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Effect of stride length on symptoms of exercise-induced muscle damage during a repeated bout of downhill running

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The purpose of this study was to assess the effects of changes in stride length on the symptoms of exercise-induced muscle damage (EIMD) during a repeated bout of downhill running in a group of 18 men and women. Muscle tenderness, plasma creatine kinase activity (CK) and maximal voluntary isometric force were measured before and after two downhill runs, with each run separated by 5 weeks. The first downhill run was at the preferred stride frequency (PSF). Participants were then randomly allocated to one of three sex-balanced groups with equal numbers of men and women: overstride (−8% PSF), understride (+8% PSF) and normal stride frequency for the second downhill run. Stride length had no effect ($P>0.05$) on muscle tenderness, CK or isometric peak force. Increases in muscle tenderness ($P<0.001$) and CK were lower ($P<0.05$) following the second downhill run, although there was no difference in the pattern and extent of the strength decrement between the two runs. There were also no differences ($P>0.05$) in muscle tenderness, CK or the relative strength loss between the men and the women. Results suggest that the symptoms of EIMD are unaffected by gender and small alterations to the normal stride pattern during constant velocity downhill running. The observation that muscle tenderness and CK were reduced following a repeated bout of similar eccentric exercise is consistent with the phenomenon known as the 'repeated bout effect' of muscle damage.

In North Wales and other mountainous areas, orienteering and fell running are common recreational and competitive activities. Apart from the obvious energy demands of running up and down steep hills for long periods of time, there are considerable mechanical stresses placed on the muscles in the hip and lower limb, particularly as a result of the repeated eccentric contractions necessary for controlling the descent on steep gradients. The phenomenon of delayed-onset muscle soreness (DOMS) resulting from damage to the muscle and/or the connective tissue is familiar to recreational and competitive fell runners alike. Damage of this type features disruption of the sarcolemma, fragmentation of the sarcoplasmic reticulum, lesions of the plasma membrane, cytoskeletal damage, swollen mitochondria, disruption of sarcomeres, disorganised myofilaments and Z-line streaming (Newham et al. 1983, Fridén & Leiber 1992).

Eccentric contractions can generate forces that are greater than isometric contractions and, depending on the velocity of contraction, several times more than concentric contractions. This feature has been causally linked to the greater soreness and damage arising from eccentric relative to concentric contractions (Newham et al. 1983, 1983a, Fridén et al. 1983). It is also well established that for a given force production, there is less motor unit recruitment for eccentric versus concentric contractions (Bigland & Lippold 1954, Motitani et al. 1988). This higher force-to-activation ratio places a high stress on the tissues involved and is considered to be a primary factor in the structural damage of the muscle fibre (Armstrong 1984, Enoka 1996).

The degree of damage also appears to be linked to the joint position or muscle length at which the eccentric contraction occurs. Eccentric or isometric exercise performed at long muscle lengths results in greater soreness and damage than the same exercise performed at short muscle lengths (Jones et al. 1989, Child et al. 1998). Conversely, after damaging exercise, the greatest decrement in force production is observed when the muscle is tested at a short compared to a long muscle length (Child et al. 1998, Saxton & Donnelly 1996).

Downhill running or walking have frequently been used to study DOMS due to the reliance on eccentric contractions in the hip and knee extensors (Byrnes et al. 1985, Schwane et al. 1983, Maughan et al. 1989, Eston et al. 1995, 1996, 1996a). In this activity, the extensors contract eccentrically over a greater range

of motion and at longer lengths than during level or uphill running (Eston et al. 1995, Baltzopoulos et al. 1996). One of the dilemmas facing the fell runner is what stride strategy to adopt during the descent. Both the fell runner and orienteer, because of the type of competition terrain and other environmental demands, appear to make a trade-off between a short and a long stride strategy. Shorter strides favour balance and posture, but increase the frequency of impact and eccentric braking necessary. Intuitively, a longer stride pattern has the advantage of increased speed on descent, although this may be at the expense of postural control. With longer strides there would be greater eccentric tension in the knee extensors caused by the body's increased acceleration due to gravitational forces. Thus, although the frequency of the eccentric contractions of the knee and hip extensors would be reduced with a longer stride pattern, it might be expected that longer stride lengths may incur greater damage to the knee extensors as a result of the increased knee flexion and consequent knee extensor length upon impact of the leading leg.

