Delayed Onset Muscle Soreness in a Bench Press Exercise

By

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OF

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ABSTRACT

Delayed onset muscle soreness (DOMS) is a common experience, yet the cause of DOMS is poorly understood and the approaches used in research to measure DOMS vary widely. The study objectives were as follows: 1) to explore the contribution of movement strategy, as measured by acceleration, to resulting DOMS; and 2) to perform a systematic comparison (magnitude and time course) of DOMS measures. METHODS: Twenty subjects were randomly assigned to use one of two movement strategies (high acceleration (HA) or low acceleration (LA)) in performing forty repetitions of a sub-maximal (75% concentric 1 RM) bench press. Acceleration of one upper extremity was measured to derive "drop and catch" acceleration magnitudes and high frequency acceleration magnitudes. Pain was evaluated on a series of 10 cm VAS' pre- and immediately post-exercise and at 12 hour intervals to 48 hours. Subjects' responses were measured at rest (R), with two levels of stretch (S1 and S2), and two levels of mechanical pressure (350 kPa with P1 and 139 kPa with P2). Effect of pain on function was also subjectively rated (Func). RESULTS: Maximum and time-specific DOMS magnitude measured by R was significantly lower than all other pain measures ($p<0.05$). Significant differences were found between S1 and S2 ($p<0.05$) but not P1 and P2. Acceleration based movement parameters, especially high frequency acceleration, were found to be significant predictors of pain ($p<0.05$). No significance was found using physical parameters or exercise parameters. CONCLUSIONS: DOMS magnitude and time course varies depending on the measure used and selection of measures should be done with careful consideration. Acceleration parameters are predictive of DOMS magnitude reflecting that within exercise shifts in neuromuscular activation strategy is a contributing factor.

PRÉCIS

This thesis is comprised of an introductory component, including a summary of the background that lead to the research objectives and a detailed description of the methodology. Two complete scientific manuscripts follow which impart the results of the study in the context of recent literature: 1) "A Comparison of Techniques for Measuring Delayed Onset Muscle Soreness After a Submaximal Bench Press Exercise"; and 2) "The Relationship Between Movement Strategy during a Bench Press Exercise and the Magnitude of Delayed Onset Muscle Soreness." Overall contributions and an overview of future directions stemming from this research project are presented following the manuscripts.

INTRODUCTION

Discomfort, soreness or pain resulting from novel eccentric exercise is termed delayed onset muscle soreness (DOMS) and is an experience familiar to virtually all of us through changes in activities of daily living, through leisure time pursuits, and even through rehabilitation. Yet, despite the prevalence of DOMS, the most fundamental question remains unanswered, that is, what mechanism(s) underlie this phenomenon? This question has been explored directly in a number of DOMS studies and reviews (e.g., Armstrong 1984; Jones et al. 1987; Clarkson and Sayers 1999; Grabiner and Owings 2002; Lieber and Friden 2002, Yu et al. 2002) and also indirectly in studies examining the relationship of DOMS to muscle damage indices (e.g., Croisier et al. 1999; Malm et al. 2000; Nosaka et al. 2002a; Pizza et al. 2002), preventative and curative measures (e.g., Paddon-Jones and Quigley 1997; Ernst 1998; Bourgeois et al. 1999; Craig et al. 1999; Barlas et al. 2000; Herbert and Gabriel 2002), factors affecting magnitude and time course (e.g., Newham et al. 1987; Vickers 2001), and the protective effect of previous exercise (Balnave and Thompson 1993; Smith et al. 1994; McHugh et al. 1999; Paddon-Jones et al. 2000; Nosaka et al. 2002b; Chen 2003; McHugh 2003). Despite the relatively large number of studies pertaining to causal factors, underlying mechanisms may not be well understood, perhaps due to the lack of consistency in the types of measures used to assess/quantify DOMS and a lack of control (e.g., range of motion, amount of work) in the application of tissue loads through exercise. There are a substantial number of pain or soreness measures (>20) used in these studies ranging from a VAS rating of "How sore are you" to pain pressure threshold using an algometer applied to the musculotendinous unit. To date there have been a limited number of studies comparing the different types of DOMS measures used.

Two general themes relating to causation of DOMS have been identified, specifically, biomechanical and structural changes in the muscle and underlying neural mechanisms (Lieber and Friden 2002). It is well accepted that unaccustomed eccentric muscle activity induces a substantially greater DOMS in comparison to concentric and isometric activity alone (Asmussen 1956; Newham et al. 1983; Nosaka and Newton 2002a; McHugh et al.

2000). For example, a recent study by Lavender and Nosaka (2005) measured pain with passively imposed extension of the elbow flexors on a 50 mm VAS following a sub-maximal bicep curl exercise session and found peak pain was 14% of maximum for concentric and 78.6% of maximum pain for eccentric, respectively. A number of mechanical aspects of exercise have been examined as potential contributors to the severity of DOMS such as muscle length (increased muscle length results in increased DOMS (Newham et al. 1988), muscle stiffness (increased muscle stiffness associated with greater DOMS (McHugh et al. 1999), sub-maximal versus maximal load (increased load associated with greater DOMS (Nosaka and Newton 2002), and number of repetitions (increased number of repetitions results in greater DOMS (Nosaka et al. 2001).

Recently, Kulig et al. (2001) introduced the examination of movement strategy as a contributing factor in the induction and magnitude of DOMS. Changes in magnetic resonance imaging (MRI) signal intensity in the elbow flexors were compared for two exercise protocols varying in movement velocity (average cadence) during the eccentric contraction (the muscle lengthening velocity was not controlled). Subjects performed a bicep curl exercise with a load established as 60% of their one repetition maximum (1 RM). A within-subjects experimental design was used whereby subjects performed a "slow" protocol on one arm with an eccentric component of 10 seconds and a "fast" protocol on the other with an eccentric component of 2 seconds. The concentric component was 2 seconds for both protocols. Total time of exercise (concentric and eccentric) was the same for both protocols with 3 x 12 (36) repetitions performed in the "fast" protocol (36 x 4 s = 144 s) and 3 x 4 (12) repetitions performed in the "slow" protocol (12 x 12 s = 144 s). DOMS at 24-hours and 48-hours post-exercise was measured. The objective of this experimental design was stated as, to "[maintain] constant velocity between the two protocols". The outcome of the study was that there was no difference between protocols with respect to overall MRI intensity reflecting that there was no difference in muscle "damage"; however, there was selective preferential activation of biceps brachii in the fast protocol and brachialis in the slow protocol relative to other elbow flexors. Fifty-eight percent (7 of 12 subjects) reported DOMS in the arm performing the fast exercise (36 reps at 60% of 1 RM) while none of the subjects reported DOMS in the arm that performed the slow exercise (12 reps at 60% or 1 RM). Based upon this, the authors concluded that movement velocity was a key factor in DOMS generation.

A number of weaknesses were identified in this study. First, in attempting to control velocity between protocols, the arm performing the fast protocol performed three times the number of repetitions, and thus, three times the amount of work (Joules) assuming that the load was constant due to constant weight. Based upon Newtonian mechanics, a constant weight does not necessarily reflect a constant load between subjects or even between repetitions or groups. The authors did not address this issue nor did they measure the actual load. Previous research has shown that the number of repetitions (work) is clearly implicated in the magnitude of DOMS, that is, the more repetitions, the greater the resultant DOMS (Nosaka et al. 2002c). Second, no comment was made in regard to experimental control of range of motion between sides and speeds. Without physical cues (end of range of motion markers), it would be possible that a speed-dependent hypermetria (target overshoot) or hypometria may exist resulting in a difference in range of motion (hence work) between protocols. Furthermore, it was not "constant" velocity (as in "isovelocity" on an "isokinetic" dynamometer) but cadence (average velocity) that was maintained between conditions. Peak velocity and acceleration magnitudes would have been very different between fast and slow protocols and, even more interestingly, different between subjects within the same condition. As such, explanatory factors other than speed may have resulted in the differences that were observed or resulted in increased uncontrolled variability leading to an inability to show differences in overall MRI signal intensity between the two cadence conditions.

Kulig et al. (2001) discussed implications of their findings with respect to exercise prescription and stated, "slower eccentric speed should allow for"…"less rapid loading of the tendon and the musculotendinous junction, thereby decreasing the potentially injurious effects of repetitive motion." This statement clearly alludes to the rate of tissue loading as opposed to the average velocity. The rate of tissue loading is best represented by limb acceleration not average speed. These authors indirectly identified acceleration as a possible contributing factor to DOMS but did not recognize it as such.

Acceleration may be defined as “the change in velocity as a function of time”. Newton’s second law of motion states that force is proportional to acceleration. Therefore, the peak forces acting on involved muscles would be proportionately greater when applying a high acceleration strategy in performing an eccentric activity versus a low acceleration strategy given the same mass. This effect can be experienced during starting and stopping motions and distinct from the point of reaching peak velocity. Based upon these well-established mechanical laws (i.e., Newtonian mechanics), it follows that increased forces resulting from higher accelerations could result in greater DOMS (Nosaka and Newton 2002; Jones et al. 1989; Lieber and Friden 2002); however, the relationship of movement related acceleration to severity of DOMS has not been examined to this point.

Until recently, accelerometry was applied primarily in areas outside the study of human motion. For example, accelerometers were used to evaluate machine vibration in industrial settings (e.g., Ghani, Choudhury, and Husni 2002) and seismic activity in geological engineering (e.g., Bouckovalas, Papadimitriou, and Achilleas 2003). As enhancements in technology have allowed for accelerometers to be designed significantly smaller and lower in cost in recent years, their application in biomechanical research has become more widespread. Accelerometers have been used to describe whole body movements as a means to assess physical activity and metabolic energy expenditure (McMurray et al. 2004; Pate et al. 2004; Mathie et al. 2004).

More pertinent to the current study is the application of accelerometry in the description of body segments in motion. Motion-dependent acceleration, which reflects the gross movement of a body segment through space, is measured and analyzed utilizing the medium frequency band of acceleration data. For example, Webber and Kriellaars (2004) positioned a uniaxial accelerometer over the L4 spinous process of subjects (n =33) to record anterior/posterior acceleration during hip flexion/extension and biceps curl before and after standardized stabilization instruction. Instruction resulted in a substantial reduction in peak lumbar acceleration magnitude. Similarly, Zedka and colleagues (1999) measured lumbar accelerations to examine reflex contributions to limb movement. Flynn et al. (2004) provides another example of this type of application whereby an accelerometer was mounted to the tibial tubercle and measured tibial acceleration at impact while subjects ran on a treadmill.

Recently, several studies have investigated the measurement and analysis of high frequency accelerations (i.e., tremor or motor unit induced vibration) during isometric muscle contraction. This application of accelerometry has established that an acceleration profile, termed a mechanomyogram (MMG) is a valid method for non-invasive assessment of the mechanical properties of motor units (Cescon et al. 2004a, 2004b; Watakabe et al. 2003). Watakabe et al. (2003) states, "a contracting muscle generates a pressure wave owing to lateral dimensional changes in active muscle fibres than can be detected with a vibration transducer on the body surface over-lying the muscle." More recently, Akataki et al. (2004) examined EMG/force and MMG/force relationships simultaneously during isometric ramp contractions in biceps brachii muscle using a uniaxial accelerometer. They concluded the motor unit activation strategy is better represented by the MMG than by the EMG. To date, this application of accelerometry had only been used to examine isometric muscle contraction.

Establishing the basis for the current study, two gaps in knowledge were identified in the literature. First, the relationship between acceleration and DOMS has not been examined to date. In order to address this deficit, we proposed to characterize movement strategy in a bench press exercise through measurement of the high and medium frequency components of acceleration. Exercise parameters known to influence DOMS were controlled (i.e., range of motion, repetitions) and acceleration parameters were used to predict DOMS magnitude. The measurement of high frequency acceleration provided insight into the contribution of neuromuscular control in the production of DOMS. The measurement of medium frequency acceleration provided insight into the contribution of instantaneous force to overall DOMS magnitude. Second, there is a lack of information on the differences in DOMS in terms of magnitude and temporal response of various measures. In the current study, a series of DOMS measures were employed and compared in the broad categories of non-provoking (intensity and function) and provoking (stretch and pressure).

OBJECTIVES

The primary objective of this study was to investigate the relationships between medium and high frequency acceleration and DOMS magnitude. Two groups of subjects performed the bench press exercise, each group with a different movement strategy. One group performed a low acceleration strategy and the other group a high acceleration ("drop-and-catch") strategy.

A secondary objective of this study was to perform a general comparison of different DOMS assessment techniques with respect to magnitude and time course.

METHODS

EXPERIMENTAL DESIGN

This experiment was cross-sectional in design, comparing subjects performing a standardized bench press applying two different acceleration strategies with a control of 1) the range of motion, 2) the number of repetitions (total work) and 3) the relative weight of the barbell. Velocity of movement was measured but not controlled between conditions.

SUBJECTS

Based on the inclusion and exclusion criteria outlined below, twenty subjects were recruited for this study and randomly assigned to one of two groups, one directed to use a low acceleration strategy (LA) and one directed to use a high acceleration strategy (HA) in a bench press exercise session. A sample size analysis was performed prior to recruitment based upon the difference in μ between the LA and HA pain ratings estimated as 2 (VAS) and a σ of 1.2 (based on means and variances reported in previous DOMS studies). The analysis used an α value of 0.05 and β value of 0.10. The minimum study size was calculated to be 8 subjects per group for a total of 16. Twenty subjects were recruited to account for attrition and data loss.

Subjects were requested not to take any anti-inflammatory or pain medications or nutritional supplements during their participation in the study. It was also requested that they not participate in any unaccustomed or vigorous physical exercise during and around the experimental period. Age (years) and sex (M/F) were documented and height (cm) and body mass (kg) were measured.

Inclusion and Exclusion Criteria

The inclusion criteria for this study were that subjects be between 18 and 45 years of age and healthy (i.e., no medical problems within the last 12 months as determined by the completion of a screening questionnaire presented in Appendix A). In order to include only subjects unaccustomed to bench press or related exercise, subjects were not to have been involved in resistance training of the upper extremities or activities with similar loading (e.g., push-ups) in the last 12-months. Beyond the definition of "healthy" as presented in the inclusion criteria, all those who had a history of injury to or had restriction in range of motion in their upper extremities were excluded. No pregnant or lactating women were accepted in this study.

Recruitment

Subjects were recruited by word of mouth. Subjects were not reimbursed for their participation in this study and were not responsible for any costs directly related to their involvement in the study.

Selection

Allocation of subjects to each of the two study groups was carried out using a pre-generated random number table resulting in an equal number of subjects in each group.

Informed consent and Ethical Approval

Subjects were required to read the study paraphrase (Appendix B) and then provide written consent prior to their participation. The Faculty of Medicine Human Ethics Committee granted ethical approval for this protocol (H2003:095).

INSTRUMENTATION AND MEASURES

Exercise equipment

A flat exercise bench and an Olympic barbell with assorted plates (masses measured using a scale Tanita BWB-800) were used to perform the bench press exercise. Figure 1 illustrates the set up for the bench press including the exercise equipment, instruments, and subject positioning.

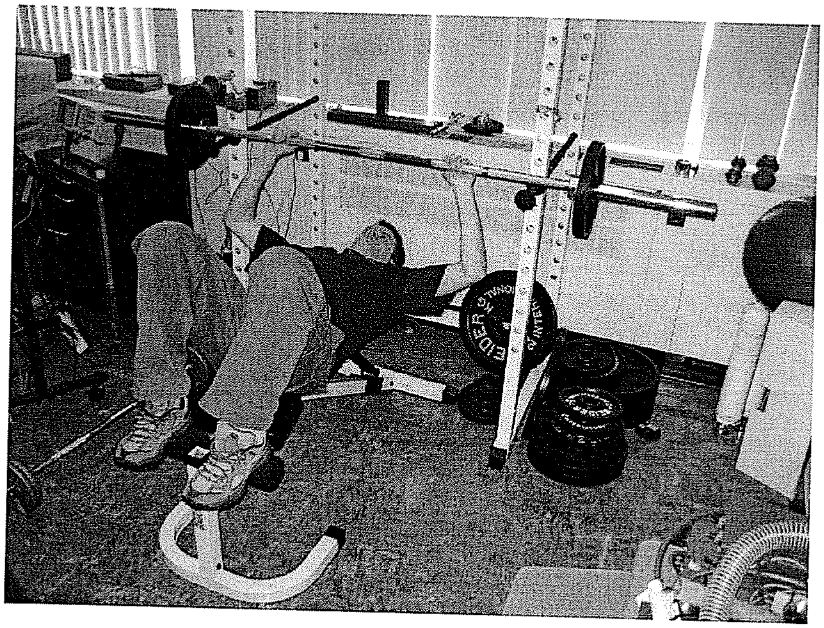

Figure 1. Exercise equipment and subject positioning for bench press.

Accelerometer and Data Acquisition

A calibrated uniaxial accelerometer (Entran EGAX piezoelectric linear accelerometer, 10g range) was affixed with tape to the posterior surface of the subject's fourth finger on the middle phalanx of the dominant hand (Fig. 2). Such positioning of the accelerometer allowed for measurement of acceleration in the vertical plane (coincident with the movement plane).

