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Articles

High-Volume Light-Load Strength Training, but Not Low-Volume Heavy-Load Strength Training Increases Corticospinal Excitability

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Purpose: To determine whether corticospinal excitability (CSE) and inhibition are differentially modulated following high-volume light-load strength training compared to low-volume heavy-load strength training. We hypothesised high-volume light-load strength training would increase CSE and low-volume heavy-load strength training would reduce intracortical inhibition.

Methods: Transcranial magnetic stimulation (TMS) was used to assess CSE, short-interval intracortical inhibition (SICI), and silent period duration (SP) following high-volume light-load strength training (n = 9), low-volume heavy-load strength training (n = 8) compared to a control group (n = 10). Twenty-seven participants completed either (1) low-volume heavy-load strength training (80% one-repetition maximum [1RM]); (2) high-volume light-load strength training (20% 1RM) or (3) a control condition. CSE, SICI and SP were measured using TMS at baseline and four time-points over a 60 min post-exercise period.

Results: CSE increased rapidly (within 5 min post-exercise) for high-volume light-load strength training and remained elevated for 60 min compared to low-volume heavy-load strength training and control groups. There were no differences following any training for reduced SICI or SP.

Conclusion: These results suggest that high-volume light-load strength training increases the excitability of corticospinal neurons and this increase is likely to be the predominant mechanism for increasing CSE for up to 60 min post training. It may be possible that a greater number of ST sessions are required to observe any differences in the excitability of the intrinsic inhibitory motor-network following high-volume light-load strength training and low-volume heavy-load strength training.

Citation

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Introduction

It is well documented that the rapid increase in muscle strength following a period of strength training is accompanied by changes in the neural control of the trained muscle [1]. To date, it remains unclear how rapidly these neural responses to strength training occur following the commencement of a strength training program. Transcranial magnetic stimulation (TMS) studies have emerged in recent years to study the effects of strength training on the synaptic activity of the cortico-cortical circuitry of the primary motor cortex and of the corticospinal tract (CST) following a bout of strength training [2-10]. Leung et al. [6] reported that a single session of heavy-load strength training (75%1RM) of the biceps brachii increased the amplitude of motor-evoked potentials (MEPs) induced by single-pulse TMS, a finding consistent with Latella et al. [5] who also showed increased MEP amplitude following both heavy-load and hypertrophy-based strength training (<75% 1RM). Recently, Mason et al. [7] and Colomer-Poveda et al. [2] reported that an acute bout of heavy-load strength training of the biceps brachii increased CSE, but interestingly, both of these studies included a light-load ST group. However, the findings of Mason et al. [7] were in contrast to Colomer-Poveda et al. [2], whereby light-light strength training increased CSE in the former study, but remained unchanged in the latter study. To add further confusion to the acute corticospinal responses to strength training, Latella et al. [4] and Selvanayagam et al. [10] both reported reduced MEP amplitude after a single session of strength training. These inconsistent findings are in agreement with a recent systematic review and meta-analysis which concluded that the acute corticospinal responses to strength training likely involve subtle changes along the entire neuroaxis from the primary motor cortex to the spinal cord, and involved increases in CSE and reduced corticospinal inhibition with a high degree of heterogeneity [8].

In contrast to single-pulse TMS, paired-pulse TMS allows for an assessment of the physiology of the intrinsic cortico-cortical connections within the primary motor cortex following strength training [11]. Preliminary evidence suggests that a single bout of strength training affects the excitability of the intracortical circuitry of the primary motor cortex towards facilitation [4,5,7]. A major caveat to the previous studies is that the authors have not determined the time-course responses or examined whether high-volume light-load strength training or low-volume heavy-load strength training differently modulate the corticospinal responses. The previous work of Mason et al. [7] and Colomer-Poveda et al. [2] only measured the responses immediately post-training. Furthermore, the findings of the systematic review by Mason et al. [8] are confounded by the heterogeneity of the strength training interventions, whereby training-load and training-volume all varied. For example, the previous work of Nuzzo et al. [9] completed high-volume (12 sets of 8, quick bursts of maximal force production per repetition), whilst Mason et al. [7] included a high (4 sets of 20) and low-volume (4 sets of 6-8) strength training group, whilst Colomer-Poveda et al. [2] had equal training volume (12 sets of 8), but different training loads (25% or 75% of maximal voluntary contraction [MVC] force). There is a need to determine if the acute motor cortical responses to strength training are due to training-volume or training-load and how these responses differ over time. Therefore, the purpose of the current study was to determine whether the time-course responses to high-volume light-load or low-volume heavy-load strength training are differentially modulated compared to a control group.

Methods

Experimental Procedures

Figure 1 depicts the experimental design and procedures undertaken by the participants. Participants assigned to either strength training group were required to undergo a maximal voluntary strength test of the elbow flexors in order to determine load-prescription for the strength training protocol. Participants from both strength training groups completed a single strength training session with either light or heavy loads. Participants assigned to the control group did not

undertake any training. TMS was applied to participants from all groups at baseline, immediately post strength training and every 20-minutes up to 60-minutes post training.

