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ORIGINAL ARTICLE

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The role of exercising muscle length in the protective adaptation to a single bout of eccentric exercise

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Abstract The purpose of this study was to determine if the protective adaptation to a single bout of eccentric exercise (repeated bout effect) is dependent on the muscle length at which the eccentric contractions are performed. Ten subjects (six men, four women) performed two bouts of 120 isokinetic eccentric contractions separated by 2 weeks (target intensity was 90% of maximum isometric strength at 70°). In the initial bout one limb exercised from 30° to 70° of knee flexion (short initial bout; SIB) and the contralateral limb exercised from 70° to 110° (long initial bout; LIB). For the repeated bout 2 weeks later, the limb that initially exercised at a short length now exercised at a long length (long repeated bout; LRB) and the limb that initially exercised at a long length now exercised at a short length (short repeated bout; SRB). Isometric strength and pain (scale 0–10) were assessed immediately post exercise and on the next 3 days. Strength loss and pain were greater following LIB versus SIB (strength loss $P < 0.01$; pain $P < 0.001$) and following LRB versus SRB (strength loss $P < 0.01$; pain $P < 0.001$). Strength loss and pain were not different between LIB and LRB. Pain was significantly greater following SIB compared with SRB ($P < 0.05$). Strength loss was not different between SIB and SRB. These results confirm that the symptoms of muscle damage are highly dependent on exercising muscle length and also demonstrate that the repeated bout effect is dependent on muscle length. Performing an initial bout of eccentric exercise at a shortened muscle length did not protect against strength loss and pain following a repeated bout at a longer muscle length. Data are given as mean (SE) unless otherwise stated.

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Introduction

The protective adaptation to a single bout of eccentric exercise has been referred to as the repeated bout effect (Nosaka and Clarkson 1995). The repeated bout effect has been demonstrated in both humans and animal models.

It has been shown to last several weeks, possibly up to 6 months (Nosaka et al. 2001a). It is apparent that the initial bout of eccentric exercise does not have to result in appreciable symptoms of damage in order to confer a protective adaptation (Clarkson and Tremblay 1998; Brown et al. 1997; Nosaka et al. 2001b). It appears that the contraction intensity must be close to maximum in the initial bout in order to confer a protective effect when the repeated bout involves high intensity contractions (Nosaka and Newton 2002). Protection is specific to the exercised muscle groups, with no evidence of a cross-transfer to contralateral muscle groups not exposed to the initial bout (Clarkson et al. 1987; Connolly et al. 2003).

Data from isolated whole muscle preparations in animals (Lieber and Fridén 1993) and voluntary contractions in humans (Newham et al. 1988) have clearly shown that the length of the muscle during eccentric contractions appears to be a critical factor in determining the extent of the subsequent damage. It has been shown that damage to rabbit tibialis anterior muscles was a function of the length to which the muscle was elongated during stimulation rather than the magnitude of stimulation (Lieber and Fridén 1993). Similarly, eccentric contractions of the elbow flexors performed at longer muscle lengths resulted in greater symptoms of damage (Newham et al. 1988). These studies support the sarcomere strain theory of myofibrillar disruption with damage occurring when sarcomeres are on the

"descending limb" of the length-tension curve (Morgan 1990). Morgan (1990) has proposed that damage occurs when sarcomeres are strained beyond myofilament overlap and do not re-interdigitate between contraction cycles. A repair process resulting in an increase in serial sarcomeres is thought to limit sarcomere strain for subsequent eccentric exercise bouts thereby limiting damage (Lynn and Morgan 1994; Lynn et al. 1998). Recently shifts in the angle-torque curve toward greater torque production at longer muscle lengths have been demonstrated for maximum voluntary contractions of the knee flexors (Brockett et al. 2001) and knee extensors (McHugh 2003) following recovery from initial eccentric bouts. These findings are consistent with a serial addition of sarcomeres.

