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Heavy-resistance exercise-induced increases in jump performance are not explained by changes in neuromuscular function

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Post-activation potentiation (PAP) is the increased involuntary muscle twitch response to stimulation following strong contraction. The enhancement to whole-body explosive muscular performance (PE) after heavy-resistance exercise is often attributed to modulations in neuromuscular function that are proposed to reflect PAP, but the evidence to support this is equivocal. We assessed the neuromuscular basis of PE using transcranial magnetic stimulation (TMS) of the primary motor cortex, and electrical stimulation of the femoral nerve. Eleven male athletes performed heavy-resistance exercise with measures of countermovement jump (CMJ) pre- and 8 min post-exercise. Pre-exercise and after the final CMJ, single- and paired-pulse TMS were delivered during submaximal isometric knee-extensor contractions to measure corticospinal excitability, short-interval intracortical inhibition (SICI), and intracortical facilitation (ICF), with motor evoked potentials recorded from rectus femoris. Twitch responses to motor nerve stimulation during and post maximum-knee-extensor contractions were studied to quantify voluntary activation (VA) and potentiated twitch ($Q_{tw,pot}$). The experimental protocol successfully induced PE (+4 ± 1% change in CMJ, $P = 0.01$), but no changes were observed for maximum voluntary force, VA, corticospinal excitability, SICI or ICF (all $P > 0.05$), and $Q_{tw,pot}$ declined ($P < 0.001$). An enhancement of muscular performance after heavy resistance exercise was not accompanied by PAP, or changes in measures of neuromuscular function.

The involuntary twitch response of a muscle to motor nerve stimulation is acutely enhanced by prior contraction of the same muscle (Vandervoort et al., 1983; Sale, 2002). This phenomenon, defined as post-activation potentiation (PAP), was originally observed in single limb models, but more recently has been cited as an explanatory factor for the observed performance enhancement of whole-body explosive tasks (e.g., jumping, sprinting, throwing) after prior heavy resistance exercise (Kilduff et al., 2007, 2008; Bevan et al., 2009, 2010; West et al., 2013a, b; Seitz et al., 2014). The use of low-volume, heavy resistance exercise as a preparation strategy for athletic performance is commonplace, and with the right combination of subject characteristics (Gourgoulis et al., 2003; Seitz et al., 2014), resistance exercise stimulus (Bevan et al., 2009, 2010), and rest interval (Kilduff et al., 2007; Bevan et al., 2009), the enhancement to muscular performance ranges between 2% and 8% (Kilduff et al., 2007; Bevan et al., 2009, 2010; West et al., 2013a, b). Although conceptually similar, the mechanisms underpinning the potentiation of the resting involuntary muscle twitch and potentiation of voluntary whole-body athletic performance likely differ, given that twitch potentiation has been observed in the absence of any enhancement of voluntary muscular performance (Folland et al., 2008) and *vice versa* (Pearson & Hussain, 2014). While the presence of twitch potentiation (hereafter referred to as PAP) and enhancement of whole-body athletic performance (hereafter referred to as PE) is well documented, a mechanistic explanation for either remains elusive.

It is likely that any enhancement or potentiation of muscular performance after a prior contraction is mediated within the neuromuscular system given its inherent relationship with explosive performance. For PAP, two principal mechanisms have been proposed; phosphorylation of myosin regulatory light chains (RLC) and an increase in the recruitment of high-threshold motor units. Phosphorylation of myosin RLCs has been demonstrated in skinned animal models (Manning & Stull, 1982; Szczesna et al., 2002) but the evidence in human muscle is unclear

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(Stuart et al., 1988; Smith & Fry, 2007). For single limb contractions *in vivo*, an increase in the recruitment of higher order motor units has been proposed based on an increase in the Hoffman spinal reflex (H reflex) after intense isokinetic plantar flexion contractions (Trimble & Harp, 1998) and maximum isometric knee-extensor contractions (Folland et al., 2008). The changes in neural function observed in single limb models are often cited as explanatory factors for the PE observed after heavy resistance exercise, but the neurophysiological responses to a whole-body heavy resistance exercise stimulus are not well studied. In addition, the presence of H reflex potentiation after single limb exercise is equivocal (Hodgson et al., 2008), and whether this contributes to a functional performance benefit is unclear (Folland et al., 2008). Changes in the H reflex could also be mediated by a number of supraspinal, spinal, and peripheral afferent inputs which might or might not contribute to an increase in high-threshold motor unit recruitment (Carroll et al., 2011). An understanding of the neuromuscular basis to PAP, and particularly PE, is therefore lacking, despite its intuitive appeal.

Transcranial magnetic stimulation (TMS) has been increasingly used in the sport and exercise sciences to assess central nervous system function (Goodall et al., 2014) and offers the potential to better understand the neural basis to PAP and PE. Stimulation of motor cortical cells with singlepulse TMS elicits a motor evoked potential in the target muscle of interest, the characteristics of which (when expressed relative to the maximum muscle compound action potential) can be studied to quantify the excitability of the brain-to-muscle pathway. Single-pulse TMS has been previously used to demonstrate acute and chronic modulations in corticospinal excitability as a result of strength training (Beck et al., 2007; Griffin & Cafarelli, 2007; Selvanayagam et al., 2011; Weier et al., 2012; Nuzzo et al., 2015) and during maximal (Butler et al., 2003) and submaximal (Williams et al., 2014) fatiguing contractions. Importantly in the context of potentiation, modulations in corticospinal excitability have been demonstrated after a single session of resistance training of the elbow flexors (Nuzzo et al., 2015) and forearm muscles (Selvanayagam et al., 2011), suggestive of a rapid plasticity of the neuromuscular system in response to resistance training. Paired-pulse TMS paradigms can be used to reveal further information about the status of facilitatory and inhibitory intracortical circuits within the brain. By varying the interval between stimuli, paired-pulse TMS can be used to measure the excitability of gamma-aminobutyric acid type A-mediated inhibitory (short-interval intracortical inhibition, SICI) and glutamate-mediated excitatory (intracortical facilitation, ICF) intracortical circuits (Chen, 2011). Paired-pulse TMS has been successfully used to reveal changes in intracortical activity as a result of resistance exercise (Weier et al., 2012; Zult et al., 2015), after a period of skill practice (Perez et al., 2004, 2007) and after fatiguing contractions (Maruyama et al., 2006; Takahashi et al., 2011). Collectively these studies demonstrate that TMS can be used to reveal modulations in the central nervous system in response to resistance training exercise, some of which are immediate in nature (Selvanayagam et al., 2011; Nuzzo et al., 2015). Considering these data, the use of single- and paired-pulse TMS paradigms to study the acute neuromuscular responses to a whole-body strength training stimulus could provide a neurophysiological explanation for the PE observed after heavy resistance exercise. The aim of this study was to assess the acute neuromuscular responses to a low-volume, heavy resistance exercise stimulus. We hypothesized that the resistance exercise would result in an acute enhancement of muscular performance, which would be concurrent with changes in neuromuscular function.

