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Research article

COMPRESSION GARMENTS AND RECOVERY FROM ECCENTRIC EXERCISE: A ^{31}P-MRS STUDY

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ABSTRACT

The low oxidative demand and muscular adaptations accompanying eccentric exercise hold benefits for both healthy and clinical populations. Compression garments have been suggested to reduce muscle damage and maintain muscle function. This study investigated whether compression garments could benefit metabolic recovery from eccentric exercise. Following 30-min of downhill walking participants wore compression garments on one leg (COMP), the other leg was used as an internal, untreated control (CONT). The muscle metabolites phosphomonoester (PME), phosphodiester (PDE), phosphocreatine (PCr), inorganic phosphate (Pi) and adenosine triphosphate (ATP) were evaluated at baseline, 1-h and 48-h after eccentric exercise using ^{31}P-magnetic resonance spectroscopy. Subjective reports of muscle soreness were recorded at all time points. The pressure of the garment against the thigh was assessed at 1-h and 48-h following exercise. There was a significant increase in perceived muscle soreness from baseline in both the control (CONT) and compression (COMP) leg at 1-h and 48-h following eccentric exercise ($p < 0.05$). Relative to baseline, both CONT and COMP showed reduced pH at 1-h ($p < 0.05$). There was no difference between CONT and COMP pH at 1-h. COMP legs exhibited significantly ($p < 0.05$) elevated skeletal muscle PDE 1-h following exercise. There was no significant change in PCr/Pi, Mg^{2+} or PME at any time point or between CONT and COMP legs. Eccentric exercise causes disruption of pH control in skeletal muscle but does not cause disruption to cellular control of free energy. Compression garments may alter potential indices of the repair processes accompanying structural damage to the skeletal muscle following eccentric exercise allowing a faster cellular repair.

KEY WORDS: Magnetic resonance spectroscopy, muscle damage, muscle metabolism, rehabilitation.

INTRODUCTION

Repeated isometric or concentric muscle contractions result in fatigue. However, the muscle quickly recovers without any long term loss of function. In contrast, unaccustomed eccentric muscle contraction frequently results in a greater loss of function which can take a number of days to recover (Proske and Allen, 2005). The muscle soreness with the loss of function after eccentric exercise is more commonly known as delayed onset muscle soreness (DOMS). Despite DOMS, eccentric exercise conveys unique benefits to clinical populations with impaired lung function (Rooyackers et al., 2003) and mitochondrial disease (Taivassalo et al., 1999) due to the low oxidative demands and improvements in muscle volume. The low oxidative demands and increase in muscle volume allow these patient

groups to participate in exercise, increase their ability to move and improve their standard of living. Understandably though, DOMS restricts the ability of eccentric exercise to be routinely used in the clinical setting. DOMS is also a prominent feature in groups prone to eccentric muscle damage such as; athletes and patients with Duchenne muscular dystrophy and idiopathic toe walking (Proske and Morgan, 2001). Strategies to limit DOMS are few.

In fact, despite significant information on the effect of eccentric muscle contraction on muscle anatomy and physiology (for review see (Proske and Allen, 2005; Proske and Morgan, 2001), there is little evidence based or basic research on the management of eccentric muscle damage. Rest, ice, compression and elevation (RICE) are routinely used as a combined management strategy for the treatment of strains (Clanton and Coupe, 1998) but there is a lack of understanding about the individual and/or collective effects of these treatments on muscle damage.

Graduated compression stockings promote blood flow from superficial veins into deep veins (Herzog, 1993). The improved blood flow and prevention of venous stasis reduce oedema and help compensate for impaired venous return in conditions such as deep vein thrombosis (Byrne, 2001) and venous insufficiency (Jonker et al., 2001; van Geest et al., 2003). Although compression is advocated in the recovery from exercise induced muscle damage (Noonan and Garrett, 1999), there is little information on the effect of compression on intracellular metabolic function. Two studies have shown that compression garments maintained muscle function and reduced perceived muscle soreness following eccentric exercise (Kraemer Bush et al., 2001a; 2001b). These studies also showed that compression garments attenuate creatine kinase (CK) release from skeletal muscle into the circulation following eccentric exercise. The retention of CK and maintenance of muscle function were attributed to the effect of the compression garments preventing oedema within the muscle (Kraemer et al., 2001a; 2001b). Despite these functional and biochemical adaptations, the effect of compression garments on intra-cellular metabolism is not known.

