©Journal of Sports Science and Medicine (2011) **10,** 635-642
http://www.jssm.org

Research article

Muscle strength and damage following two modes of variable resistance training

Saied Jalal Aboodarda [1]✉, John George [2], Abdul Halim Mokhtar [3] and Martin Thompson [4]
[1] Sports Center, [2] Department of Biomedical Imaging, and [3] Sports Medicine Unit, Faculty of Medicine, University of Malaya, Malaysia, [4] Faculty of Health Sciences, Discipline of Exercise & Sport Science, The University of Sydney, Australia

Abstract
Nautilus Machine (NM) and Elastic Resistance (ER) have gained considerable popularity among athletes and recreational lifters seeking to increase muscle strength. However, there is controversy concerning the use of ER for increasing muscle hypertrophy and strength among healthy-trained individuals. The aim of the study was to compare the effect of repeated near maximal contractions by ER/NM on indicators of muscle damage including: maximal strength decrement (MVIC), rate of muscle soreness (DOMS), concentration of plasma creatine kinase (CK) and increased high muscle signal on T2 weighted images using magnetic resonance imaging (MRI). Nine healthy male subjects completed two modalities of exercise (5 sets × 10RM ER/NM) in a counterbalance cross-over study design with three weeks "wash-out" period between experiments. The MVIC was measured and DOMS rated and recorded for 4 consecutive days while blood samples were collected on day 1, 3, 5 and 7. Prior to and forty eight hours after completion of each mode of exercise, subjects underwent MRI scanning. The average of applied forces demonstrated significantly higher value for NM compared with ER (362 ± 34.2 N vs 266.73 ± 44.6 N respectively) throughout the 5 sets of dynamic exercise (all $p < 0.05$). However, the indicators of muscle damage (T2 relaxation time, DOMS, MVIC and serum CK) exhibited a very similar response across both modes of training. Plasma CK increased significantly following both modes of training with the peak value on Day 3 ($p < 0.05$). The time course of muscle soreness reached a significant level after both modes of exercise and showed a peak value on the 2nd day ($p < 0.05$). The T2 relaxation time demonstrated a statistically significant increase following ER and NM compared with the pre-test value ($p < 0.05$). The similarity of these responses following both the ER and NM exercise training session suggests that both modes of training provide a similar training stress; despite a considerably lower external force generation during ER. The importance of these findings is underlined by the fact that exercise-induced muscle damage has been shown to be the underlying mechanism of further muscle hypertrophy.

Key words: Elastic resistance training, magnetic resonance imaging, muscle strain, muscle hypertrophy.

Introduction

It is well documented that repeated eccentric contractions or unaccustomed resistance training will cause muscle soreness, transient decrements in muscle function and an increased leakage of intracellular proteins into the vascular space (Clarkson and Hubal 2002; Howatson and van Someren, 2008; Smith, 1991). Electron-microscope observations further show rupture of the myofilaments, Z-line streaming and focal swirls, while urine analysis suggests connective tissue damage. Muscle damage following repeated eccentric contractions and/or bouts of unaccustomed exercise has been the subject of many research studies (Clarkson and Hubal, 2002; Eston et al., 2003; Linnamo et al., 2000; Newham et al., 1983). However, no investigation to date appears to have studied this phenomenon following exercise bouts with Variable External Resistance Training (VRT) devices such as the CAM-Nautilus Machine (NM) (Nautilus, Vancouver, WA) and Elastic Resistance (ER) (Hygenic Corporation, Akron, Ohio).

NM and ER have gained considerable popularity among athletes and recreational lifters seeking to increase muscle strength (Graves et al., 1989; Manning et al., 1990; Page and Ellenbecker, 2003; Wallace et al., 2006). This is due in part to the fact that VRT devices provide varying external resistance based on the muscle force generating capacity throughout the range of motion (Israetel et al., 2010; Manning et al., 1990). The ER exercise device is comprised of elastic band material that requires muscle force to transiently extend the length of the elastic band. It is an affordable and effective mode of training in therapeutic and rehabilitation settings (Hintermeister, et al., 1998; Page, et al., 1993). Despite the popularity of ER, there is controversy concerning the use of this mode of exercise for increasing muscle hypertrophy and strength among healthy-trained individuals. This stems from an unfounded assumption that an "elastic device provides a low level of external force" (Ebben and Jensen, 2002; Hodges, 2006; Newsam et al., 2005; Treiber et al., 1998) and therefore is limited in providing an appropriate resistance/stimulus for strength development. ER devices were originally commercialized as low cost therapeutic aids in muscle rehabilitation where comparatively low resistances are the norm. Reinforcing the assumption that ER devices provide low resistance, Hughes (1999) reported the resistance of an elastic device from 3.3N (yellow) to 70.1N (silver) when elastic materials were at 18% (minimum) and 159% (maximum) of deformation from resting length (un-stretched), respectively.

