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The Role of Passive Muscle Stiffness in Symptoms of Exercise-Induced Muscle Damage

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ABSTRACT

We examined whether passive stiffness of an eccentrically exercising muscle group affects the subsequent symptoms of muscle damage. Passive hamstring muscle stiffness was measured during an instrumented straight-leg-raise stretch in 20 subjects (11 men and 9 women) who were subsequently classified as "stiff" ($N = 7$), "normal" ($N = 6$), or "compliant" ($N = 7$). Passive stiffness was 78% higher in the stiff subjects (36.2 ± 3.3 N·m·rad^{-1}) compared with the compliant subjects (20.3 ± 1.8 N·m·rad^{-1}). Subjects then performed six sets of 10 isokinetic (2.6 rad·s^{-1}) submaximal (60% maximal voluntary contraction) eccentric actions of the hamstring muscle group. Symptoms of muscle damage were documented by changes in isometric hamstring muscle strength, pain, muscle tenderness, and creatine kinase activity on the following 3 days. Strength loss, pain, muscle tenderness, and creatine kinase activity were significantly greater in the stiff compared with the compliant subjects on the days after eccentric exercise. Greater symptoms of muscle damage in subjects with stiffer hamstring muscles are consistent with the sarcomere strain theory of muscle damage. The present study provides experimental evidence of an association between flexibility and muscle injury. Muscle stiffness and its clinical correlate, static flexibility, are risk factors for more severe symptoms of muscle damage after eccentric exercise.

Exercise involving predominantly eccentric actions frequently causes muscle damage, resulting in delayed-onset muscle soreness.[1,5,13] In humans, damage is usually described according to various symptoms, including strength loss, pain with activity, muscle tenderness, and elevated muscle enzyme activity.[3–5,31] Strength loss can have obvious deleterious effects on subsequent function. In addition, exercise in the presence of muscle damage involves greater metabolic stress and can result in premature fatigue.[8,9] Although muscle damage is known to occur with exercise involving eccentric actions, little is known about other etiologic factors. It is unclear why some people are more susceptible to muscle damage than others after a given bout of exercise. It is possible that flexibility, specifically muscle stiffness, is an important factor.

The injury that occurs with eccentrically induced muscle damage has been described as mechanical failure of individual myofibrils subjected to cyclic tensile loading.[1] Studies using simulated eccentric actions in animal models[2,16,19] and voluntary eccentric actions in humans[3,26] have demonstrated greater damage with contractions at long versus short muscle lengths. Consistent with these findings, Armstrong et al.[1] have suggested that damage occurs at muscle lengths on the descending limb of the length-tension curve. During eccentric actions on the descending limb of the length-tension curve, active force is decreasing while passive force is increasing. Armstrong et al. suggested that damage could result from excessive stress on the passive elements. In people with stiffer muscles, a greater passive muscle force would be expected during eccentric actions and may predispose these people to muscle damage.

The passive stiffness of skeletal muscle has been measured in humans according to the relationship between joint torque and range of motion during passive stretch.[6,7,12,14,20,21,23,24,32,33] Passive hamstring muscle

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stiffness has been shown to correlate with maximum straight-leg-raise range of motion[24] and the sit-and-reach test.[20] The straight-leg-raise and sit-and-reach tests are standard tests of flexibility, but the measurements are inherently subjective and are affected by the subject's tolerance of the discomfort of the stretch.[20] By contrast, stiffness measurements provide an objective measure of the extensibility of skeletal muscle.

A clear relationship between flexibility and muscle strain injury has not been established.[10] Conflicting results may be explained by limitations in flexibility measurements and inadequate control of confounding factors, such as exposure time, previous injury, and type of injury.[10] However, the injury induced by eccentric exercise can be studied with adequate control. Additionally, passive muscle stiffness provides a more objective measure of flexibility. Therefore, the purpose of this study was to examine the effect of passive muscle stiffness on symptoms of exercise-induced muscle damage after a bout of eccentric exercise. Such an analysis can yield important clinical information on the basic association between muscle stiffness and susceptibility to injury.

