolute changes in volume would produce a greater effect on RF muscle volume than the three vasti muscles, thus accounting for why it demonstrated the largest change in volume.

When taken in conjunction with the findings of Black et al. (5) and Hentzen et al. (18), the finding that specific force is potentially an important mechanical factor in initiating muscle injury has several wide-ranging implications. The first is in regards to motor unit recruitment during eccentric contractions. It has been suggested that eccentric contractions cause greater injury than their concentric and isometric counterparts because fewer motor units are recruited to produce a given force, leading to greater force per active area (3). It has also been suggested that an alteration in recruitment, such that a given force is spread over a greater number of motor units and/or a recruitment of a greater number of slow-twitch motor units, could represent potential mechanisms underlying the repeated bout or protective effect, where an initial bout of injurious exercise "protects" or lessens injury in subsequent exercise bouts (26). A second implication is related to the use of EMS in clinical or rehabilitative settings. Reducing the frequency of EMS has been shown to limit muscle fatigue (4). Data from the present and previous studies (5,18) demonstrate that reducing EMS frequency, for any given level of force production, limits contraction-induced muscle injury. Consequently, to maximize the effectiveness of rehabilitation, especially when the goal is to perform any type of functional task, stimulation amplitude should be increased to the maximum level tolerable to the subject to maximize muscle engaged in contraction, whereas frequency should be decreased as much as possible to limit both fatigue and muscle injury while maximizing force output.

In summary, results from the present study indicate that force per active area during eccentric contractions appears to play an important role in the initiation of exercise-induced muscle injury. The large difference in injury between the two EMS protocols is potentially the result of increased injury to fast muscle fibers because the difference in specific force between the protocols was likely in the fast fibers compared with slow fibers. Although no data regarding fiber types and fiber-type—specific injury were collected, we believe these findings, when taken in concert with those of others, suggest that high-tensile stress during eccentric contractions represents a critical factor responsible for the initiation of contraction-induced muscle injury.

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FIGURE 1.

Mean change in eccentric weight lifted during the 80 contractions of the 100- and the 25-Hz exercise protocols (panel A). Weight lifted was identical for both protocols. Mean change in eccentric weight lifted during the 100-Hz protocol (*closed circles*) and the 25-Hz protocol (*open circles*) expressed as a percent of each subject's MVIC (panel B). Values are mean (SD).

FIGURE 2. Anatomically matched axial T2 MR images from the midhigh region of the QF. Preexercise (A and C) and 72 h postexercise (B and D) images from the 100- and the 25-Hz protocols, respectively, from a representative subject.

FIGURE 3. Percent of QF muscle volume demonstrating an increase in T2 (panel A), mean change in T2 (panel B), and change in QF volume (panel C) after the 100- and the 25-Hz exercise protocols. Values are mean (SD). *Significant difference between 100 and 25 Hz ($P < 0.05$).

FIGURE 4.

Mean change in T2 (panel A) and mean change in muscle volume (panel B) for the individual muscles of the QF muscle group (RF, VM, VI, and VL). Values are mean (SD). *Significant difference for specific muscle (RF, VM, VI, and VL) between the 100- and the 25-Hz protocols ($P < 0.03$). †Significant difference among the VM and the VI and VL (DT2) and among the RF and the VI and VL (swelling) after the 100-Hz protocol ($P \leq 0.03$).

FIGURE 5.

Change in MVIC torque over 28 d after eccentric exercise at 100 Hz (*closed circles*) and 25 Hz (*open circles*). Values are mean (SD). *Significant difference between the 100- and the 25-Hz protocols ($P < 0.05$).

FIGURE 6.

Ratings of muscle soreness over 14 d after eccentric exercise at 100 Hz (*closed circles*) and 25 Hz (*open circles*). Values are mean (SD). *Significant difference between the 100- and the 25-Hz protocols ($P < 0.05$).

TABLE 1

Subject characteristics before eccentric exercise.

Subject Characteristics		
Age (yr)	26 (3)	
Height (cm)	173 (11)	
Weight (kg)	65 (12)	
Measure (Hz)	100	25
MVIC (NIm)	352 (124)	316 (113)
EMS isometric force (NIm)	98 (47)	101 (56)
EMS current (mA)	44 (8)	57 (14) *
Initial eccentric load (kg)	59 (31)	59 (31)

Values are mean (SD)

* Significant difference from the 100-Hz bout.

TABLE 2

T2 values for the QF muscle group and each individual knee extensor muscle from preeccentric and 72 h posteccentric exercise.

Muscle	100 Hz		25 Hz	
	Pre-T2 (ms)	Post-T2 (ms)	Pre-T2 (ms)	Post-T2 (ms)
QF	27.1 (2.4)	31.9 (4.4) *	26.5 (2.1)	26.9 (2.0) *
RF	29.2 (3.1)	34.5 (5.6) *	28.1 (2.2)	28.2 (2.3)
VM	26.5 (2.3)	33.5 (5.0) *	26.3 (2.4)	26.8 (2.5)
VL	26.6 (2.5)	30.1 (4.0) *	26.5 (1.8)	26.6 (1.8)
VL	27.6 (3.4)	30.5 (5.0) *	27.1 (2.7)	27.4 (2.8)

Values are mean (SD).

* Significant difference ($P < 0.05$) from pre-T2.