While muscle length is clearly an important factor in the initiation of muscle damage during eccentric contractions the role of muscle length in the initiation of a repeated bout effect is not known. If the repeated bout effect were due to reduced sarcomere strain, then an initial bout performed at a short muscle length would not be expected to provide protection for a repeated bout at a longer muscle length since this repeated bout would involve greater sarcomere strain than the initial bout. It would follow that an initial bout at a long muscle length would provide protection for a repeated bout at shorter length. Alternatively, since previous studies have demonstrated that only a few high intensity contractions are necessary to initiate a repeated bout effect (Clarkson and Tremblay 1998; Brown et al. 1997; Nosaka et al. 2001b) muscle length may not be an important factor. Therefore, the purpose of this study was to determine if the protective adaptation to a single bout of eccentric exercise is dependent on the muscle length at which the eccentric contractions are performed. It was hypothesized that an initial bout at a short muscle length would not protect against damage from a repeated bout at a longer length, whereas an initial bout at a long muscle length would protect against damage from a repeated bout at a shorter length.

Methods

Subjects

Ten subjects [six men, four women; age 31 (2) years, mass 73 (6) kg, height 173 (3) cm; mean (SE)] volunteered to participate in this study. All subjects gave written informed consent and the protocol was approved by institutional review board. The subjects were without orthopaedic injury and had not been involved in any weight training involving the quadriceps in the preceding 6 months. All subjects were asked to refrain from other exercise and not to take any pain medication during the course of the study.

The sample size for this study was based on the ability to detect a difference in isometric strength loss of 10% between the initial and repeated bouts. For example, 15% strength loss following the initial bout compared with 5% strength loss following the repeated bout (10% difference between bouts) would be significant with an alpha level of 0.05 and a beta level of 0.2 (80% power) with a sample size of eight. This calculation was based on an estimated SD of 10% for the between subject differences in strength loss between bouts. The 10% SD value was based on the inter-subject variability in previous samples performing similar repeated eccentric bouts (McHugh and Tetro 2003; McHugh et al. 2001).

Protocol

Subjects performed two bouts of 120 isokinetic (Biodex System 2, Shirley, N.Y., USA) eccentric knee extension contractions (six sets of 20 contractions) separated by 2 weeks. Target intensity was determined as 90% of maximum voluntary isometric strength (MVC) at 70° of knee flexion. Contractions were performed at 1.05 rad s^{-1} (60° s^{-1}) with subjects seated and the trunk at approximately 90° of flexion. An angular velocity of 1.05 rad s^{-1} was sufficiently slow to allow subjects to accurately produce the target torque production. For the initial bout one limb exercised from 30°–70° of knee flexion (short initial bout; SIB) and the contralateral limb exercised from 70°–110° (long initial bout; LIB). For the repeated bout the limb that exercised short now exercised long (SIB/Long Repeated Bout; LRB) and the limb that exercised long now exercised short (LIB/Short Repeated Bout; SRB). These ranges of motion (30°–70° and 70°–110°) were chosen because 70° is close to the optimal angle for knee extension torque production in most individuals. Additionally, 110° of knee flexion was close to the maximum comfortable range available with this dynamometer. The dominant limb was tested first, with the starting muscle length (short versus long) alternated between subjects. Peak torque for each contraction was recorded during the exercise.

Indices of muscle damage

Isometric strength was recorded prior to and immediately following both eccentric bouts and on each of the next three days. Isometric MVCs were performed at 30°, 50°, 70°, 90°, and 110° of knee flexion. Isometric testing was performed on the same dynamometer as eccentric testing, in the same, seated position. At each of the test angles subjects were asked to perform a maximal contraction for 3 s. Two contractions were performed at each angle with 5 s between contractions and two minutes between tests at the different angles. Peak torque was identified for each contraction and the average of the two contractions was recorded for each test angle.

Prior to eccentric exercise and on each of the subsequent 3 days, subjects were asked to report their pain level. Subjects were specifically asked to report a single

score for quadriceps pain elicited with walking. Pain ratings were recorded on a scale of 0 (“no discomfort”) to 10 (“walking with a limp”).

