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# Neuromuscular electrical stimulation via the peroneal nerve is superior to graduated compression socks in reducing perceived muscle soreness following intense intermittent endurance exercise

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## Abstract

**Purpose** A novel technique of neuromuscular electrical stimulation (NMES) via the peroneal nerve has been shown to augment limb blood flow which could enhance recovery following exercise. The present study examined the effects of NMES, compared to graduated compression socks on muscle soreness, strength, and markers of muscle damage and inflammation following intense intermittent exercise.

**Methods** Twenty-one (age  $21 \pm 1$  years, height  $179 \pm 7$  cm, body mass  $76 \pm 9$  kg,) healthy males performed a 90-min intermittent shuttle running test on three occasions. Following exercise, the following interventions were applied: passive recovery (CON), graduated compression socks (GCS) or NMES. Perceived muscle soreness (PMS) and muscle strength (isometric maximal voluntary contraction of knee extensors and flexors) were measured and a venous blood sample taken pre-exercise and 0, 1, 24, 48 and 72 h following exercise for measurement of creatine kinase (CK) and Lactate dehydrogenase (LDH) activity and IL-6 and CRP concentrations.

**Results** PMS increased in all conditions immediately, 1 and 24 h post-exercise. At 24 h PMS was lower in NMES compared to GCS and CON ( $2.0 \pm 1.6$ ,  $3.2 \pm 2.1$ ,  $4.6 \pm 2.0$ , respectively). At 48 h PMS was lower in NMES compared to CON ( $1.3 \pm 1.5$  and  $3.1 \pm 1.8$ , respectively). There were no differences between treatments for muscle strength, CK and LDH activity, IL-6 and CRP concentrations.

**Conclusions** The novel NMES technique is superior to GCS in reducing PMS following intense intermittent endurance exercise.

**Keywords** DOMS · Muscle damage · Muscle function

## Abbreviations

ANOVA	Analysis of variance
CK	Creatine kinase
CRP	C-Reactive protein
DOMS	Delayed onset muscle soreness
EIMD	Exercise-induced muscle damage
ELISA	Enzyme-linked immunosorbent assay
GCS	Graduated compression socks
IL-6	Interleukin-6
LDH	Lactate dehydrogenase
LIST	Loughborough intermittent shuttle test
LFES	Low-frequency electrical stimulation
MVC	Maximal voluntary contraction
NMES	Neuromuscular electrical stimulation
PMS	Perceived muscle soreness
RBE	Repeated bout effect
SD	Standard deviation
TENS	Transcutaneous electrical nerve stimulation
TNF- $\alpha$	Tumour necrosis factor- $\alpha$
VO <sub>2max</sub>	Maximal oxygen uptake

## Introduction

The intermittent exercise pattern of sports such as soccer, hockey and rugby, which involves rapid deceleration, change of running direction and landing after an aerial movement, can lead to high levels of exercise-induced muscle damage (EIMD) (Magalhaes et al. 2010; Thompson

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et al. 1999). EIMD is primarily caused by excessive tension being applied to sarcomeres and subsequent disruption of the muscle fibre structure (Proske and Morgan 2001; Warren et al. 2001). These events contribute to a prolonged loss of muscular force which returns to baseline in a linear fashion over a period of days (Clarkson et al. 1992; Jones et al. 1986). Damage to muscle fibres leads to the leakage of intracellular proteins, such as creatine kinase and lactate dehydrogenase (Clarkson et al. 1986), as well as an increase in perceived muscle soreness (PMS), often referred to as delayed onset muscle soreness (DOMS). Furthermore, there is an acute inflammatory response following muscle-damaging exercise with an infiltration of immune cells into the circulation (Smith 1991). This response acts to mediate the process of necrotic tissue breakdown whereby macrophages release inflammatory-related cytokines, such as interleukin-6 (IL-6) (MacIntyre et al. 1995) and consequent increase in C-reactive protein (CRP) concentration (Toft et al. 2002). Taken together, although there is large variability in such responses (Warren et al. 1999), these observations are considered to be acute markers of EIMD and can provide an indication of any subsequent recovery.

The incidence and severity of EIMD can have a profound effect on the ability to perform subsequent bouts of exercise, which is relevant during intense training as well as competitions (e.g. tournaments where games are often only several days apart). Therefore, a variety of strategies and interventions are being used as an important component of the post-exercise recovery process; including active recovery, cryotherapy, compression garments, and electro-stimulation devices (Howatson and van Someren 2008). The potential benefits purported by the use of these interventions are that of an altered haemodynamics which serves to facilitate a greater removal of tissue damaging molecules and a reduction in localised oedema.

The application of a pressure gradient by the use of compression garments causes a reduction in venous pooling and improved venous return from the lower extremities (Liu et al. 2008). Despite the popularity of compression garments, there is conflicting evidence concerning their ability to attenuate the loss of performance characterised by EIMD and DOMS. Jakeman et al. (2010) observed that the application of lower limb compression tights for 12 h following plyometric exercise reduced levels of PMS along with improved recovery of isokinetic muscle strength, squat jump and countermovement jump performance. Ali et al. (2007) and Duffield et al. (2010) also observed perceptual improvements with compression stockings following intense intermittent exercise. However, there were no improvements in exercise performance (Ali et al. 2007), skeletal muscle performance or markers of muscle damage (Duffield et al. 2010) during the recovery period.

