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Brain activation associated with low- and high-intensity concentric versus eccentric isokinetic contractions of the biceps brachii: An fNIRS study

Wei-Peng Teo[1] | **Clara Xinru Tan**[1] | **Alicia M. Goodwill**[1] | **Saqif Mohammad**[1] | **Yi-Xuan Ang**[1] | **Christopher Latella**[2,3]

[1]Physical Education and Sport Science Academic Group, National Institute of Education, Nanyang Technological University, Singapore, Singapore

[2]Neurophysiology Research Laboratory, School of Medical and Health Sciences, Edith Cowan University, Perth, Western Australia, Australia

[3]School of Medical and Health Sciences, Centre for Human Performance, Edith Cowan University, Perth, Western Australia, Australia

Correspondence
Wei-Peng Teo, Physical Education and Sport Science Academic Group, National Institute of Education, Nanyang Technological University, 1 Nanyang Walk, Singapore, Singapore 637616.
Email: weipeng.teo@nie.edu.sg

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Abstract

Studies have shown that neural responses following concentric (CON) and eccentric (ECC) muscle contractions are different, which suggests differences in motor control associated with CON and ECC contractions. This study aims to determine brain activation of the left primary motor cortex (M1) and left and right dorsolateral prefrontal cortices (DLPFCs) during ECC and CON of the right bicep brachii (BB) muscle at low- and high-contraction intensities. Eighteen young adults (13M/5F, 21–35 years) were recruited to participate in one familiarization and two testing sessions in a randomized crossover design. During each testing session, participants performed either ECC or CON contractions of the BB (3 sets × 8 reps) at low- (25% of maximum ECC/CON, 45°/s) and high-intensity (75% of maximum ECC/CON, 45°/s) on an isokinetic dynamometer. Eleven-channel functional near-infrared spectroscopy was used to measure changes in oxyhemoglobin (O_2Hb) from the left M1, and left and right DLPFC during ECC and CON contractions. Maximum torque for ECC was higher than CON (43.3 ± 14.1 vs. 46.2 ± 15.7 N m, $p = 0.025$); however, no differences in O_2Hb were observed between contraction types at low or high intensities in measured brain regions. High-intensity ECC and CON contractions resulted in greater increases in O_2Hb of M1 and bilateral DLPFC compared to low-intensity ECC and CON contractions ($p = 0.014$). Our findings suggest no differences in O_2Hb responses between contraction types at high and low intensities. High-contraction intensities resulted in greater brain activation of the M1 and bilateral DLPFC, which may have implications for neurorehabilitation to increase central adaptations from exercise.

KEYWORDS

functional near-infrared spectroscopy, motor cortex, muscle contraction, prefrontal cortex

Wei-Peng Teo and Clara Xinru Tan contributed equally to this study.

1 | INTRODUCTION

Successful performance of daily and sporting activities is governed by an individual's ability to regulate muscle forces produced by concentric (CON, muscle shortening) and eccentric (ECC, muscle lengthening) muscle contractions. For example, the ability to produce greater ECC and CON strength and power is critical for plyometric and explosive movements often seen in sprinting[1] and countermovement jumps[2] and may reduce risk of injuries in athletic populations.[3,4] In the general population, having greater ability to produce CON and ECC force and power in the lower limbs is associated with reduced risk of falls in older adults[5] and is highly involved in daily activities such as walking downhill or down a flight of stairs,[6] maintaining body posture and positioning,[7] and stopping oneself from falling[8] or performing a quick change in direction.[9] Similarly in the upper limb, having adequate CON and ECC strength is necessary for performing activities of daily living such as using the arms to sit into a chair,[10] placing a heavy object down,[10] and control or using the arms to arrest a fall.[11]

From a central and peripheral perspective, CON and ECC muscle contractions elicit different acute responses and long-term adaptations in both the central nervous system (CNS) and muscle.[12,13] At the peripheral level, studies have reported greater force output[14] at a given level of muscle electrical activity[15,16] associated with maximal ECC compared to CON muscle contractions. Further, ECC muscle contractions are more likely to induce prolonged muscle damage and delayed onset of muscle soreness (DOMS) compared to CON contractions in untrained individuals.[17,18] However, when exposed to repetitive ECC training or exercise, greater neuromuscular adaptations (i.e., strength gains, muscle volume, and neural activation) may be observed, compared to CON training.[13,19] Other studies have also shown that ECC exercises incur less metabolic and cardiorespiratory costs when similar workloads to CON exercise are performed,[20,21] which raises the possibility for ECC exercise as a suitable strategy for patients with chronic illnesses.[22,23]

At the central level, several differences in brain activation have been documented following acute bouts of ECC exercise. Using transcranial magnetic stimulation (TMS), we previously demonstrated a longer-lasting (up to 1 h post-exercise) reduction in cortical inhibition (i.e., long-interval intracortical inhibition) of the contralateral primary motor cortex (M1) following maximal ECC contractions of the right BB muscle compared to CON contractions.[24] Similarly, other studies have reported acute changes in excitatory[25,26] and inhibitory[27,28] corticospinal pathways following ECC contractions. Further, we showed that a single session of ECC strength training (three sets of 10 ECC elbow flexor exercise at 75% of 1-repetition maximum [1RM]) of the right BB resulted in acute modulation of corticospinal excitability and intracortical facilitation in left non-trained BB compared to CON training.[29]