Quadriceps electromyographic signals

During all contractions electromyographic (EMG) activity was recorded from surface electrodes placed over the rectus femoris (RF), vastus lateralis (VL) and vastus medialis (VM) muscles. EMG activity during isometric MVCs was used to assess for any potential differences in voluntary neural drive between test angles, over time following eccentric bouts. EMG activity during eccentric exercise was used to assess for any differences in neural drive between the initial and repeated bouts performed at the same intensity. The skin was shaved, cleaned and abraded prior to application of 10 mm diameter Ag/AgCl electrodes. Electrode pairs were placed along the long axis of the muscles. For RF, a pair of electrodes were placed midway along a line between the anterior superior iliac spine and the superior pole of the patella. For VL, electrodes were placed four fingerbreadths proximal to the superiolateral border of the patella along the assumed line of the fibers. For VM, electrodes were placed two fingerbreadths proximal to the superiomedial border of the patella along the assumed line of the fibers. An inter-electrode distance of 3 cm (center to center) was used and a ground electrode was placed on the patella. Care was taken to replicate this electrode placement for the repeated bout. The telemetered EMG signals were band pass filtered from 10 to 500 Hz and sampled at 1,000 Hz, with a common-mode rejection ratio of 130 dB (Telemyo, Noraxon, Scottsdale, Ariz., USA). The amplitude of the raw EMG signal was quantified by computing the root mean square (RMS) of the raw signal with a 50-ms sliding average. For eccentric contractions the average of the RMS for the duration of each contraction was calculated. RMS values were summed for the three muscles and averaged for the 20 contractions in each set. This average was then expressed relative to the average torque for that set of 20 contractions (μV [N m]$^{-1}$). These values were used to assess changes in neural drive during the eccentric bouts and between initial and repeated bouts. The amplitude of the EMG signals (RMS) during isometric contractions was compared between days to assess changes in efferent neural drive associated with strength loss and recovery.

Statistics

Repeated measures analyses of variance using univariate general linear modeling were used to determine the effects of muscle length (short versus long) and exercise bout (initial versus repeated) on isometric strength loss and pain. Repeated measures analyses of variance were also used to examine changes in EMG activity during the repeated eccentric bouts at different muscle lengths. Values given are mean (SE). A P value of less than 0.05 was considered statistically significant.

Results

Strength loss

Strength loss following the initial bout was significantly greater on the side that performed the eccentric contractions from 70° to 110° (LIB) compared with the side that performed the contractions from 30° to 70° (SIB) (side×time $P < 0.01$; Fig. 1). Peak strength loss occurred one day following LIB. Isometric strength (averaged across test angles) was 77% of baseline for LIB compared with 93% for SIB ($P < 0.05$). Prior to the repeated bout strength (averaged across test angles) was 95% of baseline ($P < 0.2$) in SIB and 88% of baseline ($P < 0.05$) in LIB. Strength loss following the repeated bout was also significantly greater on the side that performed the eccentric contractions from 70° to 110° (LRB) compared with the side that performed the contractions from 30° to 70° (SRB) (side×time $P < 0.01$; Fig. 1). Peak strength loss occurred 2 days following LRB. Isometric strength (averaged across test angles) was 80% of baseline for LRB compared with 99% for SRB ($P < 0.05$).

Strength loss following LIB was not different from strength loss following LRB (bout effect $P = 0.61$) indicating that SIB did not limit strength loss following LRB. Similarly there was no difference in strength loss between SIB and SRB (bout effect $P = 0.42$). However, in neither case was strength loss significant (SIB $P = 0.22$; SRB $P = 0.08$).

Following LIB greater relative strength loss occurred at longer muscle lengths (angle effect $P < 0.0001$; Fig. 2). This effect was most apparent on day 3 where strength

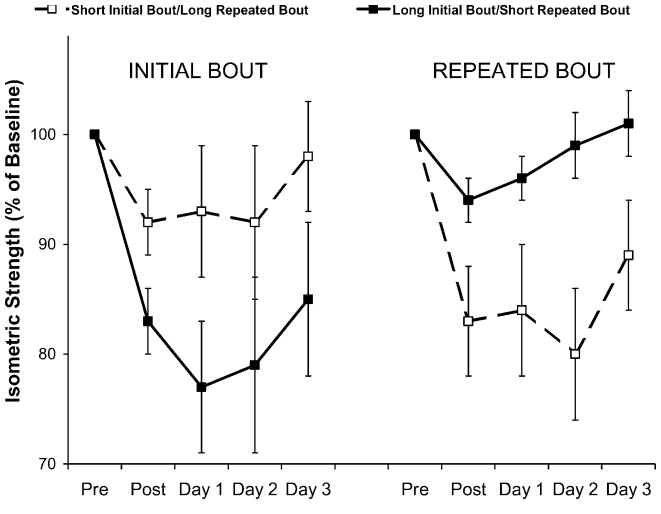

Fig. 1 Strength loss (% of baseline strength) following initial and repeated bouts of eccentric exercise. See Results section for details of statistical analyses