Materials and methods

Participants

With institutional ethical approval, 11 male athletes gave written, informed consent to participate in the study (mean ± SD, age, 23 ± 4 years, stature, 1.81 ± 0.09 m, body mass (BM), 89 ± 13 kg, predicted one repetition maximum squat, 151 ± 21 kg or 1.7 ± 0.2 kg/BM). Participants were all currently training in sports requiring explosive movements (i.e., sprinting and jumping), and had a minimum 2-year history of regular resistance training. Testing was conducted during the off-season period while participants were continuing regular strength and conditioning training.

Design

Participants visited the laboratory to complete three visits; a practice trial, followed by experimental and control trials, the order of which was randomized and counterbalanced. The practice trial consisted of habituation to the neuromuscular and functional measurements, and determination of three repetition maximum (3 RM) squat strength. Neuromuscular function was assessed using electrical stimulation of the femoral nerve, and TMS over the primary motor cortex (M1), with evoked responses recorded from the rectus femoris (RF) during isometric knee-extensor contractions. For the experimental trial, participants performed a 10-min warm-up followed by a low-volume, high-intensity strength training session (3 × 3 back squat at 80%, 90% and 100% of 3RM) with countermovement jump height (CMJ) measured pre- and post-warm-up, and 8 min post the final squat set to measure if explosive performance was enhanced by the heavy resistance exercise (Kilduff et al., 2008). A battery of neuromuscular tests were completed pre-warm-up and immediately post the final CMJ. For the control trial, participants completed the same neuromuscular assessment at the same time of day, separated by the same amount of time as the experimental trial, where they rested quietly in the laboratory. The control trial

was designed to assess any confounding effect of the neuromuscular assessment protocol on the measures studied. Prior to each visit participants were instructed to record and replicate their morning dietary intake, and to refrain from caffeine, alcohol, and strenuous exercise in the preceding 48 h. A schematic of the experimental and control trials is shown in Fig. 1.

Procedures

Preliminary visit; repetition maximum assessment

Maximum isoinertial strength was assessed in all participants by a three repetition maximum barbell back squat, with one repetition maximum estimated using a prediction equation (LeSuer et al., 1997). All participants completed a structured 10-min warm-up, which incorporated jogging, dynamic flexibility movements, mobility exercises specific to squatting and jumping, and 3 × 30 m progressive strides at 70%, 80%, and 90% of perceived maximum sprint speed. Participants then completed warm-up sets of three repetitions of back squats, beginning with an unloaded barbell and progressing to 50%, 70%, 80%, and 90% of their estimated 3RM. The load on the bar was then incremented by 2–5% until participants could not complete three repetitions. 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 successful if participants could complete the concentric phase in ≤2 s.

Countermovement jump height

An electronic photocell system (Optojump, Microgate, Bolzano, Italy) was used to measure CMJ height pre- and post-warm-up and 8 min post the final set of squats. Participants squatted to a self-selected depth and jumped for maximum height with arms akimbo to isolate the lower limb musculature. Participants were habituated to this procedure in the preliminary visit, and routinely performed tests of jumping performance in their regular training program.

Experimental trial; Heavy resistance exercise stimulus

After a 10-min warm-up replicating that performed in the preliminary visit, participants completed a low-volume, heavy resistance exercise session consisting of 3 × 3 back squats at 80%, 90%, and 100% of 3RM. The work sets were preceded by 2 × 3 warm-up sets with an unloaded barbell and 50% of 3RM. Three minutes of recovery were allocated between sets. This configuration has been previously used to acutely enhance explosive performance in athletes similar to that studied here (Bevan et al., 2009, 2010). Two maximal CMJs separated by 30 s were performed pre-warm-up, post-warm-up, and 8 min post the final squat set when, based on previous observations using a similar resistance training stimulus in a similar population, PE was expected to be maximized (Kilduff et al., 2007, 2008, 2011).

Neuromuscular function

Measures of neuromuscular function were evaluated using single- and paired-pulse TMS over the primary motor cortex, and electrical stimulation of the femoral nerve, with evoked responses recorded with surface electromyography (EMG). All measurements were taken during submaximal and maximal isometric knee-extensor contractions. After appropriate determination of stimulus intensity (details below), participants completed two practice isometric maximum voluntary contractions (MVC) of the knee-extensors, followed by three MVCs of 3–5 s in duration with electrical stimulation delivered during and 2 s post to assess voluntary activation (VA)