While TMS studies have provided evidence for excitability changes following ECC exercises/training, the neural control mechanisms, particularly at the cortical level, during ECC contractions itself has remained largely unexplored. A review published by Perrey[30] in 2018 indicated seven neuroimaging studies (two electroencephalography [EEG] studies, four functional magnetic resonance imaging [fMRI] studies, and one study using a combination of EEG and fMRI), showed a generally higher pattern of brain activation in motor-related brain regions (i.e., M1, supplementary and premotor cortices) and prefrontal cortex (PFC, involved in cognition and regulating corticomotor drive) associated with ECC compared to CON movements. However, most of these studies utilize small movements of the fingers or wrists at relatively low force outputs, which may not be truly representative of other tasks or muscle groups. Only two studies by Fang and colleagues[31,32] used a maximal BB contraction protocol and showed greater amplitude and area dimension of EEG movement-related cortical potential. To the best of our knowledge, only a handful of neuroimaging studies using EEG and fMRI have examined the central control of ECC muscle contractions during the contraction itself. However, a key limitation of these techniques is the inability to limit head movements particularly using larger muscle groups at higher contraction intensities.

To overcome the inherent limitation of current neuroimaging modalities such as EEG or fMRI, we used functional near-infrared spectroscopy (fNIRS) in this study as a neuroimaging tool during bouts of low- and high-intensity CON and ECC muscle contractions of the BB. fNIRS is a relatively new neuroimaging technique that utilizes NIR light to measure changes in cerebral hemodynamic responses in cortical brain regions.[33] Using an array of NIR light transmitter and detector optodes, NIR light emitted at specific wavelengths (650–900 nm) on the electromagnetic spectrum passes through most biological tissue, but is absorbed predominantly by oxy- and deoxyhemoglobin (O_2Hb and HHb) within the blood.[34,35] The absorption of light photons emitted from the transmitters to the detectors therefore provides an indication of the concentration of O_2Hb and HHb. Previous studies have shown that fNIRS measures to be highly reliable and valid when compared to fMRI measures.[36–38] Importantly, fNIRS is less sensitive to head movements and is portable, making it an ideal neuroimaging modality to understanding movement control.[39]

Therefore, the aim of this study is to determine the differences in contralateral M1 activation, as measured by

fNIRS, between low- and high-intensity CON and ECC contractions of the BB muscle. Additionally, based on evidence suggesting that ECC contractions may be underpinned by different motor control strategies relative to CON contractions, we seek to determine if contraction-specific activation of the bilateral dorsolateral PFC (DLPFC) is present, as a function of high and low CON and ECC contraction of the BB muscle. We hypothesize that (1) ECC BB contractions and (2) contractions at higher intensities will result in greater activation of the contralateral M1 and bilateral DLPFC. The findings from this study will provide further evidence for contraction- and intensity-specific neural control of the BB muscle, which may have implications for neurorehabilitation strategies targeting an increase in brain activation following brain injury or stroke.

2 | METHODS

2.1 | Participants

Eighteen healthy participants (13 males/5 females, 21–35 years) were recruited to participate in this study. To be included in this study, all participants had to be (1) recreationally strength trained (i.e., perform resistance training at least twice a week in the last 12 months); (2) right-handed; (3) free from upper limb injury in the past 6 months; and (4) no formal diagnosis of any neurological condition or implant in the head or neck region. Additionally, female participants that were pregnant or suspected to be pregnant were excluded. Following an explanation of the risk, benefits, and study procedure, all participants gave written informed consent prior to the commencement of the study. This study was approved by the NTU Institutional Review Board (IRB-2022-290), and all procedures were in accordance with the Declaration of Helsinki.

2.2 | Procedure

All participants recruited in this study underwent three sessions (one familiarization session and two testing sessions in a counter-balanced randomized order) at the Motor Behavior Laboratory, National Institute of Education, Nanyang Technological University. Each session lasted about 1 h and were separated by at least 7 days of rest in between. All testing sessions for each participant were scheduled at the same time-of-day to avoid any circadian effects in neuromuscular performance,[40] and were instructed to maintain their usual daily physical activity, diet, and sleep schedules.

During the familiarization session, all participants were briefed on the conduct of the study and were asked to complete the Edinburgh Handedness Inventory[41] for which all participants included were deemed as right-handed. Next, participants were setup on the Biodex System 4 isokinetic dynamometer (Biodex Medical Systems Inc.) where they were instructed to perform three maximal CON (CON_{max}) and three maximal ECC (ECC_{max}) contractions in a random order with at least 3 min rest between each contraction (refer to subsequent subsections for details). Despite only including participants that were recreationally strength-trained, the likelihood of acquiring DOMS may still be high. Therefore, following the maximal ECC/CON testing, participants were instructed to perform three sets of 10 repetitions of ECC BB contractions at 50% of ECC_{max} on the isokinetic dynamometer to not only familiarize themselves with the contraction protocol, but to elicit low level muscle damage and DOMS prior to the testing sessions, such that any subsequent ECC or CON testing sessions will result in a significantly attenuated DOMS response, known as the repeated bout effect.[42] Following the familiarization session, participants returned to the Motor Behavior Laboratory for two more testing sessions where they were randomly allocated to perform either ECC or CON contractions at three sets 8 repetitions of low- (25% ECC_{max}/CON_{max}) and three sets of 8 repetitions of high intensity (75% ECC_{max}/CON_{max}) for the second testing session, and the other contraction type in the third testing session (Figure 1). Brain activation during CON and ECC contractions, as measured by fNIRS, was recorded only in Sessions 2 and 3. Prior to all testing sessions, a 10-point likert scale was used to ascertain the level of DOMS for each participant to ensure that participants were not experiencing muscle aches and pains associated with DOMS.

