Published in final edited form as:

J Electromyogr Kinesiol. 2009 December ; 19(6): e529–e535. doi:10.1016/j.jelekin.2008.10.010.

Repeated bout effect is absent in resistance trained men:

An electromyographic analysis

Michael J. Falvo^{a,*}, Brian K. Schilling^b, Richard J. Bloomer^b, and Webb A. Smith^c

^aProgram in Physical Therapy, Washington University School of Medicine, Campus Box 8502, 4444 Forest Park Ave., Saint Louis, MO 63108, USA

^bDepartment of Health and Sport Sciences, University of Memphis, Memphis, TN, USA

^cEpidemiology and Cancer Control, St. Jude Children's Research Hospital, Memphis, TN, USA

Abstract

A prior bout of exercise is well known to confer protection from subsequent eccentric bouts (i.e. repeated bout effect; RBE), which may be fostered through neural adaptations, specifically a shift in the frequency content of the surface electromyogram (EMG). It is currently not clear whether chronically resistance trained men are capable of a RBE driven by neural adaptations. Eleven resistance trained men (23.5 ± 3.4 yrs) performed 100 eccentric actions of the barbell bench press exercise, followed by an equivalent bout 14 days later. Indirect markers of muscle damage (i.e. force production, soreness) along with surface EMG were measured before and through 48 h of recovery. Median frequency and maximal isometric force demonstrated time main effects ($p > 0.05$), but no RBE. A prior bout of eccentric exercise does not confer a RBE for indirect markers of muscle injury or elicit changes in the frequency content of the EMG signal in resistance trained men.

Keywords

Repeated bout effect; Muscle damage; EMG; Eccentric exercise; Bench press

1. Introduction

When force and velocity are held constant, the amplitude of the surface electromyogram (EMG) for eccentric actions is less than that of concentric actions (Bigland and Lippold, 1954; Moritani et al., 1988), suggestive of a reduction in recruitment and discharge rate during skeletal muscle lengthening. Coupled with characteristic higher forces observed during muscle lengthening (Lombardi and Piazzesi, 1990), eccentric actions are conducive to inducing muscle fiber injury and can lead to force loss and soreness. (For review see: Clarkson, 1997; Clarkson and Hubal, 2002; Byrne et al., 2004). The magnitude of injury and accompanying impaired muscle function appear to be attenuated through performance of prior exercise, a phenomenon referred to as the 'repeated bout effect' (RBE; Nosaka and Clarkson, 1995).

Neural adaptations secondary to strenuous eccentric exercise may be responsible, in part, for the RBE (McHugh, 2003). Said adaptations may include (1) increased motor unit activity relative to force produced, (2) increased synchrony of motor unit firing, and/or (3) altered recruitment. The latter has been reported by several investigators, reporting a shift in the frequency content (i.e. power spectrum) of the EMG during performance of the repeated bout,

suggesting a concurrent increase and decrease in slow- and fast-motor unit activity, respectively (Warren et al., 2000; Chen, 2003; Howatson et al., 2007). Increased reliance on slow motor units (i.e. reduced EMG content) may confer protection during the repeated bout by decreasing stress on susceptible fast-twitch fibers that, in turn, results in a better distribution of contractile stresses (Nosaka and Clarkson, 1995). To the knowledge of these authors, only four investigators have directly addressed, via surface EMG, whether a RBE is mediated through neural adaptations. This effect has been observed following a variety of experimental approaches including, (1) elbow- and plantar-flexor muscles, (2) initial to repeated bout time intervals of three, seven, or 14 days, and (3) preconditioning volumes equivalent or fewer than the repeated bout (Warren et al., 2000; Chen, 2003; Howatson et al., 2007). However, this has not been observed in all investigations, as McHugh et al. (2001) observed no changes in EMG frequency content 14 days following an eccentric bout of the knee flexors despite an observed RBE for torque.