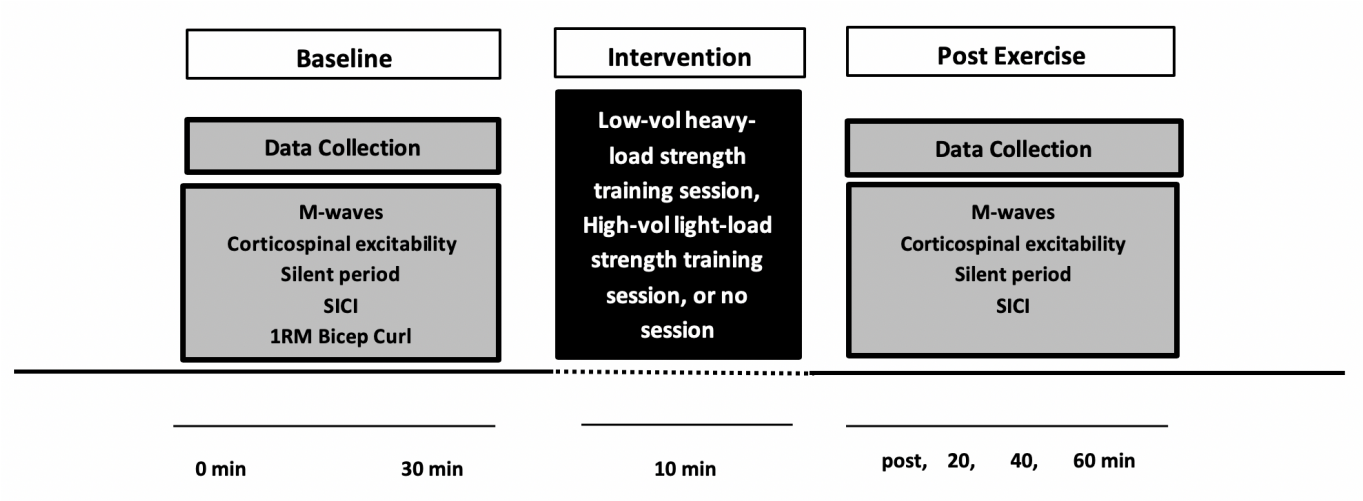

Figure 1. *Experimental design of the study. Post intervention testing was undertaken at 4 separate time points (immediately post, 20-min post, 40-min post, 60-min post).*

Participants

Twenty-seven young healthy adults (M = 19; F = 8; mean age 23.8 ± 2.8 yrs., Table 1) with no known history of neuromuscular impairment volunteered to participate in this study. Participants were randomly allocated into either a high-volume light-load strength training (n = 9), low-volume heavy-load strength training (n = 8) or to a control group (n = 10). Each participant provided written informed consent prior to participation in this study, which was approved by the Human Research Ethics Committee (ID: 10566). Participants were right-hand dominant with a Laterality Quotient Score greater than 40 [12]. Participants had not participated in a structured strength-training protocol within 12 months prior to the commencement of the study. Prior to the experimental protocol, each participant completed an adult screening questionnaire to establish readiness for TMS [13].

Training Group	Sex	Age (yrs.)	Height (cm)	BMI	1RM %	Training Load (kg)	Sets (Reps)
LVHL-ST	4 F, 4 M	24.4±2.8	171.2±9.1	0.241±0.030	80	16.5±1.5	4 (6-8)
HVLL-ST	3 F, 6 M	23.1±3.1	177 ±10.2	0.229±0.039	20	3.8±1.2	4(30)
Control	4 F, 6 M	24.3±2.5	183.2 ±8.1	0.230±0.026	0	0	0

Table 1. *Participant characteristics and strength training workload characteristics for each group.* 1RM, one repetition maximum; BMI; body mass index, CM; centimetres; F; female; LVHL-ST, low volume heavy-load strength training; HVLL, high volume light-load strength training; M; male. Data reported as Mean ± SD.

Voluntary Strength Testing

Each group completed a one-repetition maximum (1RM) strength test of a standing biceps curl which assessed the strength of the biceps brachii. Participants followed the procedures described by Siddique et al. [1]. The 1RM obtained was then used to determine the training intensity for the high-volume light-load and low-volume heavy-load strength training groups.

Strength Training Protocol

Both groups performed a similar strength training protocol using a percentage of their 1RM

measured in the voluntary strength testing protocol (see Table 1). The low-volume heavy-load strength training group performed a single bout of strength training of elbow flexors, completing 4 sets of 8 repetitions with 80% of their 1RM in time with an automatic metronome for a 3-second concentric phase and a 4-second eccentric phase [1]. The high-volume light-load strength training group also performed a single bout of strength training of the elbow flexors, completing 4 sets of 30 repetitions with 20% of their 1RM in time with a metronome for a 3-second concentric phase and a 4-second eccentric phase. Both groups had 3-minutes rest in between each set to allow adequate recovery. The control group did not perform any strength training.