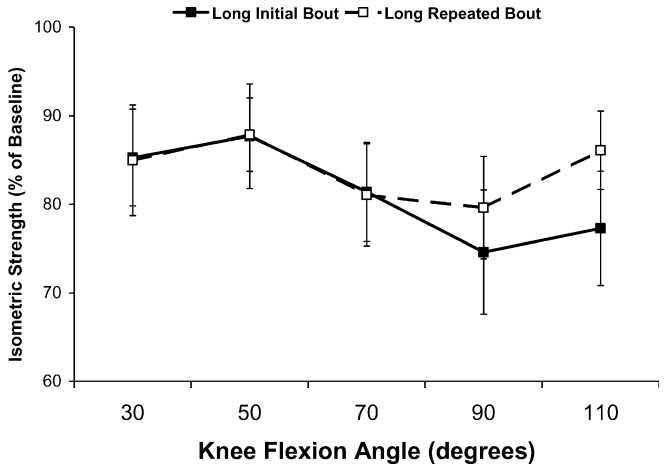

Fig. 2 Strength loss (% of baseline strength) at each knee flexion angle following initial and repeated bouts of eccentric exercise performed at a long muscle length [(long initial bout (LIB) and long repeated bout (LRB)]. Values are averaged for measurements made immediately post exercise and on each of the next 3 days. Effect of angle: LIB $P < 0.0001$, strength loss at 90° greater than at 50° $P < 0.05$; effect of angle: LRB $P = 0.32$

was 93% of baseline at 30°, 96% at 50°, 83% at 70°, 75% at 90°, and 79% at 110° (30° and 50° different from 90° and 110°, $P < 0.05$). An angle effect was not apparent for strength loss following LRB ($P = 0.32$; Fig. 2) or either bout at short muscle lengths ($P = 0.56$ and $P = 0.92$).

Pain

Pain following LIB was significantly greater than pain following SIB (side × time $P < 0.001$; Fig. 3). Peak pain occurred on day 2 for both initial bouts. No subjects had pain on either side prior to the repeated bouts. Pain was also significantly greater following LRB compared with SRB (side×time $P < 0.001$; Fig. 3). Pain following LIB was not different from pain following LRB ($P = 0.17$). Therefore the initial bout from 30° to 70° (SIB) did not protect that leg from pain following the repeated bout from 70° to 110° (LRB). Pain following SRB was less than pain following SIB ($P < 0.05$) indicating that LIB limited pain following SRB.

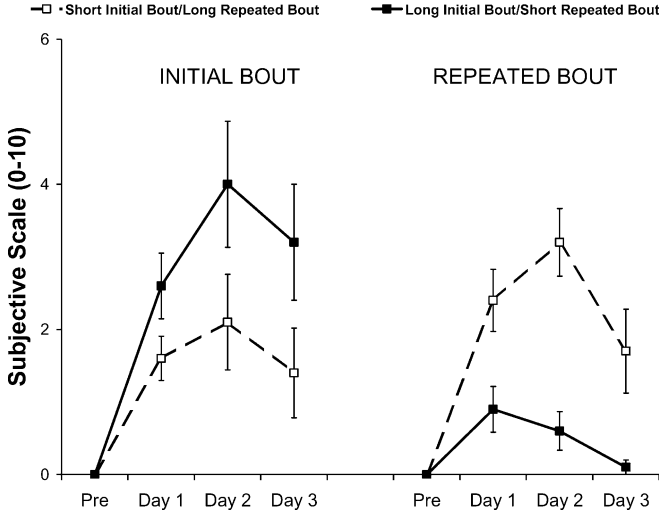

Fig. 3 Pain on a scale of 0 (no quadriceps discomfort) to 10 (walking with a limp) following initial and repeated bouts of eccentric exercise. See Results section for details of statistical analyses