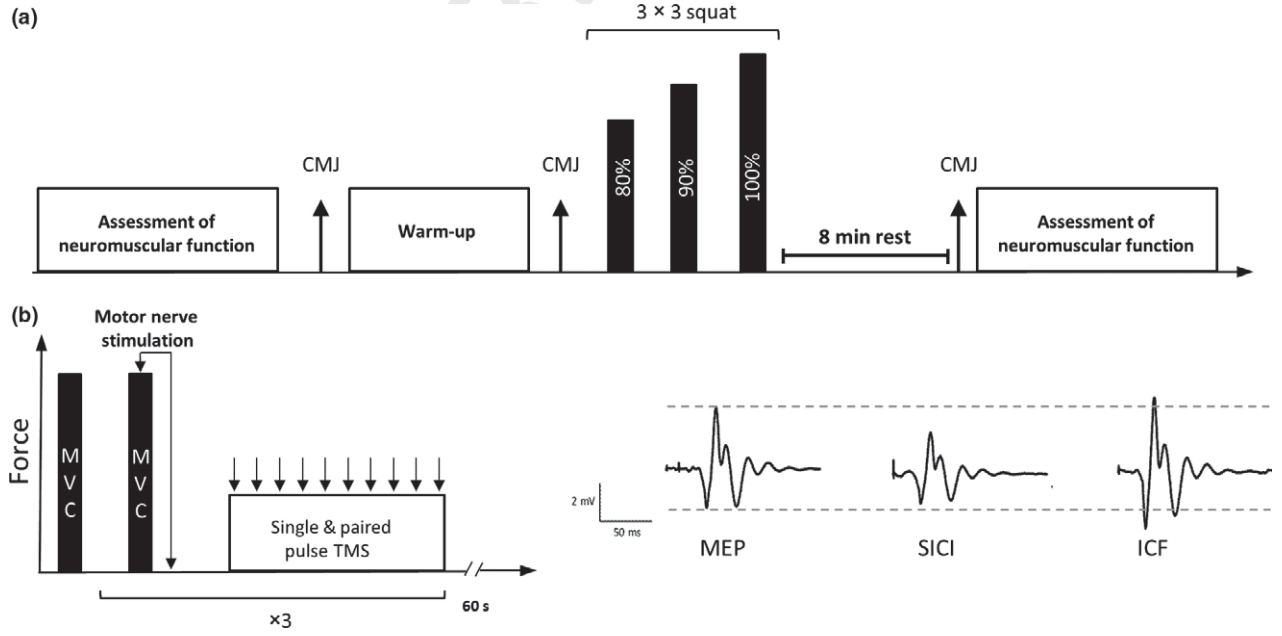

Fig. 1. Schematic of experimental trial (a), and assessment of neuromuscular function with motor evoked potential (single-pulse MEP), short-interval intracortical inhibition (SICI), and intracortical facilitation (ICF) responses (waveform averages of eight stimulations) from a representative participant (b). During the control trial the assessment of neuromuscular function was completed at the same time points, between which participants rested in the laboratory.

and potentiated quadriceps twitch force ($Q_{tw,pot}$), respectively. Subsequent to this, participants were required to maintain a submaximal contraction at 20% MVC where single- and paired-pulse TMS, and electrical motor nerve stimulation, were administered for measurement of corticospinal responsiveness, short-interval intracortical inhibition (SICI), and intracortical facilitation (ICF). Visual feedback of the target force was provided via a computer monitor. Details of these procedures are provided below.

Force and EMG recordings

Isometric knee-extensor force (N) was measured using a calibrated load cell (MuscleLab force sensor 300, Ergotest technology, Norway) affixed to a custom-built chair and attached to the participants right leg via a non-compliant strap positioned superior to the ankle malleoli, directly in line with the applied force. Participants remained seated during all contractions with the hips and knees at 90 degrees flexion. Electromyography of the rectus femoris was recorded via surface electrodes (Ag/AgCl; Kendall H897PG/F, Covidien, Mansfield, MA, USA) placed 2 cm apart over the belly of the muscle, with the reference electrode placed on the patella. The area of electrode placement was prepared by removing hair, abrading, and cleaning with an alcohol swab. Electrode position was marked with indelible ink to ensure consistent placement on repeat trials. The electrodes were used to measure the root mean square amplitude during voluntary contractions, and the evoked compound muscle action potential (M-wave) and motor evoked potential (MEP) elicited by motor nerve and motor cortical stimulation, respectively. Force and surface EMG signals were amplified (×300 and ×1000, respectively) and band-pass filtered (20–2000 Hz) using CED 1902 amplifiers (Cambridge Electronic Design, Cambridge, UK). Force and EMG signals were sampled at 200 and 4000 Hz, respectively, and stored on a computer using an analog-to-digital converter (CED 1401, Cambridge Electronic Design) for later analysis (Spike2 v7.12, Cambridge Electronic Design).

Motor nerve stimulation

Single electrical stimuli (200 μs duration) were delivered to the right femoral nerve via surface electrodes (CF3200, Nidd Valley Medical Ltd, Harrogate, UK) using a constant-current stimulator (DS7AH, Digitimer Ltd, Welwyn Garden City, UK) at rest and during voluntary contraction at 20% and 100% of maximum. The cathode was positioned over the motor nerve, high in the femoral triangle in a location that elicited the maximum quadriceps twitch amplitude (Q_{tw}) and M-wave at rest. The anode was positioned midway between the iliac crest and greater trochanter. Stimulation intensity was determined from single stimuli delivered in 20 mA stepwise increments until a plateau in Q_{tw} and M-wave were observed. The final intensity was increased by 30% to account for any activity-dependent change in axonal conduction and was not different between trials (mean ± SD current: experimental, 177 ± 50 mA, control, 173 ± 37 mA).

Transcranial magnetic stimulation

Single- and paired-pulse TMS were delivered over the left M1 via a concave double cone coil using a BiStim unit and two Magstim 200^2 stimulators (The Magstim Company Ltd, Whitland, UK). The optimal coil placement (posterior–anterior intracranial current flow) was determined at the start of each trial as the position that elicited the largest MEP in the RF (position relative to the vertex: ~1–2 cm), and was marked with indelible ink for consistent placement on subsequent trials. Active motor threshold (AMT) was determined during a 20% MVC as the minimum stimulation intensity that elicited a consistent MEP of >200 μV in three of five stimulations (Kidgell et al., 2010; Weier et al., 2012) and was not different between trials (mean ± SD stimulator output: experimental, 48 ± 7%; control, 47 ± 6%).