MATERIALS AND METHODS

The response of the hamstring muscle group to eccentric exercise was studied in 20 subjects. On the initial test day, passive hamstring muscle stiffness was measured during an instrumented straight-leg-raise stretch. A baseline measure of maximum isometric hamstring muscle strength was then made. Subjects then performed six sets of 10 isokinetic eccentric hamstring muscle actions at 60% of maximum isometric strength. On the following 3 days, maximum isometric strength, pain, and muscle tenderness were assessed. Additionally, plasma creatine kinase activity was measured in 12 subjects at baseline and on each of the following 3 days. Subjects were divided evenly into tertiles as having compliant ($N = 7$), normal ($N = 6$), or stiff ($N = 7$) hamstring muscles. Isometric strength loss, pain, muscle tenderness, and plasma creatine kinase activity were compared between subjects based on hamstring muscle stiffness. The study was approved by the institutional review board and all subjects who volunteered for the study gave informed consent. Subject characteristics are provided in Table 1. Subjects were without orthopaedic injury and had not been involved in any weight training in the preceding months.

TABLE 1
Sex, Height, and Body Mass of Subjects in the Compliant, Normal, and Stiff Groups (Mean ± SE)

Group	Sex	Age (years)	Height (cm)	Body mass (kg)
Compliant	1 man			
	6 women	30.1 ± 2.5	161.6 ± 6.4[a]	57 ± 2.9[b]
Normal	4 men			
	2 women	27 ± 1.5	177 ± 2.1	76.8 ± 2.1[a]
Stiff	6 men			
	1 woman	28.3 ± 2.5	182.1 ± 2.9	81.6 ± 2.9

[a] Significantly different from the stiff group.
[b] Significantly different from the stiff and normal groups ($P \leq 0.05$ based on Bonferroni corrections).

Measurement of Passive Hamstring Muscle Stiffness

The technique for measurement of passive hamstring muscle stiffness has been described previously.[24] In brief, a 1.05 rad (60°) straight-leg-raise stretch was applied using a hydraulically powered, motorized, rotating frame aligned with the hip joint (Scientific Stretching, Parrsboro, Nova Scotia, Canada) (Fig. 1). The ankle was attached to the moving frame by a chain in series with a load cell (Kistler Instruments, Amherst, New York). The frame was adjustable to the subject's limb length. During the stretch, the knee was braced in full extension and the contralateral limb was fixed to the table with a strap to limit pelvic rotation. Hip flexion was recorded from an electrogoniometer (Penny & Giles, Gwent, United Kingdom) attached to the frame and aligned with the hip joint. Resistance to stretch, recorded from the load cell, was corrected for the limb mass, as described previously.[12,24,25] Surface EMG signals were recorded from pairs of silver/silver chloride electrodes placed over the rectus femoris, biceps femoris, semimembranosus, and semitendinosus muscles. The skin was shaved, cleaned, and abraded before electrode placement. The raw EMG signal was recorded by telemetry (Noraxon, Scottsdale, Arizona) with a bandwidth of 0 to 500 Hz and a common-mode rejection ratio of 135 dB. Rectus femoris muscle activity was recorded to ensure the subject was not actively assisting the stretch. Hamstring muscle EMG activity was recorded to ensure that contractile force production did not contribute to the measurement of passive stiffness. Torque-range of motion curves were calculated for 0 to 1.05 rad. Passive stiffness was defined as the increase in torque from 0.35 to 0.87 rad (20° to 50°) in the absence of EMG activity. Previous studies have shown this region of

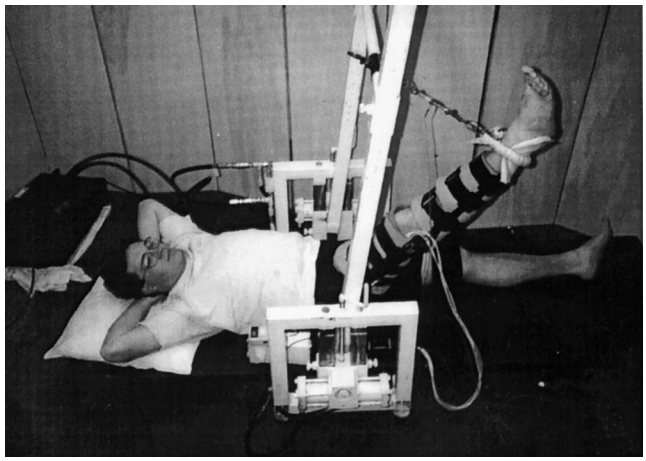

Figure 1. Instrumented straight-leg-raise stretch on a hydraulically motorized moving frame. The frame is attached to the ankle by a chain in series with a load cell. An electrogoniometer is aligned with the hip and the knee is braced in extension.

