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Research article

THE REPEATED BOUT EFFECT: DOES EVIDENCE FOR A CROSSOVER EFFECT EXIST?

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ABSTRACT

Individuals undergoing an unaccustomed exercise bout incorporating a high degree of eccentric muscle contractions commonly experience delayed onset muscle soreness. The damage manifests itself via tenderness, loss of strength, swelling, elevated muscle enzyme activity and loss of flexibility. Following an initial "damage bout," a repeated bout results in reduced symptoms. This protective effect is known as the repeated bout effect (RBE) and can last up to 24 weeks between bouts. The mechanism for this RBE is unclear and both central and local mechanisms have been suggested.In an attempt to test the central hypothesis, 12 subjects (mean age = 22.5± 4yrs, ht = 167±9cm, mass = 71.5±13.5kg) underwent an exercise protocol whereby one leg was exercised eccentrically and following complete recovery; the contralateral leg was exercised in the same manner. Subjects were required to step on and off a 46-cm step for 20 minutes at a cadence of 15 steps/minute. One leg was used to go up the step (concentric) while the opposite was used to go down (eccentric). Approximately two weeks later and following complete recovery, the protocol was repeated with the concentrically exercise leg now performing the eccentric contraction. Data analyses indicate that muscle damage was induced during both trials on the eccentrically exercised leg as evidenced by a change in tenderness (bout 1 $p < 0.05$: bout 2 $p < 0.01$), pain scores (bout 1 $p < 0.0001$; bout 2 $p < 0.01$), and strength loss (bout 1 $p = 0.001$; bout 2 $p = 0.001$) over the four day follow up period. No tenderness was evident on the concentrically exercised limbs when compared to baseline (Bout 1: $p = 0.13$, Bout 2: $p = .06$). Pain was significantly lower in bout two versus bout one ($p < 0.04$), however, we attribute this to a tolerance effect. Neither strength loss nor tenderness were significantly different between bouts. In the current study, damage was induced in both bouts in the eccentrically exercised limbs.This preliminary data does not provide evidence for a central mechanism in that an initial bout of eccentric exercise using one limb did not provide protection against damage from a repeated bout with the contralateral limb two weeks later.

KEY WORDS: Eccentric, exercise, muscle damage, protection, contralateral.

TEKARLANAN ÇALIŞAMA DÖNEMİNİN ETKİSİ: KÖPRÜ ETKİSİNİN VARLIĞININ KANITIMIDIR?

ÖZET

Yüksek derecede eksentrik kas kasılmaları içeren alışılmadık bir egzersizle karşı karşıya kalan bireyler genellikle gecikmiş kas ağrısı yaşar. Hasar esneklik kaybı, kas enzim aktivitesinde artış, şiş, kuvvet kaybı ve hassasiyet ile kendini gösterir. Başlangıç 'hasar çalışma dönemini' takiben, tekrarlanan bir çalışma dönemi semptomlarda zayıflama ile sonuçlanır. Bu koruyucu etki tekrarlanan çalışma dönemi etkisi (RBE) olarak bilinir ve 24 haftada (2 çalışma arası) sonlanabilir. RBE mekanizması açık değildir ve lokal mekanizmalar önerilmiştir. Bu merkezi hipotezi test etme girişiminde 12 denek (ortalama yaş = 22.5±4 yıl, 167±9 cm boy, 71.5±13.5 kg kitle) bir bacakta eksentrik egzersizin yapıldığı bir egzersiz protokolünü yaptı ve devamında tam dinlenimi takiben diğer bacakta aynı biçimde egzersiz yaptı. Denekler 46cm'lik bir basamakta dakikada 15 adım atacak bir ritimde 20 dakika basmak inip çıktı. Bir bacak basamak inmek (eksentrik) için