Corticospinal responsiveness, SICI, and ICF

At each neuromuscular assessment point, 8× single and 16× paired-pulse magnetic stimuli were administered to quantify corticospinal excitability, short-interval intracortical inhibition, and intracortical facilitation (8× stimuli each). Pulses were delivered during isometric knee-extensor contraction at 20% MVC in a random order, in three blocks of eight stimuli with 5–7 s separating each stimuli and 60 s in between sets. The single, or test pulse, was set at 1.2× AMT. To elicit SICI, a sub-threshold stimuli (0.7× AMT) was followed by the supra-threshold test pulse (1.2× AMT) with an ISI of 3 ms (Ortu et al., 2008; Kidgell et al., 2010; Weier et al., 2012). For ICF, an ISI of 13 ms separated the same paired-pulse configuration. Five electrical stimuli were delivered to the femoral nerve during the same strength contraction for quantification of corticospinal excitability.

Data analysis

The peak-to-peak amplitudes of the evoked M-wave and MEP responses, measured as the absolute difference between the minimum and maximum points of the biphasic waveform (Fowles et al., 2002), were quantified offline. Corticospinal excitability was quantified as the ratio between the test MEP, and the M-wave elicited from motor nerve stimuli during the same strength contraction (i.e., 20% MVC). The average of the conditioned paired-pulse MEPs were expressed relative to the averaged unconditioned MEP to quantify SICI and ICF. Additionally, the root mean square EMG amplitude (EMG_{RMS}) and average force were measured across 80 ms prior to TMS to ensure a similar level of background muscle activity was present immediately pre-stimulation for unconditioned and conditioned MEPs. The interpolated twitch technique was used to quantify VA (Merton, 1954). In brief, the amplitude of the superimposed twitch force (SIT) measured during MVC was compared with the $Q_{tw,pot}$ elicited 2 s post-MVC at rest (VA, % = (1 − [SIT/$Q_{tw,pot}$] × 100). Reductions in VA and $Q_{tw,pot}$ were considered as indicators of central and peripheral fatigue, respectively.

Statistical analysis

Descriptive statistics are presented as means ± SD. Differences in pre-stimulation muscle activity and force were assessed within trial (experimental, control) using 3 × 2 (stimulation configuration; unconditioned, conditioned SICI, and conditioned ICF, by time; pre, post) factorial repeat measures ANOVA. Differences in CMJ height between pre-warm-up, post-warm-up, and post-strength training were assessed with one-way repeat measures ANOVA with repeated planned contrasts (i.e., post-warm-up vs pre-warm-up, post-strength training vs post-warm-up) employed for pairwise comparisons. Differences between groups for all neuromuscular measures were assessed using 2 × 2 (trial; experimental and control, by time; pre, post) factorial ANOVA; with focus on the

trial × time interaction effect, which analyzes the effect of the strength training intervention relative to the control trial. The assumptions of these procedures were verified as per the guidelines of Newell et al. (2010). Statistical analysis was conducted using GraphPad Prism (GraphPad Software Inc, v5, La Jolla, California, USA).

Results

Enhancement of CMJ performance

Countermovement jump height increased from pre-warm-up (41.0 ± 4.3 cm) to post-warm-up (43.7 ± 3.9 cm, $P = 0.002$) and was further enhanced 8 min post strength training (44.7 ± 4.1 cm, $P = 0.008$, Fig. 2). The magnitude of PE from post-warm-up to post-strength training averaged 3.5 ± 1.8%.

Neuromuscular fatigue

A small decrease in MVC strength was observed after the strength training stimulus (800 ± 124 N to 774 ± 139 N) that was not different to control (774 ± 111 N to 767 ± 116 N, trial × time, $P = 0.142$; Fig. 3a). Similarly, a small reduction in voluntary activation was observed after strength training (91.2 ± 4.5% to 90.0 ± 6.2%) that was not different to control (90.2 ± 3.2% to 91.3 ± 4.0%, trial × time, $P = 0.06$; Fig. 3b). Potentiated twitch force was reduced after strength training (235 ± 65 N to 185 ± 55 N) in comparison to control (220 ± 57 N to 213 ± 51 N, trial × time, $P < 0.001$, Fig. 3c) indicating an absence of PAP and the presence of peripheral fatigue.

Corticospinal excitability, SICI, and ICF

Force and EMG_{RMS} were consistent both between stimulation configurations (unconditioned, conditioned SICI, conditioned ICF) and across time (pre, post) supporting a consistent level of muscle activation within each trial (all $P < 0.05$, Table 1). The heavy resistance training stimulus had no clear effect on measures of corticospinal excitability, or the excitability of intracortical interneurons (Table 1). Corticospinal excitability was unchanged post-strength training (Experimental, 64 ± 16% to 58 ± 13%; Control, 62 ± 9% to 66 ± 8%, trial × time, $P = 0.15$; Fig. 4a). The degree of SICI tended to increase after strength training (75 ± 15% to 66 ± 20%, time, $P = 0.07$) but the change was not different to control (73 ± 18% to 72 ± 17%, trial × time, $P = 0.20$; Fig. 4b). Intracortical facilitation was unchanged after strength training (111 ± 6% to 113 ± 16%) with no difference in comparison to control (112 ± 10% to 110 ± 10% %, trial × time, $P = 0.44$; Fig. 4c).

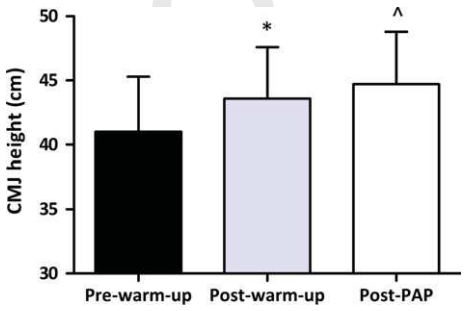

Fig. 2. Countermovement jump height pre-warm-up, post-warm-up, and 8 min post low-volume, heavy-resistance exercise. Values are mean + SD. *Different to pre-warm-up, ^Different to post-warm-up ($P < 0.05$).

