Neuromuscular fatigue and recovery after heavy resistance, jump, and sprint training

Kevin Thomas¹ Callum George Brownstein¹, Jack Dent¹, Paul Parker¹, Stuart Goodall¹, Glyn Howatson¹,²

¹Faculty of Health and Life Sciences, Northumbria University, Newcastle-upon-Tyne, United Kingdom; ²Water Research Group, School of Environmental Sciences and Development, Northwest University, Potchefstroom, South Africa

Short title:
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Address for correspondence:
Glyn Howatson,
Faculty of Health and Life Science,
Department of Sport, Exercise and Rehabilitation,
Northumbria University,
Newcastle-upon-Tyne,
NE1 8ST,
UK.
Tel: +44 191 227 4863,
Fax: +44 191 227 4713
Email: glyn.howatson@northumbria.ac.uk
Abstract

**Purpose.** Training methods that require maximal intensity efforts against light- and heavy-resistance are commonly used for athletic development. Typically these sessions are separated by at least 48 hours recovery on the assumption that such efforts elicit marked fatigue of the central nervous system (CNS), but this posit has not been well-studied. The aim of the study was to assess the aetiology and recovery of fatigue after heavy-resistance (strength), jump, and sprint training methods.

**Methods.** Ten male athletes completed three training sessions requiring maximal efforts that varied in their loading characteristics; i) heavy resistance exercise (10 × 5 back squats at 80% 1RM) (STR); ii) jumping exercise (10 × 5 jump squats) (JUMP); iii) maximal sprinting (15 × 30 m) (SPR). Pre-, post- and at 24, 48 and 72 h post-participants completed a battery of tests to measure neuromuscular function using electrical stimulation of the femoral nerve, and single- and paired-pulse magnetic stimulation of the motor cortex, with evoked responses recorded from the knee extensors. Fatigue was self-reported at each time point using a visual analogue scale.

**Results.** Each intervention elicited fatigue that resolved by 48 (JUMP) and 72 h (STR & SPR). Decrements in muscle function (reductions in the potentiated quadriceps twitch force) persisted for 48 h after all exercise. Reductions in voluntary activation were present for 24 h after JUMP and SPRINT, and 48 h after STR. No other differences in CNS function were observed as a consequence of training. **Conclusion.** Strength, jump, and sprint training requiring repeated maximum efforts elicits fatigue that requires up to 72 h to fully resolve, but this fatigue is not primarily underpinned by decrements in CNS function.

**Key words.** Neurophysiology; brain; muscle; transcranial magnetic stimulation; central nervous system
Introduction

Athletic development in a range of sports is characterized by the application of various training means and methods in order to target specific adaptations. Resistance training is a key training means employed by coaches and athletes to improve the strength, impulse and speed qualities necessary for success in sports requiring movements underpinned by high force and/or velocity. The methods by which resistance training can be employed in an athlete's training programme can vary depending on the desired adaptive outcome. For example, to target maximum strength, coaches will typically utilize heavier loads (80-95% of 1 repetition maximum (RM)) with consequent slower velocities of movement (1). Conversely, to target the ability to produce high levels of force rapidly, submaximal loads are required in order to accrue impulse quickly (2). To train acceleration and maximum velocity running characteristics, the most effective training means is practice of sprinting itself (3). Each of these training stimuli impose distinct demands on the athlete, but their specific consequences are not well-studied or understood.

Heavy resistance and high velocity training methods typically require athletes to repeatedly produce maximal efforts in order to stimulate adaptation. An inevitable consequence of this is fatigue, a symptom or percept characterised by sensations of tiredness and weakness (4). Fatigue is a complex phenomenon, and while likely underpinned by a range of physiological and psychological mediators, an often-cited posit amongst athletic development professionals is that repeated maximal efforts elicit a high degree of “neuromuscular” or “central” fatigue, requiring prolonged (>48 hours) recovery. Such a postulate has also recently been cited in the academic literature (5), further propagating this idea, despite a lack of peer-reviewed evidence.
Neuromuscular fatigue could feasibly relate to any alteration in the physiological processes governing central nervous system (CNS) or muscle function, but is typically quantified by examining voluntary and artificially-evoked forces during an isometric muscle action. Peripheral neuromuscular fatigue refers to impairments in muscle distal to the neuromuscular junction, quantified as a reduction in the resting involuntary twitch response to nervous tissue stimulation (6). Central neuromuscular fatigue is attributable to the central nervous system inadequately being able to activate muscle to the required level, quantified as a reduction in voluntary activation (6). Adjustments in CNS function can also be quantified via studying the evoked responses to motor cortical stimulation (7). Single- and paired-pulse magnetic stimulation of the motor cortex has been previously applied to understand acute and chronic adjustments in CNS function in response to strength training (8-12) and fatiguing single-limb (13-15) and locomotor exercise (16). In concert, the application of these techniques to study adjustments in neuromuscular function after athletic training could help explain the etiology of fatigue, and aid practitioners in the appropriate scheduling of, and recovery from, different training methods.

While decrements in neuromuscular function, particularly of the CNS, are widely considered when programming training stimuli, the evidence underpinning the idea that heavy strength and power sessions require >48 h recovery is incomplete. Previous studies recently demonstrated that heavy resistance exercise elicited greater acute reductions in voluntary force than a similar low-resistance, high-velocity “power” session (17), and that these heavy resistance exercise induced decrements persisted at 24 h post-exercise in elite athletes (18). Bartolomei et al. (19) recently demonstrated greater and more prolonged strength and jump performance impairments after
“hypertrophy” style training (higher volume, lower load, shorter rest periods) compared to a training stimulus targeting strength development (lower volume, higher intensity, longer rest periods). Collectively these findings suggest the acute and prolonged adjustments underpinning the fatigue experienced after resistance exercise varies between training methods, but these studies were limited by both the range of outcome measures studied, and/or a limited profile of the time-course recovery of neuromuscular function. Further study is warranted to comprehensively assess the acute and prolonged neuromuscular adjustments induced by the typical training means and methods commonly employed in the physical preparation of athletes. Such information will be of high value to practitioners when prescribing training stimuli.