the torque-range of motion curve to be linear.[12,24] The mean of two stiffness measurements separated by 60 seconds was recorded. Subjects were categorized into three groups (stiff, normal, or compliant) based on the measurement of passive hamstring muscle stiffness.

Isometric Strength and Eccentric Exercise

Subjects were seated in an upright position with the hips at 1.6 rad (90°) of flexion. The thigh of the test limb was strapped to prevent hip flexion during testing. The knee joint was aligned with the axis of rotation of the dynamometer (Biodex System 2, Biodex Medical Systems, Shirley, New York), and the leg was secured to the dynamometer arm at the ankle. The knee joint was set at 0.8 rad (45°) of flexion and limb mass was recorded. The subjects were then instructed to maximally contract the knee flexors while consistent verbal encouragement was provided to ensure maximal effort. Four 5-second contractions were performed with 10 seconds between efforts. Peak torque was recorded after correction for limb mass. Immediately after isometric strength measurements, eccentric actions were performed from 1.6 to 0 rad (90° to 0°) of knee flexion. The dynamometer arm was set to move through the selected range of motion at 2.6 rad·s^{-1} (150 deg·s^{-1}) and subjects contracted the muscle group with sufficient intensity to reach a visually displayed target strength equal to 60% of isometric strength. This fixed intensity ensured that all subjects exercised at the same relative intensity. In previous studies, six sets of 10 isotonic eccentric actions of the hamstring muscle at 100% of concentric strength[4] and one set of 10 isotonic eccentric actions of the elbow flexor at 60% of isometric strength,[31] have been shown to be sufficient to cause symptoms of muscle damage.

Pain and Muscle Tenderness Measurement

Before isokinetic exercise, and on each of the subsequent 3 days, subjects were asked to report their pain level. Subjects were asked to report hamstring muscle pain on a scale of 0 (no discomfort) to 10 (walking with a limp). Muscle tenderness was evaluated by pressing an 18-mm probe, attached to a load cell, into the muscle bellies of the respective hamstring muscles. The subjects were asked to report discomfort elicited by application of the probe. The signal from the load cell was recorded during each trial and interrupted at the point of discomfort. The force at that point was computed and subtracted from 40 N to give a tenderness value.[27] Any discomfort elicited at forces above 40 N was not included. The values from each muscle were summed for analysis.

Creatine Kinase Activity Measurement

Plasma creatine kinase activity was determined from a finger-prick blood sample. The finger was warmed, cleaned with alcohol, and dried. After puncture, the initial sample of blood was removed. A 30-μl sample was then collected in a capillary tube and immediately pipetted onto a test strip for analysis. Creatine kinase activity was analyzed by a colorimetric assay procedure (Reflotron, Boehringer Mannheim, Indianapolis, Indiana). The system uses a plasma separation principle incorporated in the reagent carrier on the test strip. Creatine kinase measurements were not made on the first eight subjects because the instrument was unavailable. Creatine kinase values were subjected to logarithmic transformations for statistical analysis.

Measurement Validity and Reliability

A separate group of subjects (6 men and 4 women) was used to test the validity and reliability of test measurements. These subjects were of similar age (32 $\pm$ 2.4 years), height (171.3 $\pm$ 3.5 cm), and body mass (77.3 $\pm$ 8.0 kg) as the subjects performing eccentric exercise. Isometric strength, pain, muscle tenderness, and creatine kinase activity were measured before six sets of 10 isokinetic concentric hamstring actions at 60% of maximum isometric strength and again on each of the following 3 days. This group served to validate the eccentric-exercise protocol by demonstrating that symptoms of muscle damage were specific to eccentric and not concentric exercise. Additionally, repeated measures in a group not experiencing muscle damage provided an indication of measurement reliability. Repeated stiffness measurements were also made on this group of subjects to test reliability.