Discussion

We hypothesized that measurement of the central nervous system responses to a low-volume, heavy

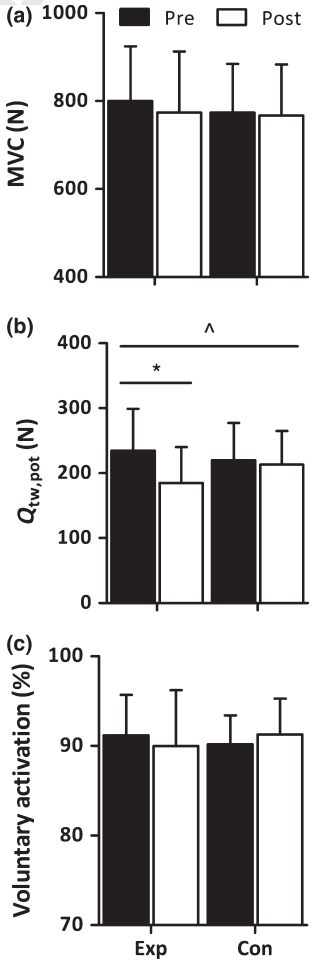

Fig. 3. Maximum voluntary contraction force (a), potentiated twitch force (b) and voluntary activation (c) pre and post low-volume, heavy-resistance exercise (Exp), and at the same time points in a control condition of passive rest (Con). *Different to pre-, ^Significant interaction effect ($P < 0.05$).

Table 1. Maximum muscle compound action potentials (M_{max}), unconditioned (MEP amplitude), and conditioned (SICI amplitude, ICF amplitude) motor evoked potentials in rectus femoris in experimental and control trials. All responses were evoked during a submaximal isometric knee-extensor contraction (20% MVC). The average pre-stimulation root mean square EMG amplitude and force for each MEP configuration was equivalent within trial. Values are mean ± SD (n = 11)

	Experimental		Control	
	Pre	Post	Pre	Post
Evoked amplitudes (mV)				
M_{max}	5.82 ± 1.94	6.21 ± 2.29	5.96 ± 2.51	5.91 ± 2.65
Unconditioned MEP	3.62 ± 1.22	3.58 ± 1.23	3.77 ± 1.86	3.58 ± 1.23
Conditioned (SICI) MEP	2.68 ± 1.00	2.34 ± 1.03	2.57 ± 1.05	2.64 ± 1.05
Conditioned (ICF) MEP	3.99 ± 1.33	4.07 ± 1.62	4.10 ± 1.80	4.16 ± 1.76
EMG_{RMS} (mV)				
Unconditioned MEP	0.076 ± 0.010	0.074 ± 0.012	0.074 ± 0.017	0.077 ± 0.018
Conditioned (SICI) MEP	0.076 ± 0.010	0.076 ± 0.012	0.073 ± 0.016	0.077 ± 0.021
Conditioned (ICF) MEP	0.083 ± 0.021	0.076 ± 0.015	0.074 ± 0.018	0.077 ± 0.021
Force (N)				
Unconditioned MEP	155.9 ± 20.8	152.6 ± 22.8	151.3 ± 18.5	151.0 ± 20.1
Conditioned (SICI) MEP	155.9 ± 21.4	152.8 ± 22.7	151.2 ± 19.2	150.6 ± 19.6
Conditioned (ICF) MEP	155.4 ± 21.4	152.6 ± 22.7	151.7 ± 18.4	150.5 ± 20.5

resistance exercise stimulus might reveal a neuromuscular basis to the subsequent acute enhancement of explosive muscular performance. Despite a significant enhancement of explosive muscular performance as a consequence of resistance exercise, we found no evidence of positive change in measures of central nervous system activation or responsiveness, the excitability of intracortical interneurons, or muscle function. The observed PE was also present in the absence of any PAP of the involuntary resting twitch response. Indeed, the data indicate a tendency for the resistance exercise to induce a small degree of muscle fatigue, despite the enhanced whole-body explosive performance. These data suggest that PAP and PE are mediated by alternative mechanisms, and that the neuromuscular basis to PE remains to be elucidated.

Enhancement of explosive muscular performance

The enhancement of CMJ performance from post-warm-up to post-strength training averaged 3.5%, which is similar to that reported in previous studies using a similar experimental approach (Kilduff et al., 2007; Bevan et al., 2009, 2010; West et al., 2013a, b). The improved jump performance suggests the resistance exercise employed was an effective stimulus to elicit an acute enhancement in whole-body, explosive performance. Previous research has suggested this enhancement in whole-body performance can be explained by the same neuromuscular mechanisms underpinning PAP, which is classically defined as an enhancement in the resting, involuntary muscle twitch response to electrical stimulation after a strong contraction (Sale, 2002). The mechanisms proposed to underpin PAP include phosphorylation of myosin RLC (Manning & Stull, 1982; Szczesna et al., 2002), recruitment of higher order motor units (Tillin & Bishop, 2009), and increases in the Hoffman spinal reflex (Trimble & Harp, 1998; Folland et al., 2008); some of which have been demonstrated (although not equivocally) in single limb models. Given that neuromuscular function is ostensibly linked to voluntary explosive movements, we hypothesized that the PE observed after heavy resistance exercise might be associated with similar mechanisms. Contrary to our hypothesis, we observed no degree of PAP, or any changes in central nervous system function in response to heavy resistance exercise, despite an improvement in voluntary explosive muscular performance.

Potentiated twitch force and voluntary activation

The resting twitch response to motor nerve stimulation exhibited no PAP after heavy resistance exercise. Indeed, the resistance training stimulus was associated with a small, significant reduction in potentiated twitch force, and there were no significant changes in measures of maximum voluntary force or voluntary activation. The recruitment of higher order motor units might be reflected in an increase in voluntary activation and has been proposed as a potential mechanism to explain the PE effect (Tillin & Bishop, 2009), but we found no evidence to support this proposal. In addition, the significant decline in potentiated twitch indicates the presence of peripheral fatigue, despite the improved jumping performance. These data suggest no neuromuscular basis to "potentiation" of whole-body explosive movements after prior heavy resistance exercise, and no positive effects on muscle function; as such the mechanisms underpinning the observed PE are likely different