The primary purpose of this study, therefore, was to assess the effects of changes in stride length during downhill running at a constant velocity on the symptoms of exercise-induced muscle damage. As it would be expected that fell runners, orienteers and other runners who regularly include hill running in their training would already have a degree of protection against muscle damage from prior bouts of downhill running, we considered that it would be more ecologically valid if participants were exposed to a bout of downhill running prior to a repeated bout in which stride length was manipulated. The study therefore involved a prior bout of downhill running in all participants at their preferred stride pattern, and a subsequent bout in which stride length was manipulated. To confirm whether a protective effect was induced by the prior bout, this factor was included in the analysis. A subsidiary purpose of the study was to examine whether gender influenced the symptoms of muscle damage after downhill running.

Methods

Participants

Eighteen physically active volunteers (9 men, age (mean±SD) 20.4±3.0 y, height 1.82±0.06 m, mass 76.4±6.0 kg and 9 women, age 19.5±1.1 y, height 1.68±0.05 m, mass 64.8±4.4 kg) gave informed consent to participate in the study. The study was approved by the Ethics Committee of the School of Sport, Health and Exercise Sciences.

Procedures

Baseline measures of muscle tenderness, isometric strength and plasma creatine kinase activity were assessed prior to and after each of two downhill running protocols, with the runs separated by a period of at least five weeks. After the first downhill run, participants were randomly allocated to one of three groups (overstride, understride and preferred stride). Each group had a balanced sex composition.

Creatine kinase activity (CK)

Plasma CK activity was determined from a fingertip blood sample. After a warm fingertip was cleaned with a sterile alcohol swab and allowed to dry, capillary puncture was made with an Autoclix lancette and a sample of whole fresh blood (32 μl) was collected into a heparinised capillary tube. The blood was then pipetted onto a test strip and analysed for CK activity via a colorometric assay procedure (Reflotron, Boehringer Mannheim, Lewes, UK). This system uses a plasma separation principle which is incorporated in the reagent carrier on the test strip. Due to large intersubject variability in CK, these values were subjected to logarithmic transformation. This reduced the heteroscedastic characteristics of the data and enabled our data to satisfy the homogeneity of variance assumption necessary for analysis of variance. This procedure has been used in previous investigations (Brown et al. 1997, Eston & Peters 1999) which have measured CK activity after damaging exercise.

Maximal voluntary isometric force

After a brief warm-up and practice of two to three maximal voluntary contractions against the lever arm of the isokinetic dynamometer (Kinetic Communicator, Chattanooga, Bicester, UK), participants performed two maximal voluntary isometric contractions of the knee extensors in the sitting position with the knee flexed at an angle of 110°. This angle was determined from a joint angle of 90°, as measured by goniometry, and entered into the dynamometer as a reference value. Force was then measured at 110° relative to this point. The axis of rotation of the dynamometer was aligned with the most prominent point of the lateral femoral condyle. To prevent extraneous movement during the maximal contractions, the thigh, hips and chest were secured to the chair with strapping. All participants were given standardised instructions to exert as great a force as possible against the lever arm of the dynamometer, and the force produced was displayed on the computer monitor in real time for feedback and motivation purposes (Baltzopoulos et al. 1991). The average maximal force produced by two trials was recorded.