kullanılırken zıt bacak basamak çıkmak için kullanıldı. Yaklaşık olarak 2 hafta sonra tam dinlenimi takiben protokol, konsentrik egzersiz yapan bacak eksentrik yapacak şekilde tekrarlandı. Verilerin analizi 4 gün takip döneminde kuvvet kaybı (1. çalışma p= 0.001; 2. çalışma p <0.001), ağrı skoru (1. çalışma p= 0.0001; 2. çalışma p <0001) ve hassasiyet (1. çalışma p= 0.05; 2. çalışma p <0.01) değişikliği ışığında her iki çalışmada eksentrik egzersiz yapan bacakta kas hasarının oluştuğunu gösterdi. Konsentrik egzersiz yapan bacakta başlangıçla karşılaştırıldığında hassasiyet yoktu (1. çalışma p= 0.13; 2. çalışma p <0.06). Ağrı 1. çalışma ile karşılaştırıldığında 2. çalışmada anlamlı (p < 0.04) olarak düşüktü, bununla birlikte, biz bunu tahammül etkisi olarak yorumladık. Ne kuvvet kaybı ne de hassasiyet çalışma dönemleri arasında anlamlı farklıydı. Sunulan çalışmada, hasar eksentrik egzersiz yapan bacaklarda her iki çalışma döneminde de oluştu. Bu ön veriler bir bacakta ilk çalışmada eksentrik egzersiz kullanımı 2 hafta sonra diğer bacakta tekrarlanan çalışmada hasara karşı korunma olarak ifade edebileceğimiz merkezi mekanizma için delil oluşturmaz.

ANAHTAR KELİMELER: Eksentrik, egzersiz, kas hasarı, korunma, kontralateral.

INTRODUCTION

Unfamiliar eccentric exercise frequently results in muscle damage, the symptoms of which include strength loss, pain, muscle tenderness, and elevation in creatine kinase (CK) activity (Belnave and Thompson, 1993; Eston et al., 1996; Mc Hugh et al., 2000; 2001). Following recovery from this initial bout a repeated bout of the same exercise results in minimal signs and symptoms of muscle damage. This has been referred to as the "repeated bout effect."(Nosaka and Clarkson, 1995) This protective effect has been demonstrated *in vivo* and *in vitro* with various types of activities using different muscle groups (Sacco and Jones, 1992; Nosaka and Clarkson, 1995). Many theories have been proposed to explain the repeated bout effect but a specific mechanism has not been identified. For a recent comprehensive review see Mc Hugh et al., (1998a). Three basic mechanisms have been proposed. They are neural (Moritani et al., 1988), cellular (Lieber and Friden, 1993) and the 'connective tissue' theory (Armstrong et al., 1991). For a comprehensive review on these proposed mechanisms see Connolly et al. (2002).

Several authors have discussed the possibility that there is a change in motor unit recruitment during the repeated bout which limits the extent of damage (Pierrynowski et al., 1987; Golden and Dudley, 1992; Mayr et al., 1995; Nosaka and Clarkson, 1995; McHugh et al., 1998b). Eccentric actions typically produce greater force but less motor unit recruitment. Specifically, Golden and Dudley (1992) suggested that the lower level of motor unit activation associated with eccentric contractions may provide the opportunity to "learn more efficient recruitment" for a repeated bout. In accordance with this Nosaka and Clarkson (1995) suggested that the neural adaptation would "better distribute the workload among fibers." Similarly, Pierrynowski and colleagues (1987) suggested that "increased synchrony of motor unit firing" may reduce myofibrillar stresses during a repeated bout. These adaptations seem plausible given the neural characteristics of eccentric muscle contractions. Indeed, recent work has demonstrated significant differences in motor unit activation and fiber type recruitment for eccentric compared to concentric exercise at the same intensity (McHugh et al., 1998b). Eccentric exercise is associated with selective recruitment of a small number of predominantly fast twitch motor units. At present, this neural control of motor unit recruitment is considered mediated by a central (nervous system) mechanism.

Eston et al., (1996) demonstrated that a prior bout of unilateral isokinetic eccentric exercise provided protection against damage following a downhill run. CK was elevated on average 580% in the group that did not have a prior bout of eccentric exercise. In contrast, CK was elevated by only 150% on average in the group that did have a prior bout of eccentric exercise. Despite the fact that only the quadriceps muscle of the dominant limb was exposed to the prior bout of eccentric exercise, whole body CK elevations were significantly blunted. It was not clear whether this effect was due to reduced damage in the dominant quadriceps or whether protection was provided to other muscle groups involved in the downhill running. This data suggests the possibility of a crossover of protection to muscles not preconditioned by eccentric exercise and thus, the possibility of a protective effect on the contralateral side. Such an adaptation would have to be mediated centrally.