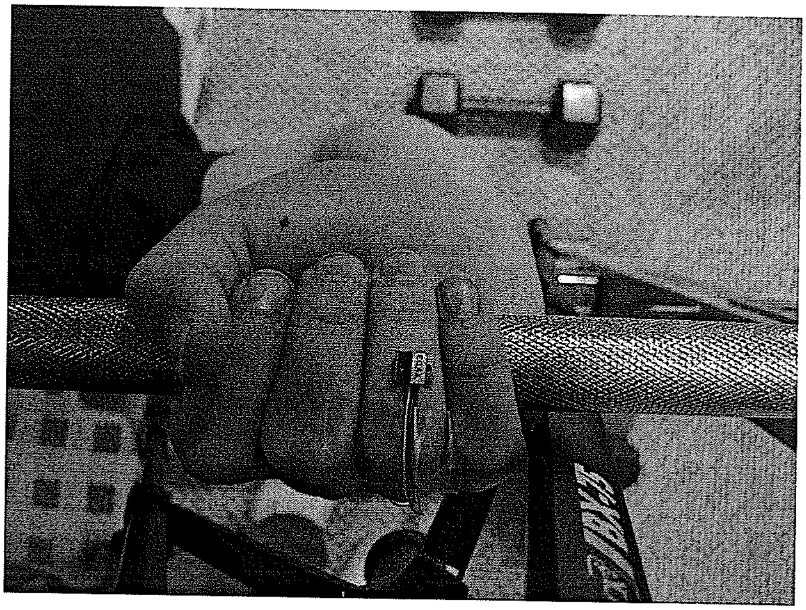

Figure 2. Placement of accelerometer on fourth finger of subject's dominant hand.

The accelerometer was connected to an amplifier with adjustable gain and offset control. The amplified accelerometer signal was acquired through a computer-based data acquisition system (Data Translation 9800) and was also visually displayed on an oscilloscope (Tektronix PM-3335 Digital Storage Oscilloscope) (Fig. 3).

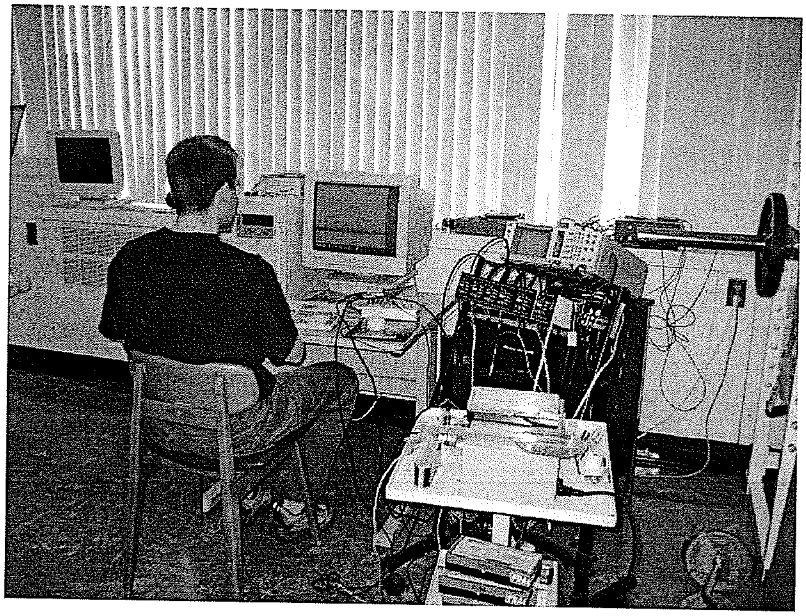

Figure 3. Data acquisition system including computer, amplifier and oscilloscope.

The accelerometer was calibrated at the beginning of each experimental session using the accelerometer orientation method (Lewis 2002; Webber and Kriellaars 2004). The amplified accelerometer signal (voltage) for each of three reference positions of the accelerometer was recorded: horizontal with gravitational acceleration equal to -1.0 g (-9.8 m/s^2), inverted with an acceleration of +1.0 g (+9.8 m/s^2), and on its side with gravitational acceleration equal to 0 g (Fig. 4). A scale factor and zero "g" offset value were derived from these values and were used to calibrate the acceleration profiles collected.

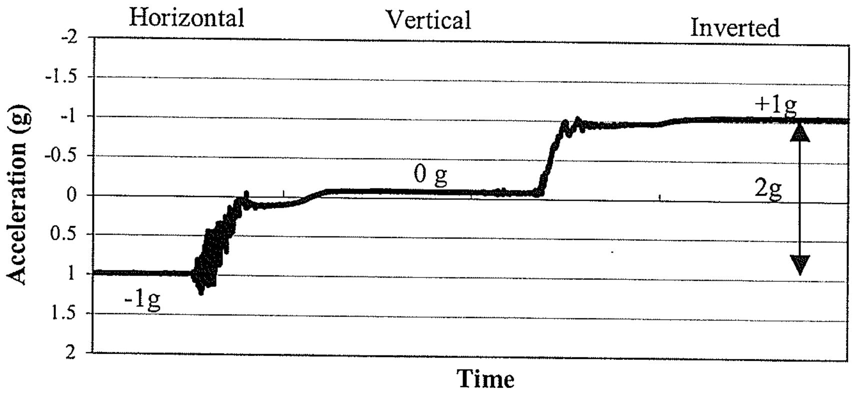

Figure 4. Calibration trial illustrating accelerations when the accelerometer is in the horizontal (-1 g), vertical (0 g), and inverted (+1g) position.

The accelerometer signal was collected at 200 Hz (Data Translation DT9800, 16 bit). Amplitude spectral density distributions derived from FFT of acceleration data show that the frequency content of acceleration in human muscle contraction ranges between 0 and 20 Hz (Watakabe et al. 2003). The Nyquist Frequency, that is, the minimum sampling frequency to preserve the frequency content of an original signal, is nominally twice the rate of the highest frequency contained in a signal being sampled (40 Hz in this case). To be able to re-create the amplitude characteristics, a sampling rate of 10 times the highest expected frequency is required. The rationale for a sampling frequency of 200 Hz was based on a value 10 times the highest frequency component resident in the accelerometer signal resulting from human movement (20 Hz).

Figure 5 presents an acceleration profile of one set of ten repetitions of the bench press movement. This acceleration profile can be separated into its frequency components using digital filtering (2^{nd} order Butterworth digital filter) into three relative frequency ranges: low, medium, and high.

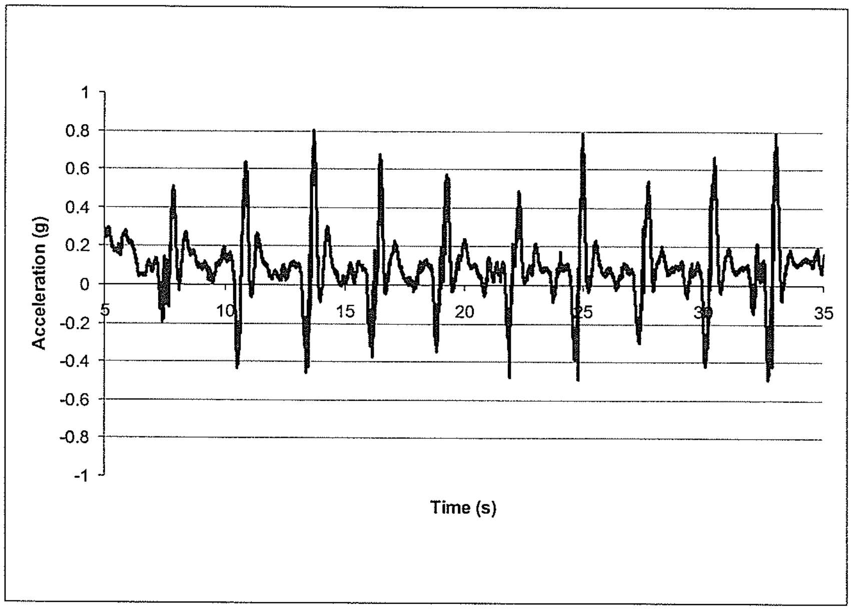

Figure 5. A representative calibrated acceleration (g, where 1 g = 9.8 m/s2) signal during ten repetitions of a bench press exercise.

Low frequency component of acceleration: Low frequencies (DC to 0.2 Hz) reflect a change in orientation of the accelerometer to the vertical axis. In the example of a "linear movement" such as a bench press with an accelerometer mounted to the subject's finger, low frequency accelerations reflect the rotation of the hand and bar as the movement is performed. With respect to this study, low frequency accelerations were filtered out in view of the fact that rotation of the barbell (dependent on the orientation of the subject's hands relative to the vertical axis) had no relevance to the study outcome.

Medium frequency component of acceleration: Medium frequencies (0.2 – 4.5 Hz) reflect the frequency of the gross repetitive motion of the accelerometer (see below for nomenclature defining phases of motion using the medium frequency signal component). This medium frequency component (the largest amplitude component of the acceleration signal) defined the basic kinematics of motion of the barbell. The medium frequency component of acceleration is termed the gross movement related acceleration. A band-pass filter, which combines a high-pass filter (a filter that maintains frequencies above a defined cutoff) with a cutoff frequency of 0.2 Hz and a low pass filter (maintaining frequencies below a defined cutoff) set to 4.5 Hz, were applied to extract the gross movement related accelerations.

Previously developed standardized nomenclature was used to describe phases of motion within the medium frequency component of the acceleration profile (Lewis 2002, Webber and Kriellaars 2004). Four major phases for each repetition can be identified and are illustrated in Figure 6. Phase 1 ($P1_{acc}$) begins at the initial extended arm position with zero velocity and zero acceleration and ends at the next zero acceleration crossing point. $P1_{acc}$ reflects the eccentric positive-acceleration phase with the bar increasing in velocity as it is lowered to the chest. Phase 2 ($P2_{acc}$) begins at the point at the zero crossing point and reflects the negative acceleration phase of the eccentric component in which the bar bell begins to "decelerate" as it is lowered to the chest. Phase 3 (P3) reflects the positive acceleration phase of the barbell away from the chest (increasing upward speed) and Phase 4 (P4) represents the "deceleration" of the bar as it is extended fully away from the chest. The four phases as depicted in Figure 6 are based on a pause being taken between eccentric and concentric components of the movement, as well as a pause between repetitions that corresponds to baseline or zero acceleration. The "drop" phase corresponds to $P1_{acc}$ and the "catch" phase to $P2_{acc}$.

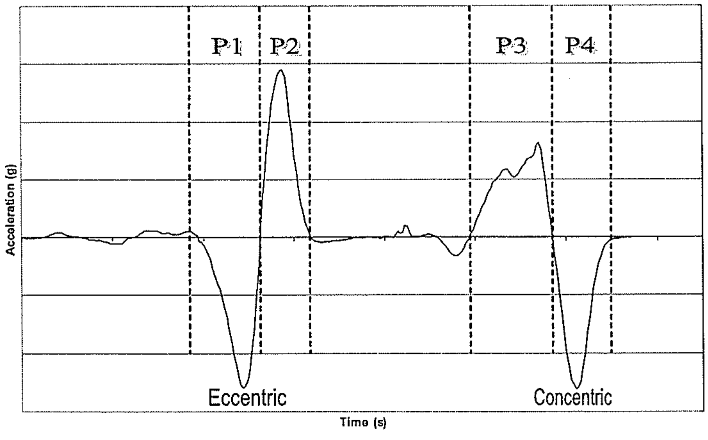

Figure 6. Phases of motion defined using the medium frequency acceleration component for a single repetition of a bench press with a pause at mid-point of the movement.

High frequency component of acceleration: The high frequency component of the signal (> 6 Hz) was also extracted through the application of a high-pass filter with a cutoff frequency set to 6 Hz to preserve frequencies above 6 Hz and eliminate the motion related components (medium frequency). The mean high frequency magnitude was calculated for the entire forty repetitions as well as for each set using the absolute values of the high frequency component across the eccentric component. In the current study, high frequency acceleration magnitude was measured in a dynamic condition and was used as an overall neuromuscular control strategy indicator as an indirect MMG (Akataki et al. 2004). Unpublished work in our lab reveals a high correlation ($r^2=0.8$ or higher) between limb-based high frequency accelerations (indirect MMG) and high frequency accelerations derived from application of an accelerometer to the primary muscle(s) involved (direct MMG).

Movement Parameters

The following variables were derived from the filtered acceleration profiles for each set (1-4) of bench press:

- Peak $P1_{acc}$ acceleration magnitude (m/s^2) derived from the medium frequency component ($P1_{acc}$-1 to $P1_{acc}$-4);
- Peak $P2_{acc}$ acceleration magnitude (m/s^2) derived from the medium frequency component ($P2_{acc}$-1 to $P2_{acc}$-4);
- Average magnitude of high frequency acceleration (m/s^2) (HF1 to HF4)
- Average repetition duration (sec) $P1_{acc}$ to $P1_{acc}$ duration; and
- Average eccentric duration (sec) demarcated as the duration of $P1_{acc}$ and $P2_{acc}$.

From the duration values, total exercise time (sec), total eccentric exercise time (sec), and percent of total exercise time in eccentric activity (%) were also determined.

Pain/Discomfort Measures

All measures of pain were based on visual analogue scales (VAS) with subjects being asked to mark an 'X' on a 10 cm (resolution of 1 mm) vertical line as their response.

Pain measures used in this study were subdivided into two categories, those that measured pain without any provocation and those with provocation. Within the latter category, two methods of provocation were used, stretch and mechanical pressure. The complete list of pain measures is as follows:

- NO PROVOCATION
  1. Pain at Rest (R)
  2. Effect of pain on the ability to function (Func)
- PROVOCATION
  - Stretch:
  3. Pain with stretch (S1)
  4. Pain with enhanced stretch (S2)
  - Mechanical pressure:
  5. Pain with pressure from pressure algometer (PA)
  6. Pain with self-applied finger pressure (P1)
  7. Pain with pressure from custom algometer (P2)

For all but the question regarding function, the VAS anchor terms were "no discomfort or pain" and "extreme discomfort or pain." "Pain at rest" (R) was measured with subjects seated with their hands resting on their lap. For the question regarding function (Func), subjects were asked to rate the impact of the exercise session discomfort on their ability to function with "completely incapacitated" as the anchor term at the upper end and "no effect" at the lower end.

"Pain with stretch" (S1) was measured with subjects positioned lying near the edge of a plinth in supine and being asked to extend their arm straight out to the side and then allow the torque produced by the weight of their limb to stretch their upper chest region (no active force was applied by the subject or the investigator). "Pain with stretch and added weight" (S2) was measured as in S1 but the subjects held a 3 kg mass in their hand to increase the torque due to weight. Immediately following S1, pectoralis major was land-marked just superior to the base of the axilla.

For “Pain with pressure algometer” (PA), the subject was positioned in supine with their dominant arm supported and 350 kPa pressure with an application slope of 50 kPa/s was applied with a Somedic® Type II Algometer with a probe area of 2 cm^2 to the tendon of pectoralis major. The algometer was calibrated prior to each session of data acquisition. As per the manufacturer’s manual, the algometer was placed on a flat surface with the pressure sensor pointing up and the 1 cm^2 probe mounted. A cylindrical brass weight is provided with the algometer and was placed over the probe. A resultant display of 100 kPa±3% indicated the algometer was properly calibrated.

Figure 7. Somedic® Type II algometer used to apply specific amount of pressure to the skin overlying the pectoralis major tendon.

The pressure algometer parameters for this study were chosen based on a pilot study that established that a moderate level of discomfort would be provoked in most individuals. This pressure served as a frame of reference for the level of pressure subjects were asked to replicate for the next measure, “Pain with self-applied finger pressure” (P1). P1 was measured with subjects being asked to re-create the previously applied algometer pressure using their third finger on the land-marked site. The final measure of pain was “Pain with pressure from a custom algometer (P2). In this study, it was not feasible to provide each subject access to a Somedic® algometer for follow-up at home nor to have subjects return at each follow-up time point. The inclusion of a measure using a standardized amount of pressure on palpation was deemed important so a “take-home” algometer was created (Figure 8). The pressure (139 kPa) was standardized by using a wooden dowel (2.11 cm^2 surface area) covered with a soft cloth attached to a carton filled with a known mass of sand. Subjects were positioned as in P1 and asked to apply the custom algometer to their dominant side with their non-dominant hand supporting the custom algometer without exerting additional pressure.

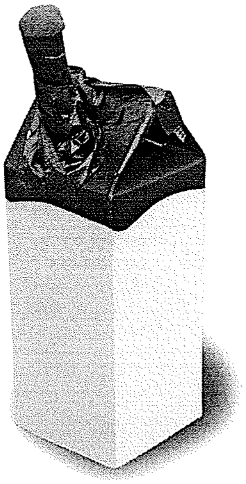

Figure 8. Custom algometer used to apply pressure to the skin overlying the pectoralis major tendon.

Pain/discomfort following exercise was categorized into post-exercise pain and DOMS. Post-exercise pain was defined as a significant difference between pre- and post-exercise VAS ratings and DOMS was defined as a significant difference between pre- and 12-48 hour measures of pain. Maximum DOMS was derived using the highest DOMS response between 12- and 48-hours.

Rating of Perceived Exertion

A standard 6 (no exertion at all) to 20 (maximal exertion) point Rating of Perceived Exertion (RPE) scale was used (Borg 1998) to assess effort required to perform the exercise.

EXERCISE PROTOCOL

Subjects completed a screening assessment/questionnaire, were weighed (mass in kg), and their height (cm) measured. Subjects were asked, prior to performing any exercise, to complete the pain questions.

The subject then observed the investigator describing and demonstrating the exercise to be performed. Those in the LA group were shown the bench press in a controlled manner with low acceleration. For those in the HA group, the bench press was performed with relatively high acceleration in a "drop and catch" manner. A brief pause between eccentric and concentric components of the bench press, as well as between repetitions, was demonstrated in both protocols.

Bench Press Exercise

Subjects were positioned lying supine with their feet on a support. They were asked to horizontally abduct their shoulders. The distance between their hands almost at end range was measured and then their hands were positioned on the barbell (shoulder width grip) to emphasize recruitment of pectoralis major through maximizing horizontal abduction/adduction in the movement rather than triceps brachii through shoulder and elbow flexion/extension (narrow grip). They were then requested to perform a set of ten repetitions of the bench press exercise with only the bar (10 kg for women, 20 kg for men) using the technique as demonstrated. Within-subject range of motion was controlled as they were asked and shown to raise the bar to almost full extension of their elbows and to lower the bar close to but not touching their chest. This "warm-up" allowed for verbal correction of their technique with relatively low load to ensure the bench press was performed with the appropriate acceleration features consistent with the overall strategy for their group assignment.