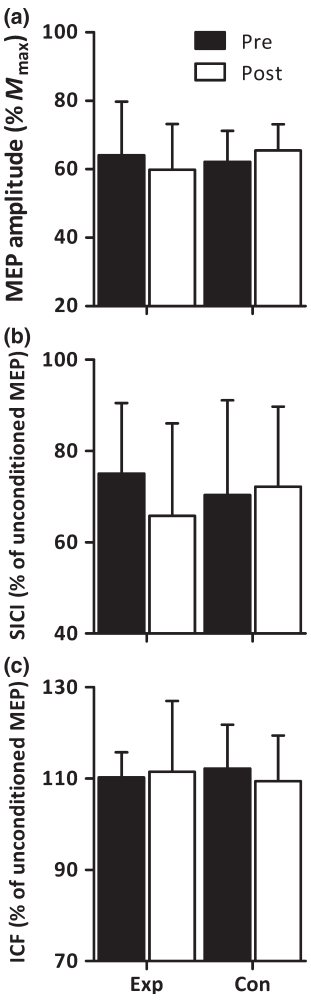

Fig. 4. Corticospinal excitability (a), short-interval intracortical inhibition (b) and intracortical facilitation (c) pre and post low-volume, heavy resistance exercise (Exp), and at the same time points in a control condition of passive rest (Con).

from those underpinning the PAP of an involuntary muscle twitch.

An alternative explanation for the disconnect between changes in potentiated twitch force and improvements in explosive performance could reside in the evolution of potentiation and fatigue following the resistance training exercise. After a strong contraction, fatigue and potentiation co-exist and exert a summative influence on the athlete's ability to express explosive force (Gossen & Sale, 2000; Kilduff et al., 2007, 2008). The PAP response to electrical stimulation, for example, is highest immediately post-MVC, when fatigue is also presumably highest, and declines over time as fatigue is resolved and potentiation dissipates (Folland et al., 2008). A significant degree of fatigue could therefore be concurrent with an improved performance if the degree of potentiation elicited by the exercise stimulus exceeds the observed fatigue such that a one-off explosive performance is enhanced. It is however difficult to reconcile this concept with the present data where measures of central activation showed no change, and PAP was absent. The prolonged effects of a low-volume, heavy resistance exercise stimulus are not known, but the magnitude of muscle fatigue observed in the present study suggests this type of preparation strategy might not be beneficial in sports requiring repeated explosive movements, as the exercise induces muscle fatigue that might impair repeated efforts once the performance enhancement effect has dissipated. Further research is required to substantiate this extrapolation and the utility of preparation strategies that employ resistance exercise in competitive scenarios requiring multiple explosive efforts.

Responses to transcranial magnetic stimulation

Using single- and paired-pulse TMS, this study is the first to probe the function of the central nervous system concurrent with PE of an explosive, athletic movement by heavy resistance exercise. The MEP recorded at the muscle in response to single-pulse TMS, when appropriately normalized to the maximal M-wave, provides information on the excitability of the brain-to-muscle pathway (Goodall et al., 2014). Paired-pulse TMS paradigms incorporate a conditioning pulse which excites cortical interneurons that subsequently inhibit or facilitate the resulting MEP, providing a measure of the status of intracortical circuits within the primary motor cortex (Chen, 2011). Attribution of changes in these measures to specific sites is problematic, as the response recorded at the muscle is subject to modulation from a range of supraspinal, spinal, peripheral afferent, and motoneuronal inputs (Carroll et al., 2011). This notwithstanding, changes in the MEP evoked by single-pulse (Beck et al., 2007; Griffin & Cafarelli, 2007; Selvanayagam et al., 2011; Weier et al., 2012; Nuzzo et al., 2015) and paired-pulse (Weier et al., 2012; Zult et al., 2015) TMS have been reported in response to resistance training exercise. For example, Weier et al. (2012) observed a 112% increase in corticospinal excitability, and a 32% reduction in SICI after 4 weeks of resistance training. Nuzzo et al. (2015) observed increases in corticospinal excitability after a single session of ballistic, isometric elbow flexor contractions. We hypothesized that single- and paired-pulse TMS might reveal similar acute changes in the central nervous system concurrent with PE of an explosive movement after heavy resistance exercise. Despite the significant degree of PE, there were no changes observed in the excitability of the brain-to-muscle pathway (MEP:M_{max}), or measures of intracortical inhibition (SICI) or facilitation (ICF). Thus, these measures were not able to explain the PE

effect, and a neural basis to the acute enhancement of explosive muscular performance after heavy resistance exercise remains to be elucidated.

What factors explain PE?

The central nervous system responses to heavy resistance training employed in this study cannot provide an explanation for the observed enhancement of jump performance, and PAP of the resting twitch was absent. This raises an obvious question; what factors contribute to the improvement in explosive performance? Firstly, absence of evidence in the measures studied here does not imply there was no change in neuromuscular function post-heavy resistance exercise. Indeed, there is some evidence to suggest that the H reflex is modulated in response to single limb contractions (Trimble & Harp, 1998; Folland et al., 2008), though this was observed in the absence of any functional benefit and has yet to be studied in a whole-body model. Additionally, the lack of specificity of the measurement method (single limb, isometric contraction) in comparison to the task (whole-body heavy resistance and explosive exercise) could obscure any modulations in neuromuscular function (discussed below in "Limitations"). A hormonal response to the heavy strength training stimulus could be an alternative explanatory factor; increases in testosterone have been reported immediately post-heavy resistance exercise and higher circulating concentrations are associated with improved physical performance (Crewther et al., 2011a, b). An increase in muscle temperature as a result of heavy resistance exercise could have facilitated jump performance (Sargeant, 1987), although the decline in potentiated twitch force observed in this study suggests there are voluntary rather than involuntary mechanisms responsible. The similarity of the squatting exercise to the countermovement jump could also be a factor; i.e., performance is enhanced not by a physiological mechanism but by acute priming through practice of the skill (Crewther et al., 2011a, b). Finally the observed improvement in explosive performance could reflect a psychological effect. That is, PE could be explained simply by an increase in the perception of readiness for explosive performance.