2.3 | Isokinetic dynamometry

To determine the maximum voluntary isokinetic torque of the BB muscle during ECC and CON contractions, a Biodex System 4 (Biodex Medical Systems, Inc.) isokinetic dynamometer was used. During each testing session, participants were setup on the isokinetic dynamometer and were instructed to perform three sets of 8 repetitions of low-intensity (25% of CON_{max} and ECC_{max}) and three sets of 8 repetitions of high-intensity (75% of CON_{max} and ECC_{max}) muscle contractions in a randomized order. To achieve the stipulated torque output, force biofeedback was provided on a screen to ensure that all participants were able to achieve the 25% and 75% CON_{max} and ECC_{max}. Prior to performing the ECC or CON BB contractions, participants were instructed to sit upright and were strapped in at the waist and shoulders to prevent any unnecessary movement of the torso during testing. The right arm was placed on an arm rest just under the elbows at chest height that allowed the arm to extend fully in an unobstructed manner. To perform the CON contraction,

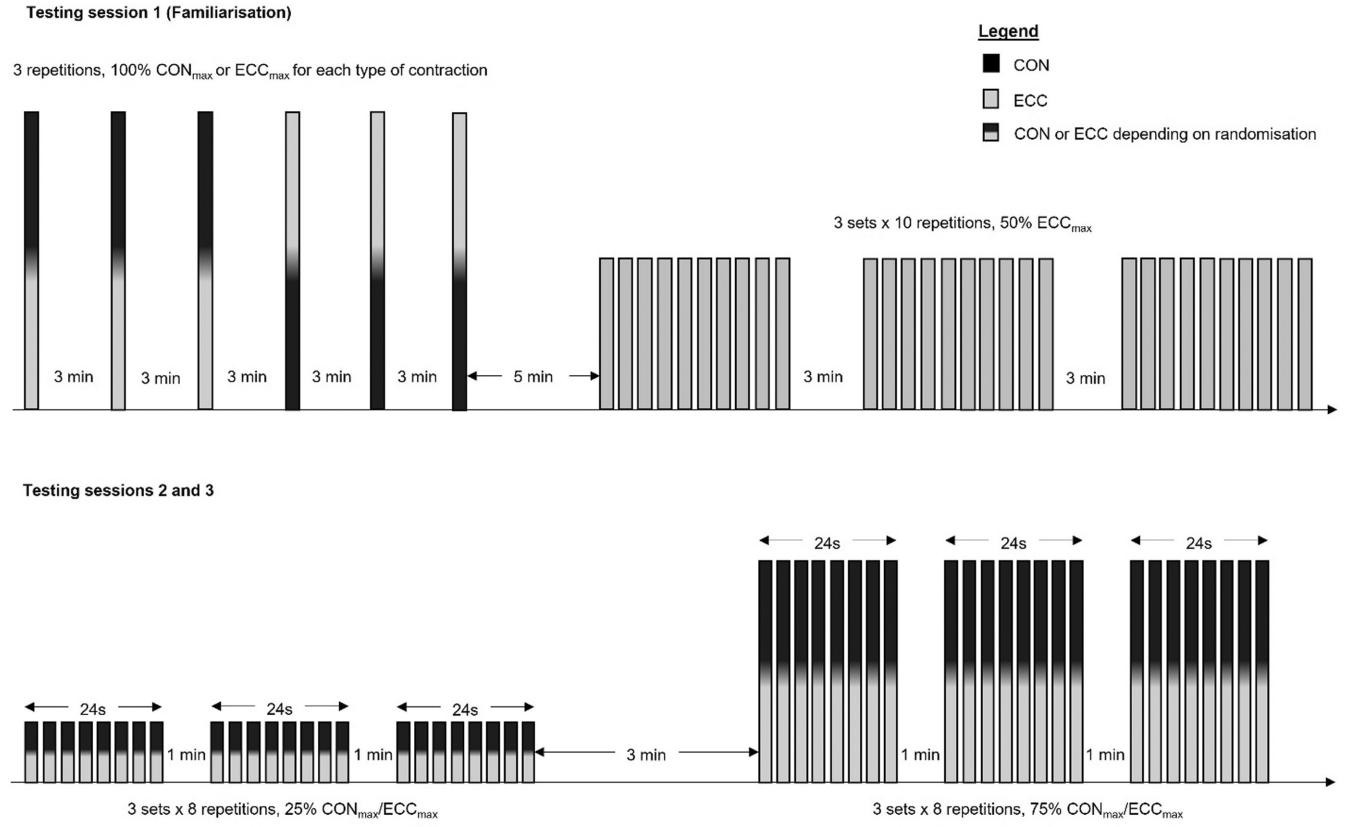


FIGURE 1 A schematic diagram of the flow for each testing session. For testing Sessions 2 and 3, participants either perform CON or ECC contractions, respectively for each session. Further, all participants would perform either three sets of 8 repetitions of low or high contractions in the order that was randomly assigned to them. CON, concentric; ECC, eccentric.

participants started with their arms fully extended at the 180° position and were instructed to move the dynamometer arm to the 90° position at a programmed speed of 45°/s. The experimenter would then physically bring the dynamometer arm back to the starting 180° position at a speed of 90°/s (total duration for each repetition = 3 s) to ensure that participants were only contracting concentrically. Altogether, each participant performed three sets of 8 repetitions with 30 s rest between sets, and a 3 min rest between low- and high-intensity contractions. The same process was applied to ECC contractions except for the starting position of the arm, whereby the arm was positioned at 90° moving toward 180° at 45°/s during ECC contractions. The experimenter would then manually move the dynamometer arm back to the 90° starting position at 90°/s to begin the next repetition.