To increase the magnitude of elastic force, two strategies have been recommended in the research literature; firstly, reducing the initial length of the elastic device (Page and Ellenbecker, 2003) and secondly, doubling or tripling the number of elastic units (using additional various elastic colour coded bands in parallel (Hodges, 2006; Simoneau et al., 2001)). These strategies have the potential to further increase the utility of ER devices by enabling significant increases in resistance. Thus ER devices that are often used as a physical therapy aid may

Received: 04 July 2011 / Accepted: 16 August 2011 / Published (online): 01 December 2011

be adapted to elicit significant strength gains with a programme of repeated near maximal contractions. However the rate of contraction is variable and the resistance is variable throughout the movement range resulting in a complex recruitment of motor units (degree of synchronization). Thus for example, the force generating capacity of the knee extensors may be compromised by activation of antagonists (biceps femoris) in a recruitment strategy that effectively contributes to knee joint stability (Carolan and Cafarelli, 1992). Whether a truly maximal contraction force can be achieved by reducing the initial length of the elastic device or using additional elastic bands is not clear. Clearly if co-activation occurs with either of these strategies then a truly maximal contraction force will not be achieved. Furthermore, the combination of co-activation, variable external resistance and difficulty in controlling velocity of contractions may result in activated muscle fibres being lengthened and hence possibly ultra-structural damage to the myofibrils. Repeated bouts of high ER contractions therefore have the potential to induce muscle damage, fatigue and delayed onset of muscle soreness (DOMS). Whether a similar protocol of repeated bouts of high resistance NM contractions elicits a similar muscle strain is not clear, although movement velocity (leg extension) and joint stability are likely to be more controlled. We are not aware of any research reporting such comparative observations between ER and NM as: (i) maximal force of contraction, (ii) fatigue with repeated high resistance contractions and (iii) delayed muscle soreness. Furthermore it is not clear whether similar forces of contraction can be achieved with an ER device compared with the NM. However, if similar forces can be achieved with an ER device this would suggest the ER device could be used as a cost effective substitute for strength training purposes (Page and Ellenbecker, 2003).

We therefore hypothesized that: (i) a similar force of contraction could be elicited with the ER device when compared with the NM by reducing the initial length of the elastic device and secondly, using additional elastic bands in parallel, (ii) a similar magnitude of decline in ER and NM contractile force generation would occur with a single session of repeated bouts of high variable resistance loading and (iii) a similar magnitude of muscle damage and DOMS with ER versus NM would be evident following a single session of repeated bouts of high resistance loading. Thus we propose that the ER device can provide similar measures of strength, fatiguability and strain as that elicited with the NM. This is of particular interest as it has been shown that muscle damage after resistance training initiates a process of muscle fiber regeneration which culminates in a greater gain in muscle hypertrophy (Eston et al., 2003; Flann et al., 2011; Howatson and van Someren, 2008).

Methods

Subjects

Nine (mean ± SD; 21.1 ± 6.2 yrs, 74.6 ± 7.2 kg, 1.72 ± 0.06 m) healthy male university students gave their signed informed consent to participate in the study. None of the subjects had a history of taking regular medications, or experiencing musculoskeletal injuries or metabolic diseases. In addition, subjects had not participated in any resistance training program or competitive sport during the previous 12 months. This study was approved by the Ethics Committee of the Sports Centre, at the University of Malaya.

Experimental design

The experimental protocol consisted of a counterbalance cross-over study design where all subjects completed two modalities of knee extension exercises with three weeks "wash-out" period between experiments. Participants attended a pre-study session to be informed of possible risks and discomfort associated with the exercise testing protocols. The subjects were then familiarized with the testing procedure by practising 10 repetition maximum (RM) knee extensions exercise with the two modes of training. The external load in each mode of exercise was either increased or decreased to enable the subject to just complete 10 RM with extreme effort, and fail when trying to complete an 11^{th} repetition. This goal, for the ER device was achieved by using different combinations of elastic colour coded bands. Commercially made elastic bands are produced in several colour-codes with each colour denoting a specific resistance (Simoneau et al., 2001). The magnitude of external force for 10 RM was recorded for each subject to avoid additional trials at the main testing sessions. The 10 RM was achieved within the first or second trial. The intraclass correlation coefficient (ICC) of external force for 10-RM NM and ER during the pre-study and main testing session was 0.93 and 0.85, respectively. The resting un-stretched length of elastic material was determined for every subject by measuring the distance from the origin of the elastic device (anchored to the base of the NM chair) to the axis (a custom made ankle cuff). In addition, the resting length of the elastic device was reduced by 30% to provide additional tensile force across the entire ROM (Hodges, 2006).

One week later, 5 subjects were randomly assigned to perform 5 sets of 10 RM knee–extension exercise by ER and the remaining 4 subjects completed the same training protocol with the NM. After a three week rest period, subjects undertook the same testing procedure with the alternate training device. The effect of the repeated ER/NM contractions was assessed by measuring the decrease in MVIC (maximal voluntary isometric contraction) and 10 RM muscle strength, subjective rating of muscle soreness (DOMS), concentration of plasma creatine kinase (CK) and increased high muscle signal on T2 weighted images compared to a control phantom using magnetic resonance imaging (MRI). All subjects refrained from any kind of physical training for the duration of the study. The sample-size in the study was estimated according to the statistical power calculations method recommended by Vincent (2005) and Hopkins (1999). If the $p = .05$ and statistical power = .80, ten subjects were required to participate in the study, and with a crossover study design, one half undertook the ER training first and the other half undertook the NM first.