Limitations

The majority of measures of central nervous system function were studied in a submaximal (20% MVC), single limb, isometric contraction at knee and hip angles of 90°. The responses of the central nervous system during contraction at a submaximal intensity might not be reflective of peri-maximal, whole-body dynamic contractions where the PE effect was elicited and observed. The submaximal intensity employed was necessary to study the excitability of intracortical interneurons as their influence is abolished at contraction strengths >25% MVC (Ortu et al., 2008). In addition, multiple stimulations are required to measure these responses, making higher contraction strengths undesirable because of potential confounding effects of fatigue. All responses to stimulation were elicited from the rectus femoris (RF). The RF muscle was chosen because of its biarticular nature and significant contribution to both hip flexion and knee extension moments during squatting and jumping movements; however, the moment arm at which force would be maximized during these dynamic movements would likely be different to the static moment arm of single limb isometric contractions at hip and knee angles of 90°. Additionally, the responses of the RF might not be reflective of all the knee-extensor musculature, nor indeed the other significant muscle groups that might contribute to hip extension moments. Optimizing the simultaneous measurement of motor evoked potentials across a range of muscle groups is, however, fraught with difficulty, which is why a specific muscle was chosen to study. A final limitation is the measurement of jump performance 8 min post for every participant. This timeframe was chosen based on previous research in a similar population (Kilduff et al., 2007, 2008); however, there is known variability in this response and as such the PE effect might not have been maximal for every participant. This notwithstanding, we did observe an enhancement in jump performance in every participant, which indicates the protocol implemented was appropriate to answer the question under study.

In conclusion, a low-volume, heavy resistance exercise stimulus can acutely enhance jumping performance in well-trained strength-power athletes. We hypothesized this enhancement might be associated with PAP, and modulations in the central nervous system responses to motor nerve and motor cortical stimulation. Despite a significant enhancement of jumping performance, there were no changes in measures of voluntary activation, corticospinal excitability, short-intracortical inhibition, or intracortical facilitation. The resting muscle twitch responses to electrical stimulation of the motor nerve revealed no PAP, but rather the presence of muscle fatigue. A neuromuscular basis to the acute enhancement of explosive muscular performance after heavy resistance exercise remains to be elucidated.

Perspective

Post-activation potentiation is the phenomenon describing the increased involuntary muscle twitch response after a strong contraction. The concept of

PAP has been used to explain the acute enhancement of whole-body, explosive muscular performance after heavy resistance exercise; such enhancement is frequently attributed to modulations within the neuromuscular system, but the evidence supporting this posit is extrapolated from single limb PAP models and the neuromuscular responses to a whole-body "potentiation" stimulus are not well studied. Here, we used motor cortical and motor nerve stimulation to probe the function of the central nervous system after a whole-body, heavy-resistance exercise stimulus. Despite a significant enhancement of jumping performance after heavy-resistance exercise, we found no evidence of modulations in measures of central nervous system activation or responsiveness and no change in the status of inhibitory and facilitatory intracortical circuits. The involuntary resting twitch response to motor nerve stimulation was not potentiated, but rather was reduced, indicative of muscle fatigue. These data are the first to explicitly test the hypothesis that the enhancement of whole-body athletic performance after heavy resistance exercise is mediated within the central nervous system. Although a plausible and oft-cited explanation, the neural basis to an enhancement of whole-body athletic performance cannot be explained by the measures studied here.

Key words: Athletic performance, intracortical facilitation, neuromuscular physiology, post-activation potentiation, short-interval intracortical inhibition, transcranial magnetic stimulation, voluntary activation.

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References

Beck S, Taube W, Gruber M, Amtage F, Gollhofer A, Schubert M. Task-specific changes in motor evoked potentials of lower limb muscles after different training interventions. Brain Res 2007: 1179: 51–60.

Bevan HR, Cunningham DJ, Tooley EP, Owen NJ, Cook CJ, Kilduff LP. Influence of postactivation potentiation on sprinting performance in professional rugby players. J Strength Cond Res 2010: 24: 701–705.

Bevan HR, Owen NJ, Cunningham DJ, Kingsley MI, Kilduff LP. Complex training in professional rugby players: influence of recovery time on upper-body power output. J Strength Cond Res 2009: 23: 1780–1785.

Butler JE, Taylor JL, Gandevia SC. Responses of human motoneurons to corticospinal stimulation during maximal voluntary contractions and ischemia. J Neurosci 2003: 23: 10224–10230.

Carroll TJ, Selvanayagam VS, Riek S, Semmler JG. Neural adaptations to strength training: moving beyond transcranial magnetic stimulation and reflex studies. Acta Physiol 2011: 202: 119–140.

Chen R. Excitatory and inhibitory effects of transcranial magnetic stimulation. Biocybern Biomed Eng 2011: 31: 93–105.

Crewther BT, Cook C, Cardinale M, Weatherby RP, Lowe T. Two emerging concepts for elite athletes: the short-term effects of testosterone and cortisol on the neuromuscular system and the dose-response training role of these endogenous hormones. Sports Med 2011a: 41: 103–123.

Crewther BT, Kilduff LP, Cook CJ, Middleton MK, Bunce PJ, Yang GZ. The acute potentiating effects of back squats on athlete performance. J Strength Cond Res 2011b: 25: 3319–3325.

Folland JP, Wakamatsu T, Fimland MS. The influence of maximal isometric activity on twitch and H-reflex potentiation, and quadriceps femoris performance. Eur J Appl Physiol 2008: 104: 739–748.

Fowles JR, Green HJ, Tupling R, O'Brien S, Roy BD. Human neuromuscular fatigue is associated with altered Na^+-K^+-ATPase activity following isometric exercise. J Appl Physiol 2002: 92: 1585–1593.

Goodall S, Howatson G, Romer L, Ross E. Transcranial magnetic stimulation in sport science: a commentary. Eur J Sport Sci 2014: 14: S332–S340.

Gossen ER, Sale DG. Effect of postactivation potentiation on dynamic knee extension performance. Eur J Appl Physiol 2000: 83: 524–530.

Gourgoulis V, Aggeloussis N, Kasimatis P, Mavromatis G, Garas A. Effect of a submaximal half-squats warm-up program on vertical jumping ability. J Strength Cond Res 2003: 17: 342–344.