Phosphorous magnetic resonance spectroscopy (^{31}P-MRS) allows non-invasive appraisal of phosphorous containing metabolites *in vivo*. Perturbations in skeletal muscle bio-energetics have been shown using ^{31}P-MRS 1-2 days after unaccustomed repeated concentric (Kemp et al., 1992) and eccentric muscle contractions (McCully et al. 1988; Yanagisawa et al., 2003). These studies reported changes in the ratio of inorganic phosphate (Pi) to phosphocreatine (PCr) to reflect non-specific muscle damage through an alteration in the creatine kinase equilibrium. The breakdown products of phospholipids can be seen on a ^{31}P-MR spectra as phosphodiesters (PDE). As a result, the appearance of PDE on a ^{31}P-MR spectrum is reported to reflect increased cell membrane turnover (Jubrias et al., 1994; Sprott et al., 2000). From our search of the literature, no studies have investigated the relationship between PDE and downhill walking induced muscle damage in healthy individuals to date.

Despite the potential of eccentric exercise in a clinical setting, little is known about how to minimise the loss of skeletal muscle function and DOMS that can accompany eccentric exercise. The purpose of this study was to observe the effects of 30-min of downhill walking upon muscle metabolism and severity of DOMS. The effect of compression garments applied after eccentric exercise was also studied. Attenuation of any changes in cellular bioenergetics or acceleration of the tissue repair process could aid recovery following eccentric exercise. The hypothesis to be tested was that wearing compression garments in the recovery from eccentric exercise could attenuate muscle metabolite changes and promote cellular repair.

METHODS

Subjects participated in a parallel design study to observe the effects of graduated compression garments following 30 minutes of downhill walking (eccentric muscle exercise). At baseline, both legs underwent ^{31}P-MRS evaluation. Subjects then completed 30 minutes of downhill walking. Immediately following exercise, subjects wore graduated compression garments on one leg. The non-compressed leg acted as an internal control. The ^{31}P-MRS evaluations were repeated 1 hour and 48 hours following the downhill walking. At all time points, the compression of the garment and perceived muscle soreness were also assessed. All values will be presented as mean ± SD unless otherwise stated.

Subjects

Eleven male recreational athletes were recruited for this study (age 21.2 ± 3.1yrs, height 1.81 ± 0.06m, weight 77.9 ± 9.1kg). Participants were requested to refrain from physical exercise and any treatment for muscle soreness or damage for 48 hours prior to and during the study. This study complied with ethical guidelines laid down for human research by the Australian NHMRC and was approved by the

University of Sydney Human Research Ethics Committee and the Royal North Shore Hospital Ethics Committee. Before taking part in the study, all subjects were made aware of the experimental procedures involved, and gave written informed consent to participate.

Eccentric exercise protocol

Participants performed a downhill walking protocol for 30 minutes on a treadmill (6 km·h^{-1}, 25% grade). The protocol was adapted from a previously reported procedure shown to induce eccentric muscle damage (Balnave and Thompson, 1993).

Compression garments

Immediately following exercise and until the end of the experiment (48-hrs), graduated compression garments were worn covering the calf and thigh on one leg (Skins ®, Skins Compression Garments, Sydney, Australia – 76% Nylon and Meryl Microfibre, 24% Roica Spandex) (Figure 1). The leg wearing the compression garment was randomly assigned (dominant v/s non-dominant). At all time points the compression of the garments on the leg was measured at the calf and thigh three times at each site using a sub bandage pressure monitoring device (Kikuhime, Soro, Germany). The mean of the three values was reported.

Figure 1. Illustration of the graduated compression garment (Skins Compression Garments, Sydney, Australia).

^{31}P-Magnetic Resonance Spectroscopy

At the baseline examination, two marks were made with indelible markers on the participants' thighs equidistant from the proximal and distal heads of the femur. Vitamin-E capsules were taped over these marks on each examination. The marks and capsules acted as reference points on the MR scout images and aided positioning of the magnetic resonance spectroscopy coil.