The possibility that the muscle damage initiated in one limb could provide protection against damage following a repeated bout in the contralateral limb has not been examined previously. Our intention was to initiate muscle damage in the quadriceps of one limb and following recovery, repeat the exercise in the contralateral limb. If the subsequent damage was less in the contralateral limb than had been observed in the previously exercised limb, this would be evidence of a central neural effect. A localized mechanism would not be plausible since no work had been previously carried out on the second limb. Thus, the purpose of the current investigation was to assess whether an unaccustomed exercise bout on one limb, resulting in muscle damage, could provide a protective effect from similar exercise when performed on the opposing limb? If so, signs and symptoms of muscle damage would be significantly decreased in the following limb following a repeated bout of exercise.

METHODS

Prior to testing, all procedures were approved by the institutional review board for use of human subjects, in accordance with the Helsinki Declaration of 1975. Twelve subjects (9 female, 3 male), 18-30 years old without knee injury, or history of, volunteered for this study (mean age = 22.5 ± 4yrs, height = 167 ± 9cm, mass = 71.5 ± 13.5kg). Knee injury history was simply determined by asking the subject about prior injury. At this time there is no conclusive evidence to suggest a gender effect, thus both males and females were recruited.

General Protocol

Each subject underwent a set of baseline measures and two exercise sessions. Baseline measurements of CK, pain (tenderness) and isometric strength at a fixed 80º at the knee were recorded. Pain was assessed using a subjective rating scale from 0-10, with 0 = no pain and 10= having difficulty walking. Within 5 days of baseline measures subjects participated in the first of two exercise sessions designed to induce muscle damage. The second session was administered two weeks following the first bout at which time all subjects appeared to have recovered fully from the first bout. Table 1 shows the data collection protocol. Detailed specifics of all data collection are presented below.

Strength Measurement

Strength (NM) was assessed isometrically in the quadriceps/hamstrings muscle group at a fixed angle (80º) on both legs. All measurements were made on an isokinetic dynamometer (LIDO 481, Chattanooga, TN, USA). Following a warm-up consisting of 5 sub-maximal contractions on each leg the subject then performed 5 maximal contractions. The average force value for the latter five repetitions was used for analysis. Strength measurements were taken at baseline, immediately after each exercise session, and every 24 hours thereafter until damage symptoms subsided.

Table 1. General protocol summary.

Day #	**1**	**6**	**7**	**8**	**9**	**10**	**24**	**25**	**26**	**27**	**28**
Variable	**Baseline**	**B1**					**B2**				
CK	4	4	4	4	4	4	4	4	4	4	4
Strength	4	4	4	4	4	4	4	4	4	4	4
Flexibility	4	4	4	4	4	4	4	4	4	4	4
Pain	4	4	4	4	4	4	4	4	4	4	4

B1 = bout 1, B2 = bout 2, CK = Creatine kinase.

Muscle Damage Exercise

Subjects performed a modified stepping protocol on a 46cm bench designed to induce delayed onset muscle soreness in the quadriceps group. This protocol was fully described by Newham et al., (1983a; 1983b) and has been used successfully by others (Gleeson et al.,

1998). The protocol required subjects to step on and off a 46cm bench. A cadence of 15 steps per minute for 20 minutes was used. Eccentric muscle damage was induced by requiring the subject to consistently lower on the same leg. We modified the protocol slightly by requiring subjects to wear a body vest weighted with 6% of body mass. In order to minimize any damage that might occur in the plantar flexor muscles of the opposite leg, subjects stepped down onto a padded mat. The damage bout was induced only once in each leg as the protective effect i.e. the 'repeated bout effect', has been extensively documented previously using similar protocols (Sacco and Jones, 1992; Nosaka and Clarkson, 1995; Eston et al., 1996).

Blood Measurement

Creatine kinase was measured via 30µL of blood taken from a finger stick and analyzed using a Reflotron Spectrophotometer (Boehringer Mannheim, Dusseldorf, Germany). This is a standard, hygienic finger stick approach that is commonly used with this measurement system. Creatine kinase was measured at the same time each day for the baseline measurements and for the four days following each exercise damage bout. While creatine kinase was collected in the current study its use as a reliable indicator has become a contentious issue in recent years (Warren et al., 1999; Nosaka and Clarkson, 1995). This is due mainly to the high degree of variability in the response between subjects and the terms 'high-responder' and 'low-responder' have been used to describe this phenomenon. Following data collection it was decided to omit this variable from the data analysis due to the high degree of variability that we experienced as damaged indices ranged from <120 $IU.L^{-1}$ to > 20000 $IU.L^{-1}$ (Thompson et al., 1997, Lin and Yang, 1999). This makes reliable statistical analysis difficult even after log transformation.