2.4 | Functional near-infrared spectroscopy

Based on the fNIRS Optode Location Decider (fOLD) toolbox,[43] we determined that our optode placement corresponded to the left and right DLPFC and the left M1 (Figure 2). The Brite24 (Artinis Medical Systems) was used to measure change in oxyhemoglobin (O_2Hb) from the contralateral left M1 (7 channels) and bilateral DLPFC regions (4 channels, 2 left DLPFC and 2 right DLPFC), making up a total of 11 channels (7 transmitters and 5 receivers, see Figure 2A). All fNIRS data were acquired with Oxysoft (Version 3.2.72, Artinis Medical Systems, Elst; sampling rate—10 Hz), and exported into MATLAB (R2021a, The MathWorks Inc.) for data preprocessing with HOMER3, a MATLAB-based toolbox.[44]

All raw fNIRS data were preprocessed in HOMER3 using the following pipeline:

1. Conversion of light intensity to optode density—(hmR_Intensity2OD)
2. Identifying motion artifacts—(hmrR_MotionArtifact; tMotion 0.5, tMask 1.0, STDEVthresh 50.0, AMPthresh 5.0)
3. Motion artifact rejection—(hmrR_StimRejection; tRange −5.0 10.0)
4. Bandpass filter—(hmrR_BandpassFilt; hpf 0.000, lpf 0.1)
5. Conversion of optode density to concentration—(hmrR_OD2Conc; ppf 6.0 6.0 6.0)

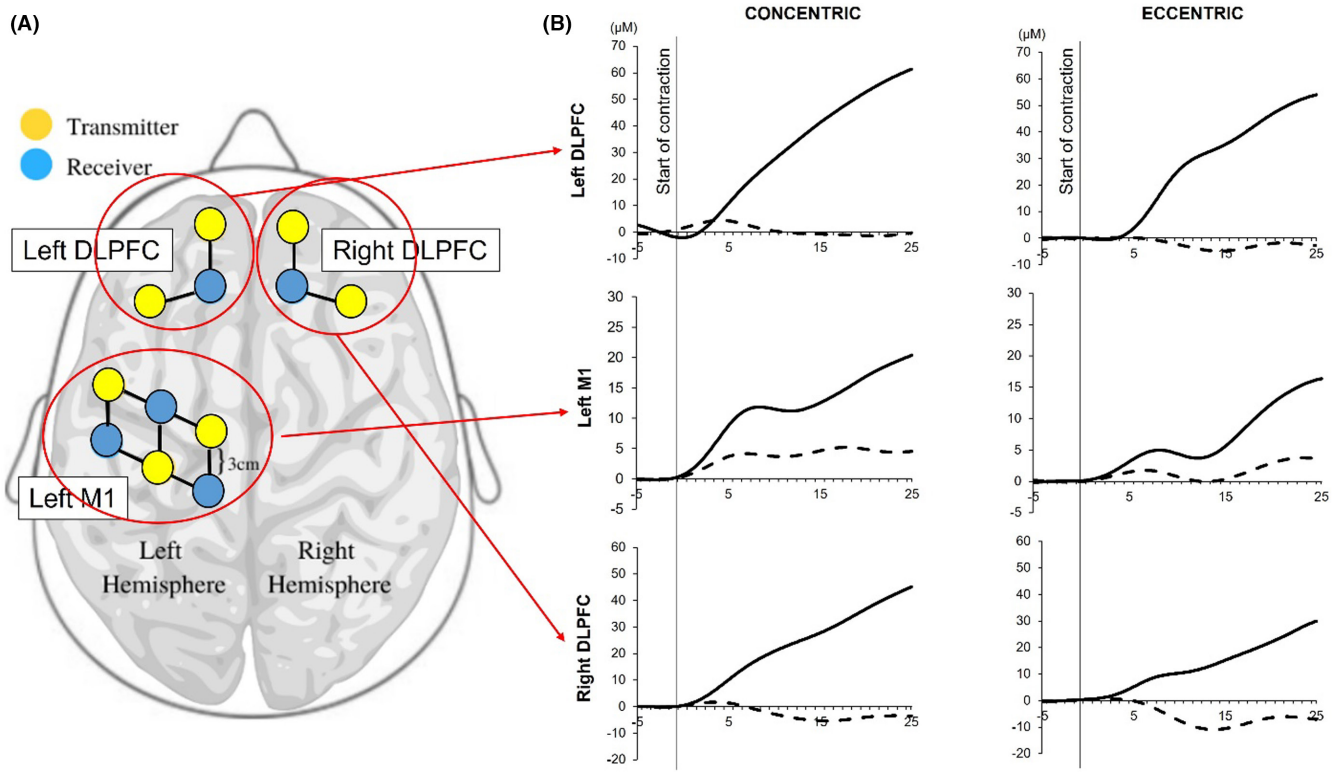
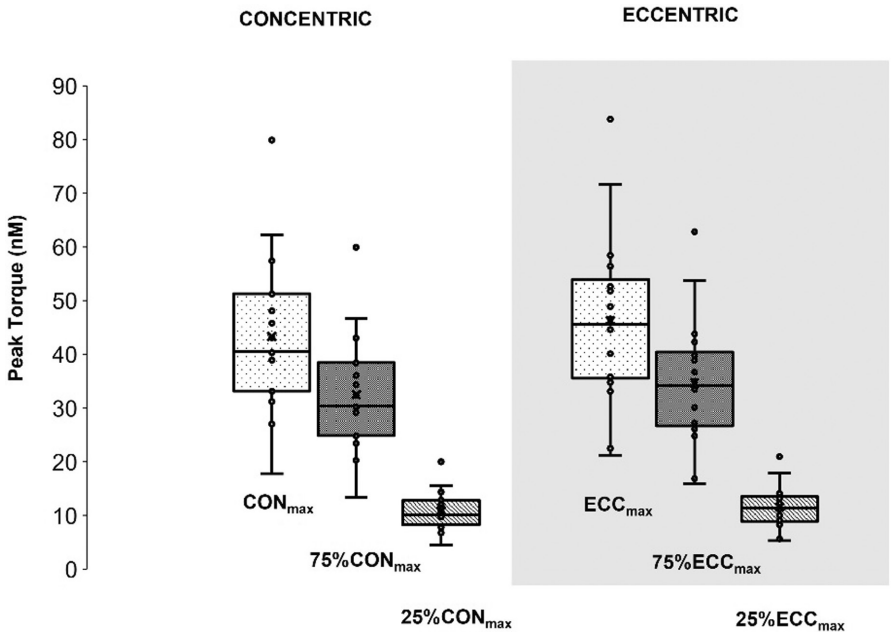


