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## Systems Physiology: Musculo-skeletal

### REPEATED BOUT EFFECT CONFERRED BY DOWNHILL BACKWARD WALKING

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#### ABSTRACT

**Nottle C, Nosaka K.** Repeated Bout Effect Conferred By Downhill Backward Walking. *JEPonline* 2005;8(1):1-10. Downhill backward walking has been shown to induce muscle damage to the triceps surae muscles, however it is not known how the muscles respond to a repeated bout of the same exercise. This study compared the changes in indirect markers of muscle damage following an initial and secondary bout of downhill backward walking. Seventeen subjects completed two 60 min bouts of downhill (-15%) backward walking (30 – 35 strides/min) separated by two weeks. A two-way repeated measures ANOVA demonstrated significant ( $p < 0.05$ ) increases in soreness (bout 1:  $6.2 \pm 2.5$ ; bout 2:  $3.1 \pm 2.1$ ) and tenderness (bout 1:  $48.0 \pm 28.9$  kPa; bout 2 –  $57.6 \pm 30.5$  kPa) following both exercise bouts, however, the severity was significantly ( $p < 0.0083$ ) lower after bout two. Plasma creatine kinase activity increased significantly ( $p < 0.05$ ) following bout one only (peak:  $497.6 \pm 401.2$  IU/L). Significant decreases ( $p < 0.05$ ) in isometric strength occurred after both bouts (bout 1: ~20%; bout 2: ~18%), but the recovery was significantly ( $p < 0.0083$ ) faster after the second bout. Significant decreases ( $p < 0.05$ ) in isokinetic strength (~17%) were evident following the initial exercise bout only. The attenuated responses following the second bout indicate that the initial downhill backward walking bout conferred the repeated bout effect. It appears that eccentric loading to the muscles during downhill backward walking exceeded that of daily activity in volume and strain, and this generated the effect.

**Key Words:** Exercise-Induced Muscle Damage, Triceps Surae, Isometric Strength, Creatine Kinase, Muscle Soreness

## INTRODUCTION

Downhill backward walking has previously been demonstrated as a model to study exercise-induced muscle damage (EIMD) in triceps surae, and is shown to produce similar symptoms to those seen in other muscle groups following eccentric exercise (1). However, the magnitude of muscle damage induced by downhill backward walking is not as large as that observed following maximal eccentric exercise (2), but similar to that following submaximal eccentric exercise (3). Since downhill backward walking utilizes the leg muscles that perform repeated submaximal eccentric exercise during daily locomotion, it appears ideal for investigation of EIMD occurring in daily exercise or recreational sporting activities. Additionally, the use of a step-together action during downhill backward walking allows the exercise model to effectively isolate triceps surae of one limb only, making it advantageous to investigations where between limbs comparisons are desired.

It is well documented that a bout of eccentric exercise confers protection against EIMD, and this adaptation is referred to as the repeated bout effect (2,8,13). The repeated bout effect is produced when the same or similar eccentric exercise is repeated following an initial exercise bout (2), and results in substantially reduced, and in some instances, absent symptoms of delayed onset muscle soreness and plasma creatine kinase (CK) efflux, and a more rapid recovery from strength loss (3,4). While EIMD and the repeated bout effect are both widely studied and published phenomena, the elbow flexors and knee extensors are the most commonly examined muscles. The repeated bout effect has also typically been examined using maximal eccentric exercise models. As the magnitude of EIMD is reported to be dependent on the intensity and duration of the exercise (5), it may be that the degree of protection conferred is also dependent on the intensity and duration of the initial exercise bout. For this reason, it is possible that the repeated bout effect conferred by submaximal eccentric exercise may differ from that by maximal eccentric exercise.

There is evidence that during slow locomotion, type I motor units are preferentially recruited during lengthening contractions (6). Since the muscle contraction velocity during downhill backward walking appears to be slow, it is possible to assume that the recruited fibers are mostly slow twitch fibers. Therefore the repeated bout effect observed following the initial bout of downhill backward walking may differ from that seen using maximal eccentric actions which are likely to recruit type II motor units. As backward downhill walking more closely replicates the intensity of contraction performed routinely in daily activity, it seems reasonable to assume that conferral of protection may already exist through the loading of daily activities in triceps surae. However, no study has investigated the repeated bout effect of downhill backward walking. Therefore, the aim of this study was to compare changes in the common indicators of EIMD following an initial and repeated bout of downhill backward walking. It was hypothesized that the repeated bout effect might not be conferred by an initial bout of downhill backward walking because triceps surae was assumed to have already adapted to eccentric loading through daily activities.