Surface Electromyography

Surface electromyography (sEMG) activity was recorded from the biceps brachii muscle using bipolar Ag-AgCl electrodes [10]. The site of electrode placement was prepared by first removing any hair and dead skin cells and cleaned with alcohol swabs prior to the placement of electrodes. The right arm of each subject was measured by marking a point two-thirds of the distance between the acromion and the lateral epicondyle [1]. The anterior point of the biceps brachii in relation to this mark was then used as the site of electrode placement. Two electrodes were placed on the belly of the biceps brachii approximately 1-2cm apart from each other, with the ground electrode placed over the bony prominence of the lateral epicondyle. sEMG signals were amplified (× 1000), band pass filtered (20–1 kHz), digitised online at 2 kHz, recorded (1 s) and analysed using Power Lab 4/35 (AD Instruments, Bella Vista, Australia).

Tr anscranial Magnetic Stimulation

Single-pulse TMS was applied using a Magstim stimulator with a standard 70mm figure-of-eight coil placed over the cortical motor representation of the biceps brachii. The motor hotspot of the primary motor cortex was determined as the site that elicited the greatest and most consistent MEP. Active motor threshold (AMT) was then established as the lowest stimulus intensity that consistently produced MEPs greater than 200 μV over 5 of 10 delivered stimuli [14]. CSE was quantified by measuring the peak-to-peak amplitude of the single-pulse MEPs [1], while corticospinal inhibition was quantified by measuring the length of the silent period (SP) of the single-pulse MEP [15]. Short-interval cortical inhibition (SICI) was assessed using paired-pulse TMS, consisting of a sub-threshold conditioning stimulus delivered at 80% of the participant's AMT closely followed by a supra-threshold stimulus at 130% of the participant's AMT with an inter-stimulus interval of 3ms [16]. The MEP amplitude of the SICI waveform was then expressed as a percentage of the unconditioned single-pulse MEP amplitude.

Maximum Compound Muscle Action Potential

Direct muscle responses were obtained from the biceps brachii muscle by supramaximal electrical stimulation (pulse width, 200 μs) of the brachial plexus at Erbs point (DS7A; Digitimer, Hertfordshire, UK). The stimuli were delivered while the participant sat in an upright position, with the elbow at 90° elbow flexion holding 5 ± 1% of maximal rmsEMG. This low level of muscle activity was used to match the conditions under which TMS was delivered. An increase in current strength was applied to Erbs point, until there was no further increase observed in the amplitude of the sEMG response (M_{MAX}).

Data and Statistical Analysis

Data were screened for normality using Mauchly's test of sphericity, specifically looking at Greenhouse–Geisser and Huynh–Feldt corrections to test the equality of variance. In addition, the Shapiro–Wilk test was used to screen for normal distribution. To ensure that there were no significant differences between groups at baseline, a one-way analysis of variance (ANOVA) was used for all dependent variables [1RM elbow flexion, rmsEMG, CSE, SP and SICI]. The amplitude of

the baseline MEP was greater in the control group compared to the high-volume light-load strength training group (CI 2.06 to 19.1, $P = 0.01$). However, the meaningfulness of *P*-values for baseline differences do not serve a useful purpose, as they do not test a useful scientific hypothesis [17]. Therefore, a 3-group (control, LVHL-ST, and HVLL-ST) × 5 time points (pre, immediately post, 20 min post, 40 min post and 60 min post) multivariate analysis, appropriate for multiple dependent variables with repeated measure, was used to determine any difference between groups for the variables of CSE, SP and SICI. If significant main effects were found, univariate and post hoc testing (Bonferroni correction) was used to analyse the percentage change from baseline, comparing group interaction by time for each dependent variable. Effect size conventions for each dependent measure followed where significant multivariate effects were found. Effect sizes of 0.2, 0.5, and 0.8 were applied to determine small, moderate and large comparative effects (Cohens *d*). The level of significance was set at $P < 0.05$. Graphpad Prism version 7.00, GraphPad Software (La Jolla, California, USA) was used for the statistical analysis. Data are reported as mean ± 95% CI in text and as mean ± SD in figures and tables.

Results

Maximal compound wave (M MAX)

Figure 2 displays the mean (± SD) M_{MAX} amplitude at baseline, immediately post, 20 minutes post, 40 minutes post and 60 minutes post. There were no differences in M_{MAX} at baseline between groups ($F_{2, 24} = 1.86$, $P = 0.17$). There were no GROUP × TIME interactions for any time point reported ($F_{8, 96} = 0.98$; $P = 0.45$, see Figure 2).