FIGURE 2 An illustration of the (A) optode locations and (B) an example of one participant's global average O_2Hb concentration change in all channels within the specific ROI during high- (solid black line) and low (dashed black line) -intensity CON and ECC contractions. CON, concentric; DLPFC, dorsolateral prefrontal cortices; ECC, eccentric.

FIGURE 3 An illustration of torque profiles under maximum 75% and 25% contraction intensity for both CON and ECC contractions. ECC_{max} was shown to be significantly greater torque production than CON_{max} ($p = 0.025$), which resulted in a significantly greater 75% and 25% contraction intensity in ECC contractions. Boxplot indicates the mean (black horizontal line), standard deviation (SD), and outliers in data points within the group. CON, concentric; ECC, eccentric.



6. Block averaging across the blocks—(hmrR_BlockAvg; trange −5.0 25.0)

Following preprocessing of fNIRS data across all channels, the channels associated with the left and right DLPFC and left M1 (see Figure 2) were averaged to provide a global value for each ROI (see Figure 2B).

2.5 | Data and statistical analyses

All fNIRS data were checked for normal distribution and heteroskedasticity using Q–Q plots, and histograms prior to analysis. To determine differences in torque profiles for CON and ECC contractions (i.e., CON_{max}/ECC_{max},

75%CON_{max}/ECC_{max} and 25%CON_{max}/ECC_{max}), paired sample *t*-tests were used. To determine differences between brain activation during CON and ECC at high (75%CON_{max}/ECC_{max}) and low (25%CON_{max}/ECC_{max}) contraction intensities, a 2×2×3 analysis of variance (ANOVA) was used to identify differences between muscle contraction type (CON vs. ECC), contraction intensity (low vs. high), and brain ROI (left DLPFC vs. right DLPFC vs. M1). A Bonferroni post-hoc *t*-test for multiple comparisons was applied to determine differences in O_2Hb between ROIs. All statistical analyses were performed using Statistical Package for Social Sciences (SPSS version 26, IBM) with an alpha level of $p < 0.05$ set as the level to determine statistical significance.

3 | RESULTS

3.1 | Torque

Torque profiles for all participants are presented in Figure 3. Overall, paired-sample *t*-test showed a significant difference between CON and ECC maximum torque, indicating a greater maximum torque during ECC compared to CON contractions (43.3 ± 14.1 vs. 46.2 ± 15.7 N m, $t[17] = 2.5$, $p = 0.025$). This resulted in a significantly greater 75%ECC_{max} (CON vs. ECC: 32.5 ± 10.6 vs. 34.7 ± 11.8 N m, $t[17] = 2.5$, $p = 0.025$) and 25%ECC_{max} (CON vs. ECC: 10.8 ± 3.5 vs. 11.6 ± 3.9 N m, $t[17] = 2.5$, $p = 0.025$), compared to the respective CON contraction intensities.

3.2 | Brain activity

Figure 4 shows the change in O_2Hb in the left M1, left DLPFC, and right DLPFC during high and low CON and ECC contractions. Mauchly's test indicated that the assumption of sphericity had been violated for the main effect of brain ROI ($X^2(2) = 15.502$, $p < 0.001$). Therefore, degrees of freedom were corrected using Greenhouse–Geisser estimates of sphericity ($\varepsilon = 0.599$). There was no significant main effect of muscle contraction type ($F_{(1,15)} = 0.894$, $p = 0.359$) or for brain ROI ($F_{(1.198,17.969)} = 2.897$, $p = 0.101$). However, a significant main effect of contraction intensity ($F_{(1,15)} = 28.561$, $p < 0.001$) was observed and we further observed a significant interaction effect between contraction intensity and brain ROI ($F_{(2,30)} = 7.769$, $p < 0.001$).