Muscle tenderness

Muscle tenderness was measured at three sites on the anterior thigh with a computer-mediated algometer (Biokinetics, Bangor, UK) (Edwards et al. 1996). This technique allowed the investigator to standardise the rate of force application at a rate of $12\ N \cdot s^{-1}$. This was plotted as force versus time during the trial as a guide for the operator. The operator tracks the guide with a plot of the actual force generated at the myometer head. Site contact was made via a cylindrical metal probe with a flat head diameter of 17 mm, whilst participants lay in a supine position. The algometer was applied at the proximal, mid and distal muscle sites following the line of the rectus femoris. Participants held a report button and were given a standard instruction to press the button when the sensation changed from discomfort to pain, i.e., at the lowest stimulus value. The force being applied at that instant was immediately recorded by the software and removed. This force (F) was subtracted from a ceiling value of 80 N, which was used as the cut-off point to indicate zero pain. Hence, tenderness (T) was calculated by the following: $80 - F = T$. A high score for tenderness was therefore recorded if the stimulus force which elicited pain was low. Whilst the method is similar to that first described by Newham et al. (1983a), and has been used successfully in previous studies from

this laboratory (Eston et al. 1996, 1996a, Eston & Peters 1999, Baker et al. 1997), the threshold value is higher in the current study, as we have observed that the limit of pressure-pain tolerance in female participants using the same algometer head was approximately 80 N in undamaged thigh muscle (Baker et al. 1997). Tenderness values from each of the three sites were summed for the analysis. The mean score of 3 trials was recorded as the participant's tenderness score.

Downhill running protocols

After the pre-test measurements described above, all participants performed two downhill runs at −12% gradient at a velocity of 188 $m \cdot min^{-1}$ (7 mph). For each run, the protocol was 5×8 min bouts separated by a 2-min recovery period of walking on a level surface. Muscle strength was reassessed immediately post, and 24 h (day 1), 72 h (day 3) and 120 h (day 5) following both runs. Creatine kinase activity and muscle tenderness measurements were also repeated on days 1, 3 and 5. The first downhill run was performed at the preferred stride frequency (PSF) for each participant. For the second downhill run, performed at least five weeks later, participants were randomly allocated to three groups: overstride, understride and normal stride pattern. Stride frequency, expressed as a proportion of the preferred stride frequency, was manipulated to elicit a shorter stride length in the understride group (PSF +8%) and a longer stride length in the overstride group (PSF −8%). In the experimental conditions, cadence was regulated for each participant using a metronome. The rationale for the specified cadence was based on prior observations of approximate changes in stride length when participants were instructed to under stride and over stride for an 8-min practice period. This stride frequency was therefore considered to be the variation in stride pattern that would occur functionally during downhill running.

Analysis of data

Data were analysed by a series of two-factor mixed model analyses of variance to explore the effects of bout, stride length, sex and time on strength, CK and muscle tenderness according to the hypothesis tested. The repeated bout effect was analysed by a bout×time ANOVA. The effects of changes in stride length were assessed by a stride length×time ANOVA on bout two data only as this was the bout in which stride length was manipulated. Gender differences were analysed by a sex×time ANOVA on bout one data only during which all participants ran at their preferred stride length. Homogeneity of covariance assumptions were tested by the Mauchly Test of Sphericity, and where necessary the critical value of F was increased according to the Greenhouse Geisser Epsilon value. This is indicated by (GG) where appropriate. Statistical significance was set at the 0.05 alpha level. Where appropriate, Scheffé post hoc tests were used to determine the location of mean differences. Data are presented as means±SEM.

Results

Creatine kinase

Changes in CK following each of the runs are shown in Fig. 1. The overall mean CK value was lower after the second downhill run, irrespective of stride pattern ($F_{1,17df}=4.75$, $P<0.05$). CK activity changed significantly over time ($F_{(GG)2,26df}=36.9$, $P<0.001$), peaking on day 1, and although it decreased significantly over the next 48 h, on day 3 the plasma CK remained higher than the pre-test value. There was also a significant interaction of bout and time on CK