Weight Determination

The weight mounted on the barbell for the bench press trial was 75% of the subject's concentric one repetition-maximum (1 RM). In several studies, it has been shown that concentric-only training does not result in muscle damage or DOMS (Armstrong et al. 1991; McHugh et al. 1999; Nosaka and Newton 2002b). Therefore, only the concentric component of the bench press was performed in the 1 RM determination process.

The amount of weight to be mounted on the barbell initially was estimated based on sex, body weight, and estimated fitness level (ascertained through the screening process). Two assistants un-racked the barbell and assisted the subject to lower the weight to just above their chest. The subject performed the concentric component of bench press to fatigue while maintaining proper technique. The assistants lowered the barbell to the patient's chest with the completion of each repetition such that only the concentric component was performed by the subject. The weight was adjusted accordingly until the subject was only able to complete one repetition. Ninety-second rest periods were imposed between attempts. The investigator recorded the amount of weight applied to the barbell and the number of repetitions completed with each attempt. 1 RM was determined for each subject within three to five attempts of the 1 RM trial.

Exercise Sets and Repetitions

Once the 1 RM process was complete, seventy-five percent of the 1 RM was determined and became the load for the bench press trial. Subjects were then instructed to perform repetitions to fatigue with this weight while maintaining proper technique and their assigned acceleration strategy. Sets separated by 90-second rest periods were repeated until forty (40) repetitions were completed.

Based on preliminary trials, it was expected that approximately eight to twelve repetitions would be completed in each set so that four to five sets would be performed, and that this would be an adequate amount of exercise to illicit measurable DOMS. Prior to the study, it was decided that if a subject was not able to achieve at least seven repetitions in the first set performed or could not perform 40 repetitions within seven sets, their results would not be included in this analysis as the weight chosen as an approximation of their 10 RM value was overestimated. Conversely, if the required 40 repetitions were performed in less than three sets, the 10 RM value was underestimated and these results would be excluded. Two of the 20 subjects recruited for this study had to be excluded on this basis (one subject performed 40 repetitions in two sets, the other in nine sets).

Immediately upon completion of the trial, subjects were asked to rate their level of exertion during this task on an RPE scale. Questions regarding pain were repeated including palpation with the algometer. Subjects were then provided with a package containing four more sets of VAS and a custom algometer to be used for follow-up approximately 12-, 24-, 36- and 48- hours post-exercise.

Measurement Sequence

All seven pain measurements were taken prior to and immediately following the exercise session in the lab. RPE was also measured immediately post-exercise. Subjects were then given a package containing four more sets of VAS' to be completed at home at 12-hour intervals up to 48 hours post-exercise (half an hour after waking in the mornings and before bed). A custom-algometer was provided for home assessment and was used as the weight in S1 and for pressure application in P2.

For the follow-up period, subjects were asked to respond to the questions on the VAS' provided as well as record the time of day and then put their responses in an envelope upon completion (prior to responding to the next question) to avoid self-monitoring of their levels of pain. Subjects recorded their responses on separate VAS' for each question. At the end of each response session, subjects were asked to seal the envelope and forward the entire series of envelopes to the investigator at the conclusion of the 48-hour period.

RESULTS

The results of this research study are presented below in the form of two manuscripts. The first manuscript compares the magnitude and time course of the spectrum of DOMS measures described above and is entitled **"A Comparison of Techniques for Measuring Delayed Onset Muscle Soreness After a Submaximal Bench Press Exercise."** The focus of the second manuscript is the examination of the relationship between movement strategy measures and DOMS and is entitled "**The Relationship Between Movement Strategy during a Bench Press Exercise and the Magnitude of Delayed Onset Muscle Soreness.**"

A Comparison of Techniques for Measuring Delayed Onset Muscle Soreness After a Submaximal Bench Press Exercise

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Keywords: pain, eccentric exercise, VAS, DOMS

INTRODUCTION

Delayed onset muscle soreness (DOMS), the pain or discomfort resulting from performing novel eccentric exercise, is an experience familiar to most people; yet, a clear understanding of the cause of this phenomenon remains elusive (Lieber and Friden 2002). The scope of current research on DOMS is broad including the study of underlying mechanisms, preventative and curative measures, factors affecting magnitude and time course, and the protective effect of previous exercise. One issue that has impacted our ability to gain an understanding of the etiology of DOMS is the methods used to assess DOMS.

The spectrum of measures used in DOMS include some that are quite vague (e.g., visual analog scale (VAS) rating of "how sore are you?") or for which no detail is given (e.g., Hart et al. 2005; Kulig et al. 2001)). A commonly used measure that incorporates some form of specific provocation is a VAS rating of soreness resulting from digital palpation by either the subject or the investigator (e.g., Nosaka et al. 2002; Zainuddin et al. 2005a; Sayer and Clarkson 2000). Referring to digital palpation by an investigator, one study stated that "since the investigator was experienced in this procedure, this palpation soreness assessment was considered to be as reliable" as two other procedures based on VAS ratings of pain and range of motion (Nosaka et al. 2002). However, measurement of the amount of pressure applied and ensuring that this pressure is consistent between patients and between time points is difficult. To address this lack of control, some studies include a commonly used measure in pain research, pain pressure threshold (PPT) whereby a quantifiable amount of pressure is applied and subjects indicate the point at which pressure becomes discomfort (e.g., Hamlin and Quigley, 2001; Paddon-Jones and Abernathy 2001; Hjortskov et al. 2005). A major limitation of PPT is that it requires patients to be present at each follow-up time point, which can make frequent or multiple follow-ups cumbersome or prohibitive. Other less frequently used measures include pain with ROM or during a functional task (e.g., with walking or squatting), pain mapping, and the McGill questionnaire (e.g., Madeleine et al. 2006; Barlas et al. 2000).

Interestingly, although a majority of DOMS studies include more than one measure of DOMS, only general trends or patterns are noted with few including a systematic and comprehensive evaluation of time course and magnitude of DOMS measures.

The objective of this study was to compare differences in magnitude and time course of pain or discomfort at rest and using two methods of provocation (stretch and pressure) after performance of a bench press exercise.

METHODS

Subjects

Twenty healthy adults participated in this study, 11 female and 9 male. Their mean (SD) age, height, and body mass were 32 (8.2) yr, 170 (10.1) cm, and 75.4 (9.1) kg, respectively. Upper extremity length (cm) was also measured from the lateral aspect of the acromion to the third metacarpophalangeal joint. To ensure bench press was, in fact, a novel exercise to the subjects, only those who had not been involved in resistance training of the upper extremities or activities with similar loading (e.g., push-ups) in the last 12-months were included in the study. All those with a history of injury to or had restriction in range of motion in their upper extremities were also excluded. Prior to their participation, all subjects signed a written informed consent according to a protocol approved by the Faculty of Medicine Human Research Ethics Board of the University of Manitoba. Subjects were asked not to take anti-inflammatory or pain medications or nutritional supplements nor participate in any unaccustomed or vigorous physical activity during the experimental period.

Exercise Protocol

Subjects were positioned lying supine on a flat exercise bench with their feet supported and their spine in a neutral position. Their hands were positioned on an Olympic barbell at a distance equivalent to the distance between their hands when their shoulders were horizontally abducted to 90° and elbows were flexed to 90°. This position emphasized recruitment of pectoralis major through maximizing horizontal abduction/adduction in the movement rather than triceps brachii through shoulder and elbow flexion/extension. A uniaxial accelerometer was mounted on the subject's middle finger and was used to measure the kinematics of the bar. The subject performed a set of ten repetitions of bench press with only the bar (women – 10 kg; men – 20 kg). The subjects were asked to raise the bar to almost full extension of their elbows and to lower the bar close to, but not touching, their chest (the exercise was described and demonstrated before the subject was positioned on the bench). This initial set served as a "warm-up" and also allowed for further instruction in proper technique to ensure safety and consistency between subjects.

The selected mass mounted on the barbell for the bench press trial was 75% of the subject's concentric one repetition-maximum (1 RM). For the 1 RM determination the subject performed the bench movement through the concentric phase while spotters "unweighted" the barbell through the eccentric phase. Based on previous studies, concentric-only training does not appear to result in severe muscle damage or DOMS (Armstrong et al. 1991; McHugh et al. 1999; Nosaka and Newton 2002a). The exclusion of the eccentric component of the 1 RM test ensured that the impact on resultant DOMS would, thus, be minimal. All 1 RM values were found within 3 to 5 attempts.

Once 75% of the subject's 1 RM was mounted on the bar as the trial load, subjects were instructed to perform a set of repetitions of the same bench press exercise to fatigue while maintaining proper technique. Sets were separated by 90-second rest periods and were continued until a total of forty repetitions were completed. The load and repetitions for this study reflect a workload commonly prescribed in exercise programs and, in preliminary trials, were found to elicit measurable DOMS. In order to control for the relative load between subjects, it was decided, prior to the study, that subjects that did not complete the required 40 repetitions within 3 to 7 sets would be excluded. Two subjects were excluded on this basis leaving the outcomes of 18 participants to be included in the analysis.

Pain Measures

All measures of pain were based on visual analogue scales (VAS) with subjects being asked to mark an 'X' on a 10 cm (resolution of 1 mm) vertical line as their response. "No discomfort or pain" and "extreme discomfort or pain" were used as the anchor terms. Pain measures were subdivided into two categories, those that measured pain without any provocation and those with provocation, specifically, stretch and mechanical pressure. Within the "No Provocation" category, "Pain at rest" (R) and "Effect of pain on ability to function" (Func) were measured. R was measured with subjects seated with their hands resting on their lap. For Func, subjects were asked to rate the impact of the discomfort resulting from the session on their ability to function with "Completely incapacitated" as the anchor term at the upper end and "No effect" at the lower end.

There were two degrees of provocation used to quantify the impact of stretch on pain following exercise. "Pain with stretch" (S1) was measured with subjects positioned lying near the edge of a plinth in supine and being asked to extend their arm straight out to the side and then relax and allow the moment of weight of their upper limb extend the limb to stretch their upper chest region (no active force was applied by the subject or the investigator). "Pain with enhanced stretch" (S2) was measured as in S1 but the subjects held a 3 kg mass in their hand to increase the moment of weight of the upper limb.

To examine the impact of mechanical pressure using different modes and degrees of application, three measures were taken. With the patient positioned in supine with their dominant arm supported, a pressure of 350 kPa pressure with an application slope of 50 kPa/s was applied to the previously land-marked tendon of pectoralis major with a calibrated Somedic® Type II Algometer with a probe area of 2 cm^2. The pressure algometer parameters were chosen based on a pilot study that established that a moderate level of discomfort would be provoked in most individuals. Subjects reported the degree of pain experienced on one of the VAS', "Pain with pressure from pressure algometer" (PA). This pressure also gave subjects a frame of reference for replicating a specified amount of pressure for the next measure, "Pain with self-applied finger pressure" (P1).

P1 was measured with subjects being asked to re-create the previously applied algometer pressure using their third finger on the land-marked site. The final measure of pain was "Pain with pressure applied with custom designed algometer" (P2). In this study, it was not feasible to permit subjects to take home an algometer or to do have subjects return at each follow-up time point. The inclusion of a measure using a standardized amount of pressure on palpation was deemed important so a take-home algometer was created using an carton filled with 3 kg of sand with a dowel extending from the carton covered in soft cloth as the probe (2.11 cm^2 surface area). The pressure applied using this instrument was calculated to be 139 kPa. Subjects were positioned as in PA and asked to apply this "custom algometer" to their dominant side with their non-dominant hand supporting the carton without exerting additional pressure.

All seven pain measurements were taken prior to and immediately following the exercise session in the lab. Subjects also indicated their rating of perceived exertion (RPE) on a standard 6 (no exertion at all) to 20 (maximal exertion) point scale immediately following the performance of the bench press exercise (Borg 1998). Subjects were then given a package containing four more sets of VAS' to be completed at home at 12 hour intervals up to 48 hours post-exercise (half an hour after waking in the mornings and before bed). The measure using the Somedic® algometer was not included for these follow-up time points. A custom-algometer was provided for home and was used as the 3 kg mass in S1 and for pressure application for P2.

For the follow-up period, subjects were asked to respond to the questions on the VAS' provided as well as record the time of day and then put their responses in an envelope upon completion (prior to responding to the next question) to avoid self-monitoring of their levels of pain. At the end of each response session, subjects were asked to seal the envelope and forward the entire series of envelopes to the investigator at the conclusion of the 48-hour period.

Pain following exercise was categorized into post-exercise pain and DOMS. Post-exercise pain was examined using pre- and post-exercise VAS ratings only. DOMS was examined based on pre-exercise ratings and each of the four ratings taken from 12 to 48 hours post-exercise. Maximum DOMS was derived using the highest DOMS response between 12- and 48-hours.

Statistical Analyses

Main effects of time and of pain measure used were determined using one-way repeated measures ANOVA. When indicated, post hoc tests were used to detect differences between the time points within measures and also between measures. The relationship between maximal pain for each measure and RPE was evaluated using Pearson correlation. Significance level was set at $p<0.05$ for all analyses.

RESULTS

Comparisons of Maximum DOMS

Figure M1-1 presents the maximum magnitude of pain experienced between 12 and 48 hours post-exercise averaged across all subjects. Maximum magnitude (Mean (SE)) of DOMS differed between measures with R eliciting the lowest pain response of 1.6 (0.5) and P2 the highest response of 5.0 (0.7).

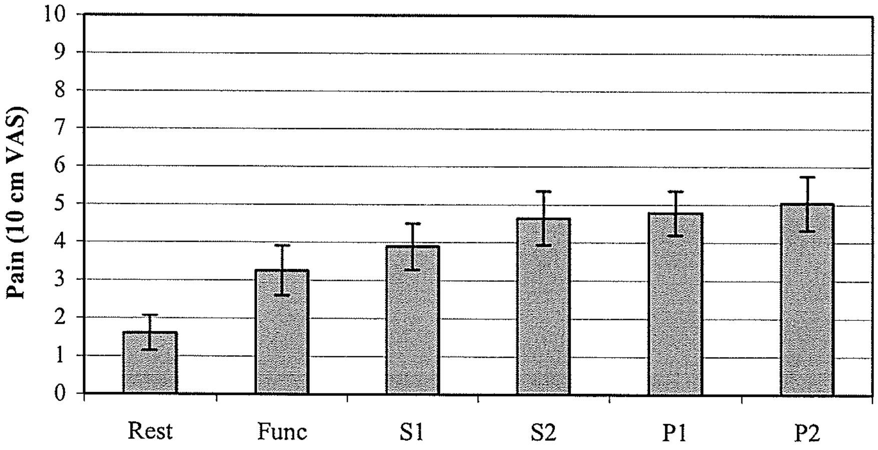

Figure M1- 1. Maximum magnitude of pain experienced between 12 and 48 hours post-exercise averaged across all subjects (significant differences discussed in text).

Maximum R was significantly lower than all measures incorporating a method of provocation. When comparing between methods of provocation, maximum DOMS as measured by S1 at 3.9 (0.6) was significantly lower than both P1 at 4.8 (0.6) and P2 at 5.0 (0.7), which were based on mechanical pressure. However, S2 at 4.6 (0.7) was not significantly different from P1 or P2.

Comparing within methods of provocation, the mean moment of the weight of the arm for S1 was estimated as 7.7 (0.3) Nm while the moment of the weight of the arm with the 3 kg additional mass for S2 was 18.7 (0.4) Nm. This additional load was reflected in a significant increase in the degree of pain (see Figure M1-1) experienced between the two levels of stretch ($p<0.004$) with S1 at 3.9 (0.6) significantly less than S2 at 4.6 (0.7). In contrast, the two chosen levels of mechanical pressure used (139 and 350 kPa for P1 and P2, respectively) were not found to illicit significantly different pain responses ($p<0.334$) with P1 at 4.8 (0.6) and P2 at 5.0 (0.7).

The maximum effect of pain on function as measured by Func (3.3 (0.7)) was similar in magnitude to S1 and was significantly greater than R. Maximum Func was significantly less than S2, P1, and P2.

Comparisons of DOMS at Measurement Interval

Figure M1-2 depicts the pain responses to the bench press exercise over time (immediate-post, 12, 24, 36, and 48) for each of the assessment techniques. R was significantly less than all DOMS measures using provocation at all time points except for S1 at 12 hours post-exercise. With respect to differences between stretch and pressure, S1 was less than both measures using pressure at all time points. S2 and P1 were significantly different at 12 hours. Beyond this comparison, however, no other significant differences between S2 and either measure of DOMS with pressure were identified.