EMG activity

Despite the marked strength loss there was no decrease in EMG amplitude during isometric tests following LIB ($P = 0.42$). EMG activity following LIB was also not different between test angles ($P = 0.72$) indicating that the angle effect on strength loss was not a function of decreased neural drive. Separate analysis of the changes in EMG amplitude over time performed at each specific angle confirmed that EMG amplitude was not less than baseline following LIB at any angle. EMG/torque increased from set 1 to set 6 during both LIB (25%) and LRB (27%) with minimal change for SIB (3%) and SRB (1%) (set × muscle length $P < 0.01$; Fig. 4). EMG/torque was not different between LIB and LRB (bout effect $P = 0.31$) or between SIB and SRB (bout effect $P = 0.12$).

Discussion

The main finding in this study was that performing an initial bout of eccentric exercise at a shortened muscle length did not protect against strength loss and pain following a repeated bout at a longer muscle length. This is surprising since previous studies have demonstrated that a repeated eccentric bout of the same intensity and

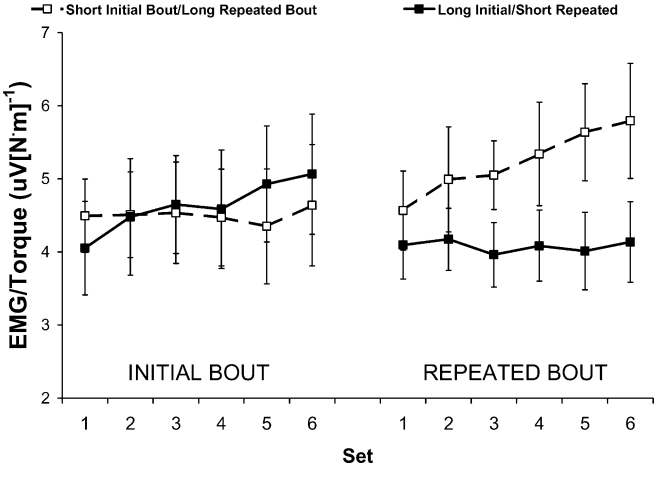

Fig. 4 EMG amplitude (RMS) averaged for the three muscles and expressed relative to knee extension torque (μV [N m]$^{-1}$). EMG/torque increased from set 1 to set 6 during both LIB and LRB with minimal change for short initial bout (SIB) and short repeated bout (SRB) (set×muscle length $P < 0.01$)

through the same range of motion as the initial bout results in less strength loss and pain compared to the initial bout (McHugh et al. 2001; McHugh and Tetro 2003). Other studies have demonstrated that as few as 10, 6, or even 2 maximal eccentric contractions of the elbow flexors are sufficient to confer a protective adaptation for a subsequent bout of 24 (Nosaka et al. 2001b) or 50 (Brown et al. 1997) maximal contractions. In these studies the initial bouts involving few contractions resulted in minimal symptoms of damage, as indicated by changes in strength, soreness and creatine kinase activity. Similarly in the present study the initial bout at a short muscle length (SIB) resulted in minimal strength loss or pain. Therefore the lack of a protective effect in the present study may be attributable to the difference in muscle length between bouts rather than the lack of symptoms following the initial bout.

It was hypothesized that an initial bout at a long muscle length would provide protection from a repeated bout at a shorter muscle length. There was some evidence with pain measurements that the initial bout performed at a long muscle length (LIB) provided some protection against damage from a repeated bout at a short length (SRB). However, this was not apparent with respect to the strength measurements. This apparent disparity in symptoms was probably due to the fact that the initial bout at a short muscle resulted in minimal symptoms (insignificant strength loss, significant but moderate pain). Therefore, it was not surprising that strength loss following SRB was not less than SIB. A larger sample size or a more injurious bout at a short muscle length (increase in the number of contractions or increase in contraction intensity) may be necessary to clearly demonstrate a protective effect due to an initial bout at a longer muscle length.