The vast amount of work within the RBE paradigm draws upon subjects with no prior resistance training experience. As trained individuals frequently experience symptoms of muscle damage (e.g. delayed onset muscle soreness) due to new training techniques or different phases (i.e. high volume) of training, this raises the question as to whether these individuals may also achieve a RBE, a question pertinent to exercise program design (Falvo and Bloomer, 2006). Newton et al. (2008) recently described the response to eccentric exercise of the elbow flexors in resistance-trained and untrained men and found those with prior training experience to be less susceptible to muscle damage than those who are untrained. Despite this level of protection, a RBE has been demonstrated in the elbow flexors of resistance-trained individuals, of which the authors attribute to neural adaptations (Howatson and van Someren, 2007; Howatson et al., 2007). This is supported not only by a shift towards greater recruitment of slow twitch motor units (Howatson et al., 2007), but also in a unique design in which following ipsilateral-only training, a RBE was observed in the untrained contralateral limb, referred to as the contralateral RBE (Howatson and van Someren, 2007).

We have previously reported that a prior bout of eccentric exercise offers no additional attenuation of impaired exercise performance independently (Bloomer et al., 2007; Falvo et al., 2007) or combined with antioxidant supplementation (Bloomer et al., 2007) in resistance-trained men. These general findings are dissimilar than that reported elsewhere in resistance-trained individuals (Howatson and van Someren, 2007; Howatson et al., 2007), but did not account for neural modification. Therefore, the present investigation was conducted to determine the existence of a RBE mediated through neural adaptations in resistance trained men. In light of our previous findings (Bloomer et al., 2007; Falvo et al., 2007), we hypothesized that such adaptations would also not be present thereby supporting the absence of a RBE in chronically resistance trained men. Parts of these results have been presented elsewhere (Bloomer et al., 2007; Falvo et al., 2007).

2. Materials

2.1. Subjects

Eleven college-age men (23.5 ± 3.4 yrs; 174.9 ± 4.8 cm; 81.6 ± 17.1 kg; mean $\pm$ SD) volunteered to participate in this investigation, which was approved by the Institutional Review Board for Human Subjects Research and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. All subjects were considered of trained status as determined by concentrically bench pressing a load greater than or equal to their body mass (1.2 ± 0.1 kg kg⁻¹), consistently performing resistance training for at least the past six months (3.6 ± 1.0 sessions wk⁻¹; 4.9 ± 3.0 yrs), with at least one session per week designated for training the pectoralis, deltoids, and triceps. We consider our operational definition of “trained” status to be unique from prior research in this area which generally describes their

subjects as having no prior resistance training history for at least the past year and is similar to that previously reported (Newton et al., 2008). In other words, based on our subjects' baseline performance as well as their frequency and history of resistance training, we believe them to be sufficiently trained. Subjects were free of any orthopedic and neuromuscular problems of the upper extremity.

2.2. Experimental design

A within-group repeated measures design was used to determine whether trained men exhibit a RBE. An exercise bout designed to induce muscle injury was performed and assessments were made prior to and following this bout (experimental protocol). After 14 days, this experimental protocol was repeated (Fig. 1). Subjects served as their own controls and comparisons were made between bouts and across time. During their first visit at which written informed consent was provided, subjects performed a one-repetition maximum test (1RM) of the concentric bench press exercise. A concentric 1RM rather than a traditional (eccentric-concentric) was chosen to minimize additional muscle trauma. Also at this time, subjects practiced the isometric performance test utilized herein and were provided verbal and visual feedback. One to two days thereafter, subjects reported to the laboratory for another brief practice session in order to reduce learning effects associated with the isometric test. These measures were found to be reliable and have been reported elsewhere (Falvo et al., 2007). Between 48 and 96 h following practice sessions, subjects began their first (bout 1) of three consecutive days of experimental testing. Day one of three consisted of baseline testing, the eccentric exercise bout, and follow-up testing (15 min post). Follow-up testing was also performed on days two and three approximately at the same time of day (± 1 h). This protocol was repeated (bout 2), in exactly the same manner as bout 1, two weeks after the completion of day three of bout 1. Subjects were instructed to maintain normal physical activity during their hiatus, but to refrain from upper body resistance exercise 48 hours prior to day one of the experimental testing (bouts 1 and 2). Fig. 1 illustrates the study timeline.

2.3. Muscle injury protocol

Subjects performed 100 repetitions (10 sets of 10 repetitions; 2 min of rest between sets) of the eccentric portion of the barbell bench press exercise, as performed on a Smith machine, at a load equivalent to 70% concentric 1RM. We adopted this intensity after thorough pilot testing on several highly trained subjects and found 100 repetitions of loads greater than 70% to be intolerable and unrealistic to complete. This protocol is unique to the literature which predominantly employs single joint movements of the elbow flexors. Therefore, it was our intent to incorporate a more realistic modality as a means of inducing muscle injury.