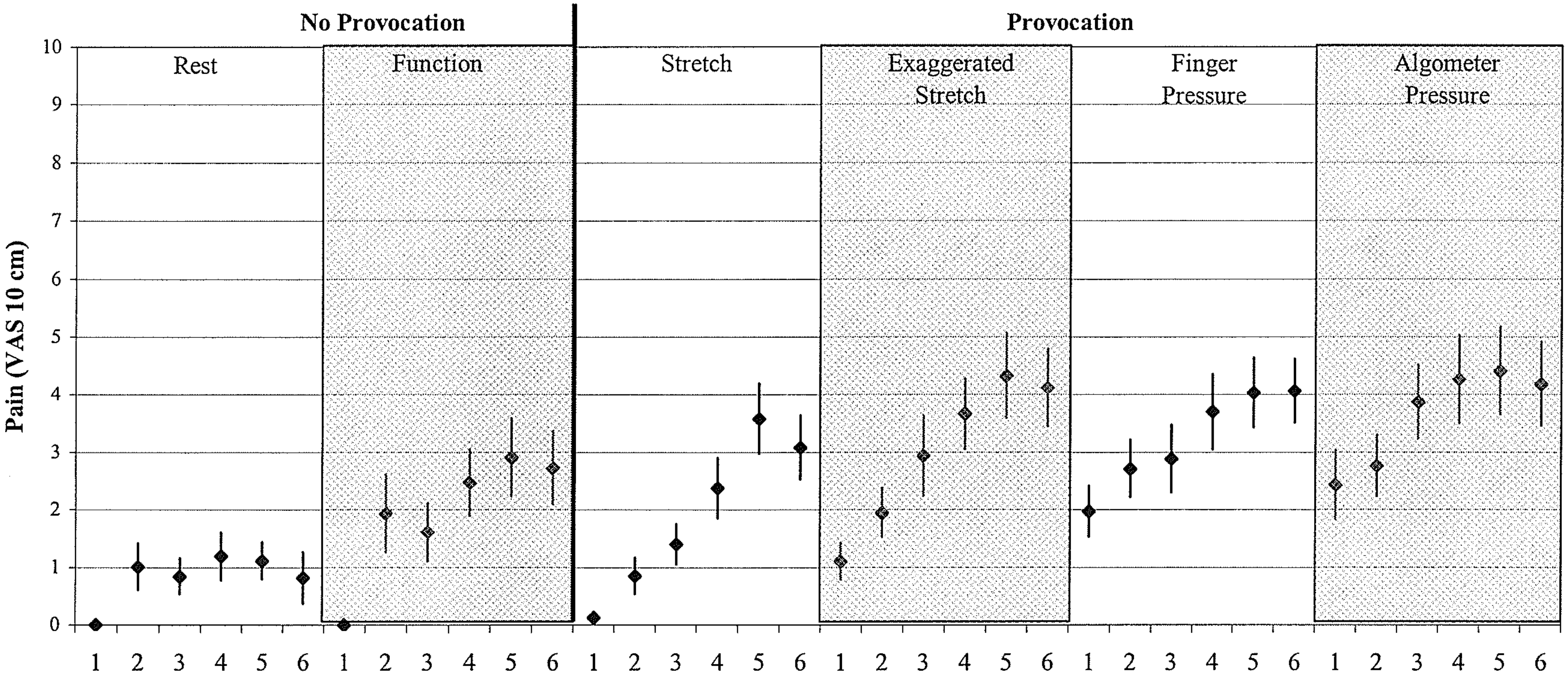

Figure M1- 2. Mean pain (±SE) reported for each measure across follow-up period (1=pre-exercise; 2=immediately post-exercise; 3=12 hours post-exercise; 4-=24 hours post-exercise; 5=36 hours post-exercise; 6=48 hours post-exercise.

Comparing DOMS between stretch measures, S1 was found to be significantly less than S2 at all time points re-confirming the finding described above with respect to maximum values that DOMS appears to be stretch-dependent. DOMS was not found to differ between the two degrees of pressure with no significance found between P1 and P2 at any time point.

Func was significantly different from all other measures at all time points with the exception of R at 12 hours and S1 at all time points.

Pre- and Post-Exercise Pain

Pre- and post-exercise, R was significantly less than all other measures using provocation. Post-exercise R at 1.0 (0.4) was significantly greater than pre-exercise R at 0 (0). Comparing discomfort with stretch, S1 was significantly less than S2 both pre- and post-exercise. Post-exercise values for both S1 and S2 at 0.9 (0.3) and 1.9 (0.4), respectively were significantly greater than pre-exercise at 0.1 (0.1) and 1.1 (0.3).

A comparison of pre- and post-exercise response to mechanical pressure was conducted between using the Somedic® algometer, self-applied finger pressure, and the custom-algometer (Figure M1-3). Pre-exercise, there were no significant differences between any of these modes of pressure application with P1 at 2.0 (0.5), P2 at 2.4 (0.6) and PA at (2.7 (0.7)). Post-exercise, there were no differences in pain response between finger and custom algometer pressure (P1 at 2.7 (0.5) and P2 at 2.8 (0.5)); however, pain with the Somedic® algometer (PA at 3.7 (0.7)) was significantly greater than either of the other measures (350 kPa versus 139 kPa and unknown for the finger palpation). None of the measures of post-exercise pain based on mechanical pressure were significantly different from pre-exercise values.

Pre-exercise, Func was less than all measures based on provocation and equal to R (0 (0)). Post-exercise, Func at 1.9 (0.7) was significantly different from any post-exercise measures. There was a significant difference between pre- and post-exercise values for Func.

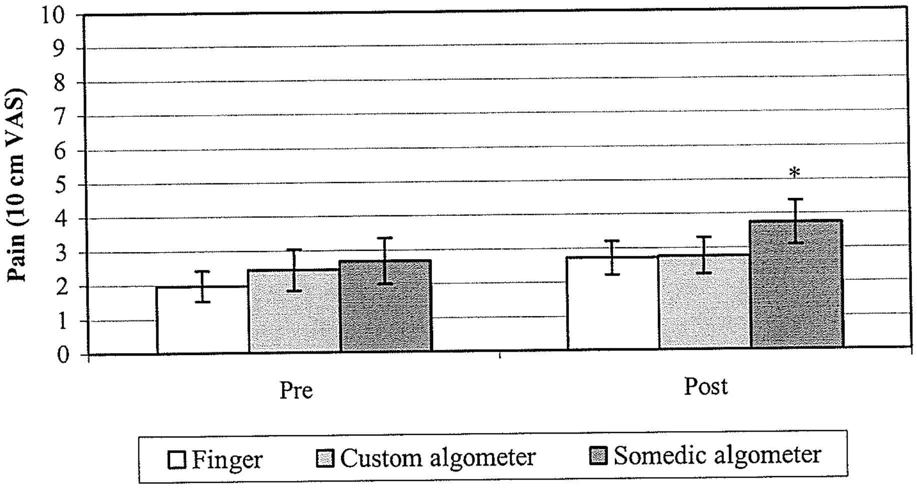

Figure M1- 3. Pre- and post-exercise VAS response to mechanical pressure for Somedic® algometer (350 kPa), finger and custom algometer pressure (139 kPa) (* = significance at $p<0.05$).

Post-exercise pain had a significant correlation to maximum DOMS for Func (r=603; $p<0.008$), S2 ($r=0.606$; $p<0.008$), P1 ($r=0.785$; $p<0.000$), and P2 (0.846; $p<0.000$) but not R or S1.

Time Effects of DOMS Within Pain Measures

All measures reflected DOMS induction by 12 hours ($P < 0.05$) except P1 that showed significance by 24 hours (Figure M1-2). Different time courses were identified between measures but all peaked at or before 48 hours. R peaked at the earliest time point, 24 hours, while P1 peaked at the latest time point, 48 hours. All other measures peaked at 36 hours.

Pain at Rest: R was significantly greater than pre-exercise levels at all follow-up time points with the exception of 48 hours where values had begun returning to baseline and were no longer significant.

Effect of pain on function (Func): Func values were greater than pre-exercise at all follow-up time points.

Pain with stretch (S1): Pain with stretch of the upper extremity (S1) was significantly higher at all time points compared to pre-exercise. S peaked at 36 hours and although began returning to baseline levels, was still significantly higher than pre-exercise at 48 hours.

Pain with enhanced stretch (S2): Pain with enhanced stretch (S2) demonstrated a similar pattern as S1 with significantly higher ratings across all post-exercise time points as compared to pre-exercise with peak pain at 36 hours. S2 was still significantly higher at 48 hours but had also begun to return to baseline levels.

Pain with self-applied finger pressure (P1): Pain with pressure applied with the subject's finger (P1) was significantly elevated from pre-exercise levels at 24, 36 and 48 hours with peak pain at 48 hours. Those subjects whose maximum P1 rating was identified as at 48 hours (5 subjects) were asked post hoc (within two weeks of completing the exercise session) whether they felt this reflected their peak pain or whether greater pain after 48 hours was experienced. Although retrospective, it is interesting to note that all five subjects indicated their pain would have been measured as the same or less if the experimental period had been extended beyond 48 hours.

Pain with pressure applied with custom algometer (P2): Pain with pressure applied using the custom algometer (P2) was not significantly different immediately post-exercise but was significantly higher for all other post-exercise time points. P2 peaked at 36 hours with pain decreasing by 48 hours but still significantly higher than pre-exercise levels.

Pain and Rating of Perceived Exertion

Correlations between RPE and post-exercise and maximum DOMS measures were performed and none were found to be significant.

DISCUSSION

The primary objective of this study was to compare magnitude and time course of pain resulting from a bench press exercise as measured using seven different approaches. These approaches included two measures with no direct provocation of pain (pain at rest and effect of pain on function) and a series of measures using either stretch (stretch and enhanced stretch) or mechanical pressure (self-applied finger pressure, pressure applied with a Somedic® and a custom algometer) to provoke a pain response. The fundamental, yet critical, finding of this study was that DOMS magnitude and time course varied depending on which pain measure was used. Pain resulting from either form of provocation (stretch or mechanical pressure) increased the magnitude of DOMS measured compared to pain measured at rest. Furthermore, DOMS magnitude and time course were found to be sensitive to the type and degree of provocation.

DOMS magnitude for all measures using stretch or pressure was significantly greater than DOMS measured at rest, comparing both maximum and time-specific values. Even though DOMS was detected using the resting pain measure, the overall sensitivity of this approach may be limited in detecting more subtle changes in discomfort across time. For example, the $75\%^{th}$ percentile for pain at rest magnitude was 1.075. In contrast, the $75\%^{th}$ percentile for measures using provocation ranged from 3.28 for pain with stretch to between 5.18 and 5.78 for pain with enhanced stretch and for the measures using pressure. Furthermore, when pre- and post-exercise points are excluded, the measure of pain using stretch demonstrates the greatest range of DOMS response with gradual and equivalent increases in reported pain at each time point. More refined examination of the sensitivity of these methods is warranted, but the evidence in this study suggests that VAS measurement of pain based on provocation allows for greater sensitivity in detecting pain response and changes in pain magnitude.

Beyond the differences in magnitude measured with and without provocation, DOMS was found to vary depending on the type and degree of provocation. Both immediate post-exercise and DOMS responses were found to be stretch-dependent with increased ratings of pain with enhanced stretch of the pectoralis region compared to stretch without the addition of the subject holding a 3 kg mass. The relationship between DOMS and mechanical pressure, however, appears to be more complex. Pre-exercise, subjects' response to different degrees of pressure (139 kPa (custom algometer), 350 kPa (Somedic® algometer), finger pressure (unknown)) was not significantly different. Yet post-exercise, pain with pressure from the Somedic® algometer was substantially higher than the other approaches. This outcome suggests that there may be a degree of pressure-dependent pain response whereby the pain response was exaggerated with greater pressure post-exercise in a way that was not detectable pre-exercise. The measure based on pressure application using the Somedic® algometer was not continued through the 12-48 hour follow-up period. Therefore, it is not possible to draw conclusions regarding the dependency of DOMS response to mechanical pressure. Future study is warranted to further examine this relationship.

Although it is not possible to ascertain the similarities or differences in pain pathways and mechanisms involved, DOMS was found to be of similar magnitude for the measures using stretch exaggerated by the 3 kg mass, and the two measures using mechanical pressure. Stretch using only the load of the upper extremity was found to be significantly less than all of these approaches. The DOMS response based on stretch is an example of allodynia whereby pain is experienced due to a stimulus that does not normally evoke pain. The pre-exercise VAS response to horizontal abduction of the upper extremity in supine reflected little or no pain whereas this same stimulus was experienced as painful in the follow-up period. Enhanced stretch and the two pressure stimuli demonstrate the hyperalgesic effect of novel eccentric exercise in that the pain reported as somewhat painful pre-exercise is reported as significantly more painful post exercise. Increased stimulation and increased sensitivity of Type III and IV muscle afferents (high-threshold receptors sensitive to mechanical (and other) stimuli) are likely the primary contributors to these phenomena. Further investigation of the allodynia and hyperalgesic effects of novel eccentric exercise using stretch- and pressure-gradients to measure pain response may further our understanding of the mechanisms underlying the DOMS response. In previous research, VAS ratings of pain at full ROM have been used (e.g., Nosaka and

Newton 2002b); but the current study is the first to introduce pain in response to loaded stretch as a DOMS measurement tool. The inclusion of two novel approaches to DOMS measurement, specifically loaded or enhanced stretch and the application of mechanical pressure with a custom algometer, provided a truly unique contribution to DOMS research. Both of these methods were developed to ensure consistency of magnitude of provocation as well as ensure feasibility of use between subjects and time points.

An investigator's, and perhaps more so, a subject's ability to replicate finger pressure between time points is difficult to control and measure. In this study, the ability of a subject to replicate a demonstrated level of pressure was examined with the comparison of pain response when subjects were asked to replicate previously applied algometer pressure (350 kPa) with their own finger. Pre-exercise, there was no statistical differences in the pain rating between the algometer pressure and self-applied finger pressure which we expected given the pressures applied were supposed to be similar. Pain response was also compared to that evoked using a lower pressure with the custom algometer (139 kPa). Interestingly, there were no significant differences between the reported levels of pain with any pre-exercise application of pressure, even though the custom algometer applied one third less pressure to the land-marked site. This finding may indicate that DOMS is not as pressure-dependent if the response is at or beyond the pain pressure threshold. This lack of difference between pain experienced may have been partly due to desensitization of the region with previous applications as they were always applied in the same order (Somedic®, finger, custom algometer). Post-exercise, subjects once again reported similar levels of pain for pressure applied with their own finger and with the custom algometer. Pressure from the Somedic® algometer, however, was rated as significantly more painful. There are many factors that may have contributed to this outcome. For example, several subjects reported that due to post-exercise fatigue experienced by both upper extremities, they felt physically unable to apply sufficient pressure post-exercise to match the amount of pressure applied pre-exercise by the Somedic® algometer. In addition, subjects may have been unconsciously resistant to applying an amount of pressure to themselves that would elicit such a significant pain response. The key point drawn from these results is that subjects did not replicate the amount of pressure pre- and post-exercise to elicit similar pain response and that findings based solely on self-application of pressure may be misleading due to inconsistency between time points.

The reliability of using an investigator to palpate has been described (Nosaka et al. 2002) and, although found to be more consistent than self-palpation, its variability is largely dependent on the skill and consistency of the investigator. Using the pain pressure-threshold (PPT) method allows for pressure to be quantified; however, like palpation by the investigator, PPT relies on the subject to return at all post-exercise time points perhaps limiting the feasibility of it's use in some studies. The application of the custom algometer as used in the current study allowed for consistent application of pressure, thus reducing the potential impact resulting from inconsistent pressure applied by the subjects themselves or the investigator between time points and between subjects. Alternately, subjects' DOMS response may not be as sensitive or as pressure dependent once the pain threshold has been reached and thus some degree of inconsistency in the pressure applied may not be a significant issue in controlling DOMS measure. An advantage of stretch as the mode of provocation is that the degree of provocation remains consistent.

A distinction was made in this study between post-exercise soreness and DOMS. It was found that all methods of provocation detected DOMS (a difference in pain at 12-, 24-, 36-, 48-hours from baseline); whereas, only the rest and stretch methods of provocation detected measurable post-exercise soreness, not those using pressure. This may have been because the palpation stimuli evoked enough of a pain response at the pre-exercise time point that the marginal difference in that experience following exercise was more difficult to discern. Alternately, this finding may demonstrate the superior sensitivity of the stretch measures to detect more subtle pain responses between time points.

DOMS not only differed in magnitude between measures but also in time course. Pain at rest peaked at the earliest time point, 24 hours, while pain with palpation using finger palpation peaked at the latest time point, 48 hours. All other pain measures peaked at 36 hours. This finding is consistent with most other research on DOMS that identifies peak pain between 24 to 48 hours (e.g., Dannecker et al. 2005; Rahnama et al. 2005; Smith et al. 1994). The relevance of this finding is that in many studies directly or indirectly examining mechanisms of DOMS, the temporal overlap (or lack there of) of peak magnitudes in muscle damage indices (e.g., torque, creatine kinase levels) and muscle soreness is used as a basis to corroborate or discard theories of causality. In demonstrating that different pain measures result in different times of peak magnitude and resolution, one must use caution in selecting measures of muscle soreness and prudence in the conclusions drawn.

One limitation of this study is that the follow-up period was not long enough to examine the complete time course to full resolution of DOMS. Based on previous studies with extended follow-up periods, resolution of DOMS has been observed to take between 8 and 10 days, depending on the method of induction, the magnitude of DOMS experienced, and the measure of DOMS used (Barlas et al. 2000; Prasartwuth et al. 2005; Smith et al. 1994; Zainuddin et al. 2005b). Particularly in studies that attempt to compare a broader spectrum of muscle damage indices (e.g., creatine kinase, maximal voluntary contraction, arm girth (swelling)) to the point that they have returned to pre-exercise levels, it would be beneficial to understand how different VAS measures reflect the resolution of soreness over time.

RPE was not significantly correlated to post-exercise pain or to maximum DOMS for any measures. Post-exercise pain, however, was significantly correlated to maximum pain for Func, and the more provocative measures, S2, P1 and P2. This has particular clinical relevance as we often prescribe exercises to patients as part of their home follow-up and base the appropriateness of the program on their response to performing the exercises in the clinic. Based on our current findings, we cannot assume that if patients report little or no exertion or report no pain if asked when at rest, that they will not experience marked DOMS. This finding emphasizes the importance of careful selection of how we choose to ask patients to relay their post-exercise response. It also reinforces the importance of using prudence in prescribing, in particular, novel exercise with an eccentric component or risk inducing DOMS, which may potentially reduce compliance.

The effect of pain on function (Func) was significantly different from all measures of DOMS at all time points with the exception of S1. Effect on function was significantly higher than pain at rest but lower than S2, P1, and P2. This suggests that individuals feel they are able to perform their normal tasks even with their resultant level of pain. This finding is interesting in the context of many studies that focus on broad measures of muscle damage including range of motion, maximal voluntary contraction, and even muscle soreness with functional movement such as quadriceps pain with squatting. No studies were identified that related changes in muscle damage indices to daily function. The apparent disassociation between effect of DOMS on function and actual ratings of DOMS found in this study identifies the need to use caution in drawing conclusions about the relationship between muscle damage indices and their impact on daily living. The most critical measure clinically is more likely what do people choose to continue to do or not do in their state of pain, weakness, limited movement, etc. The aim of a future study might be to attempt to quantify the impact of DOMS on both subjective and objective functional activity levels.