## METHODS

### Subjects And Exercise Protocol

Seventeen subjects were recruited for the study and provided written informed consent following approval from the Edith Cowan University Human Research Ethics Committee in line with the Ethics of the World Medical Association. During the experimental period, subjects were requested not to take any medication, change their diet, or perform any strenuous activity. Subjects who had participated in resistance training of the lower limbs in the past 12 months were excluded from the study.

Criterion measures were tested on 13 occasions over a 26-day period with an exercise protocol performed twice by each subject. Criterion testing was conducted 72 and 48 hours prior to downhill backward walking (baseline), and 0.5, 24, 48, 72 and 96 hours following the walk for the initial exercise bout (bout 1). For the repeated bout, measurements were taken 48 hours prior to, and 0.5, 24, 48, 72 and 96 hours post-walk (bout 2). A period of 7 days elapsed between the completion of testing for bout 1 and baseline testing for commencement of bout 2. This period was chosen to allow for recovery of all measures back to baseline levels prior to the second bout based the recovery time period assessed previously (1).

The exercise protocol consisted of 60 min of downhill backwards walking with a single limb stepping action performed on a Trackmaster (TM500) motor driven treadmill (JAS manufacturing, TX, USA). The treadmill was modified to allow the belt to rotate in reverse, and the grade was set at -15%. Subjects stepped backward with a toe-to-heel action with the left limb (exercised limb), with the right limb (non-exercised limb) then brought together with the left. Treadmill speed was determined during the initial exercise bout at the individualized pace of 30-35 strides/min, with an identical speed then used during the repeated bout. All testing was performed on both limbs, however no comparisons between limbs were conducted.

### **Criterion Measures**

Using a Cybex 6000 isokinetic dynamometer (Cybex, NY, USA), maximal voluntary isometric and isokinetic strength of the ankle plantar flexors was determined with the subject in a reclined position and the knee secured at 90° flexion. Isometric torque was measured for 5 s at a neutral (0°), dorsi flexed (-10°), and plantar flexed (10°) foot position. Three maximal efforts at each angle were performed with twenty s rest between each effort, and 60 s rest between each testing angle. The peak torque produced at each angle was recorded for further analysis. Maximal voluntary isokinetic strength was determined at the angular velocities of 30, 60 and 120 °/s throughout a full range of the subjects' ankle movement. Subjects performed three maximal repetitions with a rest interval of 60 s between each testing velocity, from which the peak torque was recorded. Plasma creatine kinase (CK) activity was determined from a 30 µL finger-prick blood sample pipetted onto a CK test strip (Boehringer-Mannheim, Indiana, USA) and analysed using a Reflotron (Boehringer-Mannheim, Indiana, USA). Muscle soreness and tenderness was determined at 5 sites over triceps surae, with the average of the 5 sites used for further analysis. Two proximal sites were marked 5 cm below the popliteal fossa over the belly of the medial and lateral gastrocnemius. Two distal sites were marked 5 cm above the insertion of medial and lateral heads of gastrocnemius over the belly of the muscle. One soleus site was marked 5 cm below the insertion of the medial and lateral heads of gastrocnemius along the midline of the limb. Muscle soreness was quantified using a 1 (normal) to 10 (very, very sore) scale, with subjects asked to report the magnitude of soreness while walking on a flat, stable surface. Muscle tenderness was determined using a myometer (Dobros) with a 1.5 cm rubber tip. The myometer was applied at the five marked sites with increasing pressure (to a ceiling value of 100 kPa [14.5 psi]), with subjects asked to report the moment pain was perceived. Thus the pressure required to elicit pain was recorded as tenderness so that an increase in tenderness is represented by a decrease in pressure.