The aim of the study was to assess the etiology and recovery of neuromuscular fatigue in response to heavy resistance, jumping, and sprinting exercise. It was hypothesised that the maximal nature of all exercise interventions would induce marked neuromuscular fatigue that would require >48 hours to resolve, and that the time-course of recovery would be similar between interventions.

Methods

Participants
Ten male participants (age 21 ± 2 years, stature, 1.82 ± 0.05 m, mass, 85 ± 12 kg) gave their written, informed consent to participate in the study, which was approved by the Northumbria University Faculty of Health & Life Sciences Ethics Committee. All participants had >3 years history of training experience utilising resistance and maximal speed methods, and were currently competing in intermittent (n = 6), or track and field (n = 4) sports at University or national standard.
Design

Participants initially visited the laboratory on two separate occasions for preliminary assessments and to habituate to the measurement tools of the study. Subsequent to this participants completed three experimental trials, each spanning four consecutive days and separated by one week, in a randomised, counterbalanced order. On the first day of each experimental trial, participants completed one of three interventions as follows: i) a heavy resistance exercise session consisting of repeated sets of back squats (STR); ii) a low-load, high-velocity exercise session consisting of repeated sets of jump squats (JUMP); iii) a maximal speed training session consisting of repeated 30 m sprints (SPR). Pre-, immediately post-, and at 24, 48 and 72 h post- a battery of assessments to measure fatigue and neuromuscular function were administered. Prior to all visits participants were instructed to refrain from caffeine (24 hours), alcohol (48 hours), and to arrive 2 h post-prandial in a fully rested, hydrated state. Participants were also instructed not to perform any exercise other than that required by the study for the duration of their participation. To account for any potential detraining-induced changes in physical fitness, a “refresh” session consisting of maintenance loads for the physical qualities under study was employed between experimental trials. An overview of the experimental trials can be viewed in Supplemental Digital Content 1.

Procedures

Practice trial

Prior to the experimental trials, participants visited the laboratory on two occasions for habituation to the measurement tools of the study (on both visits), and an assessment of 1 repetition maximum (1RM) back squat strength or jump squat
performance (on separate visits). Prior to all exercise (practice & experimental trials) participants completed a structured ten-minute warm-up, which incorporated jogging, dynamic flexibility movements, mobility exercises specific to squatting, jumping, and sprinting, and 3 × 30 m progressive strides at 70, 80 and 90% of perceived maximum sprint speed. For the assessment of maximum isoinertial strength, participants first completed warm-up sets of 3-5 repetitions of back squats (high bar position), beginning with an unloaded barbell and progressing to 50%, 70%, 80% and 90% of their estimated 1RM. The load on the bar was then incremented by 2-5% until participants could not complete 1 repetition. The technical execution of each lift required participants to descend under control (2 s tempo) to a depth where the femur was parallel to the floor. Participants then immediately reversed the movement and were instructed to maximally accelerate the bar during the concentric phase. A repetition was deemed unsuccessful if participants could not complete the concentric phase in ≤ 2 s. Maximum isoinertial strength was 126 ± 14 kg, or 150 ± 15% body mass. For jump squats, participants completed vertical jumps for maximum height, beginning with body mass (plus a wooden dowel) and incrementing by 5 kg; the first increment was achieved by replacing the dowel with a lightweight training barbell with a mass of 5 kg. Each repetition required participants to squat to a self-selected depth (approximating a half squat) and jump for maximum height. Jump height was recorded using photoelectronic timing gates (Optojump Next, Microgate, Milan, Italy) for 2 to 3 efforts at each load. When participants were unable to maintain performance within 5% of their unloaded jump height because of added resistance, the test was terminated and the highest applied load where squat jump height was maintained was used for experimental trials (mean, SD 10 ± 5 kg, with a range of 0 to 20 kg, additional load).
Experimental trials: exercise intervention

On the first day of each experimental trial, subsequent to pre-test assessment, participants completed one of three exercise prescriptions; i) heavy resistance training consisting of 10 × 5 repetitions of the high bar back squat at 80% 1RM, with 3 min recovery (STR); ii) 10 × 5 repetitions of jump squats, with 3 min recovery (JUMP); iii) 15 × 30 m maximum sprints, with 2 min recovery (SPR). For STR and JUMP participants were encouraged to maximally accelerate the load, and the velocity of each repetition was monitored using a wearable linear position transducer (PushBand, Heap Analytics, Toronto, Canada). For SPR participants began each sprint 0.5 m behind the first timing gate, and were encouraged to sprint maximally through the timing gate at 30 m. Each sprint was measured using photocell technology (TC Timing system, Brower Timing Systems, Draper, Utah, USA). For all trials participants were provided feedback on the execution of each repetition to promote a maximum effort. Post-training, participants were asked for a whole trial session rating of perceived exertion (RPE) using the 0-10 category ratio scale. While it was impossible to equate training load between the experimental trials, the configurations for STR, JUMP and SPR were designed in consultation with experienced strength and conditioning coaches to represent a “heavy” stimulus for the physical quality under stress, and were similar in duration (approximately 45 min, including the standardised warm-up).

Experimental trials: outcome measures
On each occasion participants completed a battery of assessments to measure fatigue and neuromuscular function. All outcome measures were assessed pre-, post-, and at 24, 48 and 72 h post-exercise, unless otherwise stated.