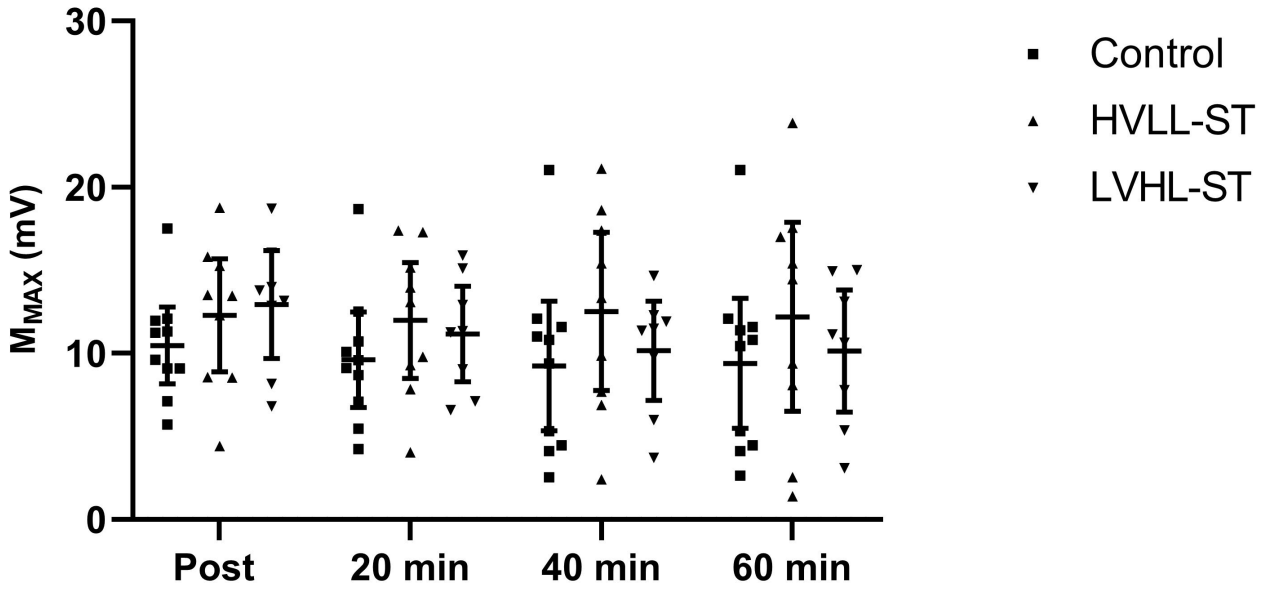

Figure 2. *Mean ± SD for M_{MAX} (mV) at pre, immediately post, 20 min post, 40 min and 60 min post training.*

Corticospinal Excitability

Table 2 and Figure 3 displays the mean (± SD) changes in MEP amplitude at baseline, immediately post, 20 minutes post, 40 minutes post and 60 minutes post. There was a significant difference in MEP amplitude at baseline between groups ($F_{2, 24} = 5.31$, $P = 0.01$). There was also a significant GROUP × TIME interaction ($F_{8, 96} = 3.2$; $P = 0.003$). Post-hoc testing showed CSE increased (73%)

immediately following a single bout of high-volume light-load strength training compared to the 2% reduction following low-volume heavy-load strength training (CI 12 to 138, P= 0.02, d = 1.54) and 0.2% reduction in the control group (CI -137 to -11, P= 0.023, d = 1.58). In a similar effect, 20-min post training, there was a 58% increase in CSE in the high-volume light-load strength training group compared to the 22% reduction following low-volume heavy-load strength training (CI 29 to 132, P= 0.003, d = 1.95); however, there was no difference between the high-volume light-load strength training and control groups despite a large comparative effect (CI -108 to 0.32, P = 0.052, d = 1.25). 40-min post strength training, CSE increased (31%) in the high-volume light-load strength training group; however this magnitude was not different to the 16% reduction following low-volume heavy-load strength training, despite a large comparative effect (CI -0.95 to 95, P = 0.055, d = 1.28) and to the 4.7% reduction in the control group (CI -102 to 30, P = 0.427, d = 0.62). Interestingly, at 60 min, CSE for the high-volume light-load strength training group was still facilitated (31%) compared to the 13% reduction following low-volume heavy-load strength training (CI 1.6 to 87, P = 0.041, d = 1.46). There was no difference between high-volume light-load strength training and the control group (CI-80 to 37, P = 0.717, d = 0.44).