The participant lay prone on a custom built board including a 10 cm P-100 pulse-receive coil (Philips Medical Systems, Best, Netherlands), allowing examination of the quadriceps muscle group (vastus medialis, vastus lateralis, vastus intermedius and rectus femoris). Subjects were then placed in a 1.5T Philips MR magnet (Philips Medical Systems, Best, Netherlands). The magnetic field was automatically shimmed twice to a localised volume (approximately 50 x 50 x 20 mm) of muscle directly above the centre of the coil to obtain a homogenous magnetic field. The P-100 coil was then manually tuned for frequency and loading.

An adiabatic pulse sequence was used to collect eight unlocalised proton decoupled (DEC) and Nuclear Overhauser Enhanced (NOE) ^{31}-P spectra (TR = 15000 ms; total acquisition time 120sec). Prior to acquisition, 10 dummy measurements were undertaken to decrease T^1 effects.

Interpretation of spectra

Interpretation of the averaged spectra was performed using the java based magnetic resonance user interface (jMRUI version 2.0, http://www.mrui.uab.es/mrui/) (Naressi et al., 2001). Fast Fourier transformed spectra were analysed using a non-linear-least-squares-algorithm [AMARES] with custom designed fitting routine (prior knowledge). Seven metabolites (eleven resonance peaks) were analysed from the ^{31}P spectra (Figure 2): β-adenosine tri-phosphate (ATP), αATP, γATP, phosphocreatine (PCr), phosphodiester (PDE), inorganic phosphate (Pi), phosphomonoester (PME). The chemical shift between PCr and Pi was used to analyse pH (Moon and Richards, 1973). The chemical shift between βATP and PCr was used to calculate intracellular free Mg^{2+} ([Mg^{2+}]) (Iotti et al., 2000). Intracellular concentrations of PCr, PME and PDE ([PCr], [PME] and [PDE] respectively) were calculated relative to βATP, assuming a resting βATP concentration of 8.2 mmol·L^{-1} and corrected for magnetic saturation (Kemp and Radda ,1994).

Perceived Muscle Soreness

Perceived muscle soreness of each thigh was assessed using a scale of 1 (no pain at all) to 10 (very, very sore).

Statistical analysis

Changes in all variables over time were analysed using a repeated measures ANOVA with a post hoc-Tukey test. An alpha level corrected paired T-test was used to assess variance between the control leg and compression garment at each time point. Statistical significance (P) was set < 0.05.