Visual analogue scales & creatine kinase

Upon arrival, and post-exercise after assessment of neuromuscular function, participants completed visual analogue scales (VAS, 100 mm scale) to record fatigue and perceptions of muscle soreness. For fatigue the VAS was anchored with the verbal descriptors “not fatigued at all” to “extremely fatigued”; participants were asked to rate their general feeling of “fatigue, tiredness, weakness and lethargy”. For muscle soreness the VAS was anchored with “no soreness” to “extremely sore”; participants preceded their rating with three repetitions of a body weight squat and were asked to rate their “muscle soreness and pain”. Subsequent to this fingertip samples of capillary blood were obtained and immediately assayed for creatine kinase (CK) concentration (Reflotron, Roche Diagnostics, Germany).

Assessment of neuromuscular function

The evoked force and electromyographic (EMG) responses of the rectus femoris (RF) to transcranial magnetic stimulation (TMS) of the primary motor cortex, and electrical stimulation of the femoral nerve, were used to assess neuromuscular fatigue, corticospinal excitability, and the status of inhibitory intracortical networks. The assessment of neuromuscular function took place subsequent to perceptual assessments and capillary blood sampling at all time points except for post-exercise, where it was conducted first in order to capture the extent of neuromuscular fatigue elicited by the exercise intervention.
A calibrated load cell (MuscleLab force sensor 300, Ergotest technology, Norway) recorded muscle force (N) during an isometric maximal voluntary contraction (iMVC) of the knee extensors. During contractions, participants sat with hips and knees at 90° flexion, with a load cell fixed to a custom-built chair and attached to the participants right leg, superior to the ankle malleoli, with a noncompliant cuff. Electrical activity from the RF and biceps femoris (BF) were recorded from surface electrodes (Ag/AgCl; Kendall H87PG/F, Covidien, Mansfield, MA, USA) placed 2 cm apart over the belly of each muscle, with a reference electrode placed on the patella. Electrode placement was marked with indelible ink to ensure consistent placement throughout the study, with the areas cleaned and shaved prior to electrode placement. The electrodes recorded the root-mean-square (RMS) amplitude for submaximal and maximal voluntary contractions, the compound muscle action potential (M-wave) from the electrical stimulation of the femoral nerve, and the motor evoked potential (MEP) elicited by TMS. Signals were amplified: gain ×1000 for EMG and ×300 for force (CED 1902; Cambridge Electronic Design, Cambridge, UK), bandpass filtered (EMG only: 20-2000 Hz), digitized (4 kHz; CED 1401, Cambridge Electronic Design) and analysed offline. Further details on these methods are provided below.

Motor nerve stimulation

Motor nerve stimulation was used for the measurement of contractile function, muscle membrane excitability and voluntary activation (VA). Single electrical stimuli were administered using square wave pulses (200 µs) via a constant-current stimulator (DS7AH, Digitimer Ltd., Hertfordshire, UK) using self-adhesive surface electrodes
(Nidd Valley Medical Ltd., North Yorkshire, UK). Electrical stimuli were first administered to the motor nerve at rest in 20 mA step-wise increments from 20 mA until the maximum quadriceps twitch amplitude ($Q_{tw}$, N) and muscle compound action potential ($M_{max}$, mV) were elicited. To ensure a consistent, supramaximal stimulus and account for any activity-induced changes in axonal excitability, the resulting stimulation intensity was increased by 30% for all subsequent stimulus. The peak-to-peak amplitude and area of the electrically evoked maximal compound action potential ($M_{max}$) was used as a measure of membrane excitability. Participants subsequently completed six iMVCs (3-5 s duration) of the knee extensors, separated by 60 s rest. For the final three iMVCs, electrical stimuli were delivered during and 2 s post contraction to assess VA and potentiated quadriceps twitch force ($Q_{tw, pot}$) respectively.

Motor cortical stimulation

Single- and paired-pulse TMS of 1 ms duration were delivered using a concave double cone coil using two linked monopulse magnetic stimulators (Magstim 200, The Magstim Company Ltd, Whitland, UK). The junction of the double cone coil was aligned tangentially to the sagittal plane, with its centre 1-2 cm to the left of the vertex. The optimal coil placement was determined at the start of each trial as the position that elicited the largest MEP in the RF, with a concomitant small MEP in the BF. The position was marked with indelible ink for consistent placement during subsequent trials. The stimulator intensity was based on active motor threshold (AMT) measured during a 10% iMVC. In order to determine AMT, the stimulator intensity was increased in 5% steps beginning at 35% of stimulator output until a consistent MEP with peak-to-peak amplitudes of $>200 \mu$V was found. Thereafter,
stimulus intensity was reduced in 1% step until an MEP of >200 µV was found in 50% of stimulations.

Corticospinal excitability & Short-interval intracortical inhibition (SICI)

Once AMT was established, the stimulator intensities required to assess the MEP response to varying TMS intensities (stimulus-response curve) were determined in order to assess corticospinal excitability. Participants held a submaximal voluntary contraction (10% iMVC) with one set of five stimuli delivered at each of 90%, 100%, 110%, 120%, 130%, 140%, 150% and 160% of AMT in a randomized and counterbalanced order, with 4-6 s between each stimulus and 15 s between each set. For SICI, ten single and ten paired-pulse TMS stimuli were administered in two sets of 10 stimuli during a 10% iMVC, for measurement of unconditioned and conditioned MEP amplitude respectively. Paired-pulse TMS consisted of a subthreshold conditioning pulse at 70% of AMT, and a suprathreshold test pulse at 120% AMT, with an inter-stimulus interval (ISI) of 2 ms. Single- and paired-pulses (× 10 each) were delivered in a pre-determined randomised order, with 4-6 s between each stimulation and a short rest between each set. This assessment was conducted pre-exercise, and at 24 hour intervals thereafter until 72 h post.