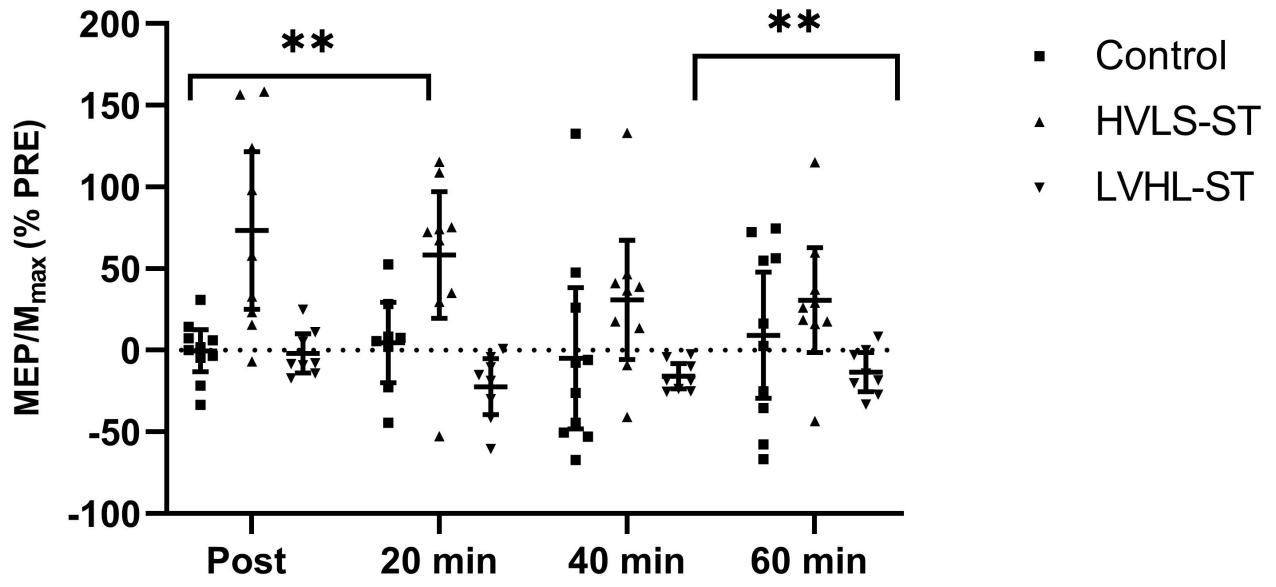

Figure 3. *Changes (mean ± SD) in corticospinal excitability for the trained biceps brachii. **Denotes a significant increase in corticospinal excitability from pre, post, 20 min post and 60 min post compared to the low-volume heavy-load strength training (LVHL-ST) and control groups (group by time effect).*

Corticospinal Inhibition

Figure 4 displays the mean (± SD) changes in SP at baseline, post, post 20, post 40 and post 60 min. There were no differences in SP at baseline between groups ($F_{2, 25}$ = 0.897, P = 0.42); however, there was a GROUP × TIME interaction ($F_{8, 100}$= 4.1; P= 0.04). Post-hoc testing showed immediately post-training, there were no differences in the reduction in SP between high-volume light-load strength training (8% reduction) compared low-volume heavy-load strength training (12% reduction) (CI -28 to 72, P = 0.561, d = 0.32) and compared to the 4% increase in the control group, despite a large comparative effect (CI -23 to 27, P = 0.99, d = 1.06). There was no difference between the low-volume heavy-load strength training and control groups, despite a large comparative effect (CI -24 to 73, P = 0.42, d = 1.46). 20-min post-training, there was an 8.5% reduction following high-volume light-load strength training, but this was not different to the 7% reduction following low-volume heavy-load strength training (CI-32 to 70, P = 0.69, d = 0.04) or the

control group (6% increase) (CI -17 to 32, P = 0.80, d = 1.42) or between the control group and low-volume heavy-load strength training (CI -24 to 77, P = 0.41, d = 1.36). 40-min post strength training, there was a 9.3% reduction following high-volume light-load strength training, but this was not different to the 6% reduction following low-volume heavy-load strength training (CI-39 to 65, P = 0.86, d = 0.29) or control group (9% increase) (CI -11 to 33, P = 0.46, d = 1.52) or between the control group and low-volume heavy-load strength training (CI -27 to 75, P = 0.48, d = 1.17). At 60-min post strength training, there was a 4.4% reduction following high-volume light-load strength training, but this was not different to the 7% reduction following low-volume heavy-load strength training (CI -35 to 81, P = 0.64, d = 0.26) or the 6% increase in the control group (CI -31 to 34, P = 0.99, d = 0.76). There were no difference between low-volume heavy-load strength training and the control group, despite a large comparative effect (CI -33 to 81, P = 0.58, d = 0.98).