Neuromuscular electrical stimulation (NMES) is a technique which initiates the skeletal muscle pump via transcutaneous stimulation of muscle fibres that in turn increases local blood flow. There are a number of studies that have investigated the potential to reduce muscle soreness using different types of electrical stimulation techniques, with conflicting results (for review see Babault et al. 2011). Vanderthommen et al. (2010) did not show any beneficial effect of low-frequency electro-stimulation (LFES; Complex) on PMS following fatiguing isometric contractions. Neither Bieuzen et al. (2012), after high-intensity intermittent running, nor Cortis et al. (2010), after submaximal running, observed any recovery benefit of NMES on PMS. Likewise, Tessitore et al. (2008) reported no reductions in muscle pain following futsal games. However, Denegar and Perrin (1992) observed favourable effects of transcutaneous electrical nerve stimulation (TENS) following maximal eccentric contractions and Tessitore et al. (2007) identified significantly lower PMS following NMES during 21 days of soccer pre-season training. Clearly, there are substantial differences in the nature of the damaging exercise as well as stimulation techniques and parameters adopted. Moreover, it is important to appreciate that many current electro-stimulation devices are relatively large and the relevant electrodes are connected to battery packs making them cumbersome and thus limited for prolonged use or when travelling.

Recently, a technique involving direct electrical stimulation of the peroneal nerve resulting in mild activation of the lower leg muscles has been observed to significantly increase blood flow in the superficial femoral vein to levels of between 50 and 70 % of the blood flow achieved during walking (Tucker et al. 2010). As a consequence of its demonstrated ability to increase whole limb blood flow (in the upper portion of the limb as well as the stimulated calf muscles), it is suggested that this technique may enhance the recovery process following an intense bout of exercise by removing or reducing any of the factors associated with muscle damage and soreness. Therefore, the aim of this study was to examine the effect of this novel NMES device on recovery following prolonged intermittent exercise. Given the contradictory evidence of the benefit of graduated compression socks in the recovery process the effect of these garments was also evaluated and compared to the novel NMES device. Both were compared to a control condition involving passive recovery. Indices of muscle damage including perceived muscle soreness, muscle strength, intracellular protein leakage (CK and LDH activity), inflammation (IL-6 and CRP) and oxidative damage (TBARS) were assessed for 72 h following a standardised intermittent endurance exercise protocol designed to replicate sports such as soccer and hockey (the Loughborough Intermittent Shuttle Test, LIST; Nicholas et al. 2000).

## Methods

### Participants

Twenty-one healthy males (mean  $\pm$  SD; age  $21 \pm 1$  years, height  $179 \pm 7$  cm, body mass  $76 \pm 9$  kg, body mass index  $24 \pm 2$  kg m<sup>-2</sup>, predicted maximal oxygen uptake ( $\text{VO}_{2\text{max}}$ )  $54 \pm 5$  ml kg<sup>-1</sup> min<sup>-1</sup>) volunteered to participate in the study. All participants were currently playing intermittent sports (soccer  $n = 6$ , field hockey  $n = 6$ , rugby  $n = 4$ , other sports  $n = 5$ ) and were currently representing their respective sports at either university, county, national or international levels. The participants were fully informed of the purposes, risks and discomforts associated with the experiment before providing written, informed consent. This study conformed to current local guidelines and the Declaration of Helsinki and was approved by Loughborough University Ethics Advisory Committee.

### Experimental design

All participants performed a modified version of the LIST (Nicholas et al. 2000), an exercise protocol designed to simulate the activity pattern characteristics of intermittent sports such as soccer. The LIST was performed on three occasions, at the same time of day, each separated by approximately 4 weeks. Following each exercise trial, one of three recovery interventions were applied, the order of which were randomly allocated. The interventions consisted of passive recovery (CON), graduated compression socks (GCS) and a NMES device. Measurements of perceived muscle soreness (PMS), muscle strength and a venous blood sample were taken pre-exercise and at 0, 1, 24, 48 and 72 h post-exercise.

### Preliminary measurements

At least 1 week before the first experimental trial, participants performed the Multi-Stage Fitness Test (Ramsbottom et al. 1988) to ascertain their predicted  $\text{VO}_{2\text{max}}$ . These  $\text{VO}_{2\text{max}}$  values were used in conjunction with the regression equation developed by Ramsbottom et al. (1988) to calculate the running speeds utilised in the LIST protocol. After approximately a 15-min recovery, participants were familiarised to the LIST protocol and, followed by a further 15-min recovery, the muscle strength testing protocol. On a subsequent visit, participants repeated the strength testing protocol.