Post-hoc analyses revealed that for ECC contractions, a significantly greater increase in O_2Hb was observed in the high-intensity compared to low-intensity contractions in the left DLPFC (53.79 ± 49.76 vs. 7.92 ± 16.33 μM, $p < 0.001$), left M1 (53.79 ± 49.76 vs. 7.92 ± 16.33 μM, $p < 0.001$), and right DLPFC (40.82 ± 36.91 vs. 4.85 ± 15.77 μM, $p < 0.001$). Similarly

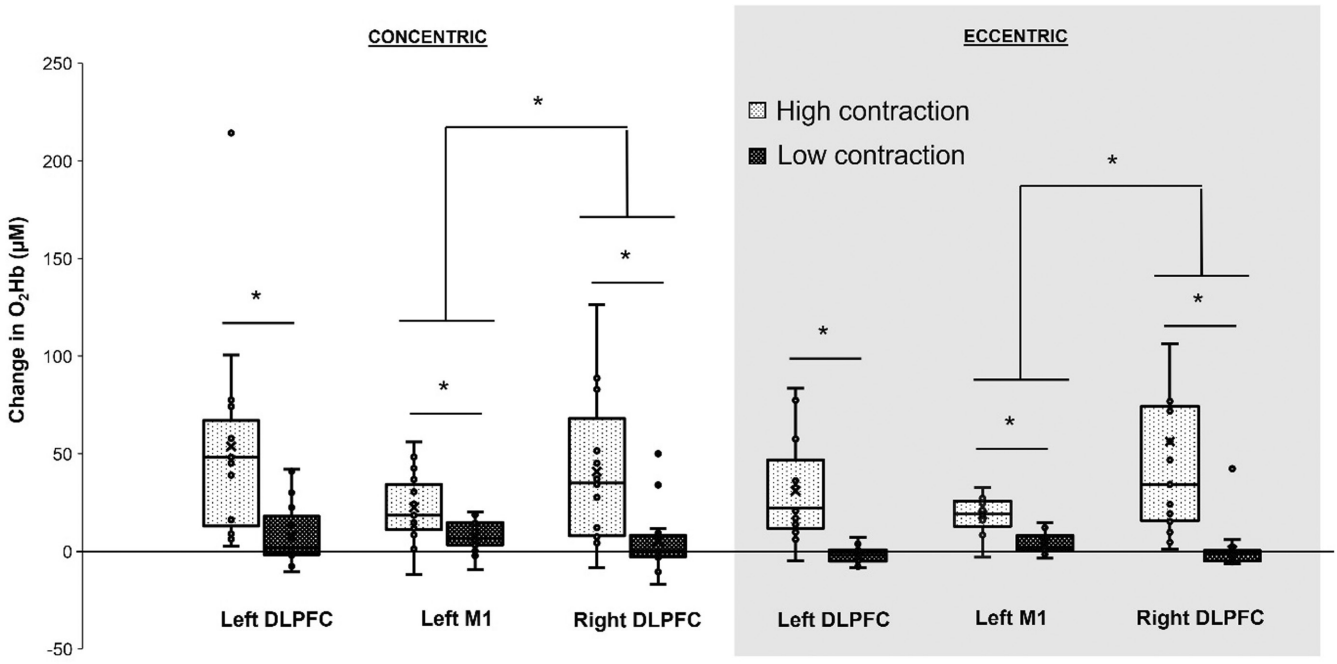


FIGURE 4 A diagram showing the changes in brain activity of the left DLPFC, left M1, and right DLPFC during CON and ECC at high and low contraction intensities. Higher contraction intensity elicits significantly greater increase in O_2Hb in all three brain regions regardless of contraction type. Furthermore, right DLPFC was significantly greater and lower, compared to the left M1, during high- and low-intensity contractions, respectively, for both CON and ECC contractions. Boxplot indicates the mean (black horizontal line), standard deviation (SD), and outliers in data points within the group. CON, concentric; DLPFC, dorsolateral prefrontal cortices; ECC, eccentric.

for CON contractions, we observed a greater increase in O_2Hb following high-intensity compared to low-intensity contractions in the left DLPFC (32.67 ± 24.25 vs. $0.57 \pm 4.00\,\mu M$, $p < 0.001$), left M1 (20.80 ± 8.71 vs. $6.65 \pm 4.81\,\mu M$, $p < 0.001$), and right DLPFC (57.23 ± 76.59 vs. $3.23 \pm 10.33\,\mu M$, $p < 0.001$).

Additionally, we observed a significantly greater increase in O_2Hb in right DLPFC activity with high-intensity contractions compared to the left M1 for both CON (right DLPFC vs. left M1: 40.82 ± 36.91 vs. $22.49 \pm 17.62\,\mu M$, $p < 0.05$) and ECC conditions (right DLPFC vs. left M1: 57.59 ± 76.59 vs. $20.80 \pm 8.71\,\mu M$, $p < 0.05$). We further observed a significantly lower O_2Hb in the right DLPFC with low-intensity contractions compared to the left M1 for both CON (right DLPFC vs. left M1: 4.85 ± 15.77 vs. $7.64 \pm 8.04\,\mu M$, $p < 0.05$) and ECC conditions (right DLPFC vs. left M1: 4.85 ± 15.77 vs. $7.64 \pm 8.04\,\mu M$, $p < 0.05$).