In the only other study on DOMS using bench press identified in the literature, subjects were told "to palpate muscles of the chest and upper arm and assign a number between 1 and 10 [Numerical rating scale] that best represented their overall ratings of soreness" (Smith et al. 1994). Based on their 26 subjects, DOMS was significantly elevated from pre-exercise levels between 24 hours (4.15 (0.24)) and 96 hours (1.8 (0.24)) with peak soreness at 48 hours (4.7 (0.24)) (Mean (SE)). The magnitude of these pain measures approximates the VAS ratings in the present study using finger self-palpation (4.8 (0.6)) and the custom algometer (5.1 (0.7)) as the methods of provocation. The variability of the ratings in the present study was greater than in the previous study even with increased control of the methods of provocation used. One reason for this outcome may be due to the heterogeneous nature of the subjects in the present study. Smith et al. (1994) used 26 men with a mean age of 20.1 years (±0.6) who had not weight trained for 3 months while the present study used men and women with a mean age of 32 (±8.2) years with no resistance training in at least one year.

One limitation of this study was that PPT, a fairly common measure in DOMS literature was not included for comparison. For this study, it was not feasible to return subjects to the lab at each time point; however, it would be useful to compare the magnitude and time course of this measure with other measures used to see if it is, in fact, beneficial to impose the requirement of patients come to the lab, or if another measure that can be done at home can suffice. Another limitation of this study was that the actual range of motion demonstrated in the two stretch conditions was not measured. Such information would allow for an even more detailed examination of the relationship of stretch, moment of the upper extremity, and perhaps muscle stiffness at different time points in the study to resulting DOMS. There may also be the potential for an ordering effect in this study as the stimuli used for the provocation measures were always applied in the same order. However, the magnitude for the final three DOMS assessments did not increase as would be expected with sensitization from repeated assessment.

Also, as noted previously, there was greater variability in the outcome of this trial as compared to other DOMS studies. The present study included male and female subjects ranging in age from 18 to 45 performing a "real-life" exercise as opposed to a group of young (<25 yrs) participants performing an exercise on a dynamometer as is common in DOMS research. Although there is not complete consensus, evidence to date suggests that there are no significant gender differences in the development of DOMS (Nie et al. 2004; Stupka et al. 2000). To date, differences in DOMS response with respect to age have been explored only in one recent study (Lavender and Nosaka 2006) in the literature which found the opposite of what was expected, that is, DOMS was significantly less in "old" men (70 yrs (1.5)) compared to "young" men (19.4 yrs (0.4)) 70) following performance of eccentric elbow flexion. Therefore, the relevance of age and sex in DOMS research remains unclear. Another possibly relevant aspect of the present subject group is that there were considerable differences with respect to current activity level and experience in exercise. As part of the screening questionnaire, subjects indicated their fitness level as poor, moderate, or excellent. Further detail gathered post hoc revealed that some subjects had a long history of involvement in upper extremity resistance training but had not been active recently (in the past year as stated in the criteria), others were currently involved in intense aerobic training programs such as triathlons, while others had no history of exercise or sports. There is incomplete evidence that suggests that athletes may be more tolerant to painful experiences (O'Connor and Cook 1999); however, no studies on the contribution of athletic experience or current fitness level have been conducted. In future studies, it would be beneficial to either control this factor or, at least, gather more detailed information that would allow for further categorization of subjects in the study.

CONCLUSIONS

The primary finding of this study was that different measures of DOMS resulted in different magnitudes of pain intensity that followed different time courses. Measures that included some method of provocation evoked higher VAS pain intensity magnitudes that were more sensitive to changes in DOMS over time. DOMS magnitude was found to be dependent on degree of stretch where as dependency of DOMS magnitude on degree of pressure was not clearly established. This study highlights the need for careful consideration in choosing DOMS measures and the use of caution in interpreting any findings in the context of other research. These findings can be used to guide in the selection of pain assessment techniques in future DOMS studies.

REFERENCES

Armstrong, R.B. 1991. Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. Med. Sci. Sports Exerc. 16: 529-38.

Barlas, P. J.A. Craig, J. Robinson, D.M. Walsh, G.D. Baxter, and J.M. Allen. 2000. Managing delayed-onset muscle soreness: lack of effect of selected oral systemic analgesics. Arch. Phys. Med. Rehab. 81: 966-972.

Borg, G. 1998. Borg RPE scale. Illinois: Human Kinetics. 120 p.

Chen, T.C. 2003. Effects of a second bout of maximal eccentric exercise on muscle damage and electromyographic activity. Eur. J. Appl. Physiol. 89: 115-121.

Danneker, E.A., H.A. Hausenblas, T.W. Kaminski, and M.E. Robinson. 2005. Sex differences in delayed onset muscle pain. Clin. J. Pain 21: 120-126.

Hamlin, M.J. and B.M. Quigley. 2001. Quadriceps concentric and eccentric exercise 2: Differences in muscle strength, fatigue and EMG activity in eccentrically-exercised sore and non-sore muscles. J. Sci. Med. Sport 4: 104-115.

Hart, J.M., C.B. Swanik, and R.Y. Tierney. 2005. Effects of sport massage on limb girth and discomfort associated with eccentric exercise. J. Athlet. Training 40: 181-185.

Hjortskov, N., M. Essendrop, J. Skotte, and N. Fallentin. 2005. The effect of delayed-onset muscle soreness on stretch reflexes in human low back muscles. Scand. J. Med. Sci. Sports 15: 409-415.

Kulig, K., C.M. Powers, F.G. Shellock, and M. Terk. 2001. The effects of eccentric velocity on activation of elbow flexors: evaluation by magnetic resonance imaging. Med. Sci. Sports Exerc. 33: 196-200.

Lavender, A.P. and K. Nosaka. 2006. Comparison between old and young men for changes in makers of muscle damage following voluntary eccentric exercise of the elbow flexors. Appl. Physiol. Nutr. Metab. 31: 218-225.

Lieber, R.L. and J. Friden. 2002. Morphologic and mechanical basis of delayed-onset muscle soreness. J. Am. Acad. Orthop. Surg. 10: 67-73.

Madeleine, P., H. Nie, and L. Arendt-Nielsen. 2006. Dynamic shoulder dynamometry: A way to develop delay onset muscle soreness in shoulder muscles. J. Biomech. 39: 184-188.

McHugh, M.P., D.A.J. Connolly, R.G. Eston, I.J. Kremenic, S.J. Nicholas, and G.W. Gleim. 1999. The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. Amer. J. Sports Med. 27: 594-599.

Nie, H., A. Kawcyznski, P. Madeleine, and L. Arendt-Nielsen. 2004. Delayed onset muscle soreness in neck/shoulder muscles. Eur. J. Pain 9: 653-660.

Nosaka, K. and M. Newton. 2002a. Concentric or eccentric training effect on eccentric exercise-induced muscle damage. Med. Sci. Sports Exer. 34: 63-69.

Nosaka, K. and M. Newton. 2002b. Difference in the magnitude of muscle damage between maximal and submaximal eccentric loading. J. Strength Cond. Res. 16: 202-208.

Nosaka, K., M. Newton, and P. Sacco. 2002. Muscle damage and soreness after endurance exercise of the elbow flexors. Med. Sci. Sports Exer. 34: 920-927.

O'Connor, P.J. and D.B. Cooke. 1999. Exercise and pain: The neurobiology, measurement, and laboratory study of pain in relation to exercise in humans. In: Exercise and Sport Sciences Reviews. J.O. Holloszy & D.R. Seals (Eds.), Vol. 27, Williams & Wilkins, 119-166.

Paddon-Jones, D. and P.J. Abernathy. 2001. Acute adaptation to low volume eccentric exercise. Med. Sci. Sports Exer. 33: 1213-1219.

Prasartwuth, O., J.L. Taylor, and S.C. Gandevia. 2005. Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. J Physiol. 567: 337-348.

Rahnama, N., F. Rahmani-Nia, and K. Ebrahim. 2005. The isolated and combined effects of selected physical activity and ibuprofen on delayed-onset muscle soreness. J. Sports Sci. 23: 843-850.

Sayers, S.P., P.M. Clarkson, and J. Lee. 2000. Activity and immobilization after eccentric exercise: I. Recovery of muscle function. 32: 1587-1592.

Smith, L.L., M.G. Fulmer, D. Holbert, M.R. McCammon, J.A. Houmard, D.D. Frazer, E. Nsien, and R.G. Israel. 1994. The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase. Br. J. Sports Med. 28: 267-271.

Stupka, N. S. Lowther, K. Chorneyko, J.M. Bourgeois, C. Hogben, and M.A. Tarnopolsky. 2000. Gender differences in muscle inflammation after eccentric exercise. J. Appl. Physio. 89: 2325-2332.

Zainudden, Z., P. Hope, M. Newton, P. Sacco, and K. Nosaka. 2005b. Effects of partial immobilication after eccentric exercise on recovery from muscle damage. J. Athlet. Training 40: 197-202.

Zainudden, Z., M. Newton, P. Sacco and K. Nosaka. 2005a. Effects of massage on delayed-onset muscle soreness, swelling, and recovery of muscle function. J. Athlet. Training 40: 174-180.

The Relationship Between Movement Strategy during a Bench Press Exercise and the Magnitude of Delayed Onset Muscle Soreness

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INTRODUCTION

Discomfort, soreness or pain resulting from novel eccentric exercise is termed delayed onset muscle soreness (DOMS) and is an experience familiar to virtually all of us through change in our activities of daily living, through leisure time pursuits, and even through the rehabilitation process. Despite the prevalence of DOMS, the mechanism(s) underlying this phenomenon remain poorly elucidated. A number of mechanical aspects of exercise have been examined as potential contributors to the severity of DOMS such as muscle length (increased muscle length results in increased DOMS (Newham et al. 1988)), muscle stiffness (increased muscle stiffness associated with greater DOMS (McHugh et al. 1999a)), submaximal versus maximal load (increased load associated with greater DOMS (Nosaka and Newton 2002)), and number of repetitions (increased number of repetitions results in greater DOMS (Nosaka et al. 2001)). One potential contributor to DOMS that has yet to be explored in a systematic manner is the influence of different movement strategies, specifically, differences in acceleration. Further, studies examining the contribution of shifts in neuromuscular activation patterns during exercise to DOMS have not been performed.

The influence of movement strategy was recently explored by Paddon-Jones et al. (2005) who studied average movement velocity and the duration and severity of several indices of muscle damage including muscle soreness. Muscle damage indices were compared between two groups, each performing 36 maximal eccentric contractions of elbow flexors on a dynamometer at one of two velocities, 0.52 (slow) or 3.14 (fast) rad/s. No difference in the magnitude of muscle soreness was identified between groups. There was a difference in time course as peak muscle soreness was measured at 24-hours for the slow group and 72-hours post-exercise for the fast group. The authors state with respect to the two groups "The only difference [was] the velocity of the damaging eccentric bout". The potential contribution of acceleration was overlooked as changes in acceleration magnitude and muscle changes in activation strategy were not measured or considered. The authors propose that their findings may be, in part, attributable to "a differential recruitment of the elbow flexors during slow and fast eccentric contractions."

Kulig et al. (2001) examined movement strategy as a contributing factor in the induction and magnitude of DOMS using MRI in response to an elbow flexion exercise protocol with two different eccentric cadences. A within-subjects experimental design was used whereby subjects performed a "slow" protocol on one arm with an eccentric component of 10 seconds and a "fast" protocol on the other with an eccentric component of 2 seconds. The concentric component was 2 seconds for both protocols. Total time of exercise (eccentric and concentric) was the same for both protocols with 3 x 12 (36) repetitions performed in the "fast" protocol (36 x 4 s = 144 s) and 3 x 4 (12) repetitions performed in the "slow" protocol (12 x 12 s = 144 s). The outcome of the study was that there was no difference between protocols with respect to overall MRI intensity; however, there was selective preferential activation of biceps brachii in the fast protocol and brachialis in the slow protocol relative to other elbow flexors. Fifty-eight percent (7 of 12 subjects) reported DOMS in the arm performing the fast exercise (36 repetitions at 60% of 1 RM) while none of the subjects reported DOMS in the arm that performed the slow exercise (12 repetitions at 60% of 1 RM). Based upon this, the authors concluded that movement velocity was a key factor in DOMS generation.

A number of weaknesses were identified in this study. First, in attempting to control velocity between protocols, the arm performing the fast protocol performed three times the number of repetitions, and thus, three times the amount of work (Joules) assuming that the given load was constant (due to constant weight). Research has shown that the number of repetitions (work) is clearly implicated in the magnitude of DOMS (Nosaka et al. 2002). Second, no comment was made in regard to experimental control of range of motion between sides and speeds. Without physical cues (end of range of motion markers), it would be expected that in the fast protocol, hypermetria (target overshoot) would be exhibited and thus would result in a greater range of motion. Furthermore, it was not "constant" velocity (isovelocity) but cadence (average velocity) that was maintained between conditions. Peak velocity and acceleration magnitudes could have been very different between fast and slow protocols and, even more interestingly, different between subjects within the same condition leading to increased variability. As such, explanatory factors other than speed may have resulted in the differences that they observed. Similarly, the uncontrolled variability could have lead to an inability to show differences in overall MRI signal intensity between conditions. Kulig et al. (2001) discuss implications of their findings with respect to exercise prescription and state "slower eccentric speed should allow for"…"less rapid loading of the tendon and the musculotendinous junction, thereby decreasing the potentially injurious effects of repetitive motion." Therefore, this study indirectly identifies acceleration as a possible contributing factor to muscle recruitment and DOMS but does not recognize it as such. This statement clearly alludes to the rate of tissue loading as opposed to the average velocity. The rate of tissue loading is best represented by limb acceleration not average speed. These authors indirectly identified acceleration as a possible contributing factor to DOMS but did not recognize it as such.

Neither of these two studies examining the influence of movement strategy and DOMS controlled for or measured acceleration. Based upon Newtonian mechanics, acceleration would impact on the level of instantaneous force generated in the muscles recruited (a fact known to contribute to DOMS magnitude). Second, no study to date has attempted to examine neuromuscular activation strategies on DOMS magnitude. Recently, the application of accelerometry to measure high frequency accelerations during muscle contraction (mechanomyograms or MMG) has been used to examine neuromuscular recruitment (Cescon et al. 2004(a), 2004(b); Watakabe et al. 2003). The extended application of this surrogate measure of neuromuscular control to further our understanding of possible underlying mechanisms contributing to DOMS magnitude is a novel contribution to the literature.

The purpose of this study was to compare the acceleration profiles of those applying a low acceleration strategy to those applying a high acceleration ("drop-and-catch") strategy while performing a bench press exercise to determine if there is a relationship to resulting DOMS. We hypothesized that, with respect to the gross movement strategy applied by a subject, peak acceleration during the eccentric–negative acceleration phase (i.e., when the barbell is slowing down toward the chest) would be positively associated the magnitude of DOMS. We also hypothesized that the high frequency component of acceleration during the eccentric-negative acceleration phase would also be predictive of the magnitude of DOMS.

METHODS

Experimental Design

This experiment was cross-sectional in design, comparing subjects performing a standardized bench press applying two different acceleration strategies (low and high) with control of range of motion (ROM), number of repetitions (total work) and relative weight of the barbell. No attempt to control velocity of movement was included, but velocity was measured as part of the acceleration profile.

Subjects

Twenty healthy adults were recruited for this study and randomly assigned to one of two groups, one directed to use a low acceleration strategy (LA) and one directed to use a high acceleration strategy (HA) in a bench press exercise session. In order to include only subjects unaccustomed to bench press or related exercise, subjects were not to have been involved in resistance training of the upper extremities or activities with similar loading (e.g., push-ups) in the last 12-months. Those with a history of injury to or who had a restriction in range of motion in their upper extremities were also excluded. Prior to their participation, all subjects signed a written informed consent according to a protocol approved by the Faculty of Medicine Human Research Ethics Board of the University of Manitoba. Subjects were asked not to take anti-inflammatory or pain medications or nutritional supplements nor participate in any unaccustomed or vigorous physical activity during the experimental period.

Exercise Protocol

Subjects were positioned lying supine on a flat exercise bench with their feet supported and their spine in a neutral position. Their hands were positioned on an Olympic barbell at a distance equivalent to the distance between their hands when their shoulders were horizontally abducted to 90° and elbows were flexed to 90°. This position emphasized recruitment of pectoralis major through maximizing horizontal abduction/adduction in the movement rather than triceps brachii through shoulder and elbow flexion/extension. A calibrated uniaxial accelerometer (Entran EGAX piezoelectric linear accelerometer, 10g range) was affixed to the posterior surface of the subject's fourth finger on the middle phalanx of the dominant hand allowing measurement of acceleration in the vertical plane (coincident with the movement plane). The accelerometer signal was acquired through a computer-based data acquisition system (Data Translation 9800) and was visually displayed on an oscilloscope (Tektronix PM-3335 Digital Storage Oscilloscope).

Subjects performed a ten-repetition warm-up through full range of motion with low load. Seventy-five percent of the subject's concentric one repetition-maximum (1 RM) was used as the trial load. Previous study has shown that as few as a two-repetition exercise session with maximal eccentric contractions results in a repeated bout effect whereby an adaptation takes place reducing resultant DOMS if the exercise is performed at a later time (Nosaka et al. 2001). In this study, we attempted to minimize the potential influence of the load determination process by including only the concentric component of the bench press in the 1 RM test with spotters "unweighting" the barbell through the eccentric phase. Concentric-only training does not result in muscle damage or DOMS (Armstrong et al. 1991; McHugh et al. 1999a; Nosaka and Newton 2002) therefore the impact of the concentric 1 RM test on resultant DOMS was likely minimal. All 1 RM values were found within 3 to 5 attempts.