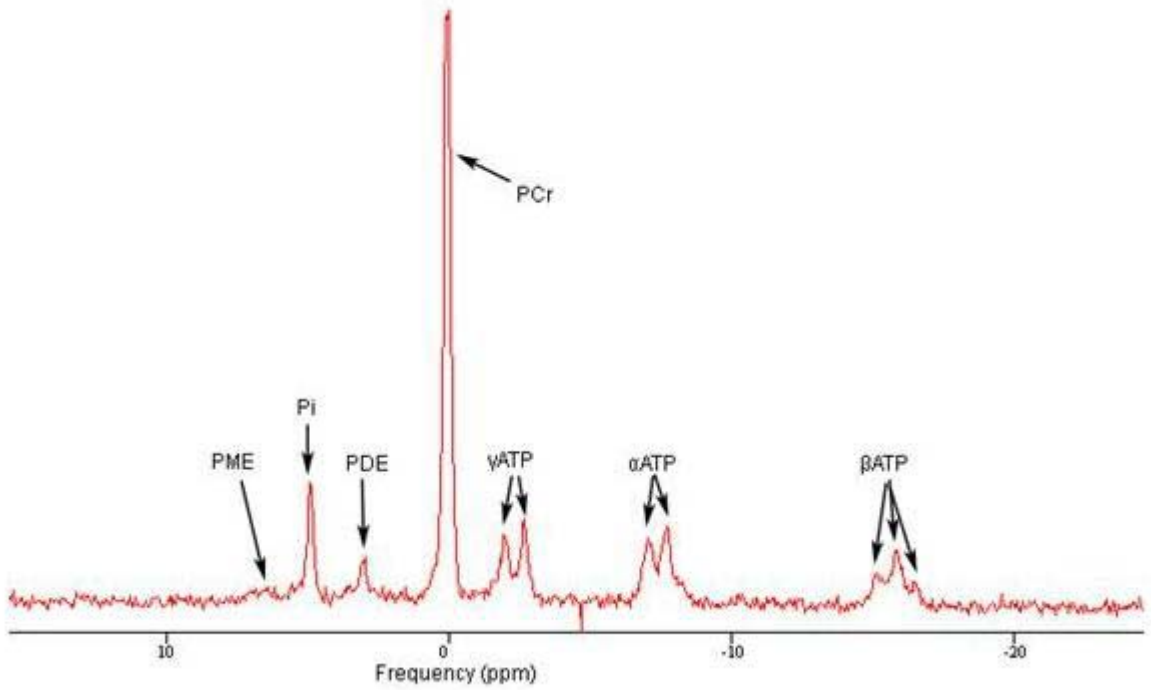

Figure 2. A resting ^{31}P-Magnetic Resonance Spectra of the quadriceps muscle group (DEC and NOE enhanced adiabatic pulse sequence; TR 15000).
Abbreviations: PME: Phosphodiester, Pi: inorganic phosphate, PDE: Phosphodiester, PCr: Phosphocreatine, γATP: γAdenosine triphosphate, αATP: αAdenosine triphosphate, βATP: βAdenosine triphosphate. The Pi peak observable in the MR spectra comprises two peaks; HPO_4^{2-} and H_2PO^-. The acidic form of the Pi peak resonates closer to the PCr peak than the more basic form, allowing determination of the intracellular pH from the resonance frequency of Pi as either the basic or acidic form of Pi dominates.

RESULTS

At baseline there was no significant difference in [PME], [PDE], PCr/Pi, pH or [Mg^{2+}] between legs. One hour and 48 hours after exercise, there was no effect of eccentric exercise or compression on [PME], PCr/Pi, or [Mg^{2+}] (Table 1).

One hour after eccentric exercise COMP showed a significant elevation in [PDE] relative to CONT ($p < 0.05$) (Figure 1). Both CONT and COMP showed no change in [PDE] relative to baseline at 1 hr or 48 hrs following eccentric exercise (Figure 3).

One hour after the completion of eccentric exercise pH was reduced to a similar extent in both the CONT and COMP legs ($p < 0.05$) (Figure 4). 48 hours after eccentric exercise pH had recovered to levels indistinguishable from baseline in both CONT and COMP legs (Figure 4). There was no significant difference in pH between legs at any time point (Figure 4).

Perceived muscle soreness was significantly elevated from baseline at 1 hour and 48 hours in both CONT (1.1 ± 1.0, 3.3 ± 2.1, 4.4 ± 2.2 respectively) and COMP legs (1.1 ± 1.1, 2.3 ± 2.2, 3.5 ± 2.4 respectively) ($p < 0.05$). There was no significant difference in perceived muscle soreness between the CONT and COMP at any time points. At both post-exercise evaluations, COMP showed a greater compression in the calf than the thigh ($p < 0.05$). Forty-eight hours after eccentric exercise, there was a reduction in calf compression (16±2 cf 17±2 mmol·Hg) ($p < 0.05$) and no change in thigh compression (10±2 cf 10±2 mmol·Hg).

Table 1. Free intracellular Magnesium ([Mg^{2+}]) and Phosphomonoester (PME) concentration and PCr/Pi were measured in Vastus Lateralis muscle at rest, 1 hour following and 48 hours following eccentric exercise. Data are expressed as means (±SD). Legs were assessed with compression garments (compression) or without (control).

	Baseline		**1-hr**		**48-hr**	
	CONT	**COMP**	**CONT**	**COMP**	**CONT**	**COMP**
[Mg^{2+}] mM	.25 (.03)	.24 (.02)	.27 (.07)	.26 (.03)	.25 (.03)	.25 (.05)
PCr/Pi	5.5 (1.5)	5.8 (1.1)	5.4 (2.6)	5.6 (1.8)	5.2 (1.3)	5.1 (1.0)
[PME] mmol·L^{-1}	.7 (.5)	.7 (.3)	.7 (.5)	.8 (.4)	.9 (.4)	.9 (.4)