With the chosen study design it was not possible to determine if an initial bout performed from 70° to 110° of knee flexion would provide protection from a repeated bout performed in the same range. This would have required an additional group of subjects. Knee extension contractions performed at the same intensity as in the present study (90% isometric MVC) from full extension to 110° of knee flexion have been shown to provide protection for a similar repeated bout (McHugh and Tetro 2003). Similar effects have been demonstrated for repeated bouts of eccentric knee flexion contractions using a similar protocol (McHugh et al. 2001). Since both of these studies (McHugh et al. 2001; McHugh and Tetro 2003) showed a clear repeated bout effect when both bouts were performed through the same joint range of motion, it is likely that a repeated bout effect would have been evident in the present study if both the initial and repeated bouts were performed at a long muscle length. However, since the symptoms of muscle damage are increased with increased exercising muscle length, it would be necessary to demonstrate that the repeated bout effect occurs when both bouts are performed at a muscle length close to the end of the functional range for the given muscle group.

Various theories have been proposed to explain the repeated bout effect and these have been categorized as neural, mechanical and cellular (McHugh 2003). A cellular adaptation whereby there is serial addition of sarcomeres between the initial and repeated bouts has been supported indirectly in training studies in animals (Lynn and Morgan 1994; Lynn et al. 1998) and in changes in the angle-torque relationship in humans (Brockett et al. 2001; McHugh and Tetro 2003). An initial bout of eccentric quadriceps contractions (six sets of ten contractions) performed from full extension to 110° of knee flexion with a target intensity of 90% of isometric MVC (same as present study) resulted in a 7% decrease in knee extension torque at 30° and a 14% increase in torque at 110° when tested 2 weeks later (McHugh and Tetro 2003). This adaptation was associated with protection from strength loss and pain following a repeated eccentric bout at the same intensity and through the same range of motion. While shifts in the angle-torque relationship demonstrated by Brockett et al. (2001) and McHugh and Tetro (2003) are consistent with longitudinal addition of sarcomeres the results could also be explained by a decrease in pennation angle. However, given that animal data indicate an addition of sarcomeres following exposure to eccentric exercise (Lynn and Morgan 1994; Lynn et al. 1998) a change in pennation angle may be a less plausible explanation for the changes in the angle-torque relationship observed by Brockett et al. (2001) and McHugh and Tetro (2003). It should be noted that others have demonstrated no change in sarcomere number with eccentric training in rabbits (Koh and Herzog 1998).

In the present study there was no evidence of such a change in the angle-torque relationship following either initial bout (SIB/LRB: bout×angle $P = 0.75$, Fig. 5a; LIB/SRB: bout×angle $P = 0.28$, Fig. 5b). The fact that the initial bout performed at a short muscle length did not result in a change in the angle-torque relationship may be attributable to the limited sarcomere strain with eccentric contractions at a short muscle length. The fact that the angle-torque curve did not shift following SIB is consistent with this bout not providing any protection for a subsequent bout at a longer muscle length. A shift in the angle-torque relationship toward greater torque production at longer muscle lengths would have been expected following LIB but such an effect was not apparent. Two possible reasons are offered to explain this result. The first explanation is that it was not possible to demonstrate increased torque production at long muscle lengths following LIB since strength had not returned to baseline prior to SRB. A second explanation relates to the fact that greater strength loss was seen at longer muscle lengths following LIB, despite the fact that the opposite effect (greater strength loss at short muscle lengths) is typically seen (Saxton and Donnelly 1996; Child et al. 1998; McHugh and Tetro 2003). Therefore, the damage caused by performing eccentric contractions at the extremes of the functional range may not result in the typical protective adaptations. As stated