Using 75% 1 RM as the trial load, subjects performed sets of bench press to fatigue while maintaining proper technique. Sets were separated by 90-second rest periods and were continued until forty repetitions were completed. In order to control for the relative load between subjects, it was decided, prior to the study, that subjects that did not complete the required 40 repetitions within 3 to 7 sets would be excluded. Two subjects were excluded on this basis.

Movement Parameters

The accelerometer signal was collected at 200 Hz (Data Translation DT9800, 16 bit). Amplitude spectral density distributions of MMGs show that "peak" accelerations in human muscle contraction range between 0 and 20 Hz frequencies (Watakabe et al. 2003).

The acceleration profile was separated into low, medium and high frequency components using digital filtering (2nd order Butterworth digital filter). Low frequencies (DC to 0.2 Hz) reflect a change in orientation of the accelerometer to the vertical axis and in this study would reflect rotation of the hand and bar as the bench press was performed. Low frequencies were filtered out, as rotation of the barbell dependent on the orientation of the subject's hands relative to the vertical axis had no relevance to the study outcome.

Medium frequencies (0.2 – 4.5 Hz) reflected the frequency of the gross repetitive motion of the accelerometer (see below for nomenclature defining phases of motion using the medium frequency signal component). This medium frequency component defines the basic kinematics of motion of the barbell. The high frequency component of the signal (>6 Hz) was extracted through the application of a high-pass filter. This component was examined in a dynamic condition and was used as a measure of overall neuromuscular control strategy (Akataki et al. 2004).

Previously developed standardized nomenclature is used below in describing phases of motion within the medium frequency component of the acceleration profile (Lewis 2002, Webber and Kriellaars 2004): Phase 1 ($P1_{acc}$) begins at the initial extended arm position with zero velocity and zero acceleration and ends at the next zero acceleration crossing point. $P1_{acc}$ reflects the eccentric positive-acceleration phase with the bar increasing in velocity as it is lowered to the chest. Phase 2 ($P2_{acc}$) begins at the point at the zero crossing point and reflects the negative acceleration phase of the eccentric component in which the bar bell begins to "decelerate" as it is lowered to the chest. Phase 3 (P3) reflects the positive acceleration phase of the barbell away from the chest (increasing upward speed) and Phase 4 (P4) represents the "deceleration" of the bar as it is extended fully away from the chest. The four phases below are based on a pause being taken between concentric and eccentric components of the movement, as well as a pause between repetitions that corresponds to baseline or zero acceleration. Subjects were instructed to perform the bench press in this manner.

Based on the filtering and analysis of the acceleration profiles for each set of bench press, peak $P1_{acc}$, peak $P2_{acc}$, average repetition duration, average eccentric duration, and average magnitude of high frequency accelerations (HF) for each set was determined. From the duration values, percent eccentric of total exercise time was also determined.

Delayed Onset Muscle Soreness Measures

DOMS was measured on a series of vertical 100 mm VAS' with "no discomfort or pain" and "extreme discomfort or pain" as the anchor terms. Measurements were taken prior to and immediately following the exercise session and at 12 hour intervals up to 48 hours post-exercise. Pain using the following five methods of provocation was measured: 1) pain at rest (R) - subjects were seated with their hands resting on their lap; 2) pain with stretch (S1) – subjects were positioned lying near the edge of a plinth in supine and asked to extend their arm straight out to the side and then let to allow the weight of their arm to stretch their upper chest region (no active force was applied by the subject or the investigator; 3) pain with stretch and weight (S2) - subjects were positioned as in S1 and were given a 3 kg mass to exaggerate the stretch; 4) pain with palpation with finger (P1) – subjects were asked to re-create a level of pressure that was demonstrated using a calibrated Somedic® Type II pressure algometer (350 kPa pressure with an application slope of 50 kPa/s) using their own finger on a land marked site at the musculotendinous junction of pectoralis major just inferior to the axilla; and 5) pain with palpation with custom designed algometer (P2)- subjects were asked to apply pressure to the previously land marked spot using a custom designed "carton algometer". It was not feasible to permit subjects to take an algometer home to do follow up measures of DOMS over the 48 hour period following their exercise session. Standardization of the amount of pressure on palpation that was applied between subjects and between time periods was deemed important; therefore, a "custom algometer" was created using a carton filled with 3 kg of sand with a dowel extending from the carton covered in soft cloth as the probe. The pressure applied using this instrument was calculated to be 139 kPa. The subject applied the custom algometer to their dominant side with their non-dominant hand allowing only the weight of the carton to exert pressure (i.e., they were asked only to support the carton, not press on it).

Statistical Analyses

Independent t-tests were performed to compare physical characteristics and movement parameters between high and low acceleration strategy groups. The predictive value of physical characteristics of the subjects, descriptors of load and work, and the medium and high components of the acceleration profile were examined through a series of stepwise regression analyses with each maximum VAS rating across 12-48 hours post-exercise for each pain and function scale as the dependent variable. Models presented below were the first models generated in the analyses that were found to be significant and R^2 values are adjusted. With respect to movement parameters, only the values from sets 1 to 4 were included in the analyses to accommodate for missing values resulting from the variability in number of sets subjects took to complete 40 repetitions. $p<0.05$ was used for all analyses.

One subject was transferred into the HA group from the LA group since the P1 and P2 accelerations were consistent with the HA group. Therefore, the sample size for any further analyses of the LA and HA groups was treated as 8 and 10 subjects, respectively.

RESULTS

Comparisons of Low and High Acceleration Groups

Means (SE) for all physical characteristics, exercise parameters, and movement related parameters for the high and low acceleration groups are presented in Table M2-1.

Table M2- 1. Physical characteristics, exercise parameters, and movement related parameters for high and low acceleration groups (Mean (SE)). Shading indicates significance between groups (p<0.05).

Parameter	Low acceleration group	High acceleration group
Sex (proportion of women)	0.75	0.40
Height (cm)	166 (2.5)	173 (3.5)
Body mass (kg)	71.4 (2.6)	78.6 (3.0)
Age (yrs)	31.6 (2.8)	32.0 (2.9)
Self-reported fitness (%Poor/Good/Excellent)	25/75/0	20/60/20
1 RM (kg)	42.0 (5.4)	45.4 (4.5)
Trial load (75% 1 RM – kg)	30.4 (3.8)	32.9 (3.3)
Trial load (% body weight)	0.43 (0.05)	0.42 (0.04)
Number of sets for 40 repetitions	5.0 (0.4)	4.5 (0.3)
External work (Joules)	7060.8 (889.5)	7772.9 (871.1)
Repetition duration (s)	4.23 (0.31)	3.03 (0.17)
Eccentric duration (s)	1.80 (0.20)	0.96 (0.05)
Total exercise time (s)	169.0 (12.3)	121.1 (6.8)
Total eccentric time (s)	72.1 (8.0)	38.5 (2.2)
Proportion eccentric time of total exercise time (%)	0.42 (0.03)	0.32 (0.02)

Physical characteristics: There were no significant differences between groups in any of the physical characteristics measured including height, weight, age, self-reported fitness level and sex (p<0.05).

Exercise parameters: No differences between groups were found with respect to number of sets to complete 40 repetitions, 1 RM, trial load, trial load as a percent of body weight, or the amount of external work performed.

Movement related parameters: The repetition duration of the LA group was significantly longer than for the HA group, 4.23 (± 0.31) and 3.03 (± 0.17) s, respectively. The eccentric component of each repetition was significantly higher and represented a significantly greater proportion of exercise time in the LA group than the HA group (mean 42±3% versus 32±2% SE). Across 40 repetitions, the LA group performed a total 72 (±8) s of eccentric exercise while the HA group performed 38 (±2) s of eccentric exercise.

Gross movement strategy was reflected in the peak accelerations identified within the positive (initial acceleration of the bar toward the chest – $P1_{acc}$) and negative ("deceleration" as the bar is slowed before contacting the chest – $P2_{acc}$) acceleration phases of the eccentric component of the bench press movement. $P1_{acc}$ and $P2_{acc}$ average values across 40 repetitions for each subject are presented in Figure M2-1 and reflect that a large and continuous range of $P1_{acc}$ and $P2_{acc}$ magnitudes were recorded. There were significant differences between the LA and HA groups with respect to $P1_{acc}$ and $P2_{acc}$ values for each set and when averaged across all repetitions (Figure M2-2). Based on visual inspection, there were no obvious patterns in P1 and P2 within a set. Also, there was a greater magnitude in the high frequency component of the acceleration profile for the HA group compared to the LA group for each set and averaged across all sets (Figure M2-3).

Pain, function, and RPE measures: Based on independent t-tests, no differences between any of the pain, function, and RPE measures were identified between the LA and HA groups.

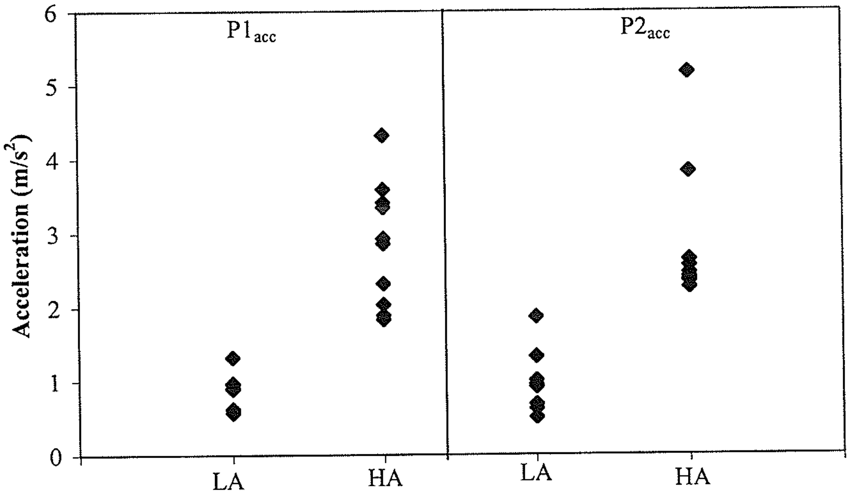

Figure M2-1. P1$_{acc}$ and P2$_{acc}$ magnitudes for each subject by group (LA = low acceleration; HA = high acceleration).

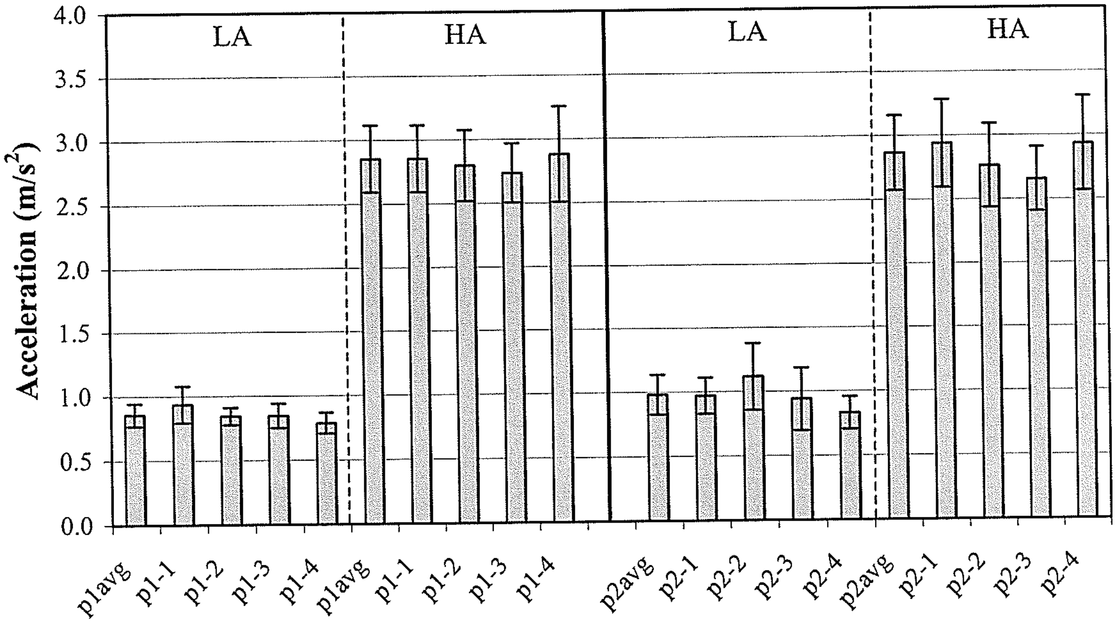

Figure M2-2. Peak positive acceleration magnitude (P1$_{acc}$) and peak negative acceleration magnitude (P2$_{acc}$) for eccentric bench press averaged across 40 repetitions and by set (1-4) for low (LA) and high (HA) acceleration groups.

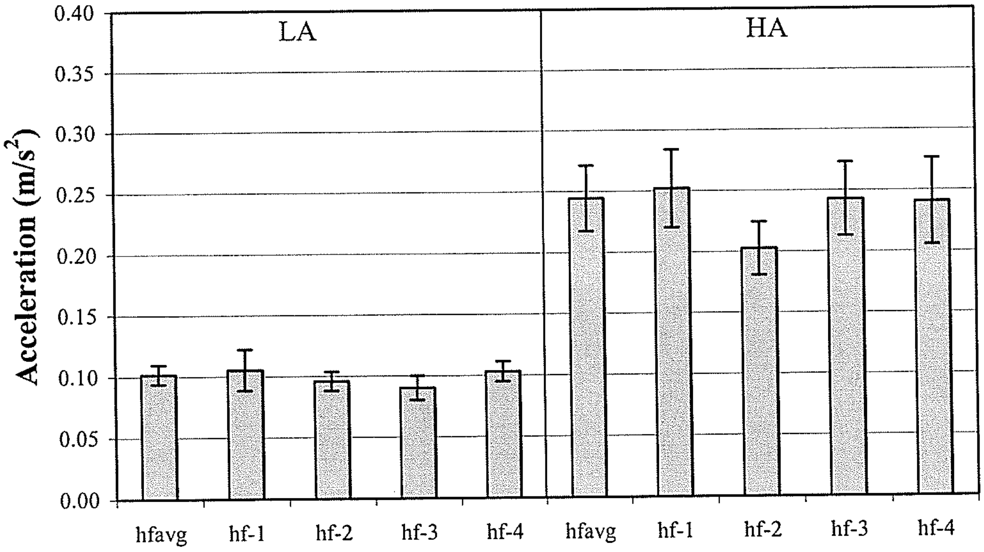

Figure M2-3. High frequency acceleration magnitude averaged across 40 repetitions and by set (1-4) for low (LA) and high (HA) acceleration groups.

Predictors of DOMS

Demographics and Physical characteristics: Age, self-reported fitness, height, weight, and sex were used as the independent variables to predict maximum DOMS for each measure and only height was found to be a significant predictor of Func (R^2=0.232; $p<0.043$) and age was found to be a significant predictor of S2 (R^2=0.271; $p<0.027$). No other significant predictors were identified.

Exercise-related variables: Variables related to time in exercise (repetition duration, eccentric duration, eccentric component as a percent of total exercise time), trial load (absolute and as a percent of body weight), and external work were not found to be predictors of any of the measures of DOMS.

Movement related parameters: Regression models found to be significant for each DOMS measure are presented in Table M2-2.

Movement-related acceleration and DOMS: Using only gross movement (medium frequency) acceleration parameters from the eccentric component of the bench press for each set ($P1_{acc}$ and $P2_{acc}$), regression equations were found to significant for S1 (DOMS with stretch) and P1 and P2 (DOMS with mechanical pressure). No significant models were found to predict R (DOMS at rest), Func (effect of DOMS on function) or S2 (enhanced stretch).

High frequency component of acceleration and DOMS: Using high frequency acceleration magnitude for each set as the independent variables, models with significant predictive value were found for all but R. HF-1 and HF-4 were included in all of these models with HF-3 included in the model predictive of DOMS with stretch.

Movement-related and high frequency component of acceleration and DOMS: Regression analyses that included both medium and high acceleration parameters yielded significant predictive models for all DOMS measures. The contributions of different types of acceleration appear to be additive; however, high frequency acceleration from sets 1 and 4 predominates, appearing in all but the model for S1. $P1_{acc}$ was included in the predictive models for the stretch measures and $P2_{acc}$ was included in those same models as well as DOMS at rest; however, the set or sets from which the included predictors were derived was not consistent between models (e.g., $P2_{acc}$ -2 included in R and S1; $P2_{acc}$ -3 included in S2).

Change in High Frequency Acceleration Component and DOMS

High frequency acceleration magnitudes for sets 1 and 4 were identified in significant models for five of the six pain measures (Table M2-2). Therefore, a new variable representing HF1 subtracted from HF4 was created, HF4-HF1. The value of HF4-HF1 ranged from -0.008831 to 0.01324 m/s^2 with a mean (SE) of –0.0001147 m/s^2 (0.0012). Eight values for HF4-HF-1 were positive and nine were negative. Post-hoc stepwise regression analyses including $P1_{acc}$, $P2_{acc}$, and HF values for sets 1 to 4 as well as HF4-HF1 were performed on each DOMS measure to examine the possible contribution of the

Table M2- 2. Prediction models for each VAS measure of pain/function based on stepwise regression analyses (NS = not significant).