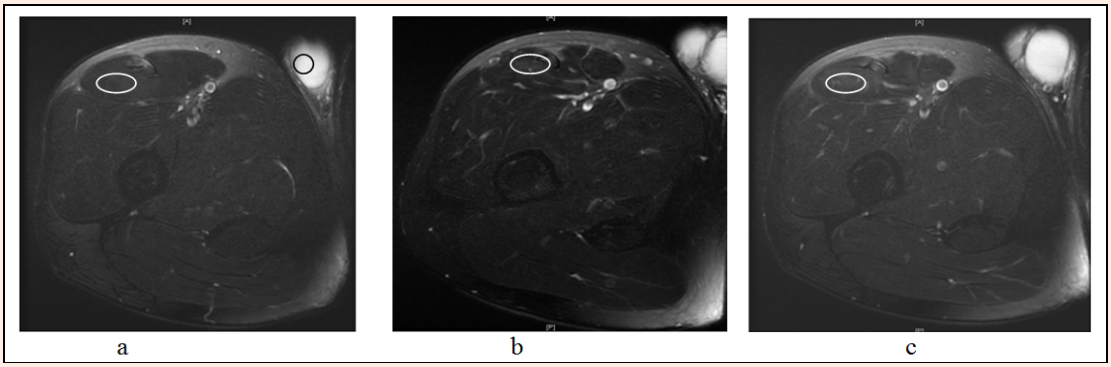

Figure 1. Sample of MRI images from one of the participants in this study. The region of interest (oval white circle) is placed in the rectus femoris muscle where T2 intensity was measured for pretest (a), after NM (b) and ER (c) trainings. The control measurement was taken from the centre of the adjacent testicle (black oval circle) as shown in Figure 1a. The ratio of the measurements forms a constant value allowing variation in display parameters.

Testing procedure

Each experimental session commenced with an MRI scan of the non-dominant thigh followed by a blood sample to determine the baseline measure of plasma CK. Warm up was performed comprising of static stretching and 5 minutes sub-maximal exercise on a cycle ergometer at a self-selected cadence. This was followed by a baseline MVIC measure of the quadriceps according to the method of Arendt-Nielsen and Mills (1988). The value taken for the MVIC was the peak force achieved in three trials each of 5 seconds duration with a 2 minute rest/recovery interval between trials.

The participants then performed 5 sets of 10 RM (ER or NM) through the assigned range of motion (80º to 180º of knee extension) with 90 second rest period between sets. To minimise possible biomechanical influences, the knee extension NM was used for both modes of exercise. Each 10 RM contractions were undertaken at a rate of one every 2 seconds with participants maintaining this frequency via the assistance of a metronome. The 10 RM is a typical training protocol for developing muscle strength and muscle hypertrophy (Kraemer et al., 1991; 1998).

The lever arm of the NM and the axis of elastic device were equipped with a force transducer (Noraxon, Scottsdale, Arizona, USA). The Myoresearch-XP data acquisition package (Noraxon, Scottsdale, Arizona, USA) was used to collect the data from the force transducer. Based on Linnamo et al., (2000b), the average of the 2nd, 3rd, and 4th repetitions of the first set, the average of the 4th, 5th, and 6th repetitions of the 2nd, 3rd, and 4th sets and the average of the 7th, 8th and 9th repetitions of the fifth sets were used for computing the magnitude of external resistance in each mode of exercise.

After completion of each experiment, the MVIC was measured and DOMS rated and recorded for 4 consecutive days while blood samples were collected on day 1, 3, 5 and 7. The DOMS was evaluated based on the method reported by Takahashi et al., (1994). In this method subjects were asked to rate their muscle soreness subjectively using a scale of 1 (normal) to 5 (very sore). Plasma CK was assayed spectrophotometrically using CK-NAC reagent kits (Thermoelectron CORP., USA). At each sampling time, a venous blood sample (5mL) was drawn from the antecubital vein into vacutainers. The blood was centrifuged for 10 minutes at 1500g to obtain a plasma sample. Duplicate samples were assayed and the mean of both measures was used for statistical analysis.

Prior to and forty eight hours after completion of each mode of exercise, subjects underwent MRI scanning. Measurements were performed with a superconducting MR unit (GE Healthcare, SIGNA 3.0T MR Systems). Images were taken from the hip joint to the upper half of the femur covering the proximal quadriceps muscles including the upper rectus femoris muscle. The T2 weighted Spin-echo pulse sequence parameters included a 15 mm slice thickness with no gap, 40cm field of view, 256 × 256 matrix. The results of our pilot study indicated that the region of interest for measuring T2 relaxation time must be selected from the same portion of muscle for all pre- and post- experiments. Accordingly, the T2 was recorded from a circular area with 12 mm diameter (and 191 pixels from the top of the femoral head) from the rectus femoris muscle. Care was taken not to include an area other than muscle, such as artery or fat. As a control, T2 values with similar size were also taken from the centre of the testicle which was always present in these upper thigh scans. Therefore, the ratio of the T2 "Muscle/Testicle" was calculated after each mode of training and compared with pretest ratio (Figure 1).

Statistical analyses

To verify the time effect (pre-test and post-test intervals) and the training mode effect (ER and NM) on the variables including peak MVIC, DOMS and plasma CK, a one way analysis of variance (ANOVA) was performed and complemented by Tukey post-hoc test. Independent sample *t*-test (within the group) was used to compare ER and NM for all measures during identical time intervals. The paired sample *t*-test was also used to identify the relative changes in T2 relaxation time following the two modes of exercise. Significance was defined as $p < 0.05$.