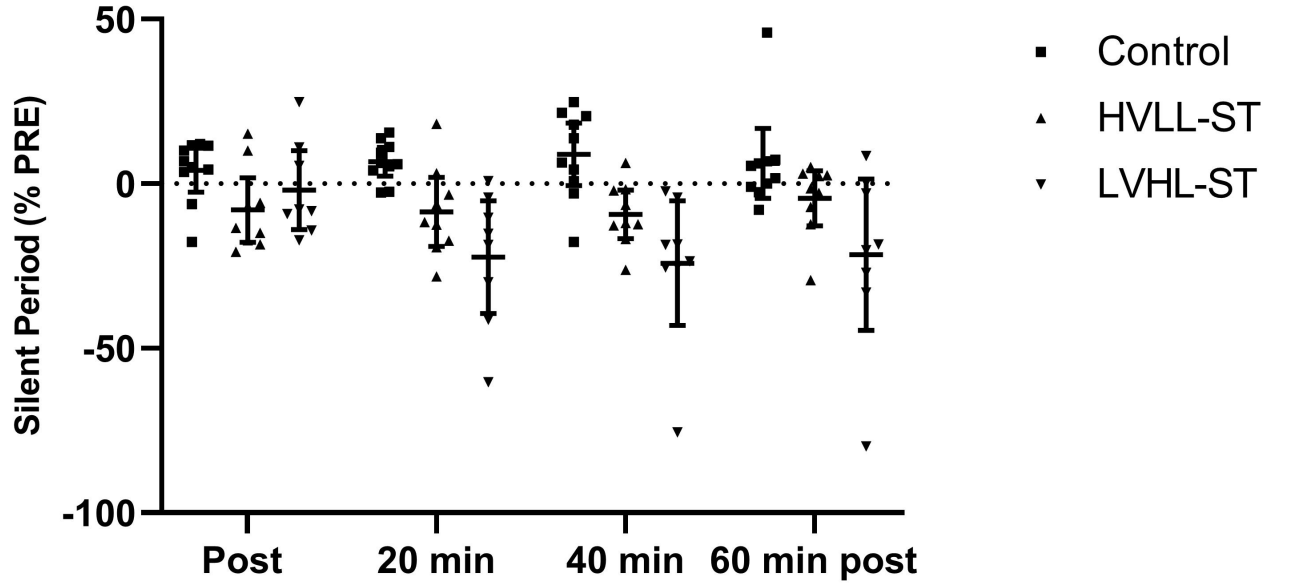

Figure 4. *Change (mean ± SD) in corticospinal silent period duration (ms) for the trained biceps brachii (mean ± SD).*

Short-interval Intracortical Inhibition

Figure 5 displays the mean (± SD) change in SICI ratio at baseline, post, post 20, post 40 and post 60 min. There were no differences in SICI at baseline between groups ($F_{2, 24}$= 0.4, P = 0.64) and there was no GROUP × TIME interaction ($F_{8, 92}$= 2.1; P= 0.203). SICI was released by 42% immediately following a low-volume heavy-load strength training, but this reduction was not different to the 24% release of SICI following high-volume light-load strength training (CI -44 to 97, P = 0.661, d = 0.26) and the 10% increase for the control group, despite a large effect size (CI -37 to 19, P = 0.792, d = 1.11). The magnitude of SICI release following high-volume light-load strength training was not different immediately post to control (CI -106 to 36, P = 0.472, d = 0.34). 20-min post-training, SICI was released by 28% following low-volume heavy-load strength training which was not different to the control group (CI -23 to 84, P = 0.357, d = 0.60), and SICI was released by 30% following high-volume light-load strength training which was not different to the 8% decrease in the control group (CI -104 to 59, P = 0.856, d = 0.33). There was no difference in the SICI release 20 min post-training between the low-volume heavy-load strength training and high-volume light-load strength training (CI -22 to 127, P =0.194, d = 0.04). 40-min post-training, SICI was released by 51% following low-volume heavy-load strength training which was not different to the 8% increase in the control group, despite a large effect (CI -26 to 58, P =0.687, d = 1.87) and to the 26% release in the high-volume light-load strength training group (CI -20 to 122, P

=0.189, *d* = 0.42). There were no differences in SICI between the high-volume light-load strength training and control groups (CI -109 to 39, *P* =0.513, *d* = 0.59). At 60-min post, SICI was released by 11% following low-volume heavy-load strength training and this was not different to the 5% increase in high-volume light-load strength training (CI -31 to 124, *P* =0.315, *d* = 0.28) and to the control group (13% increase) (CI -23 to 68, *P* =0.484, *d* = 0.47). There was no difference in SICI release 60 min post between high-volume light-load strength training and the control group (CI -103 to 56, *P* =0.809, *d* = 0.47).

	CSE (% MMAX)					**Silent Period (ms)**					**SICI (% of test response)**				
	Pre		***20***	***40***	***60***	**Pre**		***20***	***40***	***60***	**Pre**		***20***	***40***	***60***
Control	21.38 (±9.12)^	20.40 (±7.71)	22.20 (±6.13)	20.13 (±12.40)	21.53 (±8.79)	0.124 (±0.016)	0.136 (±0.014)	0.132 (±0.017)	0.144 (±0.012)	0.133 (±0.024)	35.62 (±16.95)	38.11 (±25.78)	36.81 (±15.00)	38.98 (±22.25)	35.13 (±15.72)
HVLL-ST	10.83 (±7.66)	17.46 (±11.24)^	19.28 (±14.27)^	21.33 (±24.50)	28.41 (±35.84)^	0.139 (±0.029)	0.126 (±0.025)	0.125 (±0.022)	0.124 (±0.022)	0.132 (±0.028)	44.97 (±22.24)	44.30 (±11.96)	48.22 (±14.81)	46.55 (±12.75)	42.31 (±6.32)
LVHL-ST	13.28 (±3.70)	12.93 (±3.879)	9.07 (±4.74)	8.92 (±5.08)	9.91 (±4.56)	0.132 (±0.031)	0.115 (±0.028)	0.121 (±0.030)	0.122 (±0.029)	0.123 (±0.037)	40.27 (±10.15)	54.67 (±19.42)	50.48 (±20.90)	50.69 (±15.07)	44.90 (±15.57)