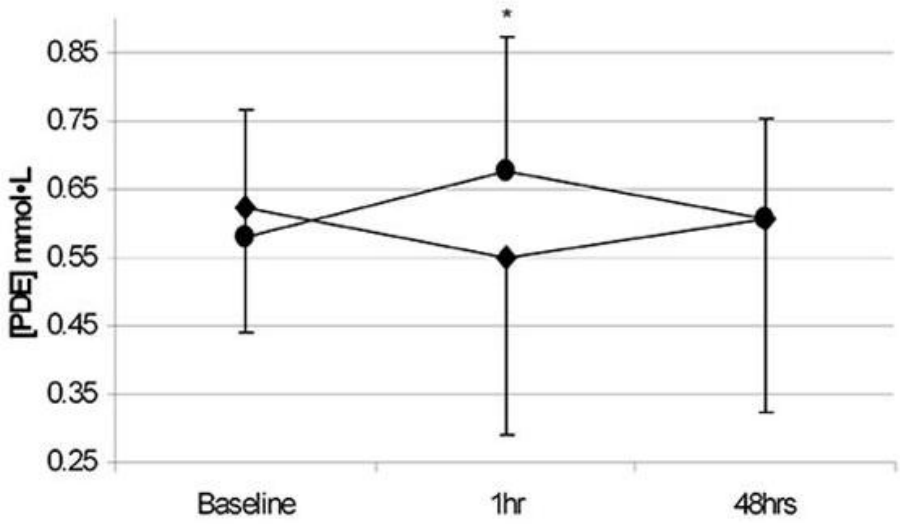

Figure 3. Skeletal muscle intracellular phosphodiester ([PDE]) at baseline, 1hr and 48hrs following eccentric muscle exercise in control (diamonds) and compression (circles). * $p < 0.05$ compared with control group.

DISCUSSION

This study has shown that compression garments resulted in a relative increase of [PDE] in the thigh one hour after eccentric exercise. One hour after exercise, pH was also significantly reduced, however compression garments showed no effect on pH. There were no observable differences in PCr/Pi, Mg^{2+} or [PME] over time or with the use of compression garments. Perceived level of muscle soreness was elevated at all time points post exercise with compression garments showing no treatment effect.

The low oxidative demand of eccentric exercise possesses unique benefits to clinical populations with impaired lung function (Rooyackers et al., 2003) and mitochondrial disease (Taivassalo et al., 1999) amongst others. The structural adaptation of the muscle to eccentric exercise can also benefit both clinical and non-clinical populations (Proske and Morgan, 2001). Despite these benefits, eccentric exercise is accompanied by DOMS 24 - 48 hours after exercise. The mechanisms for soreness characterising DOMS have been recently discussed (Proske and Allen, 2005; Proske and Morgan, 2001). These suggest that during repeated eccentric muscle contractions, sarcomeres become overstretched and eventually disrupted. The structural distortions produced by sarcomere disruptions result in membrane damage, including damage to the sarcoplasmic reticulum, transverse tubules or the sarcolemma (Proske and Allen, 2005). The resultant release of intracellular Ca^{2+} signals proteolysis and begins the breakdown of

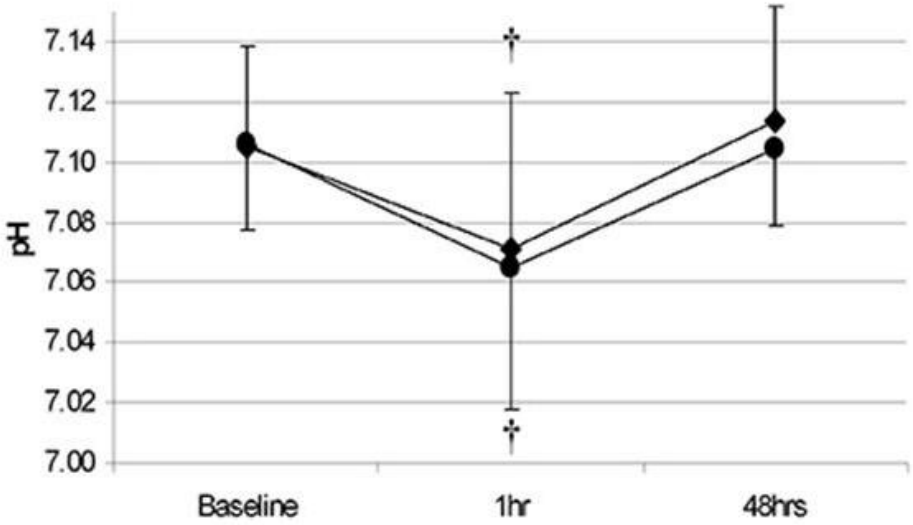

Figure 4. Skeletal muscle pH at baseline, 1hr and 48hrs following eccentric muscle exercise in control (diamonds) and compression (circles). † $p < 0.05$ compared with baseline.

damaged fibres. These processes are associated with an influx of macrophages and monocytes into the damaged area and are accompanied by oedema (Proske and Morgan, 2001).