Movement began with subjects in an arms extended position while on a traditional exercise bench. The barbell was lowered for five seconds (maintained via a computerized timing device), such that the barbell came in contact with their chest at the end of the five second period. At this time, two spotters raised the barbell back to the starting position (<1 s) and the subject immediately began lowering the bar for the next repetition. If the subject was unable to lower the barbell under control for 5 s, the bar was secured, 10% of the total weight was removed, and the next repetition immediately resumed. Standardized verbal encouragement was provided by spotters to ensure maximal effort. Percent fatigue and total work (i.e. volume-load) were compared between bouts to determine the rate of decline and total work performed, respectively. These were computed as follows;

$$\text{Total work} = \text{Load (Kg)} \times 9.81 \left(m \ s^{-1} \right) \times \text{Barbell displacement (m)} \times \# \text{ Repetitions.}$$

$$\text{Percent fatigue} = \left[\left(\text{Work}_{\text{SET 1}} - \text{Work}_{\text{SET 10}} \right) / \text{Work}_{\text{SET 1}} \right] \times 100.$$

2.4. Dependent measures

The following variables were used to indirectly determine the extent of muscle injury; (1) perceived soreness, (2) serum creatine kinase activity, (3) maximal isometric force (MIF) and rate of force development, and (4) surface electromyography parameters. Assessments were made 15 min (Post-15), 24 h (Post-24), and 48 h (Post-48) following the eccentric protocol.

2.5. Muscle soreness

Soreness was determined at each visit of the experimental protocol using a 10 cm visual analog scale (where “0” represents no pain and “10” represents intense pain). Subjects performed two repetitions of the barbell bench press exercise (20 kg) prior to recording their subjective soreness on the visual analog scale. Subjects were familiarized with this scale prior to the experimental protocol.

2.6. Serum creatine kinase activity

For each time point, 15 mL of blood was collected into Vacutainer™ tubes via antecubital venipuncture then allowed to clot at room temperature. Samples were immediately processed by centrifugation to obtain serum and stored at -80 °C for later analysis. Serum creatine kinase (CK) activity was measured spectrophotometrically using commercially available reagents (StanBio Labs, Boerne, TX). The coefficient of variation for this assay was 4.1%.

2.7. Maximal isometric force

Isometric force testing was conducted in a custom exercise rack in which the subject was in a supine bench press position as described previously McHugh et al. (2001). In brief, subjects pressed into a fixed bar, aligned at the mid-sternum level, to accommodate a 90° angle at the elbow (Fig. 2). Individual adjustments were recorded and reproduced at subsequent testing visits with extreme care to ensure consistency between all testing visits. Following two warm-ups of approximately 50% and 75% effort, subjects performed three maximal (3 s) attempts. Each warm-up and maximal attempt was separated by 1-2 min of rest. Subjects were instructed to contract as hard and as fast as possible (Sahaly et al., 2001).

Isometric force data were acquired via four load cells encased within a commercial-grade floor scale (Rice Lake Weighing Systems; Rice Lake, WI) placed beneath the exercise bench. This signal was sampled at a 1 kHz analog-to-digital conversion rate (PCI-DAS1200/JR, Measurement Computing, Middleboro, MA) and interfaced with a computer. During offline analysis, the summed signals from the four load cells were smoothed using a 4th order low-pass Butterworth digital filter (30 Hz cutoff). Force-time histories were analyzed for maximal isometric force (MIF) and rate of force development (RFD). Maximal RFD was quantified as the highest value of the slope coefficients of the tangent computed during 5 ms (Viitasalo et al., 1980). The onset of contraction was detected using a threshold criteria of 5 N and were visually inspected to ensure correct location. The attempt with the highest MIF was chosen subsequent analysis. Signal processing was performed using Datapac 2K2 software (v3.16; Mission Viejo, CA).