Results

The average of applied forces was significantly higher for

NM compared with ER (362.0 ± 34.2 N vs 266.7 ± 44.6 N respectively) throughout the 5 sets of dynamic exercise (all $p < 0.05$, Figure 2). However, no significant difference was observed between ER and NM in the pre- and post-test values for peak MVIC, plasma CK, DOMS (Table 1) and T2 relaxation time (Figure 3).

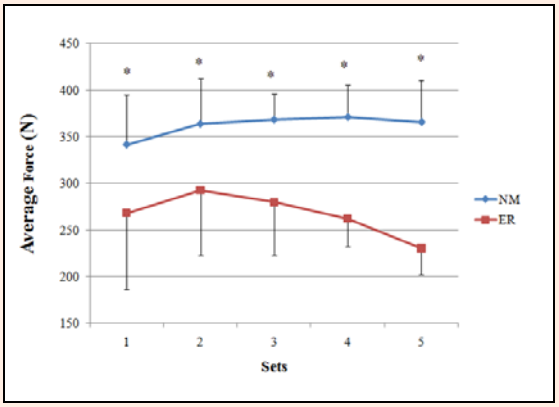

Figure 2. Average of external force employed during dynamic exercises. NM = Nautilus Machine; ER = Elastic Resistance. * = significantly greater external force lifted during NM compared with ER ($p < 0.001$).

The results addressing the time course of MVIC, DOMS and plasma CK are presented in Table 1. The data indicate that neither training protocol (ER and NM) caused a significant decrease in force generating capacity of subjects, although Figure 2 suggests there is a non-significant downward trend in repeated ER knee extension force from exercise bout 2 to exercise bout 5. Plasma CK increased significantly following both modes of training on Day 1, with the peak value on Day 3 and remained elevated up to Day 5 (all $p < 0.05$). The time course of muscle soreness reached a significant level one day after both ER and NM exercise ($p < 0.05$), with a peak value on the 2nd day and remained elevated until the 3rd day. The DOMS completely disappeared by the 4th day (all $p < 0.05$). The T2 relaxation time for the pre-test, ER and NM are graphically presented in Figure 3. The data demonstrated a significant increase in signal intensity of the rectus femoris muscle following ER and NM compared with the pre-test values (all $p < 0.05$).

Discussion

In relation to our three hypotheses: Firstly, we were able to show that a high resistance could be achieved (four exercise bouts averaging over 250N) when using an ERdevice, however the average force production of each of the 5 bouts of ER training was always significantly ($p < 0.05$) less than the NM training bouts, and this was evident for each corresponding exercise bout.

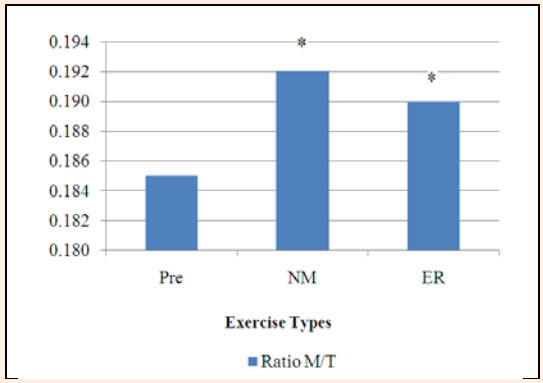

Figure 3. The T2 relaxation time for the pre-test, ER and NM. The value obtained for each mode of exercise presented the changes in the T2 of the muscle relative to the T2 of the testicle.

Secondly, a greater magnitude of decline in each ER exercise bout from the 2nd to the 5th bout was observed when compared with the average NM contractile force generation which showed little decline over the 5 exercise bouts. Calculating the effect-size correlation ($r_{Y\lambda}$) and the value of Cohen's *d* using the mean and standard deviation of the ER average force from the 2nd to the 5th exercise bout demonstrated the values of $r_{Y\lambda} = 0.50$ and $d = 1.16$ which could be interpreted as a differentiation between the ER performance compared with the NM performance. Accordingly, although the magnitude of the decline in ER average force of each exercise bout did not reach statistical significance, there appeared to be a definite trend suggesting increasing fatiguability.

Thirdly, we observed the indicators of muscle damage (T2 relaxation time, DOMS, MVIC and serum CK) exhibited a very similar response across both the NM and ER modes of training. This similarity in response to the 5 repeated bouts of quadriceps loading (10 RM) leg extensions was evident despite significantly greater forces being produced by the NM training mode compared with the ER training mode. The forces produced during the NM training mode were on average approximately 100 Newtons greater than those produced with the ER training mode. This is a large difference in muscle force generation which one might surmise as producing considerably greater strain on the quadriceps muscles when undertaking the NM training. One possible explanation

Table 1. The MVIC, DOMS and serum CK level during time course of recovery period. Data are means (±SD).