DOMS measure	HF (ß coeff)	R^2 (p)	P1$_{acc}$;P2$_{acc}$ (ß coeff)	R^2 (p)	HF;P1$_{acc}$;P2$_{acc}$ (ß coeff)	R^2 (p)	HF;P1$_{acc}$;P2$_{acc}$; HF4-HF1 (ß coeff)	R^2 (p)
R	NS	-	NS		P2$_{acc}$ -2 (-0.481) HF-1 (1.309) HF-4 (-0.985)	0.512 (0.03)	HF4-HF1 (-217.659)	0.274 (0.022)
S1	HF-3 (-0.488) HF-1 (1.654) HF-4 (-1.320)	0.607 (0.009)	P2$_{acc}$ -2 (-1.160) P1$_{acc}$ -2 (-2.037) P1$_{acc}$ -1 (1.264)	0.534 (0.023)	P2$_{acc}$ -2 (-1.160) P1$_{acc}$ -2 (-2.037) P1$_{acc}$ -1 (1.264)	0.534 (0.004)	HF4-HF1 (-348.340)	0.470 (0.002)
S2	HF-3 (-0.505) HF-1 (1.545) HF-4 (-1.225)	0.550 (0.019)	NS		P2$_{acc}$ -3 (-0.777) P1$_{acc}$ -2 (-1.024) HF-3 (-0.785) HF-1 (1.303) HF-4 (-1.083)	0.753 (0.023)	HF4-HF1 (-361.099)	0.402 (0.005)
P1	HF-4 (-1.484) HF-1 (1.259)	0.482 (0.014)	P2$_{acc}$ -4 (–0.878) P1$_{acc}$ -2 (-2.112) P1$_{acc}$ -1 (1.599)	0.502 (0.034)	HF-4 (-1.484) HF-1 (1.259)	0.482 (0.008)	HF4-HF1 (-325.420)	0.420 (0.004)
P2	HF-4 (-1.516) HF-1 (1.373)	0.501 (0.011)	P2$_{acc}$ -3 (-0.398) P1$_{acc}$ -2 (-2.626) P1$_{acc}$ -1 (1.643) P2$_{acc}$ -1 (-0.891)	0.560 (0.044)	HF-4 (-1.516) HF-1 (1.373)	0.501 (0.014)	HF4-HF1 (-393.465)	0.460 (0.002)
Func	HF-1 (1.609) HF-4 (-1.353)	0.569 (0.004)	NS		HF-4 (1.609) HF-1 (-1.353)	0.569 (0.011)	HF4-HF1 (-336.061)	0.431 (0.003)

change in high frequency magnitude between sets to DOMS magnitude. As indicated in Table M2-2, HF4-HF1 was the only variable needed for the regression models to reach significance for all maximum DOMS measures.

DISCUSSION

The primary and unique contribution of this research to existing literature on DOMS is that movement strategy, as measured by medium and high frequency acceleration magnitude, is a significant contributing factor in DOMS magnitude. Until this study, movement strategy had been examined on the basis of velocity of movement alone. Although there was some predictive value in medium frequency acceleration magnitude, the primary predictor of DOMS magnitude was the change in high frequency acceleration magnitude within an exercise bout (i.e., across sets (HF4-HF1)).

Of the acceleration parameters included in the initial regression analyses (HF, $P1_{acc}$, $P2_{acc}$), high frequency acceleration magnitude for sets 1 and 4 emerged as the most consistent factor across all DOMS measures except DOMS with stretch. Interestingly, the direction of the relationship of DOMS and high frequency acceleration differed between HF-1 and HF-4, HF-1 being positively related to DOMS and HF-4 negatively related. For those measures in which HF-1 and HF-4 were included in the significant predictive model, HF-1 demonstrated our expected outcome that with greater high frequency acceleration magnitude or tremor (reflecting decreased motor unit control) DOMS would be greater. In contrast, greater high frequency acceleration magnitudes in set 4 resulted in less DOMS.

This finding suggests that the change in neuromuscular activation strategy within an exercise bout is more important than the peak instantaneous loads (muscle forces) generated via gross movement strategy ($P2_{acc}$) in the prediction of DOMS magnitude. In seeking validation of this point, HF4-HF1 was included with the initial set of variables in regression analyses. This newly created variable (reflecting the change in high frequency acceleration across sets) was the sole variable identified by step-wise regression in the significant predictive models for all six DOMS measures. For all measures, a negative relationship was identified which means that as the difference in high frequency magnitude between sets 1 and 4 increased, maximum DOMS response decreased. In other words, the greater the shift towards increased high frequency magnitude across the exercise session, the less the DOMS experienced. High frequency acceleration magnitude was included as a surrogate measure of motor unit control or neuromuscular activation strategy and the outcome of this study suggests that the degree to which subjects were able to shift activation patterns from one set to the next is related to DOMS. There are several possible mechanisms that may underlie this shift including fatigue, a shift in the type of motor units recruited, synchronization of motor units, and altered orchestration of the muscles recruited. This finding may also reflect an effect of exercise experience and is consistent with or even contributory to the repeated bout effect phenomenon. The hypothesis that can be postulated is that there is a protective neuromuscular activation shift, which may minimize intrinsic muscle damage and thus, soreness, by minimizing between-fibre shear forces between motor units. It is clear that the relationship of DOMS to high frequency acceleration is complex, and yet, relevant in the study of causal factors of DOMS; thus, more controlled study specifically emphasizing this movement parameter is warranted.

Medium frequency or gross movement acceleration was found to play a significant role, in particular, in DOMS at rest and with either degree of upper extremity stretch included in this study. The "catch" phase of the bench press as measured by $P2_{acc}$ was the first or most significant variable to be included in the models for all three of these DOMS measures; however, not in the direction predicted. It was expected that for those with higher acceleration as the bar was slowed to the chest increased, resultant DOMS would be greater. In fact, the greater the magnitude of "catch", the less the DOMS experienced. One factor that may have influenced this result is the spectrum of subjects' previous experience with resistance training. Although all subjects met the criteria of not having been involved in resistance training for two years, some had substantial previous experience specifically in resistance training or at least, in intensive exercise programs. Based on self-report of exercise history and observation during the trial, those with more experience appeared more confident in the high acceleration or "catch" type of movement and yet experienced less pain than less experienced subjects. Although the heterogeneous sample used in this study represented a broad cross-section of the general population, studies of the contribution of age, and athletic experience in DOMS should be conducted. A recent study by Lavender and Nosaka (2006) compared changes in muscle damage indices between ten "old" (70 yrs (1.5)) versus ten "young" (19.4 yrs (0.4)) men and found the young group experienced more marked negative changes than the older group. This was attributed to physiological changes with ageing such as decreased range of motion that may limit "damaging muscle stress during lengthening muscle actions." Further study into the DOMS experience across a spectrum of ages and exercise backgrounds would be beneficial to refine DOMS research study design in general.

Medium frequency acceleration as the bar was initially lowered (P1) was included in the significant regression models for Func and Stretch (based on regressions including both high and medium frequency parameters). Similar to the relationship of DOMS to high frequency acceleration, the direction of the relationship between the magnitude of the "drop" of the bar to DOMS magnitude was not consistent across sets with greater DOMS associated with lower acceleration as the eccentric component of the bench press movement is initiated in the first set, and higher accelerations in the second set.
In this study, the number of repetitions, the average load, external work, and range of motion was controlled as there is a known relationship of these parameters to DOMS (Newham et al. 1988; Nosaka et al. 2001; Nosaka and Newton 2002). Time in eccentric exercise was greater for the subjects performing the low acceleration strategy (LA) as this strategy imposed greater exercise time and a greater proportion of eccentric exercise within each repetition and number of repetitions was identical between groups. However, time in eccentric exercise was not correlated to DOMS magnitude. No other variables were found to have significant value in predicting DOMS. These current findings are consistent with Kulig et al. 2001 and Paddon-Jones et al. 2005 that these parameters may have little or no influence on DOMS magnitude.

A series of measures was used to quantify DOMS magnitude in this study including pain at rest and pain with a series of types and degrees of provocation. Other than HF1-HF4, the variables included and their respective predictive value varied between measures. This observation reflects the importance of including multiple measures of DOMS in a well-designed research study to ensure the full nature of any relationship can be captured.

Clearly, the relationship of acceleration based movement parameters to DOMS is very complex and further research is warranted to examine these associations in more detail. One possible research direction stemming from the current study may be to examine any shifts in acceleration profile when an exercise is repeated at a second point in time. A great deal of research has been conducted into understanding what is referred to as the "repeated bout effect" whereby subjects perform similar exercise protocols at two points in time separated by a week or two and the magnitude of the resulting DOMS is significantly less following the second session as compared to the first (e.g., Chen and Nosaka 2006; Pettit et al. 2005; McHugh et al. 1999). Many theories have attempted to understand and explain this phenomenon based on studies controlling amount of external work performed but overlooking the influence of acceleration-based movement parameters in resultant DOMS. Such experiments would compare changes in medium and high frequency acceleration and DOMS within subjects over time allowing for improved control of variability resulting from between-subjects designs. Another possible direction is to perform a study with a within-subjects design whereby subjects perform a novel eccentric exercise (e.g., knee extension) on one side while measuring acceleration and then repeat the same exercise at a later time point (one week) on the opposite side. Side-to-side shifts in motor unit activation strategy and resultant DOMS can be examined to see if DOMS is tempered on the opposite leg from a shift in activation strategy based on the initial experience.

CONCLUSIONS

The role of acceleration as a contributor to DOMS magnitude has never been considered in previous literature. The findings of this study indicate gross movement acceleration, and, even more importantly, a shift in activation strategy reflected in a change in high frequency acceleration across sets was shown to be predictive of DOMS. The relationship between these parameters and DOMS is complex and further study of these parameters and, in particular, how they change within an exercise session is warranted.

REFERENCES

Akataki, K., K. Mita, and M. Watakabe. 2004. Electromyographic and mechanomyographic estimation of motor unit activation strategy in voluntary force production. Electromyogr. Clin. Neurophysiol. 44: 489-96.

Armstrong, R.B. 1991. Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. Med. Sci. Sports Exerc. 16: 529-38.

Cescon, C., D. Farina, M. Gobbo, R. Merletti, and C. Orizio. 2004a. Effect of accelerometer location on mechanomyogram variables during voluntary, constant-force contractions in three human muscles. Med. Biol. Eng. Comput. 42: 121-128.

Cescon, C., M. Gazzoni, M. Gobbo, C. Orizio, and D. Farina. 2004b. Non-invasive assessment of single motor unit mechanomyographic response and twitch force by spike-triggered averaging. Med. Biol. Eng. Comput. 42: 496-501.

Chen, T.C. and K. Nosaka. 2006. Responses of elbow flexors to two strenuous eccentric exercise bouts separated by three days. J. Strength Cond. Res. 20: 108-116.

Kulig, K., C.M. Powers, F.G. Shellock, and M. Terk. 2001. The effects of eccentric velocity on activation of elbow flexors: evaluation by magnetic resonance imaging. Med. Sci. Sports Exerc. 33: 196-200.

Lavender, A.P. and K. Nosaka. 2006. Comparison between old and young men for changes in makers of muscle damage following voluntary eccentric exercise of the elbow flexors. Appl. Physiol. Nutr. Metab. 31: 218-225.

Lewis, C. 2000. The effect of sets and repetitions on acceleration during the prone dumbbell row. Univ. of Man. M.Sc. thesis. 103 p.

McHugh, M.P., D.A.J. Connolly, R.G. Eston, I.J. Kremenic, S.J. Nicholas, and G.W. Gleim. 1999a. The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. Amer. J. Sports Med. 27: 594-599.

McHugh, M.P., D.A.J. Connolly, R.G. Eston, and G.W. Gleim. 1999b. Exercise-induced muscle damage and potential mechanisms for the repeated bout effect. Sports Med. 27: 157-170.

Newham, D.J., D.A. Jones, G. Ghosh, and P. Aurora. 1988. Muscle fatigue and pain after eccentric contractions at long and short length. Clin. Sci. 74: 553-557.

Nosaka, K. and M. Newton. 2002. Difference in the magnitude of muscle damage between maximal and submaximal eccentric loading. J. Strength Cond. Res. 16: 202-208.

Nosaka, K., K. Sakamoto, M. Newton, and P. Sacco. 2001. The repeated bout effect of reduced-load eccentric exercise on elbow flexor muscle damage. Eur. J. Appl. Physiol. 85: 24-40.

Paddon-Jones, D., A. Keech, A. Lonergan, and P. Abernethy. 2005. Differential expression of muscle damage in humans following acute fast and slow velocity eccentric exercise. J. Sci. Med. Sport 8: 255-263.

Pettitt, R.W., D.J. Symons, P.A. Eisenman, J.E. Taylor, and A.T. White. 2005. Eccentric strain at long muscle length evokes the repeated bout effect. J. Strength Cond. Res. 19: 918-24.

Watakabe, M., K. Mita, K. Atakaki, and K. Ito. 2003. Reliability of the mechanomyogram detected with an accelerometer during voluntary contractions. Med. Biol. Eng. Comput. 41: 198-202.

Webber, S.C. and D.J. Kriellaars. 2004. The effect of stabilization instruction on lumbar acceleration. Clin. Biomech. 19(8): 777-783.

OVERALL CONTRIBUTIONS

1. No previous studies have attempted a systematic comparison between multiple measures of DOMS based on the same subjects and exercise session. The outcome of this research indicates that DOMS magnitude and time course vary depending on the measure used and that measures that include some method of provocation are more sensitive to changes in pain. Based on this finding, selection of DOMS measure as well as interpretation of previous research should be done with careful consideration.

2. Two novel approaches to DOMS measurement were included in this study, specifically, pain with enhanced or loaded stretch and pain with pressure applied using a custom algometer. Both of these methods control for degree of provocation and are easy to apply in that self-application is consistent between time points and subjects without the use of expensive or complex instrumentation. Enhanced stretch may be preferable in that the results herein support the finding that DOMS is stretch-dependent whereas the relationship between DOMS and pressure is less clear.

3. Acceleration-based movement parameters have never been examined prior to this study. Beyond any other parameters measured including exercise time, eccentric time, and external work, gross movement acceleration and high frequency accelerations were found to be the most significant contributors to resultant DOMS. This finding suggests that the change in neuromuscular activation strategy within an exercise bout is more important than the peak instantaneous loads (muscle forces) generated via gross movement strategy ($P2_{acc}$) in the prediction of DOMS magnitude. The hypothesis that can be postulated is that there is a protective neuromuscular activation shift that may minimize intrinsic muscle damage and DOMS by minimizing between-fibre shear forces between motor units.

FUTURE DIRECTIONS

The current research project was fundamental in establishing acceleration-based movement strategy as a contributing factor in DOMS magnitude. This finding, coupled with demonstrating that DOMS (magnitude and time course) varies depending on the measured used, provides a basis for a number of different directions for future research:

1. The repeated bout effect is a phenomenon whereby the degree of DOMS resulting from a second bout of two separate eccentric exercise bouts is significantly less than the initial bout. A meaningful study to further our understanding of the contribution of acceleration to DOMS would be to examine shifts in acceleration profiles on the same group of subjects performing similar exercise protocols at two time points and relate this to the DOMS experienced. One clear advantage to such a study is the within-subjects design allowing for better control of variables such as previous experience in exercise and pain sensitivity. If the findings of the current study were to be corroborated, we would expect that a change in high frequency acceleration magnitude within the first exercise bout would be predictive of DOMS magnitude and would also shift to reflect the expected decrease in DOMS experienced following the second exercise bout.

2. Another interesting question to pursue may be to investigate if acceleration based adaptations following an initial eccentric exercise bout translate in to alterations in performance of that same exercise by the opposite limb. A lower extremity exercise, for example, eccentric leg press (controlling range of motion and work), could be performed on one leg while accelerations are measured and then repeated on the opposite leg at a later time point. If there is a neuromuscular adaptation that takes place that reduces DOMS after the second bout, it may be demonstrated that the acceleration profile would shift to demonstrate more effective neuromuscular strategies that result in less DOMS on the second limb.

3. The responsiveness of DOMS magnitude to stretch magnitude was demonstrated in the current study; however, possible pressure-dependency of DOMS remains unclear. A study that includes a series of measures with different degrees of pressure across the follow-up time period as well as PPT (a commonly used measure in existing DOMS literature) may further our understanding of the pain mechanisms through which DOMS is experienced.

4. Although there has been some study of the impact of exercise experience on pain sensitivity and immediate post-exercise pain, no previous literature exists that examines the influence of exercise experience on DOMS. A direction of future research could be to examine this relationship as it may have a significant bearing on all DOMS research projects and the manner in which they are designed. Included in most DOMS study design is the requirement that subjects have not specifically performed a given exercise to ensure that the exercise is truly "novel" (to reduce influence of the repeated bout effect). Although this criteria was met in the current study, the subjects recruited reported a range of exercise experience. The impact of this factor is unclear and requires further investigation in a controlled study including a detailed exercise history and even a preliminary fitness assessment.

REFERENCES

Akataki, K., K. Mita, and M. Watakabe. 2004. Electromyographic and mechanomyographic estimation of motor unit activation strategy in voluntary force production. Electromyogr. Clin. Neurophysiol. 44: 489-96.

Armstrong, R.B. 1991. Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. Med. Sci. Sports Exerc. 16: 529-38.

Asmussen, E. 1956. Observations on experimental muscular soreness. Acta Rheum. Scand. 2: 109-116.

Balnave, C.D. and M.W. Thompson. 1993. Effect of training on eccentric exercise-induced muscle damage. J. Appl. Physiol. 75: 1545-1551.

Barlas, P. J.A. Craig, J. Robinson, D.M. Walsh, G.D. Baxter, and J.M. Allen. 2000. Managing delayed-onset muscle soreness: lack of effect of selected oral systemic analgesics. Arch. Phys. Med. Rehab. 81: 966-972.

Borg, G. 1998. Borg RPE scale. Illinois: Human Kinetics. 120 p.

Bouckovalas, G.D. and A. Papadimitriou. 2003. Multi-variable relations for soil effects on seismic ground motion. Earthquake Engineering and Structural Dynamics 32(12): 1867-1896.

Bourgeois, J., D. MacDougall, J. MacDonald, and M. Tarnopolsky. 1998. Naproxen does not alter indices of muscle damage in resistance-exercise trained men. Med. Sci. Sports Exer. 31: 4-9.

Cescon, C., D. Farina, M. Gobbo, R. Merletti, and C. Orizio. 2004a. Effect of accelerometer location on mechanomyogram variables during voluntary, constant-force contractions in three human muscles. Med. Biol. Eng. Comput. 42: 121-128.