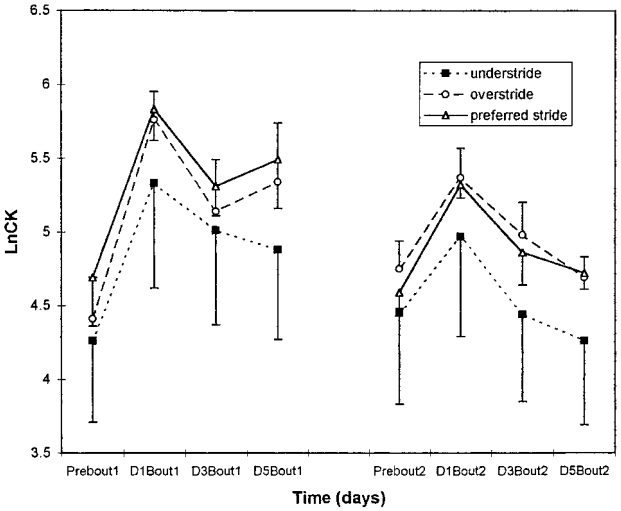

Fig. 1. Changes in plasma creatine kinase activity in 18 young men and women following an initial bout of running downhill at preferred stride length (—△—) and following a secondary bout of downhill running during which the stride length was altered by −8% (understride, ---■---), +8% (overstride, ---○---) or maintained at preferred length (—△—). Values are mean±SEM.

($F_{(GG)2,33df}=6.31$, $P<0.01$). The respective increases in CK in bout two for days 1, 3 and 5 were lower than in the first bout. The effects of stride manipulation on CK in bout two were non significant ($F_{2,15df}=0.41$, NS) with no interaction of group and time ($F_{(GG)4,33df}=0.46$, NS). There were also no differences in CK between men and women after the preferred stride length protocol ($F_{1,34df}=0.94$, NS) and no interaction of sex by time ($F_{3,102df}=0.13$, NS).

Maximal voluntary isometric force

Changes in isometric strength of the knee extensors, expressed as a percentage of the baseline measure for each bout, following the first and second bouts are depicted in Fig. 2. As expected, strength was significantly decreased relative to pre-test levels following both downhill runs ($F=26.6_{4,68df}$, $P<0.001$). This remained lower up to and including day 3, after which time values returned to their respective pre-bout values.

The overall strength retention was similar between bouts ($F_{1,17df}=0.61$, NS). The pattern of changes in strength after the two bouts of downhill running was similar, as indicated by the non-significant interaction of bout×time ($F=0.48_{4,68df}$, NS). The effect of stride manipulation on strength retention in bout two was non significant ($F_{(GG)1,8df}=3.37$, NS) with no interaction of group and time ($F_{8,60df}=1.16$, NS). There were also no differences in strength retention between men and women after the preferred stride length protocol ($F_{1,16df}=0.15$, NS) and no interaction of sex by time ($F_{1,60df}=1.09$, NS).

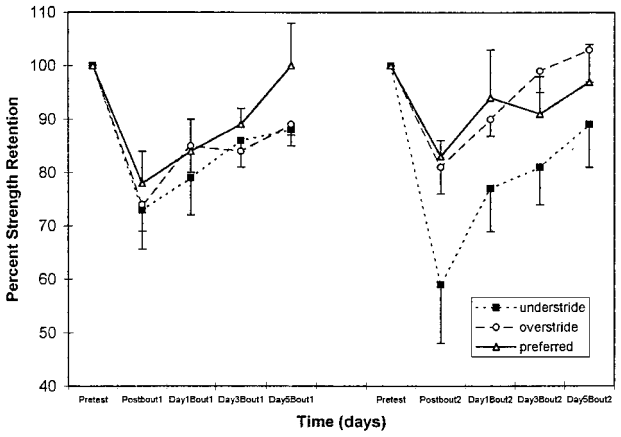

Fig. 2. Changes in percent strength retention in 18 young men and women following an initial bout of running downhill at preferred stride length (—△—) and following a secondary bout of downhill running during which the stride length was altered by −8% (understride, ---■---), +8% (overstride, ---○---) or maintained at preferred length (—△—). Values are mean±SEM.