### Experimental protocol

Participants arrived at the laboratory in the morning after an overnight fast. After remaining seated for at least

10 min, a resting blood sample was taken and PMS was assessed. Participants then performed the muscle strength test on both legs before completing an adapted version of the LIST protocol inside an exercise laboratory. This consisted of two 45-min sections of continuous intermittent exercise, separated by a 15-min rest period, according to the following pattern:  $3 \times 20$  metres at walking pace,  $1 \times 20$  metre maximal sprint, 4 s recovery,  $3 \times 20$  metres at a running speed corresponding to 75 %  $\text{VO}_{2\text{max}}$ ,  $3 \times 20$  metres at a running speed corresponding to 100 %  $\text{VO}_{2\text{max}}$ . Participants ran between markers placed 20 m apart and these varying running speeds were dictated by an audio signal. During the exercise trial, participants consumed water ad libitum and the volume ingested was recorded to estimate loss of fluid through sweat. Heart rate was measured via short-range telemetry (Polar Electro, Kempele, Finland) at the beginning of each exercise cycle. Average heart rate was then calculated over six 15-min periods. Immediately (0 h) following completion of the LIST protocol, PMS was recorded and a blood sample taken. Muscle strength was assessed within 10 min of the end of exercise. Participants then remained seated for a further hour before another blood sample was taken and PMS and muscle strength were assessed (1 h). At this point, recovery interventions were applied and the participants left the laboratory. They returned to the laboratory on a further three occasions where measurements of PMS, muscle strength and a venous blood sample were taken at 24, 48 and 72 h post-exercise. Participants were asked to refrain from vigorous physical activity, caffeine and alcohol for 24 h prior to exercise and throughout the trial until the final blood sample at 72 h post-exercise. Participants were asked to avoid heavy training sessions and to repeat a similar schedule after each exercise trial. Furthermore, participants were asked to record their pre-exercise diet for 24 h and repeat this before each subsequent exercise trial.

### Recovery interventions

The recovery interventions were applied immediately after the 1 h post-exercise measurements.

### Neuromuscular electrical stimulation (NMES)

The geko™ T-1 neuromuscular stimulation device (First-kind Limited, High Wycombe, UK), which incorporates On Pulse™ technology, was positioned on both legs according to the manufacturer's instructions. The device was applied slightly above the posterior fold of the knee joint, using the biceps femoris tendon as guidance, with the control button on the lateral side of the knee (Fig. 1). Prior to application, the area was exfoliated with an



**Fig. 1** Placement of NMES device above the posterior fold of the knee joint

abrasive pad before being wiped with an isopropyl alcohol electrode preparation pad. The device (16 grammes; 149 mm × 42 mm × 11 mm) stimulates at a frequency of 1 Hz with a pulse current of 27 mA. Pulse width ranged from 70 to 560  $\mu$ s over 7 pre-set levels. The stimulation level was selected on an individual basis and was dependent on the sensitivity of the participant but was no lower than 140  $\mu$ s (level 3) with the main subjective output being a consistent and clearly visible outward movement of the foot. Participants were encouraged to increase the stimulation to as high as was comfortably tolerable and to maintain stimulation for at least 12 h.

#### *Graduated compression socks (GCS)*

Graduated compression recovery socks (40 mmHg at the ankle to 20 mmHg at the calf; 2XU, Melbourne, Australia) were worn according to the manufacturer's sizing guide. Participants were instructed to wear them for at least 12 h, where comfort allowed, and only remove them for short periods should they require to wash.

#### *Passive recovery (CON)*

Participants returned to their daily routine without of any form of intervention strategy.

## Assessment of muscle soreness and strength

### *Perceived muscle soreness*

Ratings of perceived muscle soreness were assessed using a visual analogue scale ranging from 0 ("no pain") to 10 ("unbearable pain"). Participants rated soreness of the legs while standing in a relaxed state and were encouraged to gently palpate with the fingers major muscle groups (thigh and calf muscles) during assessment.

### *Muscle strength*

Isometric maximal voluntary contractions (MVC) of the knee extensors and flexors of both legs were assessed using an isokinetic dynamometer (Cybex Norm, Massachusetts, USA). Participants sat upright with their torso and leg secured, with a hip angle of approximately 90°. Following a warm-up set of five sub-maximal isokinetic repetitions of knee extension and flexion (1.05 rad s<sup>-1</sup>), participants completed two maximal isometric knee extension contractions at a joint angle of 60° (where 0° was set at full knee extension) each separated by 1 min. After a further 1-min rest, two maximal isometric knee flexion contractions were performed at a joint angle of 20°, each separated by 1 min. The maximal contractions lasted for 3 s and participants were given consistent verbal encouragement and visual feedback throughout each repetition. The peak value from the two contractions was recorded.