### **Data Analyses**

Each dependent variable was analysed using a two-way repeated measures ANOVA (time x bouts), with separate analysis being conducted for each limb (with the exception of plasma CK). Where a significant p value ( $p < 0.05$ ) was obtained, simple contrasts to baseline were conducted to determine those time intervals that were significantly different from baseline for each individual bout. To determine any significant differences between bouts, paired sample t-tests with a Bonferroni adjusted significance level ( $p < 0.0083$ ) were conducted between time intervals for the two exercise bouts. All results are reported as the mean ± SD.

## RESULTS

The mean age, height and weight of the recruited subjects was  $26.6 \pm 6.6$  years,  $169.6 \pm 9.0$  cm,  $72.2 \pm 14.3$  kg, respectively.

### Maximal Voluntary Strength

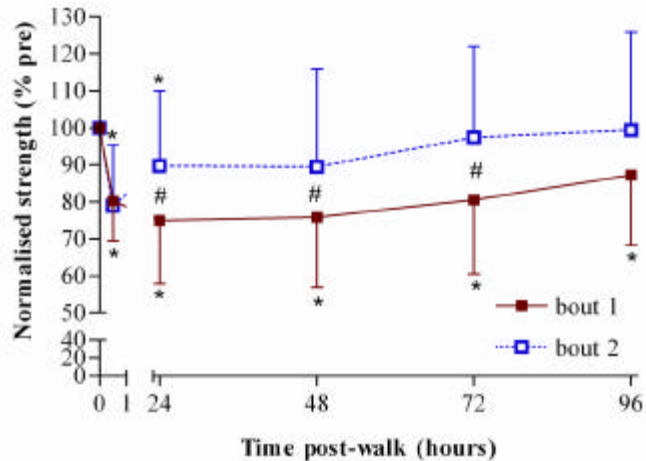
For the non-exercised limb, no significant changes ( $p > 0.05$ ) from baseline were observed for any strength variable across time. Additionally, no significant differences ( $p > 0.05$ ) between bouts were evident for any strength measure. For the exercised limb, significant ( $p < 0.05$ ) decreases from baseline were observed at all time intervals following the initial bout for maximal voluntary isometric torque at an angle of  $10^\circ$  dorsiflexion (Figure 1). Similar changes were observed for the ankle angles of  $0^\circ$  (neutral) and  $10^\circ$  plantar flexion, however significant reductions from baseline for these angles were recorded only at 0.5 and 24 hours post-walk (Table 1). No significant difference in the pre-exercise maximal voluntary isometric torque was evident between the first and second bout. Following the second bout, a significant difference ( $p < 0.05$ ) from baseline was recorded only at 0.5 and 24 hours post-walk for all testing angles. Additionally, the magnitude of decrease in isometric strength was significantly ( $p < 0.05$ ) smaller for the second bout compared to the initial bout when the foot was in either the plantar flexed (Table 1) or dorsi flexed (Figure 1) position.

The normalized changes in maximal voluntary isokinetic torque are shown in

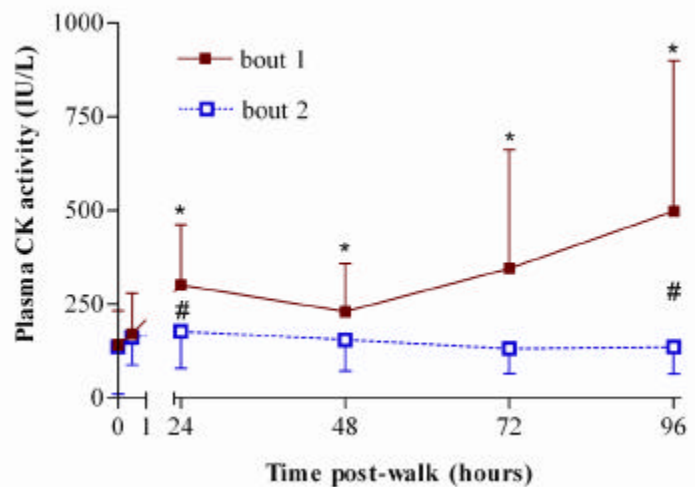
Table 1. The magnitudes of changes in isokinetic torque were similar to that the isometric torque after the initial bout (Table 1 and Figure 1). Significant decrease in isokinetic torque were found following the initial exercise bout, however no significant decreases were seen for any of the isokinetic velocities following the second bout. Although there was a trend showing attenuated responses following bout 2 compared to bout 1, no significant difference between the two bouts was observed for isokinetic strength.