Voluntary activation with TMS

Single pulse TMS was delivered during brief (3-5 s) contractions at 100%, 75% and 50% iMVC, separated by 5 s of rest, for determination of voluntary activation with TMS (VATMS). This procedure was repeated 3 times with 15 s rest between each set. The stimulation intensity was set at the stimulator output that elicited the maximum superimposed twitch force (SIT) during a 50% iMVC. The SIT force elicited from
contractions at 100%, 75%, and 50% were used to determine $V_{ATMS}$ (see data analysis section for details).

Experimental trials: “refresh session”

On the final day of each experimental trial, after all outcome measures had been completed, a “refresh” session designed to maintain the physical qualities under study over the course of the experimental period was employed. This consisted of a low-volume, high-intensity stimulus for each physical quality in a single session (3 × 5 sets of back squats at 80% 1RM, 3 × 5 maximal effort jump squats, 3 × 30 m maximal effort sprints). Previous research has demonstrated that strength qualities can be adequately maintained for prolonged periods using low doses provided the intensity of exercise remains close to maximal (20, 21).

Data analysis

Voluntary activation assessed through the interpolated twitch technique (22) was quantified by comparing the amplitude of the superimposed twitch force to the potentiated twitch (100 Hz) delivered 2 s following the iMVC at rest using the following equation: Motor point VA (%) = \[1 - \left(\frac{SIT}{Q_{tw, pot}}\right) \times 100\]. Voluntary activation using TMS ($V_{ATMS}$) was assessed during contractions at 50%, 75% and 100% iMVC using linear regression of the superimposed twitch force evoked by TMS (23), with the regression analysis confirming a linear relationship at each time-point ($r^2$ range = 0.89 ± 0.03 to 0.95 ± 0.04). The estimated resting twitch (ERT) was calculated as the y-intercept of the linear regression between the mean amplitude of the SIT force evoked by TMS at each contraction intensity. Subsequently, $V_{ATMS}$ was quantified using the equation \[1 - \left(\frac{SIT}{ERT}\right) \times 100\]. To quantify SICI, the ratio of
the average conditioned paired-pulse MEP was expressed relative to the average unconditioned MEP at 120% AMT. Recruitment curves were constructed by plotting the TMS stimulation intensity relative to AMT against the MEP amplitude averaged from the five stimulations at each intensity, expressed relative to $M_{\text{max}}$. The ratio of the MEP amplitude to the maximum M-wave was used as an index of corticospinal excitability. In order to provide a summary measure of corticospinal excitability, the summated area under the stimulus-response curve was calculated for each participant at each time point using the trapezoid integration method (24). The root mean square EMG amplitude ($\text{RMS}_{\text{EMG}}$) and average force was calculated in the 80 ms prior to each TMS to ensure a similar level of background muscle activity was present during the stimulus-response curve and SICI measurements. The peak-to-peak amplitude of evoked MEP and $M_{\text{max}}$ were measured offline.

**Statistical analysis**

Data are presented as mean ± SD. To ascertain the time-course recovery of neuromuscular fatigue within-trial, one-way repeated measures ANOVA across time were employed for STR, JUMP and SPR data. Significant main effects were followed up with Dunnett’s multiple comparison procedure, with the pre-exercise score used as the control category. To assess between-trial differences in the magnitude of neuromuscular fatigue induced by STR, JUMP and SPR, two-way (trial × time) factorial repeated measures ANOVA analysis was employed. As baseline scores did not differ between trials for any outcome measure, significant trial × time interaction effects were followed up with one-way repeated measures ANOVA, and post-hoc Tukey-adjusted pairwise comparisons at each time point to locate statistically significant between-trial differences. The assumptions underpinning these statistical
procedures were verified as per the guidelines outlined by Newell et al. (25). Data were analysed using GraphPad Prism (version 7, GraphPad Software Inc., La Jolla, CA). Statistical significance was accepted at $P < 0.05$.

Results

**Exercise responses.** All participants successfully completed the prescribed training interventions. For STR, the load lifted was $101 \pm 11$ kg. Repetition velocity decreased from $0.53 \text{ m}\cdot\text{s}^{-1}$ in set 1, to $0.44 \text{ m}\cdot\text{s}^{-1}$ in set 10 ($P < 0.05$), with a best of $0.54 \pm 0.07 \text{ m}\cdot\text{s}^{-1}$ and worst of $0.41 \pm 0.07 \text{ m}\cdot\text{s}^{-1}$ independent of set. Session RPE averaged $8 \pm 2$ for STR. For JUMP, mean repetition velocity was successfully maintained throughout the exercise ($1.61 \pm 0.17 \text{ m}\cdot\text{s}^{-1}$ in set 1 vs. $1.56 \pm 0.14 \text{ m}\cdot\text{s}^{-1}$ in set ten, $P = 0.31$, best score of $1.69 \pm 0.11 \text{ m}\cdot\text{s}^{-1}$, worst of $1.48 \pm 0.10 \text{ m}\cdot\text{s}^{-1}$) and session RPE was lower ($5 \pm 1$) than STR ($P = 0.001$). For SPR, 40 m sprint time declined from $4.40 \pm 0.14$ s in set 1 to $4.55 \pm 0.22$ s in set fifteen ($P = 0.04$), with a fastest sprint of $4.36 \pm 0.16$ s and a slowest of $4.61 \pm 0.24$ s. Session RPE after SPR ($6 \pm 2$) was not different to STR ($P = 0.18$) or JUMP ($P = 0.33$).