4 | DISCUSSION

In this study, we aimed to determine the differences in brain activation in the contralateral left M1 and left and right DLPFC during CON and ECC isokinetic muscle contractions of the BB at high and low contraction intensities in healthy adults. Overall, our results showed two key findings: (1) there were no effects of contraction type (CON or ECC) on hemodynamic responses in the contralateral M1 or the left and right DLPFC at either low or high contraction intensities; and (2) high isokinetic contraction intensities elicited greater hemodynamic responses compared with lower intensity irrespective of contraction type, which was greater in the right DLPFC compared with contralateral left M1. These findings partially supported our hypotheses that high-intensity contractions led to greater activity in all brain ROIs, but ECC contractions did not result in a greater level of brain activation compared to CON contractions.

4.1 | No effect of contraction type (CON and ECC) on cerebral hemodynamic responses

Previous studies have demonstrated several differences in neurophysiological and neuromuscular performance,[19,28,29] metabolic demands,[20,21] and motor control strategies[45,46] associated with CON and ECC muscle contractions. Namely, ECC contractions have been shown to generate greater force outputs with a lower metabolic cost.[20,21] Additionally, noninvasive brain stimulation techniques such as TMS have consistently reported different neuromodulatory effects on corticospinal excitability and inhibition following CON and ECC exercises.[24,26,27,29] In agreement with previous findings, our study showed that maximum torque produced during ECC contractions was significantly higher compared to CON contractions.[46–48] However, despite showing increased torque during ECC contractions, our results showed no significant difference in cerebral hemodynamic response in all brain ROIs between CON and ECC contractions at high or low isokinetic contraction intensities. This finding disagreed with our first hypothesis that ECC would elicit greater levels of brain activity from the M1 and bilateral DLPFC.

A review by Perrey[30] in 2018 previously highlighted several findings from seven neuroimaging (2× EEG and 5× fMRI) papers that reported; (1) longer and greater EEG-related amplitudes for ECC movement during the preparation and execution phases; (2) greater and wider area of brain activation with EEG and fMRI; and (3) weaker functional connectivity between M1 and other brain regions such as the PFC. Among the seven studies reported, two EEG studies by Fang and colleagues[31,32] reported an increase in motor-related cortical potentials and reduced EMG amplitude during ECC compared to CON contractions of the BB. While the EEG studies demonstrated a contraction-specific effect using the BB muscle, it should be noted that the temporal resolution of EEG is much higher than fMRI or fNIRS and that EEG measures neuroelectrical activity rather than cerebral hemodynamic responses. Additionally, the five other fMRI studies used muscles of the hand (i.e., first dorsal interosseous, flexor and abductor pollicis brevis muscles), motor imagery and wrist flexor/extensor muscles that arguably require greater precision and cognitive-motor control.[49–53] If we compared our study that used a larger muscle (BB) to those presented in Perrey's review, it is likely that the level of movement complexity and cognitive demand between ECC and CON contractions of the BB were not great enough to demonstrate any contraction-specific differences. It should also be noted that small, distal, hand/finger muscles used in previous neuroimaging studies may have had contributions from different descending tracts. It is relatively accepted that distal musculature relies more heavily on corticospinal tract,[54,55] while more proximal (and trunk) musculature may rely more heavily on reticulospinal tract (and other brainstem pathways).[56] This may help explain the lack of difference in M1, as the reticulospinal tract has input fibers from premotor areas,[57] which were not measured within the ROI's used. This potential difference in descending tracts could also explain why the results differed from those other neuroimaging studies.

An alternative interpretation of our findings may suggest that for the same level of hemodynamic response observed in the M1 and DLPFC regions, ECC contractions were able to elicit greater torque levels compared to CON

contractions. This could potentially indicate some level of neural efficiency that is associated with ECC contractions.[45] Indeed, studies have reported greater ECC torque concomitant with similar or less voluntary muscle activity, suggesting a higher level of neural and mechanical efficiency for ECC compared to CON contractions.[47,58] Thus, it may be that our current fNIRS data are reflective of that neural and mechanical efficiency associated with ECC contractions (i.e., similar brain activation but greater torque with ECC compared to CON contraction). However, it should be noted that as fNIRS is only reflective of metabolic demands of cortical activation, and it is unlikely that subcortical and spinal inhibitory control differences between ECC and CON[46,48] contractions were well captured with fNIRS.

4.2 | High CON and ECC contraction intensity elicits a greater increase in cerebral hemodynamic response of the left and right DLPFC, and left M1

In agreement with our second hypothesis, we showed that higher contraction intensity, regardless of contraction type, led to greater increase in O_2Hb of the left M1 and bilateral DLPFC. Interestingly, this difference was also greater in the right DLPFC compared with the left M1. Previous studies have shown that exercising at slower movement velocities, thereby increasing the amount of work done, elicits a greater increase in cerebral hemodynamic response within the M1.[59,60] For instance, a study by Formenti et al.[59] showed greater PFC activation during slower CON and ECC contractions (5 s/5 s, CON/ECC) of the knee extensors compared to faster contraction speeds (1 s/1 s, CON/ECC). Another study by Borot et al.[60] showed differences in hemodynamic response of the left M1 between ECC and CON contractions of the BB using a similar isokinetic dynamometer protocol at various angular velocities (60°/s vs. 30°/s). The authors reported higher contralateral M1 activation during ECC compared with CON contractions at 30°/s but not at 60°/s contraction speeds. We propose that our results showing greater increase in O_2Hb observed at high compared to low contraction intensity at the contralateral M1 and bilateral DLPFC would be logical. Considering that we have matched movement velocities at both contraction intensities, a higher contraction intensity (75% of peak ECC and CON torque) would potentially elicit a higher level of peripheral fatigue and work done. This has been further supported by other studies indicating a somewhat linear load/force-brain relationship, with greater level of force production resulting in an increased level of brain activation (assess using fNIRS) in simple handgrip tasks[61,62] and even during multi-joint movements such as a barbell squat.[63]