### *Circulatory markers of muscle and oxidative damage and inflammation*

Serial blood samples were taken by venepuncture (21G, Vacutainer, PrecisionGlide, BD) from antecubital veins with the participant in the seated position. Blood samples for plasma (Sodium Heparin, 10 ml, Vacutainer, BD) were placed on ice for approximately 30 min before centrifugation at 4,000 revs min<sup>-1</sup> for 10 min (4 °C). Blood samples for serum (Silica clot activator gel, 8.5 ml, Vacutainer, BD) were placed on ice and left to clot for 45 min. They were then centrifuged at 4,000 revs min<sup>-1</sup> for 10 min (4 °C). Plasma and serum were frozen and stored at -80 °C for subsequent analysis. Serum creatine kinase (CK) and lactate dehydrogenase (LDH) activity were analysed using commercially available enzymatic kits (BQ Kits Inc., San Diego, USA). Plasma Interleukin-6 (IL-6) concentrations were analysed using an in-house enzyme-linked immunosorbent assay (ELISA). Plasma C-reactive protein (CRP) concentrations were analysed using commercially available ELISA kits (R&D Systems, Minneapolis MN, USA).

**Table 1** Heart rate during the LIST in each experimental condition; passive recovery (CON), graduated compression stockings (GCS) and neuromuscular electronic stimulation (NMES)

Heart rate (beats min <sup>-1</sup> )	Recovery intervention		
	CON	GCS	NMES
0–15 min	165 ± 10	164 ± 9	165 ± 9
16–30 min	171 ± 11 <sup>a</sup>	170 ± 10 <sup>a</sup>	170 ± 9 <sup>a</sup>
31–45 min	172 ± 10 <sup>a</sup>	172 ± 11 <sup>a</sup>	172 ± 9 <sup>a</sup>
46–60 min	166 ± 10	166 ± 10	165 ± 9
61–75 min	171 ± 10 <sup>a</sup>	170 ± 11 <sup>a</sup>	170 ± 9 <sup>a</sup>
76–90 min	172 ± 10 <sup>a</sup>	171 ± 11 <sup>a</sup>	171 ± 9 <sup>a</sup>

Values are mean ± SD

<sup>a</sup> Significant difference ( $p < 0.05$ ) from 0 to 15 min in each condition

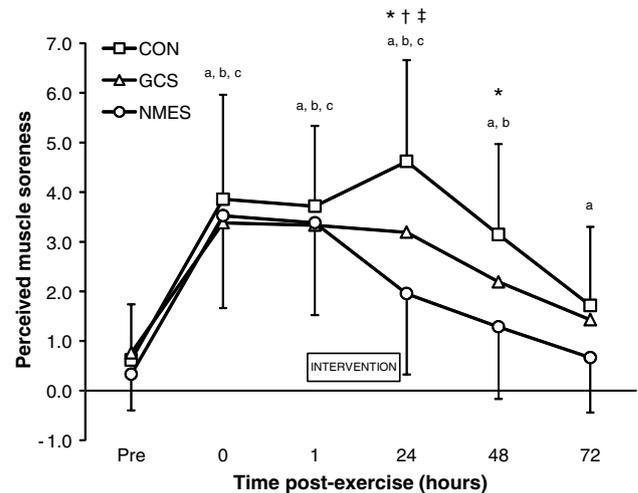
### Statistical analysis

All trial characteristics, circulatory markers, and muscle strength scores during the three trials were analysed using a two-way ANOVA, where the within-subjects factors were time and treatment. Differences in pre-exercise values between the three trials were analysed using a one-way ANOVA. Where sphericity could not be assumed, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity. Post-hoc analyses were conducted where a significant effect was identified using paired samples *t*-tests with Bonferroni adjusted significance values. PMS scores were analysed using Friedman's ANOVA for non-parametric data and where a significant effect was determined, post hoc analyses were conducted using Wilcoxon signed-rank tests. All data were analysed using the Statistical Package for the Social Sciences (SPSS 19.0, Chicago, IL, USA) and the significance level was set at  $p < 0.05$ . Values are expressed as mean ± SD.

### Results

The ambient temperature ( $20 \pm 1$ ,  $21 \pm 2$ ,  $21 \pm 2$  °C;  $p = 0.15$ ), relative humidity ( $38 \pm 10$ ,  $40 \pm 10$ ,  $37 \pm 7$  %;  $p = 0.47$ ) and estimated fluid loss ( $1.80 \pm 0.58$ ,  $1.68 \pm 0.39$ ,  $1.75 \pm 0.37$  l;  $p = 0.44$ ) were consistent between CON, GCS and NMES, respectively. There was a main effect of time on heart rate ( $p < 0.001$ ), which increased and then remained constant during each 'half' of the exercise (Table 1); however, heart rates were consistent between the three experimental trials ( $p = 0.67$ ). The duration each intervention was applied was the same between GCS ( $19 \pm 4$  h; range 9–23 h) and NMES ( $16 \pm 5$  h; range 12–23 h), respectively ( $p = 0.052$ ).