**Perceived fatigue & muscle damage responses.** All exercise interventions elicited significant perceived fatigue (Table 1) that persisted for 48 h after STR (48 h, $P = 0.002$) and SPR training (48 h, $P = 0.008$), and 24 h after JUMP training (24 h, $P = 0.02$). Between trials, both STR and SPR training resulted in greater perceived fatigue than JUMP training for up to 48 h (Figure 1, panel A). Similar patterns were also evident for perceptions of muscle soreness; all training resulted in increases in muscle soreness that were different to baseline for 48 h, and between trials - both STR (for up
to 72 h, \( P = 0.0006 \) and SPR (for up to 48 h, \( P = 0.0008 \)) elicited a greater magnitude of soreness in comparison to JUMP (Figure 1, panel B). Creatine kinase peaked at 24 h in all trials and was different to baseline for 24, 48 and 72 h for STR, JUMP and SPR respectively (Table 1). Between trials, CK was lower at 24 h in JUMP compared to both STR \( (P = 0.001) \) and SPR \( (P = 0.002) \) (Figure 1, panel C).

**Neuromuscular fatigue.** All exercise interventions resulted in declines in iMVC force that took until 72 h to fully resolve in all trials (Table 2). The magnitude of the reduction in iMVC force immediately post-exercise was higher after STR compared to JUMP \( (P < 0.001) \) and SPR \( (P < 0.001) \), a difference that persisted at 24 hours \( (P = 0.02 \) and 0.05 respectively, Figure 2, panel A). Reductions in VA were also evident immediately post-exercise for all trials, and persisted for 48 h after STR \( (P = 0.004) \), and 24 h after JUMP \( (P = 0.015) \) and SPR \( (P = 0.023, \text{Table 2}) \). Significant reductions in \( V_{\text{A}} \) were also evident post-exercise in all trials \( (all \ P < 0.05) \), but returned to baseline quicker than VA; by 48 h in STR and 24 h in JUMP and SPR (Table 2). The magnitude of reductions in VA, measured with both motor nerve and motor cortical stimulation, was not different between exercise interventions (Figure 2, panel B & C). All trials resulted in reductions in \( Q_{\text{lw,pot}} \), that took 72 h to fully resolve (Table 2). Between trials there were larger reductions in \( Q_{\text{lw,pot}} \) immediately-post STR compared to both JUMP and SPR \( (both \ P < 0.001) \), with no differences between trials thereafter (Figure 2, panel D).

**Corticospinal excitability and SICI.** Exercise resulted in no modulation of corticospinal excitability (Figure 3, stimulus-response curves) or SICI (Figure 4), both within and between trials \( (all \ P > 0.05) \). The EMG\(_{\text{RMS}} \) was also not different within
and between trials (supplementary material, Table 3). For a full list of surface EMG responses to TMS and electrical stimulation please see supplementary material, Table 3.

**Discussion**

The aim of the study was to assess the effect of strength, jump and sprint training, performed with maximal intent, on the etiology and time-course of neuromuscular fatigue and recovery. In accordance with our hypothesis, all training stimuli resulted in neuromuscular adjustments that took up to 72 h to fully resolve. For twitch force, indicative of peripheral fatigue, strength training resulted in larger post-exercise reductions compared to jump and sprint training, but the time-course recovery was similar thereafter, with marked decrements still evident at 48 h post-exercise in all trials. Reductions in voluntary activation, an indicator of central fatigue, persisted for 24 h after jump and sprint training, and 48 h after strength training, with no difference between trials in the magnitude of these reductions. Measures of CNS responsiveness and inhibition were not modulated in response to the training stimuli at any time point. Perceptual indicators of fatigue and soreness followed a similar time-course of recovery to measures of neuromuscular function, requiring up to 72 h to return to baseline, with a tendency for jump training to be less fatiguing compared to strength and sprint training. Collectively these data indicate that maximal intent, relatively high volume, strength, jump and sprint training methods elicit neuromuscular fatigue, mediated by both central and peripheral mechanisms, that requires up to 72 h to fully resolve.
Time-course of recovery of neuromuscular fatigue after training. An often-cited posit in strength and conditioning is the idea that training methods performed with maximal intent, such as those studied here, result in central fatigue, or are CNS intensive, and require 48-72 h recovery before similarly intense stimuli are imposed (26, 5, 27). To date however, the formal study of neuromuscular fatigue in the days post-training has been limited (19, 17, 18, 28, 29). Here we show that strength, jump and sprint training elicits marked neuromuscular central and peripheral fatigue, that can require up to 72 h to fully resolve, which provides some support to these previous assertions. The capacity to produce voluntary force was impaired for 48 h after all training, with decrements in MVC force of 8%, 7% and 6% on average for strength, jump and sprint training. Similarly, twitch force was reduced compared to baseline for 48 h in all trials, indicating a prolonged decrement in muscle function, with values remaining depressed by 5-6% on average at 48 h. Reductions in voluntary activation persisted for 48 h after strength training, and 24 h after jump and sprint training, suggesting heavy resistance training elicited more prolonged central fatigue than the other methods studied. At the 48 h time point the decrement in voluntary activation averaged 5%, 2% and 3% for strength, jump and sprint training respectively. Collectively, these data suggest that neuromuscular fatigue after training methods that emphasise maximal intent is persistent, and multi-factorial. This underscores the need for appropriate recovery between such sessions, alongside interventions that address the multi-factorial nature of fatigue. The data also provide some support to the assertion that training sessions that emphasise maximal intent should be separated by at least 48 h if peak performance is a priority, as the majority of variables under study took 72 h to fully resolve.
“Central” fatigue after training. Fatigue of the CNS is often implicated as a primary consideration after training modes that emphasise maximal intent, and recent reviews have called for an increased emphasis on the recovery of central and “brain” fatigue after exercise (30, 31). However, the formal study, and precise definition, of what constitutes central fatigue is limited. Here we specifically measured central fatigue as a reduction in the ability of the CNS to activate skeletal muscle. This activation deficit was evident post-training for up to 24 h after jump and sprint training, and up to 48 h after heavy resistance training. We also measured variables purported to reflect CNS excitability and inhibition, but these did not modulate with training. In contrast, the capacity to produce voluntary force was impaired for 48 h in all trials, decrements in muscle function (indicative of peripheral fatigue) persisted for 48 h in all trials, and sensory perceptions of fatigue and soreness persisted for 48-72 h post. The magnitude of central fatigue was also modest, with voluntary activation returning to within 5% of baseline in the majority of cases (n = 6, 8 & 6 respectively for strength, jump and sprint training) by 24 h post. Additionally, the magnitude of the decrement post-trial was similar to that previously observed in our lab for prolonged cycling exercise (32, 33), repeated-sprint exercise (34) and simulated intermittent-sprint exercise (35). The recovery of central neuromuscular fatigue in the days post-was also similar to that observed after simulated intermittent-sprint exercise (35). Therefore, the idea that recovery of the CNS should be prioritised after methods of training that emphasise maximal intent is debatable, but perhaps simply reflects an imprecise definition of terms. Fatigue is a symptom, or percept, characterised by sensations of tiredness and weakness (4), underpinned by a myriad of physiological and psychological mechanisms; what is commonly perceived as central fatigue by athletes and coaches is likely more accurately interpreted as fatigue *per se*. That is,
the feelings of tiredness and weakness that athletes experience in the days post-exercise are likely underpinned by a range of mechanisms relating to both central and peripheral function, and not primarily attributable to “CNS” fatigue. A caveat to this conclusion is the acknowledgement that our ability to measure aspects of CNS function, and thus infer the impact of exercise, is limited by the available measurement tools. For example, even the most widely acknowledged measure of central fatigue - a reduction in voluntary activation of skeletal muscle – has questionable validity (36). This notwithstanding, our data suggest that the fatigue experienced after the training methods under study is multi-factorial and not primarily underpinned by central mechanisms.