2.8. Surface electromyography

Pairs of round Ag-AgCl surface electrodes (Ambu Blue Sensor SP, 20 mm interelectrode distance) were affixed bilaterally on the pectoralis major, anterior deltoid, long and lateral heads of the triceps according to SENIAM guidelines (Freriks et al., 1991). A ground electrode was placed on the styloid process of the ulna for signal noise reduction. Electrode positions were carefully recorded and marked with permanent ink for each subject to ensure identical placement in subsequent testing sessions. Prior to electrode placement, skin was shaved, vigorously abraded, and cleaned with alcohol. EMG was recorded during MIF attempts and

processed using a Myopac Jr (RUN Technologies; Mission Viejo, CA) with eight dual lead channels. As these electrodes were passive, pre-amplification was not necessary. This system has a common mode rejection of 90 dB, a band-pass filter (10-1000 Hz), and input impedance of 10 M Ω . Data were collected at 1 kHz and synchronized with the force signal.

During offline analysis, raw EMG signals were high-pass filtered using a 4th order Butterworth digital filter (5 Hz cutoff), rectified and integrated, summed for all muscles uni- and bi-laterally, and expressed relative to force for the same contraction (McHugh et al., 2001). This allowed us to compute the electrical activity per unit of force ($\mu\text{V/N}$), expressed relative to the integrated isometric curve for the contraction. EMG amplitude was also quantified by computing a root mean square (RMS), 50 ms time constant, of the raw signal. RMS values were averaged over the entire length of the contraction for analysis. Both $\mu\text{V/N}$ and RMS were computed for each side (left and right) as well as averaged for a bilateral measures. Frequency content was determined by zero-padding the raw EMG signal before running a 512-point Fast Fourier Transform (FFT) with Hamming window for the whole contraction. The FFT was squared to determine the median frequency (MDF), and was averaged across the three attempts. MDF is reported to be sensitive to detect surface EMG changes induced by eccentric actions (Felici et al., 1997). These variables ($\mu\text{V/N}$, RMS, MDF) were chosen to replicate prior studies in this area (Warren et al., 2000; McHugh et al., 2001; Chen, 2003; Howatson et al., 2007).

2.9. Statistical analysis

Dependent variables were analyzed using a two-way repeated measures analysis of variance (ANOVA), with bout and time (pre, post, 24, and 48) as factors. Greenhouse-Geisser corrections were applied to those significance tests that failed to meet the assumption of sphericity. Assumptions of normality were tested and met with the exception of the CK data which was log transformed. Pairwise comparisons were made using Bonferroni contrasts and effect sizes were calculated using Cohen's *d*. Total work and percent fatigue during initial and repeated bouts were compared using a paired *t*-test. Statistical significance was established at $p \leq 0.05$ prior to analyses. Data are presented as mean $\pm$ standard deviation (SD). Statistical analyses were performed using SPSS (v14; SPSS Inc., Chicago, IL).

3. Results

All subjects successfully completed 100 repetitions during bouts 1 and 2. Total work and percent fatigue during bout 1 (work; fatigue: 24565.12 Nm; 11.79%) and bout 2 (28066.43 Nm; 7.77%) were statistically similar ($p = 0.25$; $p = 0.37$). Significant interactions were found for perceived muscle soreness ($p = .002$) and RMS ($p = .044$) and are depicted in Figs. 3 and 4. Further analysis demonstrated between-group differences at Post-24 ($p = .002$; $d = 1.48$) and Post-48 ($p = .002$; $d = 1.54$) for perceived muscle soreness. However, no such differences were observed for RMS. One-way repeated measures ANOVA were conducted for each bout on RMS and only bout 1 was significant ($p = .026$). Pairwise comparisons revealed a significant difference in RMS values from Pre to Post-15 ($p = .007$; $d = 0.46$).

No other dependent variable demonstrated significant interaction or bout differences ($p > 0.05$). Time main effects were noted for MIF ($p < .0001$) and RFD ($p = .001$). For both MIF and RFD, muscle function was significantly impaired from baseline through Post-24 (Figs. 5 and 6). MIF also remained significantly depressed at Post-48, albeit moderately ($d = 0.57$).

Time main effects were also noted for MDF ($p = .001$) and $\mu\text{V/N}$ ($p = .032$). Pairwise comparisons indicated a significant increase in the frequency content from Pre to Post-15 ($p = .001$; $d = .057$). No significant pairwise comparisons were noted for $\mu\text{V/N}$. Surface EMG-related variables are presented in Table 1.