Variables		Pre-exercise	Day 1	Day 2	Day 3	Day 4	Day5	Day 7
CK (IU/L)	NM	147 (26)	410 (136) †		705 (185) †		574 (127) †	279 (45)
	ER	167 (54)	331(119) †		595 (147) †		455 (133) †	252 (54)
MVIC (N)	NM	1073 (233)	838 (164)	897 (172)	918 (129)	987 (204)		
	ER	1082 (225)	864 (192)	862 (223)	905 (241)	977 (254)		
DOMS	NM	1 (0)	1.2 (.4) †	2.4 (.5) †	1.9 (.3) *	1 (0)		
	ER	1 (0)	1.3 (.5) †	2.8 (.4) †	1.8 (.4) *	1 (0)		

The MVIC, serum CK level and the DOMS within time course of recovery period. NM = Nautilus Machine; ER, Elastic Resistance.
* = significantly different from pre-exercise values ($p < 0.05$). † = significantly different from pre-exercise values ($p < 0.001$).

for this difference in the ER average forces compared with the NM average forces, despite the measures of muscle strain and damage being similar, could be due to considerable activation of antagonists during the ER mode. Unfortunately we do not have a complete set of EMG data to support this postulate. The downward trend in forces produced in the ER mode from the 2nd leg extension exercise bout to the 5th leg extension bout further suggests a possible greater fatiguability when undertaking the ER training mode, although this suggestion may also be a consequence of a progressively increased activation of antagonists.

The similar responses indicative of muscle strain in both NM and ER training protocols was surprising considering the greater external load employed during NM (26.31%) compared with ER (Figure 2). In previous studies, muscle damage following intensive resistant exercise has been attributed to the mechanical stress on the contractile apparatus during eccentric contraction (Moritani et al., 1988). It has been shown that a given external load is always distributed over less number of active muscle fibers during an eccentric contraction (Warren et al. 1993). Thus the forces generated per cross-sectional area of active muscle are very high. This mechanical strain has the potential to disrupt contractile proteins in recruited muscle fibers (Cutlip et al., 2008; Tee et al., 2007). Therefore it seems axiomatic that the NM training would cause greater disruption to the active muscle and associated connective tissue.

The underlying mechanism for these findings is unknown. However, a potential explanation for this result has been offered by Cronin and colleagues (2003). They reported an increase in electromyographic activity of the quadriceps muscle in the late eccentric phase of motion during ER exercise. They have speculated that increasing segment velocity at the beginning of the eccentric phase, due to the recoil of force from the elastic device, would increase segment momentum which requires a greater muscle force to decelerate the load at the end of the eccentric phase. Linked with this speculative explanation is scientific evidence that demonstrates exacerbation of muscle damage when a given load is administered to the muscle at the end of eccentric phase (Lieber and Fraiden, 1993). Thus the lengthened quadriceps muscles should have experienced a greater mechanical strain in decelerating the lower leg motion at the end of eccentric phase during the ER exercise. One limitation of our study is that we did not quantify segment velocity and moment of force at different phases of motion.

Immediate and prolonged changes in maximal isometric force after exercise-induced muscle damage have been proposed as an effective means of evaluating the magnitude and time course of muscle damage (Byrne et al., 2004; Clarkson and Hubal, 2002; Cutlip et al., 2008). In the present study, the Peak MVIC decreased the day after both modes of exercise (18.19 % vs 17.52 % for NM and ER, respectively); though, the rate of decrease was statistically non-significant. A plausible explanation for the limited decrease in MVIC strength could be the relative active state of the quadricep muscles which are used frequently during day-to-day locomotion (Howatson and van Someren, 2008). The magnitude of strength loss in knee extensors after exercise-induced muscle damage has been shown to be less (around 35% loss) and recovery has been usually faster (4-7 days) than that observed for the elbow flexors (Byrne and Eston, 2002 ; Komi and Viitasalo, 1977).

The prolonged muscle strength decrement following intensive resistance training has been associated with both an inflammatory response and delayed muscle soreness (Byrne et al., 2004). Clarkson and Hubal (2002) have reported a consistent relationship between muscle soreness, plasma CK concentration and the prolonged force decrement. However a second group of investigators have identified a weak relationship between central inhibition via perceived soreness contributing to a loss of strength (Hubal et al., 2007). Accordingly, the prolonged impairment of maximal force production could be attributed to other mechanisms such as disruption of fast twitch (Type II) muscle fibers and the compensatory recruitment of slow twitch (Type I) muscle fibres (Bosco et al., 2000), disruption of Ca^{2+} homeostasis, impaired excitation-contraction coupling and disturbance of impulse propagation due to ischemia (Komi and Tesch, 1979). The data in our investigation appears to be in agreement with the latter school of thought, because the greatest MVIC strength loss was observed on the 1st day of recovery, when the DOMS and plasma CK had not attained their peak levels (Table 1). It is not an unusual observation to find a delayed DOMS and plasma CK response which may suggest a progressive deterioration in the muscle fibre membrane leading to CK leakage and an influx of fluid into the muscle fibre. Thus muscle oedema and the associated pressure then stimulates group III & IV nocioceptors and precipitates the sensation of DOMS.

The concentration of plasma CK demonstrated an increasing trend 24 hrs after termination of each mode of exercise training with the peak concentrations evident 48 hrs after training. Considering that our subjects were relatively untrained we anticipated a marked increase in plasma CK following both the ER and NM training sessions. However the magnitude of the plasma CK response (595 to 795 IU/litre) was relatively very low compared with reports of 20,000 IU/litre following high-force eccentric contractile work (Byrnes et al., 1985; Schwane et al., 1983).The low plasma CK response of the present study may reflect a limited eccentric contraction component of the ER and NM exercise bouts.