### Plasma CK activity

Significant increases in plasma CK activity from baseline were evident following the initial exercise bout at all time intervals 24 to 96 hours post-walk (Figure 2). Plasma CK activity did not increase



**Figure 1.** Changes in maximal voluntary isometric torque At  $10^\circ$  dorsiflexion from pre-exercise level (100%) 0.5, 24, 48, 72, and 96 hours following the first (bout 1) and second (bout 2) downhill backward walking exercise. (\*  $p < 0.05$  difference to baseline; #  $p < 0.0083$  difference between bouts).



**Figure 2.** Plasma CK activity before (0) and 0.5, 24, 48, 72, and 96 hours following the first (bout 1) and second (bout 2) downhill backward walking exercise. (\*  $p < 0.05$  difference to baseline; #  $p < 0.0083$  difference between bouts).

significantly following the second exercise bout. The reduced responses in the second exercise bout resulting in a significant difference ( $p < 0.0083$ ) between the bouts at 24 and 96 hours post-walk.

**Table 1. Changes (% baseline) in maximal voluntary isometric and isokinetic strength (mean  $\pm$  SD) 0.5, 24, 48, 72 and 96 hours following the first (1) and second bout (2) of downhill backward walking (\*  $p < 0.05$  significantly different from baseline; #  $p < 0.0083$  significantly different between bouts)**

		<i>Time Post-walk (hrs)</i>				
	<b>Bout</b>	<b>0.5</b>	<b>24</b>	<b>48</b>	<b>72</b>	<b>96</b>
<b>0° neutral (isometric)</b>	<b>1</b>	81.5 $\pm$ 14.4*	79.1 $\pm$ 22.3*	82.1 $\pm$ 23.3	85.6 $\pm$ 25.1	90.4 $\pm$ 19.7
	<b>2</b>	80.5 $\pm$ 14.9*	88.7 $\pm$ 19.7*	89.4 $\pm$ 27.8	96.2 $\pm$ 25.9	95.8 $\pm$ 26.5
<b>10° plantarflexion (isometric)</b>	<b>1</b>	78.4 $\pm$ 15.2*	75.0 $\pm$ 19.6*	79.5 $\pm$ 22.9	84.5 $\pm$ 25.4	94.5 $\pm$ 18.5
	<b>2</b>	84.2 $\pm$ 13.3*	90.2 $\pm$ 18.6*#	96.0 $\pm$ 27.6#	103.4 $\pm$ 27.2	100.4 $\pm$ 19.8
<b>30°/sec (isokinetic)</b>	<b>1</b>	83.5 $\pm$ 20.6*	81.1 $\pm$ 26.2*	80.2 $\pm$ 33.2*	83.7 $\pm$ 32.1	87.3 $\pm$ 28.4
	<b>2</b>	91.7 $\pm$ 24.0	88.3 $\pm$ 18.6	96.6 $\pm$ 130.6	101.4 $\pm$ 36.8	102.5 $\pm$ 33.4
<b>60°/sec (isokinetic)</b>	<b>1</b>	81.9 $\pm$ 12.8*	77.2 $\pm$ 22.6*	75.6 $\pm$ 28.6*	86.0 $\pm$ 28.5	96.7 $\pm$ 27.6
	<b>2</b>	89.4 $\pm$ 20.5	90.3 $\pm$ 20.2	89.8 $\pm$ 29.3	101.0 $\pm$ 28.3	97.8 $\pm$ 30.7
<b>120°/sec (isokinetic)</b>	<b>1</b>	84.9 $\pm$ 22.7*	82.8 $\pm$ 21.0*	79.7 $\pm$ 26.7	86.4 $\pm$ 27.3	96.1 $\pm$ 30.0
	<b>2</b>	88.4 $\pm$ 19.9	90.1 $\pm$ 17.3	88.4 $\pm$ 27.1	95.2 $\pm$ 24.8	97.6 $\pm$ 32.9

### Soreness and Tenderness

Following both exercise bouts significant increases ( $p < 0.001$ ) in soreness were recorded at all time intervals 24 to 96 hours for the exercised limb, with peak soreness scores of 6.2  $\pm$  2.5 (bout 1) and 3.1  $\pm$  2.1 (bout 2) being recorded 48 hours post-walk (Figure 3). Compared to the first bout, the magnitude of muscle soreness was significantly ( $p < 0.01$ ) ( $p < 0.0083$ ) smaller following the second bout, being approximately 2 points lower than those following the initial bout. No significant ( $p > 0.05$ ) increases in soreness were observed for the non-exercised limb at any time interval.