Differential effect of strength, jump and sprint training. A number of differences were observed between trials that indicated the jumping training stimulus elicited less fatigue, and took less time to recover from. These included differential effects on iMVC and twitch force, the creatine kinase response, and perceptions of fatigue and muscle soreness, in comparison to heavy resistance exercise and sprinting. However, whether these differences could be primarily attributed to differences in the force-velocity requirements of the differing sessions is debatable. Both the heavy resistance (back squat to parallel depth) and sprinting stimuli required greater displacement of load (external or body mass) in comparison to power training (jumping from a half squat). The ostensibly increased work required during STR and SPR (and associated metabolic demand), and the increased potential for muscle damage at longer muscle lengths, could explain the differences observed between trials independent of differences in the force-velocity demands of the exercise. Equating the training stimulus between trials is an impossible endeavour, and therefore any between-trial
comparisons should be interpreted with caution. However, the relatively lower stress and quicker recovery observed after jumping compared to heavy resistance training is not without precedent. Howatson et al. (18) previously observed strength training (consisting of 4 × 5 heavy back squat, split squat and push press) elicited reductions in iMVC for up to 24 h, whereas the same session conducted with lower loads and higher repetition velocities elicited no reduction in iMVC. Additionally, Linnamo et al. (29) previously demonstrated a higher degree of acute neuromuscular fatigue following heavy load vs. light load “explosive” bilateral leg extension resistance training. These previous data, and the current study, indicate that training methods that emphasise the ability to generate impulse to accelerate relatively light loads might require less recovery time than heavy resistance or maximal sprint training, a finding that has implications for the scheduling of such activities.

Corticospinal excitability and short intracortical inhibition. There were no discernible adjustments in corticospinal excitability nor short intracortical inhibition at any time point in response to all exercise interventions. Corticospinal excitability has been shown to modulate acutely with single limb fatiguing exercise (13-15) and ballistic isometric exercise (9), and chronically after single limb (8, 12) and whole body (10) resistance training programmes. Short intracortical inhibition has similarly been demonstrated to be modulated after a period of resistance training (10), and acutely during locomotor exercise (16) and after fatiguing isometric knee extensor exercise (37). Of importance, these acute adjustments seem to quickly resolve upon exercise cessation (37, 16); this could explain why, in the present study, we did not observe any differences post-exercise as the measurement of these variables was delayed in comparison to previous work. The finding that neither corticospinal
excitability nor short intracortical inhibition were modulated with recovery in the days post-exercise concurs with previous studies from our laboratory studying the etiology and recovery of neuromuscular fatigue after simulated and competitive intermittent-sprint exercise (38, 35). Thus, while measures of CNS excitability and inhibition might be modulated during and immediately post-exercise, or chronically in response to longer-term training, they do not systematically differ from baseline in the days post-fatiguing exercise.

In addition to an inability to match training stimuli between trials, the ecological validity of both the imposed sessions, and the measurement protocols, could also be questioned. Considering the primary variables under study (i.e. indicators of neuromuscular fatigue), we deliberately chose to study a high volume of exercise for each training stimulus, and limited each to a single exercise that required a significant contribution from the quadriceps muscle group, and where possible were biomechanically similar (e.g. back squats vs. jump squats). For these reasons, the applicability of the results to regular athletic development training, which typically involves lower volumes and higher variation of exercises within sessions, is questionable. There are of course unlimited configurations of exercise selection, sets, repetitions and recovery durations that could be manipulated, and consequently any decisions on the exercise intervention employed in a study of this nature could be questioned. Additionally, adjustments in neuromuscular function as a consequence of exercise were studied during single-limb, isometric knee extensor muscle actions. This assessment set-up is required to measure neuromuscular fatigue, however these adjustments might not fully reflect decrements in the type of dynamic knee extensor function required of the exercise modes under study, and athletic performance more
generally. These limitations notwithstanding, the data do provide new information on
the nature of fatigue and recovery after resistance and speed training; an area of
research that is under-studied, and in need of further investigation.