Table 2. *Mean (±SD) corticospinal excitability (CSE), silent period, and short-interval cortical inhibition (SICI) for control and training groups up to 60 min following a single session of strength training .*†^* *AMT = active motor threshold. *CSE = Corticospinal excitability.*HVLL-ST = high-volume light-load strength training. *LVHL-ST = Low-volume heavy-load strength training. *M_{MAX} = maximal compound action potential. *ms = Milliseconds. * SICI = Short-interval cortical inhibition. †MEP and Silent Period measured at 130% of AMT. ^ significant group × time effect ($P < 0.05$).

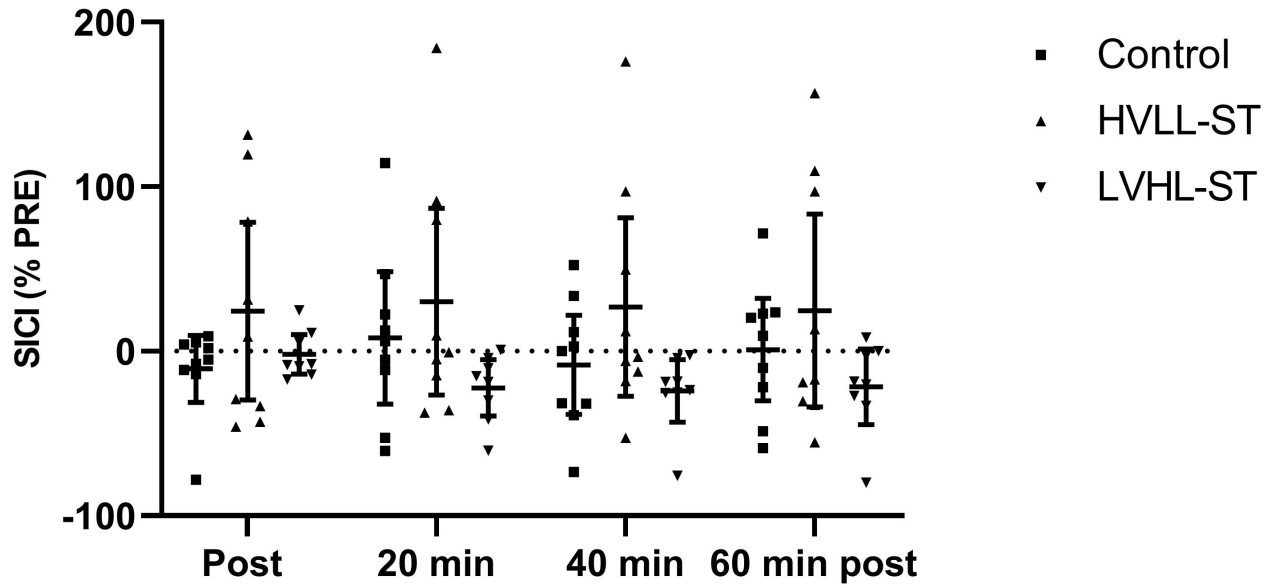

Figure 5. *Change (mean ± SD) in SICI for the trained biceps brachii.*

Discussion

This study mapped the early corticospinal responses following a single session of high-volume light-load strength training and low-volume heavy-load strength training of the elbow flexors. It was hypothesized that both modes of strength training would increase intracortical excitability, but by different mechanisms; specifically, that low-volume heavy-load strength training would reduce SP and SICI, whilst high-volume light-load strength training would increase CSE. The main findings showed that a single bout of high-volume light-load strength training led to a sustained increase in CSE when compared to low-volume heavy-load strength training, but both modes of training had no effect on intracortical inhibition.

The current study adds new knowledge by showing that the increase in CSE following strength training is volume dependent and is sustained over a 60-min period. The mechanism underlying this increase in CSE is not clear, but the data suggest that the training-load used (20% 1-RM) and the training-volume (120 reps) during high-volume light-load strength training did not induce fatigue (see Figure 3) and, given that there was no post MEP depression, the combination of completing the strength training exercise to external pacing coupled with a higher number of repetitions may be responsible for the increase in CSE. As with previous strength training studies, the high-volume light-load strength training group was required to complete their repetitions in time to a metronome [6,7]. Accordingly, the enhanced neural response to high-volume light-load strength training could be related to the higher auditory afferent input from the auditory cortex that is then synchronized with the activation of corticospinal cells during the strength training exercise. This persistent afferent input could lead to increased synaptic efficacy according to Hebbian principles [18]. However, a caveat to this interpretation is that the low-volume heavy-load strength training group were also required to pace their repetitions and we have recently shown that isometric strength training and listening to an audible cue has no effect on CSE following chronic strength training [1]. Interestingly, in the present study, some participants in the low-volume heavy-load strength training group were unable to keep time with the required repetition rate (3 s concentric, 4 s eccentric) due to the heavy-load. At a minimum, this may have limited the centrally challenging nature of the low-volume heavy-load strength training exercise and it may explain why we did not observe any acute modulation of CSE. Thus, it is likely that the acute adjustments in CSE following high-volume light-load strength training are due to the increase in training-volume, rather than training-load or the contraction intensity. Further, the sustained increase in CSE following high-volume light-load strength training could simply be an attempt by the nervous system to circumvent any muscular fatigue developed throughout the exercise trial. For example, strength training is typically sufficient to induce increases in lactate [19], which has been linked with increases in CSE [20]. Higher training-volume with lighter-loads generates greater increases in lactate than lower training-volumes [21], which may suggest that the high-volume nature and the increased time under tension in the high-volume light-load strength training, may have increased lactate levels, driving an increase in CSE.