Griffin L, Cafarelli E. Transcranial magnetic stimulation during resistance training of the tibialis anterior muscle. J Electromyogr Kinesiol 2007: 17: 446–452.

Hodgson MJ, Docherty D, Zehr EP. Postactivation potentiation of force is independent of h-reflex excitability. Int J Sports Physiol Perform 2008: 3: 219–231.

Kidgell DJ, Stokes MA, Castricum TJ, Pearce AJ. Neurophysiological responses after short-term strength training of the biceps brachii muscle. J Strength Cond Res 2010: 24: 3123–3132.

Kilduff LP, Bevan HR, Kingsley MI, Owen NJ, Bennett MA, Bunce PJ, Hore AM, Maw JR, Cunningham DJ. Postactivation potentiation in professional rugby players: optimal recovery. J Strength Cond Res 2007: 21: 1134–1138.

Kilduff LP, Cunningham DJ, Owen NJ, West DJ, Bracken RM, Cook CJ. Effect of postactivation potentiation on swimming starts in international sprint swimmers. J Strength Cond Res 2011: 25: 2418–2423.

Kilduff LP, Owen N, Bevan H, Bennett M, Kingsley MI, Cunningham D. Influence of recovery time on post-activation potentiation in professional rugby players. J Sports Sci 2008: 26: 795–802.

LeSuer DA, McCormick JH, Mayhew JL, Wasserstein RL, Arnold MD. The accuracy of prediction equations for estimating 1-RM performance in the bench press, squat, and deadlift. J Strength Cond Res 1997: 11: 211–213.

Manning D, Stull J. Myosin light chain phosphorylation-dephosphorylation in mammalian skeletal muscle. Am J Physiol Cell Physiol 1982: 242: C234–C241.

Maruyama A, Matsunaga K, Tanaka N, Rothwell JC. Muscle fatigue decreases short-interval intracortical inhibition after exhaustive intermittent tasks. Clin Neurophysiol 2006: 117: 864–870.

Merton PA. Voluntary strength and fatigue. J Physiol 1954: 123: 553–564.

Newell J, Aitchison T, Grant S. Statistics for sports and exercise science: a practical approach. Harlow: Pearson Education, 2010.

Nuzzo JL, Barry BK, Gandevia SC, Taylor JL. Acute strength training increases responses to stimulation of corticospinal axons. Med Sci Sports Exerc 2015: ???: ???–???.

Ortu E, Deriu F, Suppa A, Tolu E, Rothwell JC. Effects of volitional contraction on intracortical inhibition and facilitation in the human motor cortex. J Physiol 2008: 586: 5147–5159.

Pearson SJ, Hussain SR. Lack of association between postactivation potentiation and subsequent jump performance. Eur J Sport Sci 2014: 14: 418–425.

Perez MA, Lungholt BK, Nyborg K, Nielsen JB. Motor skill training induces changes in the excitability of the leg cortical area in healthy humans. Exp Brain Res 2004: 159: 197–205.

Perez MA, Wise SP, Willingham DT, Cohen LG. Neurophysiological mechanisms involved in transfer of procedural knowledge. J Neurosci 2007: 27: 1045–1053.

Sale DG. Postactivation potentiation: role in human performance. Exerc Sport Sci Rev 2002: 30: 138–143.

Sargeant AJ. Effect of muscle temperature on leg extension force and short-term power output in humans. Eur J Appl Physiol Occup Physiol 1987: 56: 693–698.

Seitz LB, de Villarreal ES, Haff GG. The temporal profile of postactivation potentiation is related to strength level. J Strength Cond Res 2014: 28: 706–715.

Selvanayagam VS, Riek S, Carroll TJ. Early neural responses to strength training. J Appl Physiol 2011: 111: 367–375.

Smith JC, Fry AC. Effects of a ten-second maximum voluntary contraction on regulatory myosin light-chain phosphorylation and dynamic performance measures. J Strength Cond Res 2007: 21: 73–76.

Stuart DS, Lingley MD, Grange RW, Houston ME. Myosin light chain phosphorylation and contractile performance of human skeletal muscle. Can J Physiol Pharmacol 1988: 66: 49–54.

Szczesna D, Zhao J, Jones M, Zhi G, Stull J, Potter JD. Phosphorylation of the regulatory light chains of myosin affects Ca^{2+} sensitivity of skeletal muscle contraction. J Appl Physiol 2002: 92: 1661–1670.

Takahashi K, Maruyama A, Hirakoba K, Maeda M, Etoh S, Kawahira K, Rothwell JC. Fatiguing intermittent lower limb exercise influences corticospinal and corticocortical excitability in the nonexercised upper limb. Brain Stimul 2011: 4: 90–96.

Tillin NA, Bishop D. Factors modulating post-activation potentiation and its effect on performance of subsequent explosive activities. Sports Med 2009: 39: 147–166.

Trimble MH, Harp SS. Postexercise potentiation of the H-reflex in humans. Med Sci Sports Exerc 1998: 30: 933–941.

Vandervoort AA, Quinlan J, McComas AJ. Twitch potentiation after voluntary contraction. Exp Neurol 1983: 81: 141–152.

Weier AT, Pearce AJ, Kidgell DJ. Strength training reduces intracortical inhibition. Acta Physiol (Oxf) 2012: 206: 109–119.

West D, Cunningham D, Bevan H, Crewther B, Cook C, Kilduff L. Influence of active recovery on professional rugby union player's ability to harness postactivation potentiation. J Sports Med Phys Fitness 2013a: 53: 203–208.

West DJ, Cunningham DJ, Crewther BT, Cook CJ, Kilduff LP. Influence of ballistic bench press on upper body power output in professional rugby players. J Strength Cond Res 2013b: 27: 2282–2287.

Williams PS, Hoffman RL, Clark BC. Cortical and spinal mechanisms of task failure of sustained submaximal fatiguing contractions. PLoS ONE 2014: 9: e93284.

Zult T, Goodall S, Thomas K, Hortobágyi T, Howatson G. Mirror illusion reduces motor cortical inhibition in the ipsilateral primary motor cortex during forceful unilateral muscle contractions. J Neurophysiol 2015: 113: 2262–2270.