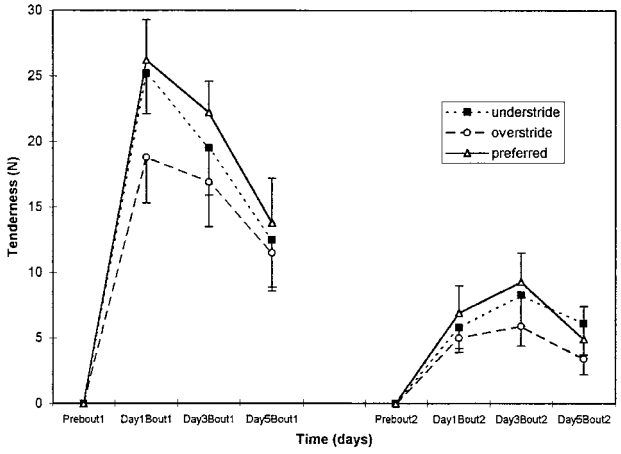

Fig. 3. Changes in muscle tenderness in 18 young men and women following an initial bout of running downhill at preferred stride length (—△—) and following a secondary bout of downhill running during which the stride length was altered by −8% (understride, ---■---), +8% (overstride, ---○---) or maintained at preferred length (—△—). Values are mean ±SEM.

Tenderness

Fig. 3 shows the changes in muscle tenderness that occurred following the first and second running bouts. Tenderness values changed significantly over time ($F=63.5_{3,51df}$, $P<0.001$), with values throughout the experimental periods being higher than baseline following both runs. The overall tenderness value was, however, significantly lower after the second bout of downhill running ($F_{1,18df}=86.8$, $P<0.001$). There was also a significant interaction of bout and time on tenderness ($F_{3,51df}=35.7$, $P<0.001$). For the initial run, tenderness peaked on day 1 and, whilst declining thereafter, remained elevated relative to pretest levels on days 3 and 5. In contrast, tenderness peaked on day 3 following the second bout, although this peak in muscle tenderness was much lower than that observed following the initial bout. The effect of stride manipulation on tenderness in bout two was non significant ($F_{2,15df}=0.46$, NS) with no interaction of group and time ($F_{6,45df}=0.51$, NS). Whilst the difference was not statistically significant, it was notable that males tended to report less tenderness after the preferred stride length protocol, with overall mean values of 33±12 N and 40±17 N for the males and females, respectively ($F_{1,16df}=4.01$, $P=0.06$). However, the relative changes in tenderness for the males and females were similar, as indicated by the non-significant interaction of sex by time ($F_{3,48df}=1.09$, NS).

Discussion

This study assessed the effects of changes in stride length during downhill running on the symptoms of exercise-induced muscle damage. A secondary objective of the study was to examine the extent to which symptoms of muscle damage were influenced by gender and a prior bout of downhill running undertaken 5 weeks previously.

The protocol consisted of an intermittent bout of downhill running, as used in previous studies (Eston et al. 1995, 1996, 199a), which was repeated after a 5-week interval. Both runs were effective in producing significant increases in tenderness, an increase in circulating CK activity and a reduction in muscle strength. The magnitude and timing of the changes in CK activity following the first run, with mean peak values of 360 $U \cdot l^{-1}$ (prior to logarithmic conversion) on day 1, was in agreement with previous studies which have used downhill run protocols to induce damage (Byrnes et al. 1985, Schwane et al. 1983, Eston et al. 1995, 1996, 1996a).

As far as we are aware, there is no published research related to the effects of stride manipulation on the symptoms of muscle damage induced by downhill running. Previous studies which have manipulated stride length have focussed on the effects on running economy. These studies determined that on a level gradient running economy is optimal at the preferred stride length (Hogberg 1952, Cavanagh & Williams 1982). For reasons stated earlier, it was hypothesised that the overstriding group in the current study would present greater evidence of exercise-induced muscle damage. Conversely, however, it was the understriding group which appeared to suffer the greatest overall decrement in strength, although it should be noted that this difference was just outside statistical significance. We must therefore conclude that stride manipulation, of the magnitude applied in

this study, had no effect on muscle tenderness, CK activity or knee extensor strength measures in individuals who have some degree of protection from a prior bout. It is possible that the change in preferred stride length (±8%) was not great enough to induce a significant change in the parameters measured. However, it is pertinent to acknowledge that this magnitude of change may be expected functionally, and any greater change to stride length would be unnatural and could lead to instability and acute musculo-skeletal injury during the descent.