In line with reports of tissue regeneration following muscle damage (Smith 1991), the elevated phosphodiester (PDE) following eccentric exercise and compression may be representative of increased skeletal muscle membrane turnover. The lipid metabolites glycerophosphocholine (GPC) and resembling phosphodiesters (PDE) are breakdown products of phospholipids (Schmidt et al., 1952). Visible at 2.99 ppm (relative to PCr at 0ppm), elevated PDE is representative of a higher rate of membrane turnover, indicating changed phospholipid metabolism (Sprott et al., 2000). Elevated PDE is seen in fibromyalgia (Jubrias et al., 1994; Sprott et al., 2000), inflammatory, mitochondrial and metabolic myopathies (Argov et al., 1998; Kemp et al., 1993; Matthews et al., 1991). In these diseases, skeletal muscle can end up in a state of repair as the tissue alters the balance of pro- and anti-inflammatory mediators. The impaired metabolic capacity of the tissue results in an activation of the inflammatory pathways, increasing muscle cell membrane turnover and consequently increasing the appearance of PDE on a ^{31}P MR spectra. As such, the observation of PDE in these diseases lends support to the concept that PDE is representative of increased membrane turnover. Consequently, in these healthy individuals we suggest that the increase in PDE is representative of an accelerated inflammatory and repair timeframe. From our understanding, this is the first serial observation of PDE in healthy skeletal muscle following muscle damage. As a consequence we are unable to compare our observations with previous studies. However, it could also be reasoned that changes in PDE could be a consequence of under perfusion or representative of a change in the total nucleotide pool as discussed below.

Vasoconstriction could produce an increase in PDE, as the supply of oxygen to the muscle and the ability to clear protons is reduced, initiating a stress response and the breakdown of muscle cells. A previous study has observed the effect of drug induced vasoconstriction on skeletal muscle metabolism. This study observed the onset of myalgia 48-hrs after administration of Bryostatin (an anti-neoplastic agent and protein kinase C activator) and was accompanied by an increase in PDE, observed using ^{31}P-MRS (Hickman et al., 1995). The authors observed changes constant with vasoconstriction (proton retention and increased intracellular ADP) and attributed vasoconstriction to the elevation in PDE. Although the present study shows reduced pH at 1-hr, in line with proton retention and vasoconstriction, this was observed in both the control and compression legs suggesting that the compression garments were not inducing vasoconstriction. Furthermore, there were no changes in PCr/Pi, which would indicate changes in resting intracellular ADP and would be expected to accompany vasoconstriction. Combined, the observation of no effect of the compression garments on pH and the stability of PCr/Pi over time supports the concept that compression garments do not cause vasoconstriction.

As absolute quantification is not possible using ^{31}P-MRS it is feasible that changes in the relative quantity of metabolites may result in an over- or under-estimation of the reference metabolite. Using ^{31}P-MRS, relative metabolite quantities, such as [PDE], are expressed relative to an assumed ATP concentration of 8.2 mmol·L^{-1} (Kemp and Radda, 1994). However, eccentric exercise has been shown to result in the release of CK into the circulation (Sayers and Clarkson, 2003). A reduction in CK content in muscle may result in a reduction of metabolites such as creatine phosphate and ATP as a consequence of reduced ability to maintain the CK equilibrium. Therefore, it is possible that the effective change in the intracellular concentration of PDE is not due to an increase in PDE itself; rather it represents a change in β-ATP peak area or a loss in the nucleotide pool. However, although being measured in the same manner as PDE, the ratios of PCr/β-ATP or PME/β-ATP did not follow a similar trend. Furthermore, a previous study has reported that following eccentric exercise the release of CK into the circulation from skeletal muscle is attenuated with the use of compression garments (Kraemer et al., 2001a). Retention of CK, and resultant maintenance of the CK equilibrium with compression garments, would produce an effective reduction in [PME]. The present study showed an increase in [PDE], supporting the hypothesis that there was an increased cellular membrane turnover independent of any changes in CK or the nucleotide pool.