There were no differences in pre-exercise values between the three experimental interventions: PMS ( $p = 0.35$ ), isometric knee extensor torque ( $p = 0.85$ ), isometric knee



**Fig. 2** Perceived muscle soreness before and following the LIST with three different recovery interventions: passive recovery (CON), graduated compression stockings (GCS) and neuromuscular electronic stimulation (NMES). *a* Significant difference ( $p < 0.05$ ) from pre in CON; *b* significant difference ( $p < 0.05$ ) from pre in GCS; *c* significant difference ( $p < 0.05$ ) from pre in NMES; *asterisk* significant difference ( $p < 0.05$ ) CON versus NMES; *†* Significant difference ( $p < 0.05$ ) GCS versus NMES. *‡* Significant difference ( $p < 0.05$ ) CON versus GCS. Values are mean ± SD

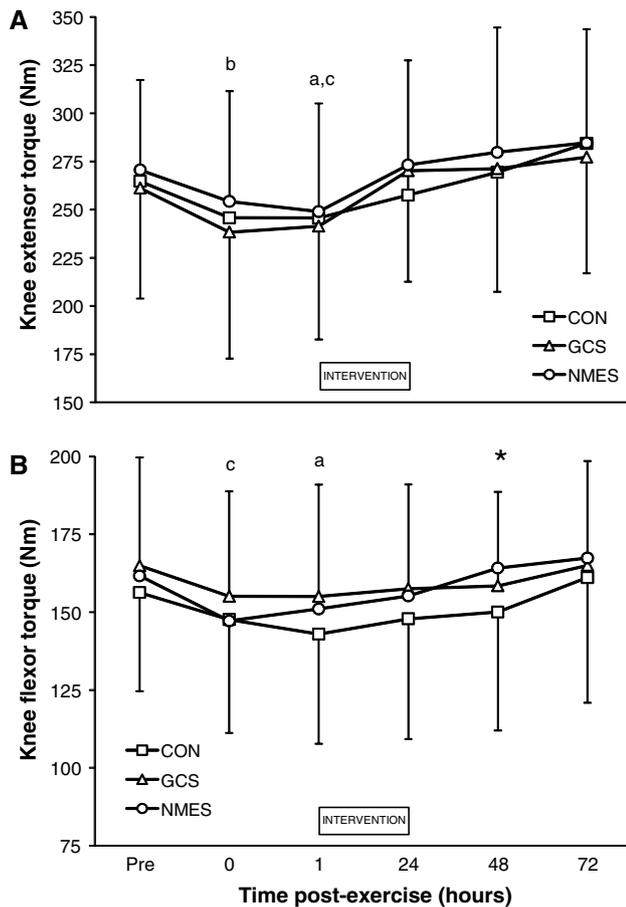
flexor torque ( $p = 0.71$ ), CK ( $p = 0.74$ ), LDH ( $p = 0.51$ ), IL-6 ( $p = 0.91$ ), CRP ( $p = 0.93$ ).

### Perceived muscle soreness

A difference in PMS scores was found between treatments ( $p < 0.001$ ). Post-hoc analyses revealed that PMS increased above baseline in all conditions immediately, 1 and 24 h post-exercise (Fig. 2). PMS remained elevated compared to baseline in CON at 48 and 72 h post-exercise and in GCS at 48 h after exercise. At 24 and 48 h PMS was lower in NMES compared to CON. At 24 h PMS was also lower in NMES compared to GCS and in GCS compared to CON.

### Muscle strength

There was a main effect for time ( $p < 0.001$ ) but no main effect for treatment ( $p = 0.16$ ) and no time × treatment interaction ( $p = 0.71$ ) for isometric knee extensor torque, which decreased compared to baseline in CON by  $7.2 \pm 9.3$  % 1 h post-exercise, in GCS by  $8.7 \pm 12.8$  % immediately post-exercise and in NMES by  $8.0 \pm 13.2$  % 1 h post-exercise (Fig. 3a). Following the intervention, knee extensor torque had increased from 1 h post-exercise back to baseline levels by 24 h in GCS and NMES but not in CON, which had returned to baseline levels by 48 h. There were, however, no differences between treatments at any time point. There was a main effect for time ( $p < 0.001$ ) and a main effect for