Serum creatine kinase activity demonstrated no significant interaction or main effects. This response is illustrated in Fig. 7. It should be noted that blood was taken immediately following the damaging protocol (Post-0), and therefore represents a different time point than the aforementioned markers which occurred approximately 15 min (Post-15) following the damaging protocol.

4. Discussion

Findings from this investigation generally support our hypothesis in that the RBE is absent in resistance trained men. Although this effect was noted for perceived muscle soreness, Nosaka et al. (2002) have highlighted the poor relationship between soreness and other indirect markers of damage such as maximal force production, suggesting soreness does not accurately reflect the extent of injury. Soreness was also not correlated with MIF in our study at any time point ($r = -0.15$ to 0.16). This is further supported in the present study by the lack of significant between-bout differences observed for all other markers of muscle injury. From this, we cannot support that a RBE is realized in these resistance trained men, which may be attributable to the lack of any noticeable neural adaptation. The ensuing discussion will predominately focus on the absence of these neural adaptations as we have previously discussed the response and recovery to this protocol elsewhere (Bloomer et al., 2007; Falvo et al., 2007).

Similar to our findings, McHugh et al. (2001) observed no significant differences in EMG activity between initial and repeated bouts. In their study, 60 eccentric actions of the knee flexors were performed on an isokinetic device at ~60% of isometric strength. However, unlike our study, these authors were able to demonstrate a RBE for isometric strength despite no evidence for neural adaptation. It is not clear from this investigation if their subjects had any prior resistance training experience. Nevertheless, other investigations have demonstrated a RBE for several markers of injury in both untrained (Warren et al., 2000; Chen, 2003) and trained (Howatson et al., 2007) individuals, attributing such effects to decreases in the frequency content of the surface EMG. In these studies, a 30% decrease in MDF was observed in the anterior tibialis muscle following 50 maximal eccentric actions repeated one week after an initial bout (Warren et al., 2000). Following 30 maximal eccentric actions of the elbow flexors performed three days after the initial bout, Chen (2003) reported a 20% decrease in MDF. As highlighted by Howatson et al. (2007), these investigations may be confounded by the short duration between bouts whereby residual damage may have affected motor unit recruitment.

Utilizing a longer between bout interval, Howatson et al. (2007) demonstrated a 10% decrease in MDF ($d = 0.48$) following 45 maximal eccentric actions of the elbow flexors performed 14 days after an initial bout of either 10 or 45 maximal eccentric actions. From this, it was determined that low or high volume maximal eccentric actions confer protection, via neural adaptation, during a subsequent bout of equal or greater volume. Unlike the large effect size found in 16 men from Howatson et al. (2007), the present study found no differences in MDF with its largest effect ($d = 0.28$) at Post-24. Perhaps with a larger sample size this small-medium effect may have become significant. Interestingly, these authors described their subjects as being “familiar with resistance training, but not accustomed to eccentrically-biased exercise” (Howatson et al., 2007, p. 558). Unfortunately, no further description was provided to better qualify the trained status of these subjects. These results are attractive taken in light of the highly trained status of the subjects in the present investigation and question the role of prior training history.

Our statistical analysis did demonstrate a significant bout $\times$ time interaction for RMS, which is a parameter used to represent total neural activity. Previous studies in this area have found no difference in RMS between bouts (Warren et al., 2000; McHugh et al., 2001; Chen, 2003;

Howatson et al., 2007), and we believe this result is trivial. Separate analyses within each bout demonstrated a time effect only during bout 1 and no significant effect for bout 2. The effect in bout 1 was specific to a significant difference between Pre and Post-15 which may be considered marginal ($d = .46$). In addition, other investigators have demonstrated that RMS does not follow a consistent pattern or provide reliable information following eccentric exercise-induced muscle injury (Felici et al., 1997; Ahmadi et al., 2007). These authors support the use of MDF as a superior measure for reflecting the changes in EMG during recovery from muscle injury.

In comparison with related literature (Warren et al., 2000; McHugh et al., 2001; Chen, 2003; Howatson et al., 2007), the present study did not quantify EMG parameters during repeated isokinetic contractions of the injured muscle of interest, rather during an isometric contraction. Perhaps this may be considered a limitation; however, other investigators have utilized isometric assessments to quantify MDF and RMS subsequent eccentric exercise (Felici et al., 1997; Linnamo et al., 2000; Ahmadi et al., 2007). Felici et al. (1997) sufficiently demonstrated that following strenuous eccentric exercise, EMG parameters (e.g. MDF) are reliably obtained during isometric assessment and MDF is a suitable marker for the early and non-invasive detection of muscle injury. From this, we believe our methods were tenable.