Corticospinal inhibition is measured by recording the SP of the single-pulse TMS MEP and is facilitated by the neurotransmitter gamma-aminobutyric acid-$_B$ ($GABA_B$), whilst changes in SICI are exclusively examined using paired-pulse TMS and are primarily modulated via changes in receptor activity of the inhibitory neurotransmitter gamma-aminobutyric acid-$_A$ ($GABA_A$) [16]. A reduction in either of these inhibitory processes seems to be significant in the expression of muscular strength [11] and representative of a change in the balance of excitation within the inhibitory micro-circuits of the M1. The current results show that the indices of corticospinal inhibition (SP and SICI) remain unchanged following an acute training session of either high-volume light-load strength training or low-volume heavy-load strength training of the upper limb. Whilst this finding is in agreement with previous studies that have trained biceps brachii, whereby there were no changes in inhibition [4], it is in contrast to Mason et al. [7] who reported reduced SP in the biceps brachii following an acute bout light-load strength training or heavy-load strength training. However, the training-volume for the light-load strength training group in the study by Mason et al. [7] was less than that of the

current study.

Overall, we found no change in SICI following either type of strength training. Taken together with the results of the SP, the present findings suggest a single session of either high-volume light-load strength training or low-volume heavy-load strength training is not sufficient to reduce the efficacy of the $GABA_A$ and $GABA_B$ receptor–mediated inhibitory motor network. The finding that high-volume light-load strength training had no effect on the inhibitory circuits is consistent with our previous finding [7], but the fact that low-volume heavy-load strength training had no effect is in contrast. We hypothesized that low-volume heavy-load strength training would result in a sustained release in inhibition as previous research has shown that graded force production leads to a release in inhibition [22] and low-volume heavy-load strength training results in greater sensory feedback [23]. Further, sufficiently challenging the nervous system is important in maximizing the neural responses to strength training [6]. Therefore, there must be other characteristics of strength training, which remain unclear, that are necessary to reduce inhibition. In light of this, a recent systematic review concluded that the excitatory circuits of the primary motor cortex were the most susceptible to modulation following acute strength training. Thus, our findings are well aligned with this [8] and that of Colomer-Poveda et al. [2]

There are several limitations to this study which need to be considered when interpreting the main findings. First, there are limitations associated with the interpretation of MEPs as a result of single-pulse TMS, because MEP amplitude is influenced by the excitability of corticospinal and intracortical neurons that are activated by TMS; the synaptic efficacy between intracortical and corticospinal neurons; the synaptic efficacy between the CST and motor neuron pool; and the excitability of the motoneurons themselves [24]. TMS and the MEP alone are unable to reveal the site of adaptation to strength training. Additional techniques such as transmastoid stimulation and cortical voluntary activation measures would strengthen this study as they could confirm whether the change in MEP was due to increased excitability of corticospinal axons or a change in voluntary drive to the muscle as a result of central fatigue. Both these measures were not obtained. In order to detect any meaningful neural response to acute strength training, the task in which the TMS measures are obtained should be specific to the training-task employed [2].

Conclusion

High-volume light-load strength training increased CSE, whilst low-volume heavy-load strength training had no effect. The increases in CSE were not accompanied by decreases in corticospinal inhibition as determined by the SP and SICI ratio. These results suggest that only high-volume light-load strength training increases the excitability of corticospinal neurons. Although it could be considered that a greater number of strength training sessions may be required to observe the difference in the excitability of the intrinsic inhibitory motor network, there could also be other mechanisms at play that warrant investigation. For example, changes in cortical voluntary activation, using TMS (TMS_{VA}), may provide greater insight into mechanism associated with neural drive to trained muscles and investigating the changes in agonist-antagonist TMS responses, are likely to provide additional or different mechanisms that might be differentially modulated following different load-volumes of strength training. Thus, additional studies, examining different neural phenomena are required to determine the dose-response relationship following both high-volume light-load strength training and low-volume heavy-load strength training on the corticospinal responses to strength training.

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Declaration of Competing Interest

None of the authors have potential conflicts of interest to be disclosed.

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