MRI has been recognized as the direct and noninvasive methods of assessing damage (edema) in human muscle (Clarkson and Hubal, 2002; Järvinen et al., 2007). A positive correlation between the extent of ultrastructural injuries of muscle and the increase in MRI signal intensity has been reported (Takahashi et al., 1994). The T2 relaxation time in the current study demonstrated a statistically significant increase in the intensity of MRI signals which is indicative of muscle damage following both modes of training. The increase in the T2 signal is related to the accumulation of fluid in the damaged muscle due to degradation of both structural and functional proteins together with an increase in capillary permeability (McCully et al., 1992; Takahashi et al., 1994). A num-

ber of investigators have noted that the increase in T2 relaxation time is approximately in accordance with higher concentrations of degraded proteins (e.g. CK) in the blood, muscle soreness and muscle oedema (Foley et al., 1999). The current study observed a concurrence between peak plasma CK activity, peak rating of muscle soreness and T2 relaxation time which was significantly higher than the pretest measurement following both types of training.

These results suggest similar potential of the two training devices in creating sufficient muscle strain to induce muscle damage. The importance of these findings is underlined by the fact that exercise-induced muscle damage has been shown to be the underlying mechanism of further muscle hypertrophy (Eston et al., 2003; Flann et al., 2011; Howatson and van Someren, 2008). Previous research has shown that the inflammatory response following chronic exposure to exercise induced muscle damage is characterized by an infiltration of neutrophils and macrophages (Cutlip et al., 2009). These inflammatory cells produce cytokines and chemokines which activate local pathways assisting the repair of damaged tissue. Phagocytic macrophages also invade damaged tissue in order to digest damaged structural and functional proteins and promote regeneration. During muscle adaptation, satellite cells (quiescent muscle precursor cells) are activated, proliferate and finally fuse with the existing myofibre. Furthermore, developmental myosin heavy chain isoforms are expressed in injured fibers during this time period, and this has been suggested to comprise the developmental program (Cutlip et al., 2009).

Considering that an elastic resistance device has long been accepted as an affordable, portable and versatile exercise training aid compared with other training equipment such as the NM (Page and Ellenbecker, 2003), the present data support the use of an ER device as a cost effective mode of training for achieving further muscle strength and hypertrophy in healthy individuals. This is contrary to many previous investigations that have rejected the potential of utilizing an ER device in athletic settings, because of a perception that an ER device does not provide an adequate training stress. (Hopkins et al., 1999; Hostler et al., 2001)

Conclusion

In theory, exercise induced muscle soreness, increased levels of plasma CK, increased MRI T2 signal and prolonged strength loss indicate the moderate to intense nature of the training protocol. The similarity of these responses following both the ER and NM exercise training session suggests that both modes of training provide a similar global training stress; despite a considerably lower external force generation during ER. It is possible that there may have been large variations of contraction velocity during ER despite the subject's adherence to the timing of one contraction every 2 seconds. Such variations in contraction velocity may not only contribute to the difficulty of performing the contraction, but may also accelerate the onset of fatigue with no consistent recruitment pattern of the fast and slow twitch muscle fibres. Furthermore, this may have implications for quantifying the intensity of the training session as the magnitude of the training load when it may be better to express this in terms of the rate of work performed. However this approach requires accurate timing of all phases through which the range of motion of the limb is exercised.

The data in the present study suggest elastic training is a viable mode of resistance exercise that can provide a training stimulus that is significantly greater than that employed in rehabilitation settings. However, the results of the present study point to the need for further research on the effectiveness of the ER device in developing muscle strength and hypertrophy with an extended training programme.

Acknowledgment

For this investigation a research grant was provided by University of Malaya, Malaysia (PS008/2008C).

References

Arendt-Nielsen, L. and Mills, K. (1988) Muscle fibre conduction velocity, mean power frequency, mean EMG voltage and force during submaximal fatiguing contractions of human quadriceps. *European Journal of Applied Physiology and Occupational Physiology* **58**, 20-25.

Bosco, C., Colli, R., Bonomi, R., Von Duvillard, S.P. and Viru, A. (2000) Monitoring strength training: neuromuscular and hormonal profile. *Medicine & Science in Sports & Exercise* **32**, 202-208.

Byrne, C. and Eston, R. (2002) The effect of exercise-induced muscle damage on isometric and dynamic knee extensor strength and vertical jump performance. *Journal of Sports Science* **20**, 417-425.

Byrne, C., Twist, C. and Eston, R. (2004) Neuromuscular Function after Exercise-Induced Muscle Damage: Theoretical and Applied Implications. *Sports Medicine* **34**, 49-69.

Byrnes, W.C., Clarkson, P.M., White, J.S., Hsieh, S.S., Frykman, P.N. and Maughan, R.J. (1985) Delayed onset muscle soreness following repeated bouts of downhill running. *Journal of Applied Physiology* **59**, 710-715.

Carolan, B. and Cafarelli, E. (1992) Adaptations in coactivation after isometric resistance training. *Journal of Applied Physiology* **73(3),** 911-917.

Clarkson, P. and Hubal, M. (2002) Exercise-induced muscle damage in humans. *American Journal of Physical Medicine & Rehabilitation* **81(Suppl. 11),** 52-69.