We have considered several other possibilities to account for the absence of neural adaptations and/or a RBE in our sample. One interpretation may be that the protocol employed herein is relatively novel to the related literature (i.e. multi-joint bench press) and does not involve focal damage as seen in other investigations. Similarly, this protocol induced injury submaximally rather than through maximal eccentric isokinetic actions. We argue that this is not the first experiment to utilize a multi-joint chest press for inducing injury (Smith et al., 1994) nor is it the first to accomplish this through submaximal means (Pierrynowski et al., 1987; Semark et al., 1997; Byrne and Eston, 2002). Furthermore, Newton et al. (2008) recently compared the response to 60 maximal eccentric actions of the elbow flexors in resistance-trained and untrained men. As expected, the percent decline in MIF was significantly smaller in those who were trained. Interestingly, the percent decline following the eccentric bout (25%) was very similar to that observed in the present investigation (27%) and also demonstrated a similar pattern of recovery. In addition, following 60 maximal eccentric actions of the elbow flexors, subjects reported peak muscle soreness values that were comparable with the present study. This demonstrates that a submaximal multi-joint and maximal single-joint means of inducing and assessing muscle injury demonstrated very similar MIF recovery profiles and perceived muscle soreness.

Additionally, given the sensitivity of MIF assessment for detecting muscle injury (Warren et al., 1999), we are confident that 100 eccentric repetitions were sufficient to induce considerable muscle trauma in our trained subjects. As seen in Fig. 5, MIF was significantly reduced from baseline at Post-15 (-27%), Post-24 (-13%), and Post-48 (-6%). Incomplete recovery by 48 hours following the exercise bout has previously been reported following multijoint bench press (Smith et al., 1994) and squat exercises (Byrne and Eston, 2002) as well as isolated elbow flexor exercises (Newton et al., 2008). Despite this, we did not note a time main effect for serum creatine kinase activity (Fig. 7). Although we expected this measure of sarcolemma disruption to exhibit such an effect, this may be attributable to creatine kinase being a poor surrogate for the magnitude of muscle injury (Warren et al., 1999; Friden and Lieber, 2001).

Given the absence of a RBE for any measured variable, it is likely that adaptations associated with the RBE are already present in resistance trained men (Bloemer et al., 2007). During the conception of this study, our working hypothesis was that this form of high- volume and load eccentric exercise would elicit unique adaptations in our subjects as they did not experience such stimuli in their own training. This, however, was not supported. Future investigation may

consider including other assessments to more definitively refute the presence of a RBE in trained men, such as histochemical and electron microscopy analyses, as well as examining different muscle groups and/or protocols.

Acknowledgments

The authors would like to thank Ms. Andrea Creasy and Mr. Cory Leatherwood for their assistance in data collection. This work was directly supported by the National Strength and Conditioning Association (NSCA) Graduate Research Grant and Jarrow Formulas. Additional support was provided by Grant Number T32HD007434 from the National Institute of Child Health and Human Development. The content is solely the responsibility of the authors and does not necessarily represent the official views of the NICHD, NIH, NSCA, or Jarrow Formulas.

Biography

Michael Falvo is a Doctoral Candidate in Movement Science with an emphasis in Biocontrol at Washington University in St. Louis. He completed a M.S. in Exercise and Sport Science at the University of Memphis where he studied the response to high load eccentric exercise. His current research efforts involve understanding the supraspinal and spinal effects of resistance training in health and disease.

Brian Schilling is the Exercise Neuromechanics Laboratory Director in the Department of Health and Sport Sciences at the University of Memphis. His primary interests are the acute and chronic mechanical effects of resistance training, in particular for persons with neuromuscular disease. His current research is focused on exercise interventions in persons with Parkinson's disease, as well as technology for providing exercise devices to this population.

Rick Bloomer holds a PhD in Exercise Physiology from The University of North Carolina at Greensboro and is an Assistant Professor within the Department of Health and Sport Sciences at The University of Memphis. His research focus is centered on oxidative stress and frequently involves the use of antioxidant agents.