Cescon, C., M. Gazzoni, M. Gobbo, C. Orizio, and D. Farina. 2004b. Non-invasive assessment of single motor unit mechanomyographic response and twitch force by spike-triggered averaging. Med. Biol. Eng. Comput. 42: 496-501.

Chen, T.C. 2003. Effects of a second bout of maximal eccentric exercise on muscle damage and electromyographic activity. Eur. J. Appl. Physiol. 89: 115-121.

Chen, T.C. and K. Nosaka. 2006. Responses of elbow flexors to two strenuous eccentric exercise bouts separated by three days. J. Strength Cond. Res. 20: 108-116.

Clarkson, P.M. and S.P. Sayers. 1999. Etiology of Exercise-induced muscle damage. Can. J. Appl. Physiol. 24: 234-248.

Craig, J.A., J. Bradley, D.M. Walsh, G.D. Baxter, and J.M. Allen. 1999. Delayed onset muscle soreness: Lack of effect of therapeutic ultrasound in humans. Arch. Phys. Med. Rehabil. 80: 318-323.

Croisier, J.-L., G. Camus, I. Venneman, G. Deby-Dupont, A. Juchmes-Ferir, M. Lamy, J.-M Crielaard, C. Debu, and J. Duchateau. 1999. Effects of training on exercise-induced muscle damage and interleukin 6 production. Musc. Nerve 22: 208-212.

Danneker, E.A., H.A. Hausenblas, T.W. Kaminski, and M.E. Robinson. 2005. Sex differences in delayed onset muscle pain. Clin. J. Pain 21: 120-126.

Ernst, E. 1998. Does post-exercise massage treatment reduce delayed onset muscle soreness? A systematic review. Br. J. Sports Med. 32: 212-214.

Flynn, J.M., J.D. Holmes, and D.M. Andrews. 2004. The effect of localized leg muscle fatigue on tibial impact acceleration. Clin. Biomech. 19: 726-732.

Ghani, A.K., I.A. Choudhury, and U.L. Husni. 2002. Bearing failure prognostic model based on damage mechanics and vibration monitoring. J. of Mat. Proc. Tech. 127(1): 17-22.

Grabiner, M.D. and T.M. Owings. 2002. EMG differences between concentric and eccentric maximum voluntary contractions are evident prior to movement onset. Exp. Brain Res. 145: 505-511.

Hamlin, M.J. and B.M. Quigley. 2001. Quadriceps concentric and eccentric exercise 2: Differences in muscle strength, fatigue and EMG activity in eccentrically-exercised sore and non-sore muscles. J. Sci. Med. Sport 4: 104-115.

Hart, J.M., C.B. Swanik, and R.Y. Tierney. 2005. Effects of sport massage on limb girth and discomfort associated with eccentric exercise. J. Athlet. Training 40: 181-185.

Herbert, R.D. and M. Gabriel. 2002. Effects of stretching before and after exercising on muscle soreness and risk of injury: systematic review. BMJ 325: 1-5.

Hjortskov, N., M. Essendrop, J. Skotte, and N. Fallentin. 2005. The effect of delayed-onset muscle soreness on stretch reflexes in human low back muscles. Scand. J. Med. Sci. Sports 15: 409-415.

Jones, D.A., D.J. Newham, and P.M. Clarkson. 1987. Skeletal muscle stiffness and pain following eccentric exercise of the elbow flexors. Pain 30: 233-242.

Jones, D.A., D.J. Newham, and C. Torgan. 1989. Mechanical influences on long-lasting human muscle fatigue and delayed-onset pain. J. Physiol. 412: 415-427.

Kulig, K., C.M. Powers, F.G. Shellock, and M. Terk. 2001. The effects of eccentric velocity on activation of elbow flexors: evaluation by magnetic resonance imaging. Med. Sci. Sports Exerc. 33: 196-200.

Lavender, A.P. and K. Nosaka. 2005. Changes in fluctuation of isometric force following eccentric and concentric exercise of the elbow flexors. Eur. J. Appl. Physiol. Epub.

Lavender, A.P. and K. Nosaka. 2006. Comparison between old and young men for changes in makers of muscle damage following voluntary eccentric exercise of the elbow flexors. Appl. Physiol. Nutr. Metab. 31: 218-225.

Lewis, C. 2000. The effect of sets and repetitions on acceleration during the prone dumbbell row. Univ. of Man. M.Sc. thesis. 103 p.

Lieber, R.L. and J. Friden. 2002. Morphologic and mechanical basis of delayed-onset muscle soreness. J. Am. Acad. Orthop. Surg. 10: 67-73.

Madeleine, P., H. Nie, and L. Arendt-Nielsen. 2006. Dynamic shoulder dynamometry: A way to develop delay onset muscle soreness in shoulder muscles. J. Biomech. 39: 184-188.

Malm, C., P. Nyberg, M. Engstrom, B. Sjodin, R. Lenkie, B. Ekblom, and I. Lundberg. 2000. Immunological changes in human skeletal muscle and blood after eccentric exercise and multiple biopsies. J. Physiol. 529: 243-262.

Mathie, M.J., A.C.F. Coster, N.H. Lovell, and B.G. Celler. 2004. Accelerometry: providing an integrated, practical method for long-term, ambulatory monitoring of human movement. Physio. Meas. 25: R1-R20.

McHugh, M.P. 2003. 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. Med. Sci. Sports 13: 88-97.

McHugh, M.P., D.A.J. Connolly, R.G. Eston, I.J. Kremenic, S.J. Nicholas, and G.W. Gleim. 1999a. The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. Amer. J. Sports Med. 27: 594-599.

McHugh, M.P., D.A.J. Connolly, R.G. Eston, and G.W. Gleim. 1999b. Exercise-induced muscle damage and potential mechanisms for the repeated bout effect. Sports Med. 27: 157-170.

McHugh, M.P., D.A.J. Connolly, R.G. Eston, and G.W. Gleim. 2000. Electromyographic analysis of exercise resulting in symptoms of muscle damage. J. Sports Sci. 18: 163-172.

McMurray, R.G., K.B. Ring, M.S. Treuth, G.J. Welk, R.R. Pate, K.H. Schmitz, J.L. Pickrel, V. Gonzalez, M.J. Almedia, D.R. Young, and J.F. Sallis. 2004. Comparison of two approaches to structured physical activity surveys for adolescents. Med. Sci. Sports Exerc. 36(12): 2135-2143.

Newham, D.J., D.A. Jones, and P.M. Clarkson. 1987. Repeated high-force eccentric exercise: effects on muscle pain and damage. J. Appl. Physiol. 63: 1381-1386.

Newham, D.J., D.A. Jones, G. Ghosh, and P. Aurora. 1988. Muscle fatigue and pain after eccentric contractions at long and short length. Clin. Sci. 74: 553-557.

Newham, D.J., K.R. Mills, B.M. Quigley and R.H. Edwards. 1983. Pain and fatigue after concentric and eccentric muscle contractions. Clin. Sci. (Lond) 64: 55-62.

Nosaka, K. and M. Newton. 2002a. Concentric or eccentric training effect on eccentric exercise-induced muscle damage. Med. Sci. Sports Exerc. 34(1): 63-69.

Nosaka, K. and M. Newton. 2002b. Difference in the magnitude of muscle damage between maximal and submaximal eccentric loading. J. Strength Cond. Res. 16: 202-208.

Nosaka, K., M. Newton, and P. Sacco. 2002a. Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. Scand. J. Med. Sci. Sports 12: 337-346.

Nosaka, K., M. Newton, and P. Sacco. 2002b. Responses of human elbow flexor muscles to electrically stimulated forced lengthening exercise. Acta Physiol. Scand. 174: 137-145.

Nosaka, K., M. Newton, and P. Sacco. 2002c. Muscle damage and soreness after endurance exercise of the elbow flexors. Med. Sci. Sports Exer. 34: 920-927.

Nosaka, K., K. Sakamoto, M. Newton, and P. Sacco. 2001. The repeated bout effect of reduced-load eccentric exercise on elbow flexor muscle damage. Eur. J. Appl. Physiol. 85: 34-40.

O'Connor, P.J. and D.B. Cooke. 1999. Exercise and pain: The neurobiology, measurement, and laboratory study of pain in relation to exercise in humans. In: Exercise and Sport Sciences Reviews. J.O. Holloszy & D.R. Seals (Eds.), Vol. 27, Williams & Wilkins, 119-166.

Paddon-Jones, D. and P.J. Abernathy. 2001. Acute adaptation to low volume eccentric exercise. Med. Sci. Sports Exer. 33: 1213-1219.

Paddon-Jones, D., A. Keech, A. Lonergan, and P. Abernethy. 2005. Differential expression of muscle damage in humans following acute fast and slow velocity eccentric exercise. J. Sci. Med. Sport 8: 255-263.

Paddon-Jones, D. and B.M. Quigley. 1997. Effect of cryotherapy on muscle soreness and strength following eccentric exercise. Int. J. Sports Med. 18: 588-593.

Paddon-Jones, D., M. Muthalib, and D. Jenkins. 2000. The effects of a repeated bout of eccentric exercise on indices of muscle damage and delayed onset muscle soreness. J. Sci. Med. Sport 3: 35-43.

Pate, R.R., K.A. Pfeiffer, S.G. Trost, P. Ziegler, and M. Dowda. 2004. Physical activity among children attending preschools. Pediatrics. 114(5): 1258-63.

Pettitt, R.W., D.J. Symons, P.A. Eisenman, J.E. Taylor, and A.T. White. 2005. Eccentric strain at long muscle length evokes the repeated bout effect. J. Strength Cond. Res. 19: 918-24.

Pizza, F.X., T.J. Koh, S.J. McGregor, and S.V. Brooks. 2002. Muscle inflammatory cells after passive stretches, isometric contractions, and lengthening contractions. J. Appl. Physiol. 92: 1873-1878.

Prasartwuth, O., J.L. Taylor, and S.C. Gandevia. 2005. Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. J Physiol. 567: 337-348.

Rahnama, N., F. Rahmani-Nia, and K. Ebrahim. 2005. The isolated and combined effects of selected physical activity and ibuprofen on delayed-onset muscle soreness. J. Sports Sci. 23: 843-850.

Sayers, S.P., P.M. Clarkson, and J. Lee. 2000. Activity and immobilization after eccentric exercise: I. Recovery of muscle function. 32: 1587-1592.

Smith, L.L., M.G. Fulmer, D. Holbert, M.R. McCammon, J.A. Houmard, D.D. Frazer, E. Nsien, and R.G. Israel. 1994. The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase. Br. J. Sports Med. 28: 267-271.

Stupka, N. S. Lowther, K. Chorneyko, J.M. Bourgeois, C. Hogben, and M.A. Tarnopolsky. 2000. Gender differences in muscle inflammation after eccentric exercise. J. Appl. Physio. 89: 2325-2332.

Vickers, A.J. 2001. Time course of muscle soreness following different types of exercise. BMC Musc. Disorders 2:5.

Watakabe, M., K. Mita, K. Atakaki, and K. Ito. 2003. Reliability of the mechanomyogram detected with an accelerometer during voluntary contractions. Med. Biol. Eng. Comput. 41: 198-202.

Webber, S.C. and D.J. Kriellaars. 2004. The effect of stabilization instruction on lumbar acceleration. Clin. Biomech. 19(8): 777-783.

Yu, J., C. Malm, and L. Thornell. 2002. Eccentric contractions leading to DOMS do not cause loss of desmin nor fibre necrosis in human muscle. Histochem. Cell. Biol. 118: 29-34.

Zainudden, Z., P. Hope, M. Newton, P. Sacco, and K. Nosaka. 2005b. Effects of partial immobilication after eccentric exercise on recovery from muscle damage. J. Athlet. Training 40: 197-202.

Zainudden, Z., M. Newton, P. Sacco and K. Nosaka. 2005a. Effects of massage on delayed-onset muscle soreness, swelling, and recovery of muscle function. J. Athlet. Training 40: 174-180.

Zedka, M., A. Prochazka, B. Knight, D. Gillard, M. Gauthier. 1999. Voluntary and reflex control of human back muscles during induced pain. J. Physiol. 15: 520, 591-604.

APPENDIX A: SCREENING ASSESSMENT AND QUESTIONNAIRE

The Relationship Between Movement Strategy and
Delayed Onset Muscle Soreness in a Sub-Maximal Bench Press Exercise
University of Manitoba 2005

Contact: Dr. Dean Kriellaars
787-2505

Name: ______________________________
Date: ______________________________
Date of Birth: __________________________ (mm/dd/yy)
Height (cm): ___________________________
Weight (kg): ___________________________

Exercise history

1. Which arm do you throw a ball with (R or L): __________
2. Have you performed a bench press in the last year (please circle):

   Yes No

3. Have you performed push ups or a similar exercise in the last year (please circle):

   Yes No

4. Have you injured either/both arm(s) or shoulder(s) in the last two years (please circle):

   Yes No

5. Do you have pain/discomfort in your arms or shoulders at rest or with activity (please circle):

   Yes No

6. Do you have any restriction in movement of your arms or shoulders (please circle):

   Yes No

7. What is your current level of fitness (please circle):

   Poor Fair Excellent

8. Do you have any cardiovascular problems (e.g., dizziness, high blood pressure, pain in chest) or any other medical conditions (e.g., arthritis) which might affect your ability to participate in the study?

   ____

9. Are you currently pregnant or breastfeeding (please circle):

   Yes No

APPENDIX B: PARAPHRASE

Changes in delayed onset muscle soreness with different levels of acceleration during the eccentric component of a bench press exercise

University of Manitoba 2005

Contact: Dr. Dean Kriellaars
Phone: 787-3505

Delayed onset muscle soreness (DOMS) is the discomfort or "stiffness" felt following the performance of novel exercise involving lengthening muscle contractions. One mechanism thought to be involved in DOMS is how an exercise is performed. During an exercise session, we propose to use a device called an accelerometer mounted on your finger to evaluate the performance of a bench press. How the exercise is performed will be compared to the level of discomfort resulting from the exercise. Through this, we hope to gain better understanding of the contributing factors of DOMS enabling us to more effectively prescribe exercise programs in both the rehabilitative and training settings.

PROCEDURE

All subjects are being recruited for this study via word of mouth. You will be asked to attend one exercise session (day of the week and time will be flexible to accommodate your schedule as much as possible). Total time for each of these sessions will not exceed one hour. Other than this session, you will be requested not to perform any other exercises outside of your normal daily activities.

At the exercise session, you will complete a screening assessment/questionnaire. You then will be weighed and your height will be measured. Range of motion of your shoulders will also be observed. You will then be asked a series of questions regarding any pain or discomfort you feel in your upper extremities at rest, with movement of your arms, and with a moderate pressure applied to the tendon of your pectoralis major muscle (near the under arm region). Prior to performing any exercise, you will observe a demonstration of the bench press to be performed.

At the beginning of the exercise session, you will be asked to perform a set of ten repetitions as you previously observed in the demonstration with the bar only. Input from the investigator may be provided at this point as to how you are performing the bench press to ensure safe and proper technique.

An amount of weight to be mounted on the barbell will be selected accounting for your weight, age, and estimated fitness level. You will be asked to perform one set of repetitions of bench press involving only the movement of the bar away from you. You will be assisted to return the bar to just above your chest. You will be asked to continue until you are unable to complete any further repetitions. This process will be repeated until a weight is mounted that you are only able to lower once before getting fatigued. A portion of this "maximum weight" will then be mounted on the bar and you will be asked to perform a series of sets to fatigue. There will be a 90-second rest period between sets and you will be requested to perform 40 repetitions in total.

Following your completion of the exercise, another series of questions regarding your level of discomfort will be asked. You will be given a package of questions and scales to complete approximately every 12 hours up to 48-hours post-exercise. You will be asked to mark down your responses and enclose them in an envelope to be forwarded to us via mail (at no expense to you).

The exercise session above will be stopped, if:

- You wish to stop for any reason;
- You exhibit signs of severe pain or discomfort; or
- You use unsafe technique in the performance of the exercise.

In this study, you will be "randomized" into one of two study groups described below. "Randomized" means that you are put into a group by chance, like flipping a coin. You will have an equal chance of being placed in any group. Both groups will be asked to perform a bench press exercise but will be given different instructions regarding the pace of the movement. Subjects will be randomly placed in a group by assigning random numbers to a list of all subjects with those having the lowest ten numbers being placed in one group and the highest in the other.

Participation in the study will be for 2 days (one exercise session with a written follow-up four times in 48 hours).

The researcher may decide to take you off this study if you are unable to perform the exercise properly (i.e., safely).

Approximately six months following your participation in the study, aggregate results will be provided to you for your interest.

RISKS

The risk associated with the bench press exercise sessions are minimal including:

1. After the exercise sessions, you are expected to feel some discomfort in the muscles involved in performing the bench press. This discomfort will likely peak 24-48 hours after the session and last for approximately five days resolving on its own.
2. Minor discomfort may be associated during the exercise session (as with any form of exercise). However, if obvious pain or severe discomfort arises at any time during the session, the session will be discontinued.
3. Many studies involving the performance of submaximal eccentric (lengthening) contractions have been conducted on different joints with trained and untrained subjects with no report of injury. Of the several hundred studies performed

involving eccentric (lengthening) muscle contractions, there has been one published report on six anecdotal cases of extensive muscle damage *possibly* resulting from the exercise performed for the research being conducted. However, maximal voluntary contraction was required in these cases which will not be used in the study herein.

You will not be identified in any published report of the results of this study. You will not be paid to participate in this project, your participation is voluntary and you are free to withdraw at any time and for any reason. You are not responsible for any costs directly related to this study.

Any and all information provided for this study will be kept confidential. If you have any questions or do not understand any aspect of this form, please contact:

Dr. Dean Kriellaars
School of Medical Rehabilitation
University of Manitoba
787-3505