Cronin, J., McNair, P. and Marshall, R. (2003) The effects of bungy weight training on muscle function and functional performance. *Journal of Sports Sciences* **21**, 59-71.

Cutlip, R.G., Baker, B.A., Hollander, M. and Ensey, J. (2009) Injury and adaptive mechanisms in skeletal muscle. *Journal of Electromyography and Kinesiology* **19**, 358-372.

Ebben, W.E. and Jensen, R.L. (2002) Electromyographic and Kinetic Analysis of Traditional, Chain, and Elastic Band Squats. *The Journal of Strength & Conditioning Research* **16**, 547-550.

Eston, R., Byrne, C. and Twist, C. (2003) Muscle function after exercise-induced muscle damage: Considerations for athletic performance in children and adults. *Journal of Exercise Science and Fitness* **1**, 85-96.

Flann, K., LaStayo, P., McClain, D., Hazel, M. and Lindstedt, S. (2011) Muscle damage and muscle remodelling: no pain, no gain? *Journal of Experimental Biology* **15**, 674-679.

Foley, J.M., Jayaraman, R.C., Prior, B.M., Pivarnik, J.M. and Meyer, R.A. (1999) MR measurements of muscle damage and adaptation after eccentric exercise *Journal of Applied Physiology* **87**, 2311-2318.

Graves, J.E., Pollock, M.L., Jones, A.E., Colvin, A.B. and Leggett, S.H. (1989) Specificity of limited range of motion variable resistance training. *Medicine & Science in Sports & Exercise* **21**, 84-89.

Hintermeister, RA, Bey, MJ, Lange, GW, Steadman, JR and Dillman, CJ (1998). Quantification of Elastic Resistance Knee Rehabilitation Exercises. *Journal of Orthopaedic and Sports Physical Therapy* **28(1),** 40-50.

Hodges, G.N. (2006) *The effect of movement strategy and elastic starting strain on shoulder resultant joint moment during elastic resistance exercise.* Master Thesis. School of Medical Rehabilitation, Faculty of Medicine, University of Manitoba.

Hopkins, J.T., Christopher, D.I., Michelle, A.S. and Susan, D.B. (1999) An electromyographic comparison of 4 closed chain exercises. *Journal of Athletic Training* **34**, 353-357.

Hopkins W.G. (2006) Estimating sample size for magnitude-based inferences. *SportScience* **10**, 63-70. Available from URL: http://sportsci.org/resource/stats/ssdetermine.html

Hostler, D., Schwirian, C.I., Campos, G., Toma, K., Hagerman, F.C. and Staron, R.S. (2001) Skeletal muscle adaptation in elastic resistance training young men and women. *European Journal of Applied Physiology* **86**, 112-118.

Howatson, G. and van Someren, K.A. (2008) the prevention and treatment of exercise induce muscle damage. *Sports Medicine* **38**, 483-503.

Hubal, M.J., Rubinstein, S.R. and Clarkson, P.M. (2007) Mechanisms of variability in strength loss after muscle-lengthening actions. *Medicine & Science in Sports & Exercise* **39**, 461-468.

Hughes, C.J., Hurd, K., Jones, A. and Sprigle, S. (1999) Resistance properties of Thera-Band® tubing during shoulder abduction exercise. *Journal of Orthopaedic and Sports Physical Therapy* **29**, 413-420.

Israetel MA, McBride JM, Nuzzo JL, Skinner JW, Dayne AM (2010) Kinetic and kinematic differences between squats performed with and without elastic bands. *Journal of Strength and Conditioning Research* **24(1),** 190-194.

Järvinen TAH, Järvinen TLN, Käriäinen M, Ä ärimaa V, Vaittinen S, Kalimo H, Järvinen M (2007) Muscle injuries: optimising recovery. *Best Practice & Research Clinical Rheumatology* **21,** 317–331.

Komi, P.V. and Tesch, P.A. (1979) EMG frequency spectrum, muscle structure, and fatigue during dynamic contractions in men. *European Journal of Applied Physiology and Occupational Physiology* **42**, 41-50.

Komi, P.V. and Viitasalo, J. (1977) Changes in motor unit activity and metabolism in human skeletal muscle during and after repeated eccentric and concentric contractions. *Acta Physiology Scandinavia* **100,** 246-254.

Kraemer, W., Gordon, S., Fleck, S., Marchitelli, L., Mello, R., Dziados, J., Friedl, K., Harman, E., Maresh, C. and Fry, A. (1991) Endogenous anabolic hormonal and growth factor responses to heavy resistance exercise in males and females. *International Journal of Sports Medicine* **12**, 228-235.

Kraemer, W.J., Duncan, N.D. and Volek, J.S. (1998) Resistance training and elite athletes: adaptations and program considerations. *Journal of Orthopedic and Sport Physical Therapy* **28**, 110-119.

Lieber, R. and Fraiden, J. (1993) Muscle damage is not a function of muscle force but active muscle strain. *Journal of Applied Physiology* **74**, 520-526.

Linnamo, V., Bottas, R. and Komi, P. (2000) Force and EMG power spectrum during and after eccentric and concentric fatigue. *Journal of Electromyography and Kinesiology* **10,** 293-300.

Linnamo, V., Newton, R.U., Häkkinen, K., Komi, P.V., Davie, A., McGuigan, M. and Triplett-McBride, T. (2000 b) Neuromuscular responses to explosive and heavy resistance loading. *Journal of Electromyography and Kinesiology* **10**, 417-424.