Webb Smith is an Exercise Specialist and Human Performance Lab Director at St. Jude Children's Research Hospital in Memphis, TN. He completed a Master's degree in Exercise and Sport Science at the University of Memphis. His research interests are cardiovascular function and physiology, exercise prescription for improved performance/function, and physiological adaptations to exercise/physical work in childhood cancer patients and survivors.

References

- Ahmadi S, Sinclair PJ, Foroughi N, Davis GM. Electromyographic activity of the biceps brachii after exercise-induced muscle damage. *J Sports Sci Med* 2007;6:461–70.
- Bigland B, Lippold OC. The relation between force, velocity and integrated electrical activity in human muscles. *J Physiol* 1954;123:214–24. [PubMed: 13131257]
- Bloomer RJ, Falvo MJ, Schilling BK, Smith WA. Prior exercise and antioxidant supplementation: effect on oxidative stress and muscle injury. *J Int Soc Sports Nutr* 2007;3:4–9.
- Byrne C, Eston RG. The effect of exercise-induced muscle damage on isometric and dynamic knee extensor strength and vertical jump performance. *J Sport Sci* 2002;20:417–25.
- Byrne C, Twist C, Eston RG. Neuromuscular function after exercise-induced muscle damage. *Sports Med* 2004;34:49–69. [PubMed: 14715039]
- Chen TC. Effects of a second bout of maximal eccentric exercise on muscle damage and electromyographic activity. *Eur J Appl Physiol* 2003;89:115–21. [PubMed: 12665973]
- Clarkson PM. Eccentric exercise and muscle damage. *Int J Sports Med* 1997;18:S314–7. [PubMed: 9391847]
- Clarkson PM, Hubal MJ. Exercise-induced muscle damage in humans. *Am J Phys Med Rehab* 2002;81:S52–63.
- Falvo MJ, Bloomer RJ. Review of exercise-induced muscle injury: relevance for athletic populations. *Res Sports Med* 2006;14:65–83. [PubMed: 16700405]
- Falvo MJ, Schilling BK, Bloomer RJ, Smith WA. Efficacy of prior eccentric exercise in attenuating impaired exercise performance after muscle injury in resistance trained men. *J Strength Cond Res* 2007;21:1053–60. [PubMed: 18076261]
- Felici F, Colace L, Sbriccoli P. Surface EMG modifications after eccentric exercise. *J Electromyogr Kinesiol* 1997;7:193–202.
- Friden J, Lieber RL. Serum creatine kinase level is a poor predictor of muscle function after injury. *Scand J Med Sci Sports* 2001;11:126–7. [PubMed: 11252462]
- Freriks, B.; Hermens, H.; Disselhorst-Klug, C.; Rau, G. The recommendations for sensors and sensor placement procedures for surface electromyography. Vol. 8. Springer; 1991. p. 101-5.1991
- Howatson G, Van Someren K. Evidence of a contralateral repeated bout effect after maximal eccentric contractions. *Eur J Appl Physiol* 2007;101:207–14. [PubMed: 17534644]
- Howatson G, Van Someren K, Hortobagyi T. Repeated bout effect after maximal eccentric exercise. *Int J Sports Med* 2007;28:557–63. [PubMed: 17373600]