In conclusion, this study has demonstrated that training methods requiring repeated
maximal intensity efforts elicit marked neuromuscular fatigue that requires up to 72 h
to fully resolve. The observed neuromuscular fatigue was of both a central and
peripheral origin, with a faster recovery of central, compared to peripheral,
neuromuscular fatigue. The data provide partial support for the idea that training
methods that emphasise maximal intent to express force or velocity should be
separated by at least 48 h, but the recovery of central nervous system function is not
necessarily the primary aim of this period. Rather, the residual fatigue experienced by
athletes after such training is multi-factorial, and thus development of appropriate
monitoring and rest/recovery strategies that reflect this is warranted. Further research
is required to further probe the consequences of maximal intensity training using
novel measurement tools, and stimuli that more accurately reflect the day-to-day
practice of different athletic groups.

Acknowledgements

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Association research grants programme. The authors would like to thank Mr Joe
Kupusarevic, Mr Sam Orange, Mr Alan Toward, and Mr Sam White for their
assistance with data collection.

Conflict of Interest
The authors have no conflict of interest to declare. The results of the study do not constitute endorsement by ACSM. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.


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Tables & Figures

**Table 1.** Within-trial differences in fatigue and perceptions of muscle soreness measured using visual analogue scales (100 mm scale), and creatine kinase (CK), measured pre- and in the 72 h post-strength, jump, and sprint training. Values are mean ± SD. * = significant difference within-trial from pre-test score.

**Table 2.** Within-trial differences in isometric maximum voluntary contraction strength and measures of neuromuscular fatigue pre-, post, and 24, 48, and 72 hours post-strength, jump and sprint training. Values are mean ± SD. * = significant difference from pre-test score within trial.

**Figure 1.** Between-trial differences in fatigue (A), muscle soreness (B) and creatine kinase (C) measured pre-, post- and 24, 48 and 72 hours post-strength, jump, and sprint training. Between trial differences indicated by * = difference between strength and jump; # = difference between jump and sprint; ^ = difference between strength and sprint (all $P > 0.05$). Individual responses are plotted, with lines representing the mean score.

**Figure 2.** Between-trial differences in isometric maximum voluntary contraction force (A), voluntary activation measured with motor nerve (B) and motor cortical (C) stimulation, and quadriceps potentiated twitch force (D) Between trial differences indicated by * = difference between strength and jump; # = difference between jump and sprint; ^ = difference between strength and sprint (all $P > 0.05$). Individual responses are plotted, with lines representing the mean score.
Figure 3. Motor evoked potential (expressed relative to Maximum M-wave) stimulus-response curves measured above and below active motor threshold (AMT, 100%) pre-, and 24, 48 and 72 hours post-strength (A), jump (B) and sprint (C) training. Values are mean ± SD. A reference line is included at 60% to assist comparison between trials.

Figure 4. Short intracortical inhibition (SICI) expressed as the ratio between conditioned and unconditioned motor evoked potentials pre-, and 24, 48 and 72 hours post-strength, jump and sprint training. Individual responses are plotted, with lines representing the mean score.

Supplemental digital content

Supplemental digital content 1.pdf. Schematic of experimental protocol. Pre-exercise and at 24, 48 and 72 h post participants completed the battery of assessments in the same order. After the pre-exercise assessment participants completed one of three exercise interventions: i) heavy resistance training consisting of 10 × 5 repetitions of the high bar back squat at 80% 1RM, with 3 min recovery (STR); ii) 10 × 5 repetitions of a jump squat, with 3 min recovery (JUMP); iii) 15 × 30 m maximum sprints, with 2 min recovery (SPR). Participants were encouraged to complete every repetition with maximal intensity. Immediately post-exercise, central and peripheral neuromuscular fatigue were evaluated within 2 min of exercise cessation. Pre-exercise and at 24 h intervals thereafter, single-pulse transcranial magnetic stimulation (TMS) were administered during a submaximal isometric contraction at various percentages (90 to 160%) of active motor threshold (AMT) for
the assessment of corticospinal excitability. Paired-pulse TMS were administered during submaximal contraction for assessment of short intracortical inhibition.
Figure 1.
Figure 2
Figure 3

A, B, C show changes in MEP/Mmax (%) with increasing stimulation intensity (%AMT) over time. Legends indicate 'Pre', '24', '48', and '72'.
Figure 4
Table 1. Within-trial differences in fatigue and perceptions of muscle soreness measured using visual analogue scales (100 mm scale), and creatine kinase (CK), measured pre- and in the 72 h post-strength, jump, and sprint training. Values are mean ± SD. * = significant difference within-trial from pre-test score.