For the exercised limb, significant increases in tenderness were recorded following both the initial ( $p < 0.001$ ) and the repeated ( $p < 0.01$ ) exercise bouts with peak tenderness readings of 48.0  $\pm$  7.0 48.0  $\pm$  28.9 kPa and 57.6  $\pm$  7.4 57.6  $\pm$  30.5 kPa recorded 48 hours post-walk respectively (Figure 4). Although tenderness scores tended to be lower on the second bout, the differences between bouts was only significant ( $p < 0.0083$ ) at the final 96 hour post-walk time interval. No significant ( $p > 0.05$ ) changes in tenderness were reported following either exercise bout for the non-exercised limb.

### DISCUSSION

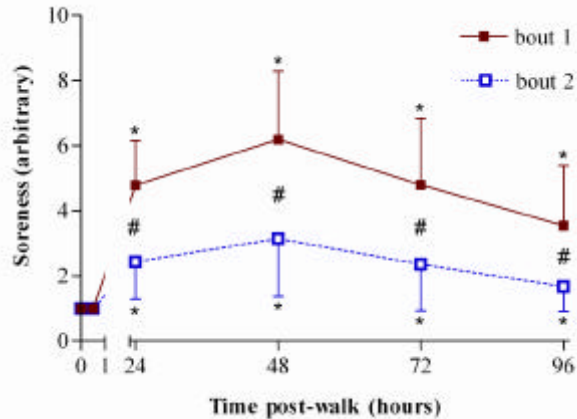
The present study showed significantly smaller decreases and faster recovery of maximal voluntary strength (Figure 1, Table 1), smaller increases in plasma CK activity (Figure 2), and attenuated development of DOMS (Figure 3) and tenderness (Figure 4) following the second bout of downhill backward walking compared to the initial bout. The attenuated changes in the measures following the second bout seem to be conferred by the first bout of downhill backward walking, and are consistent with the repeated bout effect shown in previous studies utilizing other muscle groups such

as elbow flexors (2) and knee extensors (3). This is the first study to demonstrate the repeated bout effect for triceps surae. It was hypothesized that the repeated bout effect might not be conferred by an initial bout of downhill backward walking because triceps surae was assumed to have already adapted to eccentric loading through daily activities. Yet the muscle damage observed following the initial bout suggested that no protection had been conferred. As a result of this, it was questioned whether triceps surae would demonstrate the repeated bout effect similar to that which has been shown in other muscle groups. Despite previous work, it could not be assumed that triceps surae would respond the same to repeated eccentric loading, particularly given that the exact stimulus for the repeated bout effect is unknown. However, the results of the present study suggest that triceps surae are not the exception.

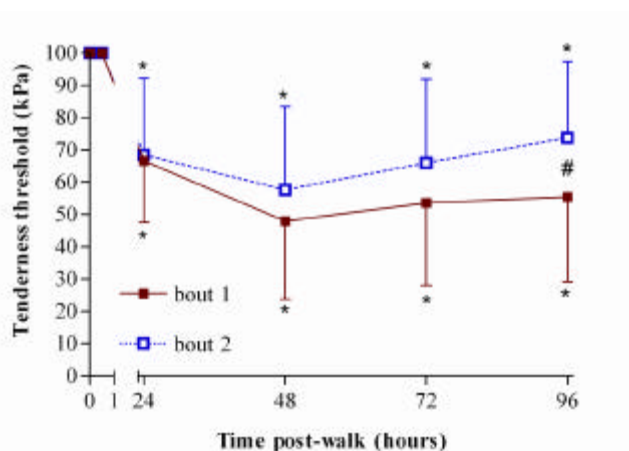
Neural, mechanical, and cellular adaptations have been proposed for the repeated bout effect (4), however debate still exists with regards to the primary causal mechanism. It should be noted that decreases in maximal voluntary isometric strength from baseline occurred following both exercise bouts, and the magnitude of strength loss immediately after exercise was similar between the bouts (Figure 1). Some studies have reported an attenuated strength loss immediately after the second exercise bout compared to the initial bout (3,7).