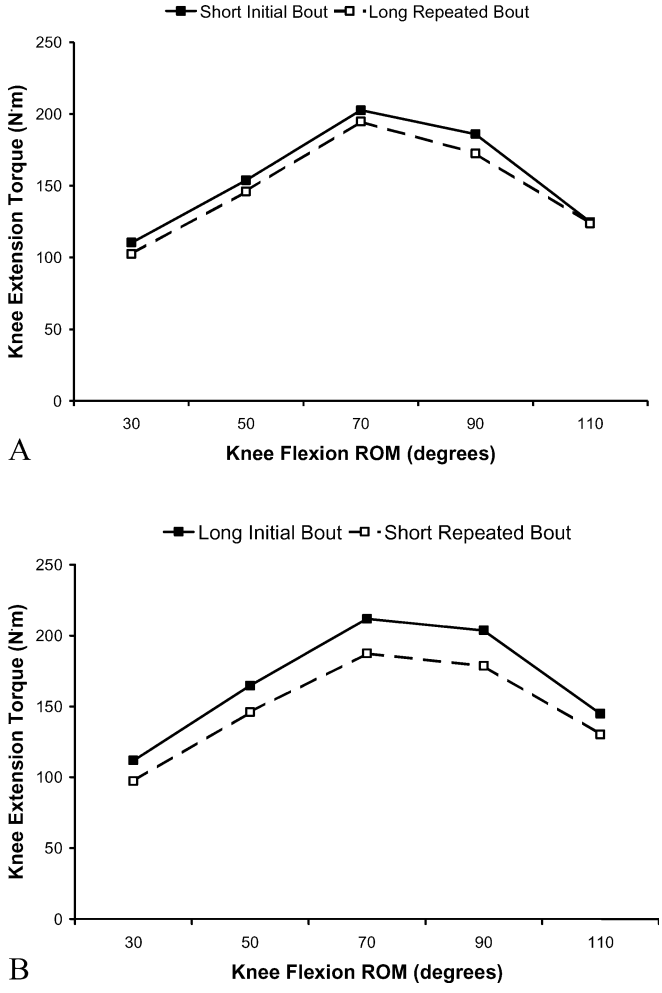

Fig. 5 Changes in the angle-torque relationship based on isometric knee extension measurements made prior to SIB versus LRB (**a**) and LIB versus SRB (**b**). There was no significant change in the angle torque curve for either condition (bout×angle $P = 0.75$ for SIB versus LRB and $P = 0.28$ for LIB versus SRB). Overall strength was significantly lower for SRB versus LIB (bout effect $P < 0.05$) indicating incomplete recovery

previously this possibility needs to be further investigated.

An immediate rightward shift in the length-tension curve following damaging eccentric contractions has been attributed intact sarcomeres adopting a shorter length for a given muscle length to accommodate the increased length of the strained disrupted sarcomeres (Wood et al. 1993; Jones et al. 1997; Whitehead et al. 2001). This effect results in greater relative strength loss at short versus long muscle lengths (Saxton and Donnelly 1996; Child et al. 1998; McHugh and Tetro 2003). For example, following eccentric quadriceps contractions performed from full extension to 110° strength loss was apparent at 30°, 50°, 70°, and 90° but not at 110° (McHugh and Tetro 2003). Surprisingly, and as stated previously, this was not the case in the present study, where strength loss following LIB was greater at longer muscle lengths. The EMG measurements during isometric MVCs indicated that this effect could not be explained by preferential inhibition at longer muscle length. It is possible that disruption of structures other than the sarcomere may have contributed to strength loss at longer muscle lengths. For example, excitation-contraction coupling failure contributes to strength loss following eccentric exercise (Warren et al. 2001) and this effect may have been more apparent at longer muscle lengths. However, further work is necessary to elucidate the mechanism(s) for this angle dependent effect.

The results of this study confirm previous findings demonstrating that the symptoms of muscle damage are highly dependent on exercising muscle length (Newham et al. 1988; Lieber and Fridén 1993; Brooks et al. 1995; Hunter and Faulkner 1997; Child et al. 1998). Strength loss (averaged across days and test angles) was 19% following the initial bout performed at the long muscle length, while it was only 6% following the initial bout performed at the short muscle length. However, it is notable that strength loss following LIB was highly variable; two subjects had no strength loss (7% and 5% above baseline), while one subject had 51% strength loss (averaged across all measurements following eccentric exercise). Increased susceptibility to damage at longer muscle lengths is thought to be due to decreased myofilament overlap and therefore greater sarcomere strain (Morgan 1990). In the present study the lower torque production at 110° compared with 70° during isometric MVCs prior to the initial eccentric bout was primarily due to the decrease in myofilament overlap with increasing muscle length. Therefore, subjects demonstrating a greater difference in torque production between 70° and 110° would be expected to experience greater sarcomere strain during eccentric contractions in this range and subsequently would be expected to experience greater symptoms of damage. In support of this notion, the difference in isometric torque production between 70° and 110° has been shown to be related to the subsequent strength loss following eccentric knee extension contractions performed through the full range of motion (McHugh and Tetro 2003). A similar effect was apparent in the present study. The relative difference in torque production between 70° and 110° (range 13–50% decrease from 70° to 110°) during isometric MVC testing prior to LIB was inversely correlated with strength (percentage of baseline) on the days following LIB ($r = -0.87$, $P < 0.001$; Fig. 6). The difference in torque production between 70° and 110° was not significantly related to pain averaged across the three days following LIB ($r = 0.57$, $P = 0.09$) but was positively correlated with pain on day 3 ($r = 0.73$, $P < 0.05$). While significant correlations with such a small sample size must be interpreted with caution, these findings indicate that susceptibility to muscle damage following eccentric contractions may be related to the length-tension relationship. Future research is required to determine if the high variability in symptoms of damage following an experimentally controlled eccentric bout are attributable to inter-individual differences in the length-tension curve for the muscle group being studied. In practical terms