Manning, R.J., Graves, J.E., Carpenter, D.M., Leggett, S.H. and Pollock, M.L. (1990) Constant vs variable resistance knee extension training. *Medicine & Science in Sports & Exercise* **22,** 397-401.

McCully K, Vandenborne K, DeMeirleir K, Posner J, Leigh JJ (1992) Muscle metabolism in track athletes, using 31P magnetic resonance spectroscopy. *Canadian Journal of Physiology and Pharmacology* **70**, 1353-1359.

Moritani, T., Muramatsu, S. and Muro, M. (1988) Activity of motor units during concentric and eccentric contractions. *American Journal of Physical Medicine* **123**, 214-224.

Newham, D., McPhail, G., Mills, K. and Edwards, R. (1983) Ultrastructural changes after concentric and eccentric contractions of human muscle. *Journal of the Neurological Sciences* **61**, 109-122.

Newsam, C., Leese, C. and Fernandez-Silva, J. (2005) Intratester reliability for determining an 8-repetition maximum for 3 shoulder exercises using elastic bands. *Journal of Sport Rehabilitation* **14**, 35-47.

Page P., Lamberth J., Abadie B., Boling R., Collins R. and Collins R (1993) Posterior rotator cuff strengthening using Theraband® in a functional diagonal pattern in collegiate baseball pitchers. *Journal of Athletic Training* **28(4),** 346-354.

Page, P. and Ellenbecker, T. (2003) *The Scientific and Clinical Application of Elastic Resistance*. Human Kinetics.

Schwane, J.A., Johnson, S.R., Vandenakker, C.B. and Armstrong, R.B. (1983) Delayed-onset muscular soreness and plasma CPK and LDH activities after downhill running. *Medicine and Science in Sports and Exercise* **15**, 51-56.

Simoneau, G.G., Bereda, S.M., Sobush, D.C. and Starsky, A.J. (2001) Biomechanics of elastic resistance in therapeutic exercise programs. *Journal of Orthopaedic and Sports Physical Therapy* **31**, 16-24.

Smith, L.L. (1991) Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Medicine & Science in Sports & Exercise* **23**, 542-551.

Takahashi, H., Kuno, S., Miyamoto, T., Yoshioka, H., Inaki, M., Akima, H., Katsuta, S., Anno, I., Itai, Y. (1994) Changes in magnetic resonance images in human skeletal muscle after eccentric exercise. *European Journal of Applied Physiology and Occupational Physiology* **69**, 408-13.

Tee, J.C., Bosch, A.N. and Lambert, M.I. (2007) Metabolic consequences of exercise-induced muscle damage. *Sports Medicine* **37**, 827-836.

Treiber, F.A., Lott. J., Duncan, J., Slavens, G. and Davis, H. (1998) Effects of theraband and lightweight dumbbell training on shoulder rotation torque and serve performance in college tennis players. *The American Journal of Sports Medicine* **26,** 510-515.

Vincent W.J. (2005) *Statistics in kinesiology*. Third Edition, Human Kinetics. 147-151.

Wallace B.J., Winchester J.B. and McGuigan M.R. (2006) Effects of elastic bands on force and power characteristics during the back squat exercise. *Journal of Strength and Conditioning Research* **20,** 268-272.

Warren, G.L., Lowe, D.A., Hayes, D.A., Karwoski, C.J., Prior, B.M. and Armstrong, R.B. (1993) Excitation failure in eccentric contraction-induced injury of mouse soleus muscle. *Journal of Physiology* **468,** 487-499.

Key points

- Exercise induced muscle soreness increased levels of plasma CK, increased MRI T2 signal and prolonged strength loss indicate the moderate to intense nature of the training protocol.
- The similarity of these responses following both the Elastic Resistance and Nautilus Machine exercise training session suggests that both modes of training provide a similar training stress; despite a considerably lower external force generation during ER.
- The data in the present study suggest elastic training is a viable mode of resistance exercise that can provide a training stimulus greater than that employed in rehabilitation settings.

AUTHORS BIOGRAPHY

Saied Jalal ABOODARDA

Employment

Sports center, University of Malaya, Malaysia

Degree

MSc, PhD Candidate

Research interests

Strength and conditioning, elastic resistance training, electromyography, neuromuscular, hormonal and morphological adaptation.

E-mail: SaiedJalal@siswa.um.edu.my

John GEORGE
Employment
Prof., Department of Biomedical Imaging Faculty of Medicine
Degree
FRCR, MBBS
Research interests
Musculoskeletal radiology.
E-mail: johng_rad@um.edu.my

Abdulhalim MOKHTAR
Employment
Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia
Degree
MD, MSc
Research interests
Sports medicine, sports injury and rehabilitation.
E-mail dranzdaxx@yahoo.com

Martin THOMPSON
Employment
Associate Prof., Faculty of Health Sciences, Discipline of Exercise & Sport Science, The University of Sydney, Australia
Degree
PhD, MSc
Research interests
Muscle function and fatigue, prolonged exercise and environmental physiology.
E-mail: martin.thompson@sydney.edu.au

✉ **Saied Jalal Aboodarda**
Sport Center, University Malaya, Kuala Lumpur, 50603 Malaysia