Warren et al. (7) explained that the smaller strength loss immediately after eccentric exercise may be due to a neural adaptation in which an increased activation of slow motor units and a concomitant decrease in activation of fast units occurs. It has been speculated that more efficient recruitment of motor units reduces the stress on some muscle fibers that are susceptible to muscle damage (3). However, the similar strength loss between the bouts in the present study does not appear to suggest that this is the case for triceps surae when performing backward downhill walking.

It is important to note that the first bout of downhill backward walking induced considerable muscle damage as indicated by prolonged strength loss (Figure 1 & Table 1), increases in plasma CK activity (Figure 2), and development of DOMS (Figure 3). This suggests that the eccentric muscle actions during downhill backward walking exceeded the level of daily activities to which muscle fibers were normally accustomed. As the repeated bout effect has been demonstrated to occur following only a small number of eccentric actions (8), it was surprising that no protection had been conferred by the



**Figure 3. Muscle soreness before (0), and 24, 48, 72, and 96 hours following the first (bout 1) and second (bout 2) downhill backward walking exercise. (\*  $p < 0.05$  difference to baseline; #  $p < 0.0083$  difference between bouts).**



**Figure 4. Muscle tenderness before (0), and 24, 48, 72, and 96 hours following the first (bout 1) and second (bout 2) downhill backward walking exercise. (\*  $p < 0.05$  difference to baseline; #  $p < 0.0083$  difference between bouts)**

eccentric actions performed during daily activities. Although the actual lengthening velocity of the eccentric actions during downhill backward walking was not measured in the present study, the velocity did not appear to be fast compared to the velocity in other activities of the triceps surae such as running or jumping. Also, as subjects were walking with a controlled stepping action, it could be assumed that the magnitude of load on the muscle did not exceed that which occurs during running or jumping (9). Thus it would appear that muscle strain during the volume of contractions (1800 – 2300 contractions) was the main stimulus in inducing the muscle damage following the initial exercise bout, because the toe-to-heel stepping action during downhill backward walking was likely to exceed the range of motion of the joint in normal locomotion.

As it has been shown that slow motor units are preferentially recruited during slow lengthening actions (6), it seems reasonable to assume that most of the damaged fibers from the downhill backward walking were slow twitch fibres. It is possible that some slow twitch muscle fibers have adapted to the eccentric load during the downhill backward walking, but others have not. Furthermore, it has been shown that eccentric muscle actions at a long muscle length induce greater magnitude of muscle damage than at a short muscle length (10). It may be that the toe-to-heel actions during stepping back stretched the muscle more than usual and induced muscle damage to a population of slow twitch fibers that would generally be resistant to eccentric exercise-induced muscle damage.

Fatigue, excitation-contraction [E-C] coupling failure and physical disruption of muscle fibres are reported to account for strength loss immediately after exercise, while loss of contractile protein is reported to prolong strength loss (11). Since no recovery of strength was evident from 0.5 to 24 hours after the first bout (Figure 1, Table 1), it may be that E-C coupling failure and physical disruption rather than fatigue were responsible for the strength loss observed using this exercise model. The faster recovery of strength from 0.5 to 24 hours after the second exercise bout would suggest a more rapid recovery of E-C coupling failure and repair to the force bearing elements within the muscle. It is possible that this faster repair of physical disruption reduces the degree of contractile protein loss and allows for the significantly faster rate of recovery of strength following the second bout (Figure 1) as has previously been demonstrated in other muscle groups (2,8).