Muscle Tenderness and Pain

Muscle tenderness scores were assessed using a standard manual muscle myometer. Measurements were recorded for the vastus medialis oblique (VMO), the rectus femoris (RF) and the vastus lateralis (VL). All measurements are reported in Newtons (N). Force was applied via the probe through a 1cm-diameter head until the subject indicated pain or discomfort. At this point the force value (N) was recorded. Baseline values for tenderness were declared to be at ≥ 70N once force application reached 70N. Therefore, decreasing force scores indicated increasing tenderness, a reflection of muscle damage. We used a baseline value of 70N as force application greater than 70N would likely have caused localized damage. Patient discomfort (pain) was also measured every day using a visual scale. Pain was assessed using a subjective rating scale from 0-10, with 0 = no pain and 10= having difficulty walking. Subjects were simply presented the scale with '0' on the right and arrows proceeding to '10' on the left and asked to identify their overall discomfort or pain in daily movements.

Data were analyzed using a 2 x 5 (bout x time) repeated measures ANOVA (strength, pain, and tenderness) with alpha set at .05. Data for each variable for each day are also presented as mean plus or minus standard deviation. Data were analyzed using SPSS software (V10.05).

RESULTS

Overall results do not indicate a protective effect of a prior bout of exercise on a contralateral limb. Data indicate a significant effect of the exercise session over time for pain, tenderness and strength loss for both bout one and bout two ($p<0.05$). This is evidence that damage was indeed induced. Mean pain scores increased significantly from '0' at baseline to a peak of '6.00 ± 0.62' and '4.90 ± 0.53' for bout one and two, respectively ($p<0.05$). Pain scores peaked on day two for both groups (see Figure 1).

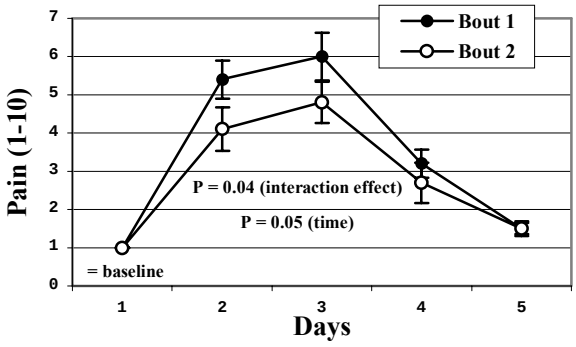

Figure 1. Pain values from baseline to day 4 (mean ±SE).

Data also indicate that there was a significant difference in pain response between bout one and two ($p<0.04$), with bout two being lower. Tenderness

values decreased significantly for both groups over time from a baseline value of 70N to 50.4 ± 3.9 N and 52.9 ± 2.7 N, for bout one and two respectively ($p<0.01$) [see Figure 2].

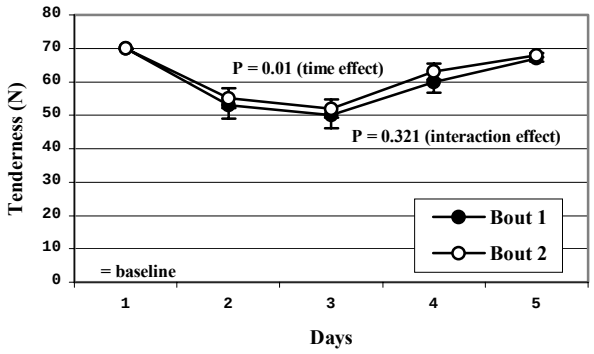

Figure 2. Tenderness changes from baseline to day 4 (mean ± SE).

Tenderness scores did not differ significantly between bouts one and two ($p= 0.321$). Mean strength scores, expressed as a percent of contralateral leg at baseline, decreased significantly over time ($p=0.001$ for both bouts) but did not differ significantly between bouts one or two ($p=0.539$). See figure 3 for percent strength loss over time.

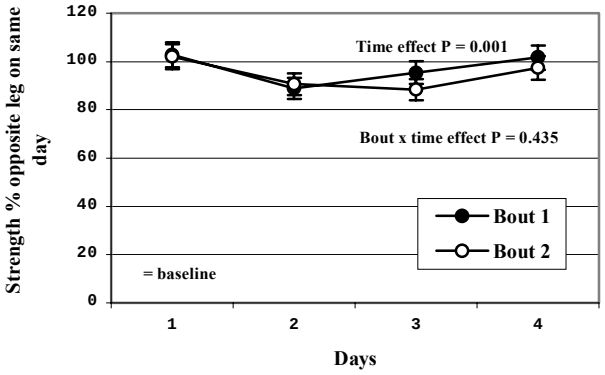

Figure 3. Strength changes from baseline to day 4 (mean ± SE).