Combined, these observations suggest that the increase in [PDE] 1 hour following eccentric exercise is a result of an alteration of the inflammatory and repair processes as opposed to vasoconstriction or as a loss of CK. This would follow the increased repair processes and raised [PDE] observed in myalgia, fibromyalgia (Jubrias et al., 1994; Sprott et al., 2000), inflammatory, mitochondrial and metabolic myopathies (Argov et al., 1998; Kemp et al., 1993; Matthews et al., 1991). Increased muscle repair would also account the superior effects of wearing compression garments

upon muscle function following eccentric exercise (Kraemer et al., 2001a). However, additional studies showing muscle performance and direct biochemical effects of compression garments on the muscle in recovery from eccentric muscle damage are necessary to confirm these hypotheses.

Following eccentric exercise, previous studies have used the ratio of PCr and Pi as a marker of muscle damage, concluding that the ratio of these metabolites can reflect non-specific damage in normal muscle (McCully et al., 1988). Interestingly we showed no change in PCr/Pi at any time point. As a consequence of the CK equilibrium, changes in PCr/Pi can be representative of variations in intracellular ADP ([ADP]). In healthy skeletal muscle, variation in the concentration of [ADP] controls the availability of intracellular free energy, directly determining the rate of mitochondrial ATP synthesis. As such, PCr/Pi indicates altered muscle metabolism through an increased resting oxidative flux or a reduced sensitivity of the mitochondria to changes in cellular free energy. Our study supports previous work (Yanagisawa et al., 2003) and suggests that 30minutes of downhill walking does not cause an alteration in resting oxidative flux or change the sensitivity of the mitochondria to cellular free energy.

Although there were no changes in PCr/Pi throughout the study, a reduction in the intracellular pH was observed 1hour after exercise in both legs. Proton efflux from skeletal muscle is achieved by a combination of sodium hydrogen exchange, bicarbonate influx and lactate efflux. Intracellular pH is also dependent upon tissue temperature. Skeletal muscle intracellular pH decreases with increasing temperature, through increased thermal activity of the Lohmann reaction (Binzoni et al., 2000). However, based on the Lohman reaction, a decrease in intracellular pH as a result of temperature would be accompanied by an increase in [ADP] (Binzoni et al., 2000), or an effective reduction in PCr/Pi. Again, we did not observe any change in PCr/Pi over all time points. The disassociation of changes in cell pH and PCr/Pi suggests that there is impaired control of muscle cell pH following eccentric exercise which is not temperature dependent. It is possible that an alteration in cell pH could be the consequence of the structural reorganisation of the sarcomere.

The prevention of venous stasis with compression garments could possibly aid proton removal from the muscle. However, studying proton removal at rest using ^{31}P-MRS is not particularly profitable. A more accurate determination could be gained from observation of the response of intracellular pH in the minutes immediately following exercise. To date, no studies have looked at the effect of compression garments on proton efflux.

Subjective reports of muscle soreness showed an increase from baseline 1-hr and 48-hrs following eccentric exercise in both CONT and COMP. Interestingly, there was no significant difference between CONT and COMP at any time. It should also be noted that the perceived muscle soreness was not significantly different between 1-hr and 48-hr which is contrary to previous observations (Kraemer et al. 2001a; 2001b). However, subjective reports of muscle soreness have been reported to be a poor indicator of cellular damage (Nosaka et al., 2002). Furthermore, the pressure of the garment on the muscle may directly affect perceived muscle soreness. Further work investigating the relationship between compression garments and subjective responses is warranted.

CONCLUSIONS

This study shows that eccentric exercise can cause disruption to skeletal muscle pH control and induce cellular metabolic changes consistent with inflammatory and repair processes. Thirty minutes of downhill walking did not influence control of the CK equilibrium. The data suggests that wearing compression garments in the recovery from eccentric exercise may alter the inflammatory response to damage and accelerate the repair processes inside of the muscle. However, further studies are warranted to confirm any alteration in muscle repair/recovery consequent to wearing of compression garments, determine the mechanisms, and understand the functional benefits underlying both eccentric exercise and the use of compression garments in healthy and clinical populations.

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KEY POINTS

- Eccentric exercise results in reduced cellular pH 1 hour after exercise.
- Graduated compression garments may influence indices of muscle membrane repair and turnover at one-hour recovery from eccentric exercise.
- Graduated compression garments do not change perceived muscle soreness.

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