Additionally, the potential effects of stride manipulation on strength during the second downhill run may have been masked by the protective effect of the initial bout. The overall effects of this may have minimised additional damage in the subsequent bout. However, all participants received the same prior bout of downhill running. There is no reason to suggest that any group, in which stride length was manipulated, may have received a greater protective effect from the prior bout. In addition, we considered that some degree of protection would be present in people who run on hilly terrain.

The protective adaptation has been referred to as the 'repeated bout effect' (Nosaka & Clarkson 1995) and has been well documented (see McHugh et al. (1999) for review). This protective adaptation has been reported to last for approximately six weeks (Nosaka et al. 1991). In the present study, it is evident that the protective effect of a single bout of downhill running remained at five weeks, particularly with regard to the muscle tenderness and creatine kinase activity. Although the overall strength loss in the second bout was lower, this was not statistically significant and so a protective effect for strength cannot be inferred over this length of time.

Although the mechanism underlying the repeated bout effect is unknown, many theories have been proposed to explain this protective adaptation. In general, the protective effect has been attributed to neural (Nosaka & Clarkson 1995, Golden & Dudley 1992, Mair et al. 1994), connective tissue (Clarkson & tremblay 1988, Ebbeling & Clarkson 1989) or cellular adaptations (Fridén et al. 1983, Byrnes et al. 1985, Mair et al. 1994, Ebbeling & Clarkson 1989, Armstrong et al. 1983, Lynn & Morgan 1994). The 'neural theory' predicts that damage from the initial bout results from the high stress on a relatively small number of active fast twitch fibres. In the repeated bout, it is hypothesised that there is an increase in the motor unit activation and/or a shift to slow twitch fibre activation. This distributes the contractile stress over a larger number of fibres. The connective tissue theory predicts that muscle damage occurs when there is disruption to the non-contractile connective tissue elements which destroys myofibrillar integrity. The theory suggests that remodelling of the intermediate filaments and/or an increase in intramuscular connective tissue provides a degree of protection from a second bout. The cellular theory predicts that muscle damage is the result of irreversible sarcomere strain during eccentric contractions. It is believed that some sarcomeres may be stretched beyond myofilament overlap with a loss of contractile integrity. Following an initial bout, it is hypothesised that there is an increase in the number of sarcomeres connected in series. This reduces the sarcomere strain during a subsequent bout and limits the amount of muscle damage. It is unlikely that one set of adaptations can explain the repeated bout effect under all circumstances and more likely that an interaction of the various neural, connective tissue and cellular mechanisms is responsible, with the relative importance of each mechanism being task-dependent.

In this study, the response of each of the dependent measures to each of the two downhill runs was similar in males and females. It has previously been observed that higher levels of CK activity are observed in men after intensive exercise (Hortobagyi & Denahan 1989). However, this is most likely attributable to the higher proportion of muscle tissue relative to body mass, and is unlikely to indicate greater damage in the male groups. In the present study, although mean peak CK activity values were higher for the males on day 1, the difference was non significant ($P>0.05$).

Conclusion

In terms of training regimens, these results have implications for both fell runners and orienteers, where the competitive environments dictate changes in stride pattern. It would appear that downhill running per se offers an appropriate training effect for this group of athletes, irrespective of gender. The potential consequences of different stride patterns within this competitive environment will not be compromised as long as the runner's training schedule includes specific downhill elements. In conclusion, it would appear that muscle damage and delayed onset muscle soreness are not affected by small alterations to the preferred normal stride pattern during downhill running, in men and women who already possess a degree of protection against exercise-induced muscle damage. These findings are within the context of participants already having been exposed to a prior bout of downhill running at their preferred cadence, which appears to offer relatively long-lasting protective effects on the symptoms of muscle damage from a subsequent bout.

Key words: exercise-induced muscle damage; DOMS; stride length; downhill running.

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