DISCUSSION

Data from the current investigation, while preliminary, suggests no crossover effect for the 'repeated bout effect'. While the data indicates that pain scores were lower in bout two, we feel this is a result of subjects being more familiar with the discomfort associated with the testing, having experienced the pain with bout one. No other variables suggest any significant crossover effect. Furthermore, evidence of damage is clearly evident in both the tenderness and pain variables for both bouts one and two. Prior work by Eston et al., (1996) demonstrated that a prior bout of unilateral isokinetic eccentric exercise provided protection against damage following a downhill run. As aforementioned, CK was elevated by 580% in the group that did not have a prior bout of eccentric exercise. In contrast, CK was elevated by only 150% in the group that did have a prior bout of eccentric exercise. Our measurements of CK proved to be unreliable and were discarded. This variability problem has been reported previously and appears to a function of individuals being high or low responders (Evans et al., 1986; Newham et al., 1988; Eston et al., 1996). Regardless, the work by Eston et al., (1996) demonstrated muscle damage following the downhill bout in both groups. This was evidenced by strength, tenderness and CK data. While tenderness increased in both groups it occurred to a greater extent in the group that did not receive a prior isokinetic bout suggesting some protective effect. In the Eston et al., (1996) protocol, only the quadriceps muscle of the dominant limb was exposed to the prior bout of eccentric exercise. Therefore, it is difficult to know how much protection may have been afforded the contralateral limb. Their CK data indicates a blunted response suggesting reduced damage in the opposing limb. However, since data was collected only on the dominant limb we are unsure about the contralateral limb response. The work by Eston et al., (1996) may simply serve to confirm the 'repeated bout effect' in the previously exercised limb.

The current study attempted to more directly answer the question of a crossover effect. By damaging only one leg, allowing full recovery and then damaging the other we should get a better indication of any protective or crossover effect. This does not appear to have occurred. We successfully induced muscle damage in both legs as evidenced by pain and tenderness scores, indicating no crossover effect. In the current study strength data are expressed as percent of contralateral leg at baseline for each day. The pattern of strength loss was significant in the damaged leg for each bout with a decrease in strength apparent over 4 days. There was no difference in the strength loss between damaged legs for either bout one or two. Again, this suggests no crossover or protective effect.

As aforementioned, three basic mechanisms for the RBE have been proposed. They include neural (Moritani et al., 1988), cellular (Lieber and Friden, 1993) and the 'connective tissue' theory (Armstrong et al., 1991). The neural theory predicts that the initial damage is the result of high stress on a small number of fast twitch fibers. The resultant repeated bout effect purportedly stems from an increase in motor unit activation in subsequent bouts. The connective theory suggests that muscle damage occurs as a result of non-contractile elements being disrupted. Consequent filament remodeling with increased connective tissue then occurs to induce the repeated bout effect. The cellular theory suggests the initial damage occurs from excessive sarcomere strain during the eccentric action. The repeated bout is then the result of reduced sarcomere strain in the subsequent bout. While the current study does not directly advocate any of the proposed mechanisms, it does suggest that the mechanism is centrally mediated and does question the neural theory.

CONCLUSIONS

Our findings suggest that both the connective tissue theory and the cellular theory may have more acceptability as they suggest a more localized response than does the neural theory. However, it is possible that the repeated bout effect occurs through an interaction of cellular, connective and neural adaptation. It is further likely that the extent of the response varies as a function of intensity, specificity of contraction, trained state of the muscle and the muscle group involved.

Further work in this area is warranted and should consider a greater degree of damage than was induced in the current study, use of an upper body model or a model that revisits the initially damaged muscle before the contralateral limb.

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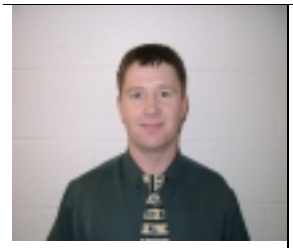

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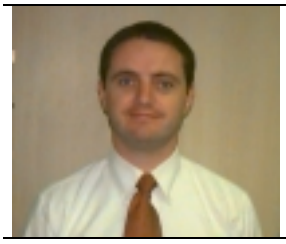

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