- Linnamo V, Bottas R, Komi PV. Force and EMG power spectrum during and after eccentric and concentric fatigue. *J Electromyog Kinesiol* 2000;10:293–300.
- Lombardi V, Piazzesi G. The contractile response during steady lengthening of stimulated frog muscle fibres. *J Physiol* 1990;431:141–71. [PubMed: 2100305]
- McHugh MP. Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise. *Scand J Sci Med Sports* 2003;13:88–97.
- McHugh MP, Connolly DAJ, Eston RG, Gattman EJ, Gleim GW. Electromyographic analysis of repeated bouts of eccentric exercise. *J Sports Sci* 2001;19:163–70. [PubMed: 11256821]
- Moritani T, Muramatsu S, Muro M. Activity of motor units during concentric and eccentric contractions. *Am J Phys Med* 1988;66:338–50. [PubMed: 3324772]
- Newton MJ, Morgan GT, Sacco P, Chapman DW, Nosaka K. Comparison of responses to strenuous eccentric exercise of the elbow flexors between resistance-trained and untrained men. *J Strength Cond Res* 2008;22:597–607. [PubMed: 18550979]
- Nosaka K, Clarkson PM. Muscle damage following repeated bouts of high force eccentric exercise. *Med Sci Sports Exerc* 1995;27:1263–9. [PubMed: 8531624]
- Noasaka K, Newton M, Sacco P. Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. *Scand J Med Sci Sports* 2002;12:337–46. [PubMed: 12453160]
- Pierrynowski MR, Tudus PM, Plyley MJ. Effects of downhill or uphill training prior to a downhill run. *Eur J Appl Physiol* 1987;56:668–72.
- Semark A, Noakes TD, St Clair Gibson A, Lambert MI. The effect of a prophylactic dose of flurbiprofen on muscle soreness and sprinting performance in trained subjects. *J Sports Sci* 1997;17:197–203. [PubMed: 10362386]
- Sahaly R, Vanderwalle H, Driss T, Monod H. Maximal voluntary force and rate of force development in humans - importance of instruction. *Eur J Appl Physiol* 2001;85:345–50. [PubMed: 11560090]
- Smith LL, Fulmer MG, Holbert D, McCammon MR, Houmard JA, Frazer DD, et al. The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase. *Brit J Sport Med* 1994;28:267–71.
- Viitasalo JT, Saukkonen S, Komi PV. Reproducibility of measurements of selected neuromuscular performance variables in man. *Electromyogr Clin Neurophysiol* 1980;20:487–501. [PubMed: 7250028]
- Warren GL, Lowe DA, Armstrong RB. Measurement tools used in the study of eccentric contraction-induced injury. *Sports Med* 1999;27:43–59. [PubMed: 10028132]
- Warren GL, Hermann KM, Ingalls CP, Masselli MR, Armstrong RB. Decreased EMG median frequency during a second bout of eccentric contractions. *Med Sci Sports Exerc* 2000;32:820–9. [PubMed: 10776902]

Fig. 1.
Investigation timeline.

Fig. 2.
Maximal isometric force (MIF) assessment.

Fig. 3. Perceived muscle soreness: a significant bout $\times$ time interaction was found ($p = .002$). These significant differences were manifested at Post-24 ($d = 1.48^*$) and Post-48 ($d = 1.54^*$). Note that soreness peaked within each bout at Post-24.

Fig. 4. Total neural activity: RMS demonstrated a significant bout $\times$ time interaction ($p = .044$). One-way repeated measures ANOVA revealed that only bout 1 demonstrated a significant difference from Pre to Post-15 ($d = 0.46^+$).

Fig. 5.

Maximal isometric force (MIF): A significant main effect for time was observed for MIF ($p < .0001$). MIF at Pre was significantly higher than all other time points. Compared to Pre, MIF was significantly reduced 27%, 13%, and 6% at Post-15, Post-24, and Post 48, respectively. No differences were observed between bouts.

Fig. 6. Maximal rate of force development (RFD): A significant time main effects was observed for RFD ($p = .001$). RFD was significantly depressed from baseline at Post-15 and Post-24, but recovered by Post-48.

Fig. 7.

Creatine kinase activity: serum creatine kinase activity demonstrated no significant interaction or main effects. Please note that serum samples were obtained immediately after the eccentric protocol (Post-0) which is approximately 15 min earlier than performance measures (Post-15) as shown in Figs. 3-6.

Table 1

EMG parameters: data are presented mean $\pm$ SD. Time main effects were observed for MDF ($p = .001$) and $\mu V/N$ ($p = .032$). Only MDF exhibited significant pairwise differences from Pre to Post-15 ($d = .057^{\dagger}$)

	Bout 1				Bout 2			
	Pre	Post-15	Post-24	Post-48	Pre	Post-15	Post-24	Post-48
MDF	61.9 $\pm$ 11.4	66.1 $\pm$ 11.5	62.7 $\pm$ 12.9	61.7 $\pm$ 11.1	60.1 $\pm$ 9.5	64.4 $\pm$ 8.1	59.5 $\pm$ 9.9	59.4 $\pm$ 7.3
$\mu V/N$	4.1 $\pm$ 2.1	4.6 $\pm$ 2.6	5.1 $\pm$ 3.4	4.7 $\pm$ 3.2	4.5 $\pm$ 3.3	5.5 $\pm$ 4.0	5.0 $\pm$ 3.3	4.6 $\pm$ 2.8