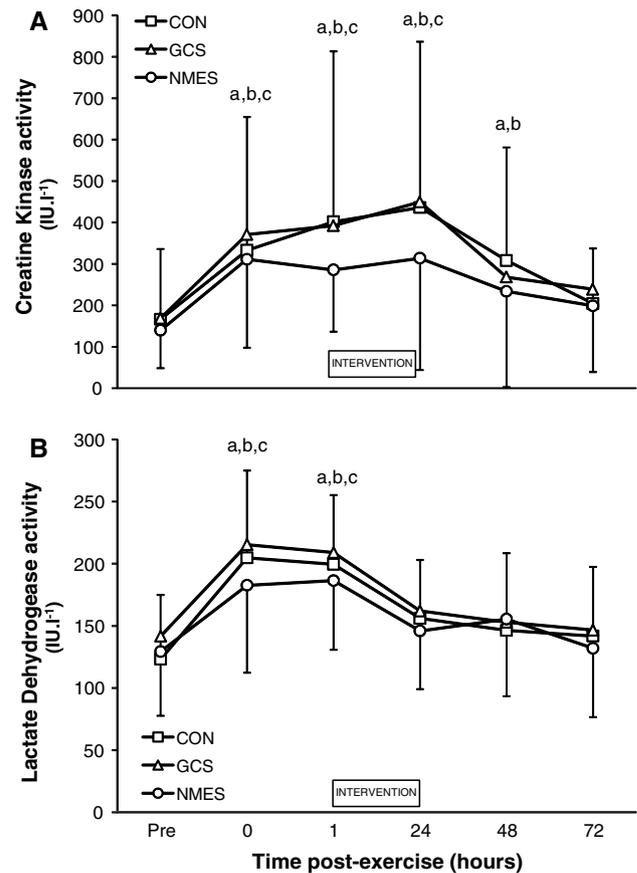
**Fig. 3** Isometric knee extensor (a) and flexor (b) torque before and following the LIST with three different recovery interventions: passive recovery (CON), graduated compression stockings (GCS) and neuromuscular electronic stimulation (NMES). *a* Significant difference ( $p < 0.05$ ) from pre in CON; *b* significant difference ( $p < 0.05$ ) from pre in GCS; *c* significant difference ( $p < 0.05$ ) from pre in NMES; *asterisk* significant difference ( $p < 0.05$ ) CON versus NMES. Values are mean  $\pm$  SD

treatment ( $p = 0.02$ ), but no time  $\times$  treatment interaction ( $p = 0.42$ ) for isometric knee flexor torque, which decreased compared to baseline in CON by  $8.4 \pm 13.0$  % 1 h after exercise and in NMES by  $8.7 \pm 11.3$  % immediately post-exercise (Fig. 3b). Knee flexor torque was constant compared to baseline at all time points in GCS. The only difference between groups was at 48 h where knee flexor torque was higher in NMES compared to CON ( $p = 0.01$ ).

Circulatory markers of muscle and oxidative damage and inflammation

#### Creatine kinase and lactate dehydrogenase activities

There was a main effect for time ( $p < 0.001$ ) but no main effect for treatment ( $p = 0.19$ ) and no time  $\times$  treatment



**Fig. 4** Creatine kinase (a) and lactate dehydrogenase (b) activity before and following the LIST with three different recovery interventions: passive recovery (CON), graduated compression stockings (GCS) and neuromuscular electronic stimulation (NMES). *a* Significant difference ( $p < 0.05$ ) from pre in CON; *b* significant difference ( $p < 0.05$ ) from pre in GCS; *c* significant difference ( $p < 0.05$ ) from pre in NMES. Values are mean  $\pm$  SD

interaction ( $p = 0.50$ ) for serum CK activity (Fig. 4a), which increased in all conditions at 0 and 1 h post-exercise and remained elevated compared to baseline in CON and GCS at 24 and 48 h, but only at 24 h in NMES before returning to baseline at 72 h in all conditions. There were, however, no differences between treatments at any time point. There was a main effect for time ( $p < 0.001$ ) but no main effect for treatment ( $p = 0.78$ ) and no time  $\times$  treatment interaction ( $p = 0.56$ ) for serum LDH activity (Fig. 4b) which increased in all conditions at 0 and 1 h post-exercise, before returning back to baseline after 24 h. There were, however, no differences between treatments at any time point.

#### Interleukin-6 and C-reactive protein concentrations

There was a main effect for time ( $p < 0.001$ ) but no main effect for treatment ( $p = 0.90$ ) and no time  $\times$  treatment

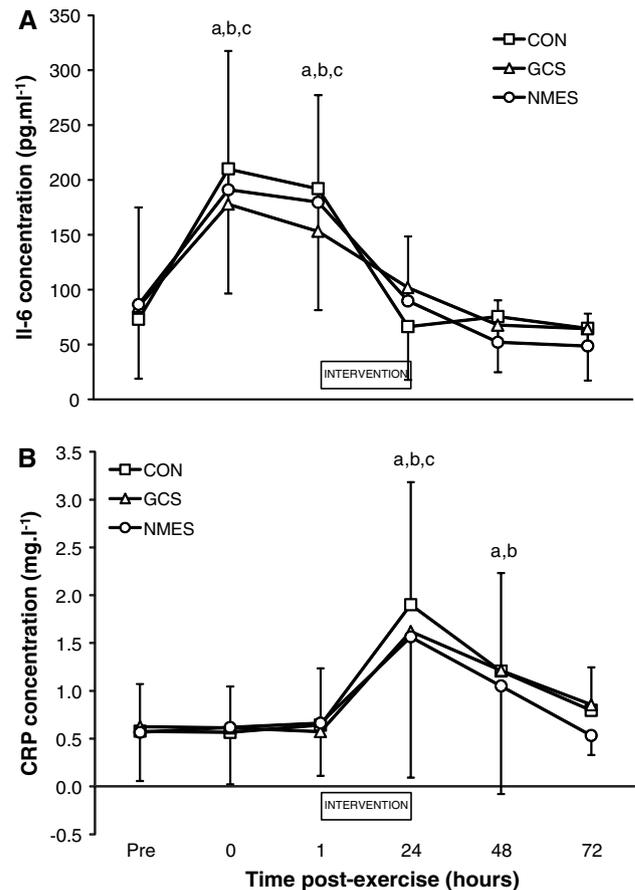
interaction ( $p = 0.57$ ) for plasma IL-6 concentration, which increased compared to baseline in CON, GCS and NMES immediately and 1 h post-exercise (Fig. 5a) before returning to baseline at 24 h in all conditions. There were, however, no differences between treatments at any time point. There was a main effect for time ( $p < 0.001$ ) but no main effect for treatment ( $p = 0.63$ ) and no time  $\times$  treatment interaction ( $p = 0.45$ ) for plasma CRP concentration, which increased compared to baseline in CON, GCS and NMES 24 h post-exercise (Fig. 5b). CRP remained significantly higher than baseline in CON and GCS at 48 h before returning to baseline at 72 h in all conditions. There were, however, no differences between treatments at any time point.