However, in contrast to the studies by Formenti et al.[59] and Borot et al.,[60] we did not observe any contraction-specific effects on hemodynamic response in the M1 at low and high contraction intensities when movement velocity was matched. While it is unclear as to why the results of our study are in contrast to those by Formenti et al.[59] and Borot et al.,[60] we speculate that the temporal control of movement (i.e., control of movement speed and velocity), may exert a strong influence on the M1 and other areas upstream of the M1, such as the premotor cortex and supplementary motor area.[64,65] For example, we previously showed that the depression in motor-evoked potential (MEP) amplitude following exercise (a phenomenon known as post-exercise corticomotor depression), ascertained using single-pulse TMS over the M1, was more significant following self-paced index finger flexion-extension movement rate (slower movement) compared to the same task at performed at maximal voluntary rate (faster movement).[66] This is perhaps due to greater attentional demands resulting in a greater post-exercise depression in corticospinal excitability that is associated with slower, more controlled, movements. Hence, it is plausible that performing tasks at slower movement velocity (as used in Formenti and Borot, but not in our study) could have resulted in greater utilization of close-loop motor control as compared to faster movement[64,65] resulting in greater M1 activation, which may be further amplified by the muscle lengthening nature of ECC contractions.

Considering that different movement strategies are likely to underpin the motor control of CON and ECC movements, we hypothesized that an increase in bilateral DLPFC activation would be observed in ECC compared with CON muscle actions, and that this difference would be more pronounced at high intensities. Previous studies have postulated that higher PFC activity during imagined[50] and real[49] ECC contractions are due to higher attentional resources and cognitive control required to regulate force during ECC actions. We however observed no differences in neural activity during ECC and CON muscle actions. This could be partially explained by the intensity of the contractions used in our study compared to prior fMRI studies, which were primarily body weight movements using small muscle groups or motor imagery, to account for limitations of head movement inside the scanner. In our current study, we controlled the intensity of the contractions using force feedback to reach a desired percentage of maximal CON or ECC torque. This biofeedback could have added additional elements of cognitive demand, potentially masking any differences from contraction type alone. Moreover, high intensity contractions overall showed greater neural activity in the PFC, which

may have required greater effort and attentional resources to meet the desired torque level, regardless of the contraction type.

4.3 | Limitations and future considerations

To this end, we acknowledge certain limitations of our study. First, we must acknowledge that the lack of torque- and/or EMG-matched conditions for CON and ECC contractions limits the interpretation of our findings. Indeed, the difference in torque levels for both CON and ECC in this study may have resulted in the inability to discriminate brain activation associated with CON and ECC contractions. Second, as there were no short separation channels used during fNIRS recording, we were not able to discern if extra-cerebral hemodynamic changes (i.e., change in scalp blood flow) due to increased cardiovascular parameters (i.e., increased blood pressure and heart rate) would have affected our findings.[63] However, we have ensure that all participants were securely strapped into the seat to control for any extraneous trunk and neck movements, which could potentially affect changes in the circulatory system. Moving ahead, future studies involving fNIRS measures during aerobic or resistance exercise should, at least, account for any changes in cardiovascular parameters to ensure that any intra- and extra-cerebral hemodynamic changes may be controlled for. Finally, prefrontal, premotor, and supplementary motor regions have functional connections with the M1 and DLPFC. Whole brain analysis examining the magnitude of contributions across these regions, with comparisons to less active regions, will be important for future research to extend our understanding of dynamic brain networks involved in motor control.

4.4 | Conclusion

In conclusion, our findings showed that fNIRS was able to detect intensity-specific differences in brain activation of the contralateral M1 and bilateral DLPFC. However, when matched for movement velocity during CON and ECC elbow flexion/extension, no differences in hemodynamic response were observed between CON and ECC contractions at high and low contraction intensities. The implication of our findings is that future studies will need to carefully consider the testing methodology of ECC and CON contractions, such that training/testing intensities are matched in order to truly elucidate the intrinsic motor control mechanisms of CON and ECC muscle actions. Nonetheless, our data support the role of higher exercise intensities to elicit central changes in brain activity. This finding will have future implications for planning of exercise and neurorehabilitation interventions, whereby greater exercise intensities may be necessary to increase brain activation in clinical populations such as those with neurological or neurodegenerative disorders.

4.5 | Perspective

The findings from our results suggest that when matched for movement velocity, no differences in brain activation between CON and ECC contractions in the contralateral M1 and bilateral DLPFC regions were observed. However, a greater increase in brain activity was found in all brain ROIs in the high-intensity contraction condition. Considering our results to those found by Formenti et al.[59] and Borot et al.,[60] it is likely that contraction velocity and intensity, may be a significant consideration when utilizing ECC exercises to elicit neural adaptations. Therefore, future interventions that are adopting ECC exercises for the purpose of neurorehabilitation will need to purposefully consider the role of muscle contraction velocity and intensity to optimize any physiological and physical effects of ECC exercise on the brain and muscle.

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CONFLICT OF INTEREST STATEMENT

There are no potential conflicts of interest with all co-authors.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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