It has been suggested that an adaptive response during the repair process from the initial bout of damage may result in structural re-organization allowing the muscle fibers to become more resistive to damage in the repeated bouts. The proposed remodelling includes removal of weakened sarcomeres, strengthening of the cell membrane, and longitudinal addition of sarcomeres (12,13). Results from human models have shown that the further beyond resting length the muscle is strained, the greater the symptoms of muscle damage (3). The stretching caused by such contractions resulting in “popping” of the sarcomeres within the muscle, which causes disruption to the titin filament, the anchor point of the myosin filament to the Z-disc (14). This disruption to the filament inhibits the protein interaction necessary for force generation between sarcomeres and therefore limits force generation of the affected muscle (15). The disruption to sarcomeres has also been postulated to account for observations of a shift in optimum angle following eccentric exercise (16). Following this ‘popping’, remodelling of the sarcomeres and other intermediate filaments within the muscle may protect the muscle against the repeated bout of eccentric exercise.

The extent to which sarcomere remodelling is responsible for the repeated bout effect however is unclear, because previous investigations examined optimum angle changes following only a single exercise bout, and showed that the changes lasted only two days post-exercise (12,17). As optimum angle is said to shift to the right after eccentric exercise, meaning that maximal force generation occurs at a greater joint angle (12), it could be assumed that any adaptation or remodelling within the

exercised muscle as a result of this shift would occur at the longer muscle length. In the present study however, a difference between bouts was observed when the foot was at both a dorsi-flexed (long) and plantar-flexed (short) position, with no difference between bouts at the neutral angle. It seems unlikely that this observation would have occurred if sarcomere remodelling or a shift in optimum angle were responsible for the adaptation, with protection at either a shortened or lengthen, not both more reasonable. This assumption is based on previous adaptations demonstrated following eccentric exercise where a period of pre-exercise concentric training had been performed (18,19). To fully understand the degree to which sarcomere remodelling and a shift in optimum angle contribute to the repeated bout effect in terms of strength loss, it would therefore be necessary to specifically examine this variable following repeated eccentric exercise.

In accordance with previous investigations (20), a significant reduction in plasma CK efflux was recorded following the repeat bout of exercise compared with the initial bout (Figure 2). As the increase in enzyme activity following exercise is generally thought to reflect the existence of muscle membrane damage (5), the large plasma CK activity after the first exercise bout suggest some muscle fibers became necrotic. The results of previous studies that directly quantified the histological damage following repeated eccentric exercise bouts support this idea (21). It is likely that the smaller CK response reflects a reduced severity of muscle damage and cellular disruption following the repeated exercise in comparison to the initial exercise bout (22).

The present study also demonstrated attenuated responses in DOMS and tenderness of triceps surae after the second exercise bout. This is consistent with a number of studies that also evaluated soreness during active movements such as walking (3,23). Throughout the literature, little explanation is given as to why an attenuation of soreness and/or tenderness occurs with a repeated exercise bout. Neural adaptation is a frequently proposed mechanism in relation to 'protection' of strength losses with an improved ability to repair injury and a decreased inflammatory response cited as possible mechanisms for a reduction in soreness (4). This is supported by the faster recovery of strength and smaller increases in plasma CK activity after the second bout compared to the first bout. It seems reasonable to assume that the attenuated soreness and tenderness reflect the smaller magnitude of muscle degeneration and inflammation in the second bout.

The exercise protocol used in the current investigation was selected as a model for examining the magnitude of muscle damage that would be likely to occur during daily activity. The degree of muscle damage however would suggest that the exercise was still 'novel'. Having said this, the downhill backward walking exercise model is easily adaptable and further investigations altering either the treadmill grade, the walking time or speed, may yield different results. Given that the repeated bout effect appears to be a consistent feature of various skeletal muscles, examining the degree of protection conferred by daily activity may help in the understanding of the stimuli responsible for the adaptation.

## **CONCLUSIONS**

In conclusion, the repeated bout of the downhill backward walking resulted in less indication of muscle damage compared to the initial bout. Since the initial bout of downhill backward walking induced changes in all criterion measures, it seems reasonable to assume that the eccentric actions during the walking exceeds that in daily activities. It would appear that the damaged fibers in the first bout adapted to the eccentric actions, thus muscle damage was attenuated after the second bout. The repeated bout effect demonstrated in the present investigation further validates the downhill backward walking as an effective model for exercise-induced muscle damage in triceps surae, and supports the eccentric training potential of backward walking.

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