Statistical Power

An effect size was calculated for differences in the dependent variables (strength loss, pain, tenderness, creatine kinase activity) between subjects with compliant and stiff hamstring muscles. At an alpha level of $\leq$0.05, there was 80% power to detect a 37% difference in strength loss between subjects with compliant and stiff hamstring muscles. Additionally, there was 80% power to detect a six-point difference in pain, a 19-N difference in tenderness, and a 36% difference in increased creatine kinase activity. However, creatine kinase activity was tested with nonparametric statistics because of the small uneven group sizes.

Statistics

A mixed-model analysis of covariance was used to test changes in isometric strength, pain, and muscle tenderness between stiff, normal, and compliant subjects, with sex and body mass as control variables. An alpha level of $\leq$0.05 was required for statistical significance. Post hoc tests, with Bonferroni corrections, were used to test specific differences between stiff and compliant subjects. Greenhouse-Geisser corrections were applied to significant F ratios that did not meet Mauchly's sphericity assumption. Probability values that have been corrected are denoted by the subscript $_{GG}$. Given the small, uneven group sizes for analysis of creatine kinase activity (compliant = 5, normal = 3, stiff = 4), nonparametric tests were used to analyze changes in creatine kinase values

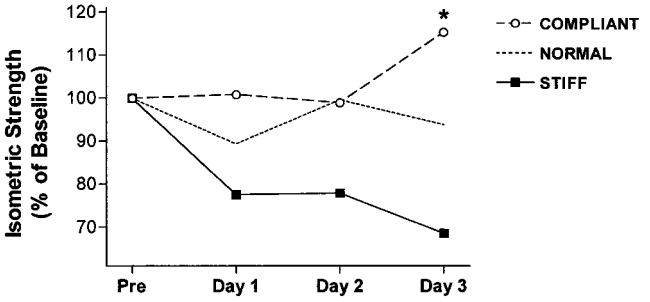

Figure 2. Isometric hamstring muscle strength loss after eccentric exercise expressed as a percentage of the baseline (before eccentric exercise). Means are adjusted for sex and body mass; $P \leq 0.05_{GG}$ for the main effect of stiffness on strength loss. The asterisk indicates significantly greater strength loss in the stiff group compared with the compliant group ($P \leq 0.05$).

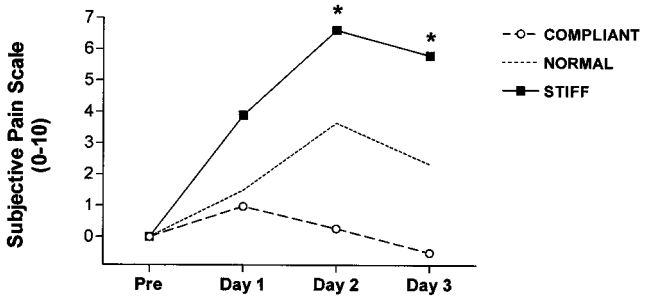

Figure 3. Changes in pain after eccentric exercise. Pain values are arbitrary units on a scale of 0 to 10. Means are adjusted for sex and body mass. The asterisks indicate significantly greater pain in the stiff group compared with the compliant group ($P \leq 0.05$).

subjected to logarithmic transformation (CK_{log}). Differences in percentage increase in CK_{log} activity between compliant, normal, and stiff subjects were analyzed by Kruskal-Wallis H tests. Where significant H values were found, post hoc Mann-Whitney U tests were used to compare the compliant and stiff groups. Bonferroni corrections were applied to P values from Mann-Whitney U tests. One-way repeated measures analysis of variance was used to test changes in isometric strength, pain, muscle tenderness, and creatine kinase activity in the subjects who had performed concentric exercise. Intraclass correlations $(3,1)$[29] were calculated to test measurement reliability in this group.