## Discussion

The main finding of this study is that the use of a novel NMES device reduced perceived muscle soreness following intense intermittent exercise. Moreover, NMES was superior to GCS in reducing perceived muscle soreness. NMES provided approximately 30 and 60 % greater reductions in PMS compared to GCS and CON, respectively. Indeed, after just 24 h when NMES was used the level of soreness was that of the control condition at 72 h. However, there were no effects of either intervention on physiological markers of muscle damage (CK, LDH, IL-6 and CRP) and there were no differences in the rate of recovery of muscle strength (MVC).

The intermittent shuttle-running protocol used in the present study resulted in more moderate levels of soreness and associated physiological outcome measures (CK activity, MVC etc.) than those previously observed (Magalhaes et al. 2010), presumably due to the fact that our participants were well accustomed to performing intermittent sports. Similar levels of soreness were observed by Pruscino et al. (2013) who also recruited relatively well-trained hockey players.

There are a number of studies that have investigated the potential to reduce muscle soreness using different types of electrical stimulation devices, with conflicting results (Babault et al. 2011). The precise mechanism underpinning the sensation of soreness is still not fully understood, but most likely involves multifactorial processes, each presiding at different time points throughout the period following exercise. Initial stimulation of nociceptors by products of tissue breakdown (Proske and Morgan 2001) and swelling/oedema (Friden et al. 1988) is thought to occur within the first days of damage. It is probable that NMES has an effect at this early phase through an analgesic effect of stimulation of afferent nerve fibres (DeSantana et al. 2008) as well as a reduction in oedema brought about by the increased



**Fig. 5** Interleukin-6 (a) and C-reactive protein (b) concentration before and following the LIST with three different recovery interventions: passive recovery (CON), graduated compression stockings (GCS) and neuromuscular electronic stimulation (NMES). *a* Significant difference ( $p < 0.05$ ) from pre in CON; *b* significant difference ( $p < 0.05$ ) from pre in GCS; *c* significant difference ( $p < 0.05$ ) from pre in NMES. Values are mean  $\pm$  SD

microvascular blood flow (Tucker et al. 2010). Thereafter, leukocytes typically infiltrate damaged muscle cells over the subsequent 2 days, initiating a complex activation of inflammatory cytokines that co-ordinate phagocytic macrophages in the removal of cellular debris (Tidball 2005). These macrophages also release prostaglandins which stimulate nociceptors (Smith 1991). The sharp rise in IL-6 and CRP levels following exercise is a typical indicator of an inflammatory response, although other work has suggested that circulating IL-6 may not actually reflect an inflammatory response (Malm et al. 2004). The time course of CRP with the expected peak value at approximately 24 h after exercise provides an essential insight into the inflammatory cascade of cytokines and leukocytes associated with recovery from muscle damage (Paulsen et al. 2012). In the current study, there was no significant difference in plasma IL-6 and CRP response following exercise between passive recovery and GCS. Similarly, recent research reveals

that following the same LIST exercise protocol, there was no recovery benefit on the response of inflammatory cytokines tumour necrosis factor- $\alpha$  (TNF- $\alpha$ ), IL-6 or CRP, between whole leg GCS and passive recovery (Pruscino et al. 2013). As such it may be considered that NMES, although increasing limb blood flow did not specifically enhance the removal of the inflammatory associated factors and suggests that the sensation of muscle soreness might be dissociated from the inflammatory response. However, it has to be considered that one of the main drivers of CRP production is IL-6 release which peaks immediately after exercise before declining normally within a few hours (Cox et al. 2007), i.e. before the NMES device was actually used in the present study. It is, therefore, worth considering that perhaps alternative measures of the inflammatory response could be made (e.g. prostaglandins), which are directly responsible for the soreness sensation. Moreover, it is also worth considering whether reducing the inflammatory response is actually a necessity given its role in muscle cell repair and regeneration (Paulsen et al. 2012), where delivery of neutrophils and macrophages (by an enhanced blood flow) may actually be beneficial.