<table>
<thead>
<tr>
<th></th>
<th>Strength</th>
<th>Jump</th>
<th>Sprint</th>
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<tbody>
<tr>
<td><strong>Fatigue (mm)</strong></td>
<td></td>
<td></td>
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<tr>
<td>Pre-</td>
<td>16 ± 13</td>
<td>14 ± 11</td>
<td>16 ± 6</td>
</tr>
<tr>
<td>Post-</td>
<td>63 ± 16*</td>
<td>44 ± 15*</td>
<td>56 ± 11*</td>
</tr>
<tr>
<td>24 h</td>
<td>52 ± 19*</td>
<td>28 ± 15*</td>
<td>51 ± 21*</td>
</tr>
<tr>
<td>48 h</td>
<td>56 ± 19*</td>
<td>30 ± 16</td>
<td>40 ± 16*</td>
</tr>
<tr>
<td>72 h</td>
<td>26 ± 16</td>
<td>22 ± 17</td>
<td>27 ± 13</td>
</tr>
<tr>
<td><strong>Muscle soreness (mm)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-</td>
<td>16 ± 13</td>
<td>15 ± 13</td>
<td>18 ± 9</td>
</tr>
<tr>
<td>Post-</td>
<td>47 ± 22*</td>
<td>34 ± 10*</td>
<td>39 ± 17*</td>
</tr>
<tr>
<td>24 h</td>
<td>61 ± 22*</td>
<td>25 ± 11*</td>
<td>68 ± 17*</td>
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<td>48 h</td>
<td>63 ± 23*</td>
<td>33 ± 21*</td>
<td>52 ± 21*</td>
</tr>
<tr>
<td>72 h</td>
<td>40 ± 29</td>
<td>20 ± 21</td>
<td>31 ± 18</td>
</tr>
<tr>
<td><strong>CK (IU·L⁻¹)</strong></td>
<td></td>
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<tr>
<td>Pre-</td>
<td>185 ± 98</td>
<td>253 ± 114</td>
<td>265 ± 142</td>
</tr>
<tr>
<td>24 h</td>
<td>863 ± 659*</td>
<td>569 ± 340*</td>
<td>946 ± 531*</td>
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<tr>
<td>48 h</td>
<td>733 ± 673</td>
<td>547 ± 328*</td>
<td>622 ± 357*</td>
</tr>
<tr>
<td>72 h</td>
<td>440 ± 333</td>
<td>356 ± 205</td>
<td>484 ± 270*</td>
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</table>
**Table 2.** Within-trial differences in isometric maximum voluntary contraction strength and measures of neuromuscular fatigue pre-, post, and 24, 48, and 72 hours post-strength, jump and sprint training. Values are mean ± SD. * = significant difference from pre-test score within trial.

<table>
<thead>
<tr>
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<th>Strength</th>
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<th>Sprint</th>
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<tbody>
<tr>
<td></td>
<td>(N)</td>
<td></td>
<td></td>
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<tr>
<td><strong>iMVC</strong></td>
<td></td>
<td></td>
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<tr>
<td>Pre-</td>
<td>691 ± 78</td>
<td>693 ± 78</td>
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<tr>
<td>Post-</td>
<td>548 ± 61*</td>
<td>611 ± 52*</td>
<td>614 ± 66*</td>
</tr>
<tr>
<td>24</td>
<td>600 ± 78*</td>
<td>630 ± 63*</td>
<td>627 ± 72*</td>
</tr>
<tr>
<td>48</td>
<td>637 ± 90*</td>
<td>644 ± 77*</td>
<td>650 ± 83*</td>
</tr>
<tr>
<td>72</td>
<td>678 ± 102</td>
<td>686 ± 77</td>
<td>682 ± 78</td>
</tr>
<tr>
<td><strong>VA (%)</strong></td>
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<tr>
<td>Pre-</td>
<td>92.4 ± 2.9</td>
<td>92.2 ± 2.7</td>
<td>92.3 ± 2.6</td>
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<tr>
<td>Post-</td>
<td>84.5 ± 5.8*</td>
<td>84.8 ± 6.1*</td>
<td>86.1 ± 4.7*</td>
</tr>
<tr>
<td>24</td>
<td>87.6 ± 3.3*</td>
<td>89.4 ± 3.8*</td>
<td>88.1 ± 3.5*</td>
</tr>
<tr>
<td>48</td>
<td>88.2 ± 4.4*</td>
<td>89.9 ± 3.8</td>
<td>89.5 ± 3.3</td>
</tr>
<tr>
<td>72</td>
<td>91.4 ± 3.2</td>
<td>92.0 ± 3.2</td>
<td>91.1 ± 2.9</td>
</tr>
<tr>
<td><strong>VA&lt;sub&gt;TMS&lt;/sub&gt; (%)</strong></td>
<td></td>
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<td></td>
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<tr>
<td>Pre-</td>
<td>94.7 ± 2.5</td>
<td>94.0 ± 2.4</td>
<td>94.2 ± 2.0</td>
</tr>
<tr>
<td>Post-</td>
<td>86.9 ± 5.7*</td>
<td>89.1 ± 4.7*</td>
<td>87.7 ± 5.3*</td>
</tr>
<tr>
<td>24</td>
<td>90.7 ± 5.7*</td>
<td>91.5 ± 5.1</td>
<td>91.2 ± 4.0*</td>
</tr>
<tr>
<td>48</td>
<td>92.8 ± 4.1</td>
<td>93.3 ± 4.4</td>
<td>92.5 ± 3.4</td>
</tr>
<tr>
<td>72</td>
<td>93.2 ± 3.5</td>
<td>94.5 ± 3.3</td>
<td>94.2 ± 2.0</td>
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</tbody>
</table>
Supplemental digital content 1. Schematic of experimental protocol. Pre-exercise and at 24, 48 and 72 h post participants completed the battery of assessments in the same order. After the pre-exercise assessment participants completed one of three exercise interventions: i) heavy resistance training consisting of 10 × 5 repetitions of the high bar back squat at 80% 1RM, with 3 min recovery (STR); ii) 10 × 5 repetitions of a jump squat, with 3 min recovery (JUMP); iii) 15 × 30 m maximum sprints, with 2 min recovery (SPR). Participants were encouraged to complete every repetition with maximal intensity. Immediately post-exercise, central and peripheral neuromuscular function were evaluated within 2 min of exercise cessation. Pre-exercise and at 24 h intervals thereafter, single-pulse transcranial magnetic stimulation (TMS) were administered during a submaximal isometric contraction at various percentages (90 to 160%) of active motor threshold (AMT) for the assessment of corticospinal excitability. Paired-pulse TMS were administered during submaximal contraction for assessment of short intracortical inhibition.