this would imply that individuals with a greater operating range of motion for force production may be somewhat protected from eccentric contraction-induced damage.

Surface EMG signals were recorded from the quadriceps to assess differences in neural drive between eccentric bouts at different muscle lengths and between initial and repeated eccentric bouts. EMG values were averaged across the VL, VM and RF and expressed relative to torque. A limitation in this approach was that it did not account for potential differences in neural drive between the three muscles during eccentric exercise or between bouts. However, this provides a gross estimate of quadriceps activation relative to torque production. EMG/torque increased during both bouts performed at a long muscle length but did not change during either bout performed at a short muscle length (Fig. 4). This is consistent with previous findings during damaging eccentric contractions in the hamstrings (McHugh et al. 2000). Since EMG/torque only increased for the eccentric bouts that resulted in symptoms of damage (LIB and LRB) this effect may reflect increased motor unit activation to compensate for decreased force generating capacity secondary to myofibrillar disruption.

In conclusion, this was the first study to examine the role of muscle length in the protective adaptation to a single bout of eccentric exercise (repeated bout effect). The main finding was that performing an initial bout at a short muscle length did not protect that muscle group from symptoms of damage following a repeated bout at a longer muscle length. The practical significance of these results pertains to strategies aimed at reducing the negative effects of unfamiliar predominantly eccentric exercise. Preconditioning exercise bouts to avoid or limit subsequent exercise-induced muscle damage should not be performed in a range of motion that is less than the range of motion that the muscles will be exposed to in subsequent bouts. For example, a squat training session where the squats are performed to parallel will not protect against damage following a subsequent training session involving deeps squats. The muscle-length-dependent effects demonstrated here support the theory that muscle damage is due to sarcomere strain that results from reduced myofilament overlap during eccentric contractions. Furthermore, the lack of protection when the repeated bout was performed at a greater muscle length supports the notion that the repeated bout effect is dependent on a reduction in sarcomere strain during subsequent eccentric exercise.

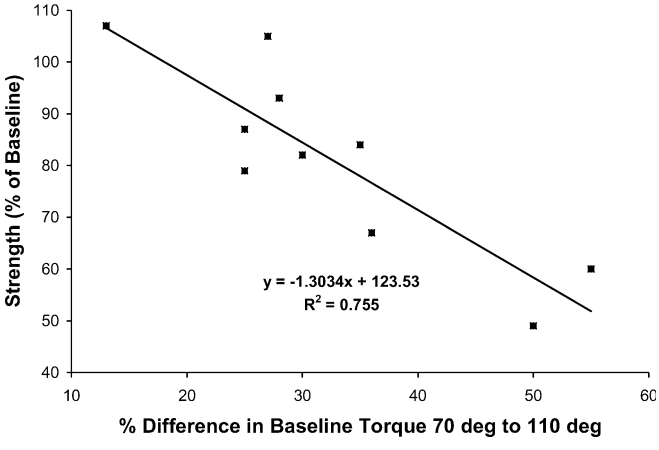

Fig. 6 Relationship between the percentage difference in knee extension torque between 70° and 110° during baseline isometric testing (pre-LIB) and strength loss following LIB (average of values from testing post-exercise, day 1, day 2 and day 3). Inverse linear relationship $r = -0.87$, $P < 0.001$

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