RESULTS

Passive Hamstring Muscle Stiffness

The subjects were divided into three groups based on passive hamstring stiffness: compliant ($N = 7$; 6 women, 1 man), 20.3 ± 1.8 N·m·rad^{-1} (mean ± SEM); normal ($N = 6$; 2 women, 4 men), 27.1 ± 0.4 N·m·rad^{-1}; and stiff ($N = 7$; 1 woman, 6 men), 36.2 ± 3.3 N·m·rad^{-1}. In one subject, rectus femoris muscle EMG activity was present during the first straight-leg-raise stretch, but no activity was present during the second stretch. The stiffness value was computed from the second stretch only. No other subjects exhibited detectable EMG activity in the rectus femoris and hamstring muscles from 0 to 0.87 rad during straight-leg-raise stretches. There was no significant difference between the stiffness value for the first and second stretch across stiffness groups. Passive stiffness was significantly related to sex ($P \leq 0.05$) and body mass ($P \leq 0.05$). Therefore, the effect of passive muscle stiffness on symptoms of muscle damage was analyzed on means adjusted for sex and body mass (analysis of covariance).

Symptoms of Muscle Damage

Eccentric exercise resulted in significant strength loss ($P \leq 0.05_{GG}$), pain ($P \leq 0.05$), muscle tenderness ($P \leq 0.05_{GG}$), and elevated creatine kinase activity ($P \leq 0.05$). Strength loss ($P \leq 0.05_{GG}$), pain ($P \leq 0.05$), muscle tenderness ($P \leq 0.05_{GG}$), and elevated creatine kinase activity ($P \leq 0.05$) were significantly affected by passive muscle stiffness. Stiff subjects experienced greater strength loss than compliant subjects on day 3 (Fig. 2). Stiff subjects also reported significantly greater pain than compliant subjects on days 2 and 3 ($P \leq 0.05$) (Fig. 3). In addition, muscle tenderness was higher in the stiff subjects compared with the compliant subjects on days 2 and 3 ($P \leq 0.05$) (Fig. 4). Creatine kinase measurements were made on five compliant subjects, three normal subjects, and four stiff subjects. Larger increases in CK_{log} activity were observed in the stiff compared with the compliant subjects on day 1 (0.6% versus 26%; $P \leq 0.05$). Similar differences in creatine kinase activity were seen on day 2 (3.4% versus 40.1%) and day 3 (12% versus 37.9%), but these did not attain statistical significance.

Measurement Validity and Reliability

Isometric strength was unaffected by concentric exercise and showed good reliability (intraclass coefficient correla-

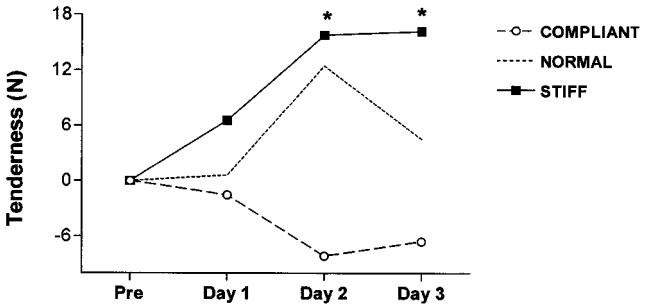

Figure 4. Changes in muscle tenderness after eccentric exercise. Tenderness values are summed for each muscle and means are adjusted for sex and body mass. Negative values are a function of the means adjustment and do not represent real values. The asterisks indicate significantly greater tenderness in the stiff group compared with the compliant group ($P \leq 0.05$).

tion [ICC], 0.93). Similarly, pain and muscle tenderness were unaffected by concentric exercise. Only two subjects reported hamstring muscle pain after concentric exercise (pain level = 1 and 2), and no subjects had muscle tenderness. Therefore, intraclass coefficient correlations were not calculated on these variables. The CK_{log} activity was slightly increased after concentric exercise (day 1, 10%; day 2, 7%; day 3, 4%; $P \leq 0.05$). The CK_{log} measurements showed good reliability (ICC, 0.87). Repeated passive stiffness measurements were within 5% of the initial measurement and demonstrated good reliability (ICC, 0.95).

DISCUSSION

Strength loss, pain, muscle tenderness, and elevated creatine kinase activity define the delayed-onset muscle soreness syndrome and provide indirect evidence of muscle damage. These symptoms of damage differed according to the subject's passive hamstring muscle stiffness. Greater strength loss, pain, muscle tenderness, and elevated creatine kinase activity in stiff compared with compliant subjects indicates that subjects with stiffer hamstring muscles experienced greater muscle damage, despite exercising at the same relative intensity.