Reductions in muscle function are a well-documented consequence of damaging exercise. As expected there was a small but significant decline in MVC following exercise. Although there was no significant effect of either GCS or NMES on knee extensor torque, knee flexor torque was higher at 48 h compared to CON suggesting that NMES was effective in restoring muscle function. This is despite a large body of evidence demonstrating that NMES is an ineffective method of reducing the decline in torque production. For example, Vanderthommen et al. (2010) compared low-frequency electrical stimulation (LFES) with active and passive recovery strategies following submaximal isometric knee extensions in untrained males, identifying no effect of the recovery mode on peak torque production. Similar findings by Martin et al. (2004) following downhill running suggest that LFES had no recovery effect on voluntary or electrically evoked torque production when compared with active or passive recovery interventions. A further study demonstrated no benefits of 20 min electrical stimulation on MVC or neuromuscular parameters following fatiguing exercise (Bieuzen et al. 2012). Uniquely, the participants in the current study underwent a considerably longer period of NMES (16 h), which might help explain the small but seemingly beneficial effects on knee flexor torque.

As expected there were increases in serum LDH and CK activity following the LIST. Although there was a tendency for CK to be lower in NMES after 24 h this did not reach statistical significance. There are very few studies that have investigated the effect of NMES on CK activity. Vanderthommen et al. (2007) reported lower CK activity 72 h after maximal eccentric knee flexor

contractions with the use of continuous low frequency non-tetanic stimulation of the hamstrings. Beaven et al. (2013), using the same device as in the present study, observed a greater decline in CK activity 36 h after a pre-season rugby match, however NMES was combined with full leg compression garments, so direct comparison to the present study is difficult. Other recovery modalities are equally contradictory in their findings. For instance, Duffield et al. (2010) and Jakeman et al. (2010) did not observe any effect of compression garments on CK activity following intense exercise. Furthermore, a recent meta-analysis of cold water immersion suggests quite variable responses for CK activity (Leeder et al. 2011) and coupled with the fact that CK activity generally has a high level of variability (Clarkson et al. 2005), it has been suggested that the severity of soreness is not affected by the level of muscle damage, and has little correlation with other commonly used indirect markers of muscle damage such as CK and loss of muscle function (Nosaka et al. 2002).

#### Limitations to the study

As with any repeated measures experimental trial, the repeated bout effect (RBE) must be considered. RBE is the protective adaptation in response to a single bout of damaging exercise (Nosaka and Clarkson 1995) which has been shown to remain for several weeks and possibly up to 6 months (Nosaka et al. 2001). In the present study a 4-week period between trials was considered to be sufficient to allow participants to return to a normal baseline status, which is largely supported by all baseline outcome measures not being different. An order effect analysis was also conducted by performing the statistical test on PMS, but instead of using condition, the independent variable was test order. There was no significant effect of order and thus we can be confident that there was no RBE. However, despite our best intentions there was no guarantee that individuals were still 'protected' from previous trials. This was especially the case since our participants were engaged in regular intermittent sport training and competition during the study (several were national and international standard hockey players). However, the ecological validity of using well-trained games players cannot be underestimated. The relatively moderate perceived muscle soreness scores (~5) in the control condition suggests that whilst inducing some degree of soreness, the damage was not necessarily as severe as can be achieved during intense eccentric muscle contractions. Consequently, any potential benefit of the NMES treatment is likely to be harder to detect.

A further limitation, as is always the case with studies investigating such novel interventions, is that it was not possible to blind participants to the interventions. As such

there may be a psychological effect from simply wearing the device. Moreover, the interventions (NMES and GCS) were not controlled as to the duration they were worn. Whilst every attempt was made to do this, because of the randomised nature of the study and the unusual nature of the stimulation intervention, there were occasions where participants were unable/unwilling to maintain a matched duration (e.g. compression socks may have been worn during sleep without any problems, whereas the device was switched off during sleep). Of the 21 participants, 11 did match the intervention duration. In any case, this may also further highlight the enhanced benefit of the NMES device since despite a slightly shorter duration, greater benefits were perceived.

### Practical benefits

The practical benefits of these findings may be that athletes and competitors feel better recovered and more willing and able to perform subsequent training sessions or repeated tournament games. Moreover, the geko™ T-1 device is highly portable due to its size and can be worn for prolonged periods of time with minimal interference during daily activities. Indeed, Taylor et al. (2014) reported that following instruction, athletes were able to easily apply a similar device themselves which could be significant during periods where access to athletes by sports science support staff may be limited. The ease of wear of the device also makes it ideal for use during travel following competition, where athletes can often find themselves in confined spaces for lengthy periods of time and the resultant reductions in blood flow and consequent delay in recovery [together with the increased risk of DVT; Tucker et al. (2010)] can be offset.

### Conclusion

We report positive perceptual benefits of a novel NMES device following intense, sport-specific, intermittent endurance exercise. Despite this, major physiological benefits such as the recovery of the functional (MVC) and biochemical markers (e.g. CK) were not as apparent, which may suggest that the device has an analgesic effect. Furthermore, there were no negative effects on recovery.

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