Few studies have identified other etiologic factors involved in muscle damage. However, a recent study indicated that prior concentric exercise or warm-up lessened subsequent symptoms of muscle damage.[28] Nosaka and Clarkson[28] found that fatiguing concentric exercise or cyclic passive motion before a bout of eccentric exercise dramatically reduced the subsequent symptoms of damage. These effects were attributed to reduced muscle stiffness after the concentric exercise or the passive motion. Although actual stiffness measurements were not made, previous research has shown reduced passive stiffness after fatiguing concentric actions[22] and repeated passive stretches.[21] The results of Nosaka and Clarkson are supported by the present study, which indicated that stiffer muscles are more susceptible to damage after eccentric exercise.

Greater symptoms of muscle damage in subjects with stiffer muscles may be explained by tendon-aponeurosis mechanics during eccentric actions. We propose that the strain imposed by active lengthening of stiff muscles is transferred from a rigid tendon-aponeurosis complex to the muscle fibers, resulting in myofibrillar strain. In compliant muscles, the tendon-aponeurosis complex is able to absorb lengthening, thereby limiting myofibrillar strain. This theory is supported by studies that observed muscle fiber shortening[11] or constant fiber length[30] despite lengthening of the muscle-tendon unit during eccentric actions. Griffiths[11] demonstrated muscle fiber shortening in cat plantarflexors during simulated eccentric actions in isolated muscle-tendon units and during the eccentric phase of walking. These findings were attributed to lengthening in the tendon. It has been suggested that tendons act as mechanical buffers to protect muscle fibers from abrupt length changes.[11] Similarly, Roberts et al.[30] demonstrated that tendon-aponeurosis compliance in turkey lateral gastrocnemius muscles allowed muscles fibers to maintain a relatively constant length during the stance phase of running, thus decreasing muscular work.

A theoretical explanation of the present results assumes that passive muscle stiffness reflects tendon-aponeurosis extensibility. It has been suggested that passive muscle stiffness primarily reflects the extensibility of the connective tissue elements in parallel with the muscle fibers (parallel elastic component).[17,18,24] However, aponeurosis strain does occur with lengthening of isolated muscle-tendon units,[15] with greatest strain in the region closest to the muscle fibers.[34] It is also reasonable to assume that, in a given muscle, parallel elastic component stiffness would be related to stiffness in the tendon-aponeurosis complex. Stiff subjects presumably had greater tendon-aponeurosis stiffness given that their passive muscle stiffness was 78% higher than in compliant subjects.

A limitation of the present study was that stiffness groups differed in terms of sex and body mass. Stiff subjects (6 men and 1 woman) had 43% higher body mass and 78% higher stiffness. A large component of the between-group differences in stiffness probably reflected differences in muscle cross-sectional area. However, it is unlikely that cross-sectional area accounted for the entire between-group difference. Analyses were controlled for both sex and body mass and indicated that the observed effects primarily reflected the role of stiffness. Despite limited statistical power, significant differences were detected between stiff and compliant subjects in each of the markers of muscle damage.

Clinical Significance

The present study provides experimental evidence of a positive association between flexibility and muscle injury. Specifically, the results indicate that more-flexible people are less susceptible to exercise-induced muscle damage. Exercise in the presence of muscle damage involves a greater metabolic stress and limits exercise intensity and duration.[8,9] Therefore, more flexible people may be able to exercise at a higher intensity or for a greater duration on the days after a bout of eccentric exercise. It is not known if stretching to improve flexibility before eccentric exercise will limit subsequent muscle damage. However, there is some evidence indicating that preexercise warm-up may reduce subsequent symptoms of muscle damage.[28]

SUMMARY

The present study demonstrated a significant effect of passive muscle stiffness on symptoms of muscle damage after eccentric exercise. Subjects with stiffer hamstring muscles experienced greater strength loss, more pain, greater muscle tenderness, and higher creatine kinase elevation on the days after eccentric hamstring muscle exercise. These effects are attributed to differences in sarcomere mechanics during eccentric actions in stiff and compliant muscles. Therefore, muscle stiffness and its clinical correlate, static flexibility, are risk factors for more severe delayed-onset muscle soreness.

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