



ORIGINAL RESEARCH

# Therapeutic Lower Extremity Power Training Alters the Sensorimotor Cortical Activity of Individuals With Cerebral Palsy



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## KEYWORDS

Frog game;  
Lower extremity;  
Magnetoencephalography;  
Neuroimaging;  
Optimization;  
Rehabilitation

**Abstract Objective:** To utilize magnetoencephalographic (MEG) brain imaging to examine potential changes in sensorimotor cortical oscillations after therapeutic power training in individuals with cerebral palsy (CP).

**Design:** Cohort.

**Setting:** Academic medical center.

**Participants:** Individuals with CP (N=11; age=15.9±1.1 years; Gross Motor Function Classification System I-III) and neurotypical controls (NTs; N=16; age=14.6±0.8 years).

**Interventions:** Participants with CP underwent 24 (8 weeks; 3 days a week) sessions of high-velocity lower extremity power training on a leg press. The NTs underwent single baseline MEG assessments.

**Main Outcome Measures:** Pre-post bilateral leg press 1-repetition maximum and peak power production were used to assess the muscular performance changes. The 10-m walk and 1-minute walk tests were

**List of abbreviations:** 1RM, 1-repetition maximum; CI, confidence interval; CP, cerebral palsy; ERD, event-related desynchronization; GABA, gamma-aminobutyric acid; GMFCS, Gross Motor Function Classification System; MEG, magnetoencephalography; NT, neurotypical.

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used to assess mobility changes. During MEG recordings, participants used their right leg to complete a goal-directed isometric target-matching task. Advanced beamforming methods were subsequently used to image the strength of the sensorimotor beta oscillatory power.

**Results:** Before the therapeutic power training, the participants with CP had stronger beta sensorimotor cortical oscillations compared with the NT controls. However, the beta sensorimotor cortical oscillations were weaker and approximated the controls after the participants with CP completed the therapeutic power training protocol. There also was a link between the amount of improvement in leg peak power production and the amount of reduction in sensorimotor cortical oscillations seen after therapy.

**Conclusions:** Therapeutic power training appears to optimize the sensorimotor cortical oscillations of individuals with CP, and these neuroplastic changes partly contribute to improvements in the leg peak power production of individuals with CP. Therapeutic power training might provide the key ingredients for beneficial neuroplastic change.

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Cerebral palsy (CP) is a nonprogressive perinatal neurologic condition that is caused by a defect or lesion to 1 or multiple areas of the brain.<sup>1</sup> Commonly the lesions affect the periventricular white matter, which results in decreased transmission of information along the thalamocortical and corticospinal tracts.<sup>2,3</sup> Because of the location of the lesions, motor and sensory impairments occur, and as the individual matures physically, motor skills and mobility may become compromised partly due to alterations in the musculoskeletal system (ie, weakness, contractures, spasticity, stiffness).<sup>2,4</sup> A large body of the literature has focused on the use of strength training as a means for improving the mobility in individuals with CP.<sup>5-7</sup> The premise for this treatment approach has been supported by studies that have shown the musculoskeletal architecture is more likely composed of shorter muscle fibrils and reduced cross-sectional area.<sup>8,9</sup> Nevertheless, the strength training outcomes have been mixed with several studies showing minimal mobility improvements.<sup>10-12</sup> The consensus is that although many patients with CP may lack strength, maximal strength is not necessary to perform many activities of daily living. Rather, deficiencies in the ability to rapidly recruit the available motor units and rate coding of the respective motor units may play a larger role in the mobility deficits seen in individuals with CP.<sup>13</sup>

Realizing these neurophysiological deficits, the current therapeutic trends have shifted from strength training toward power training. Power training involves the production of rapid muscular contractions performed at submaximal force production levels,<sup>14</sup> whereas traditional strength training involves heavier loads moved at slower velocities. High-velocity power training provides for earlier activation of the motor units and rapid force production,<sup>15</sup> conceivably leading to better functional mobility compared to strength training.<sup>16</sup> The prior studies that have employed therapeutic power training have shown that individuals with CP demonstrate larger mobility improvements that are accompanied by changes in the musculoskeletal architecture.<sup>17,18</sup> Although it is recognized that these clinically relevant changes are accompanied by muscular plasticity, less consideration has been given to the nervous system per se.

Numerous magnetoencephalographic (MEG) brain imaging experiments have well established that the sensorimotor cortical oscillatory activity at the beta frequency is tightly connected with the production of a motor action.<sup>19-24</sup> Specifically, the spectral power of the cortical oscillations at the beta

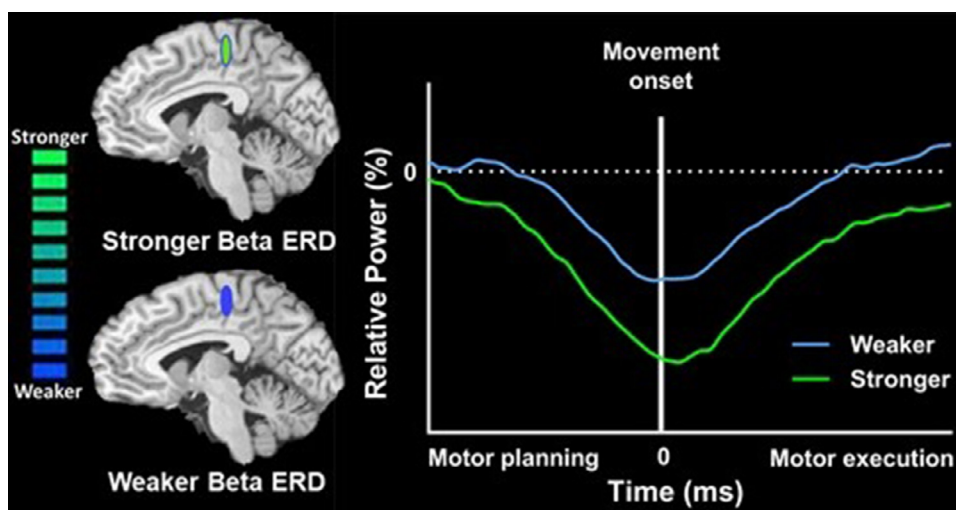
frequency are known to decrease prior to the onset of movement, and this decrease is sustained throughout the movement. This reduction in the spectral power is commonly referred to as the beta event-related desynchronization (ERD). A stronger beta ERD refers to a greater reduction in the spectral power relative to the baseline, whereas a weaker beta ERD indicates less of a reduction in the spectral power relative to the baseline (see [figure 1](#)). Physiologically, a stronger ERD (ie, power reduction) may indicate that fewer pyramidal neurons in the cortical area of interest are oscillating at the beta frequency, whereas a weaker ERD (ie, less of a power reduction) suggests more pyramidal neurons are oscillating in the beta range. Past work has shown that the sensorimotor beta ERD is weaker after neurotypical youth practice a leg isometric force motor task, which is presumed to indicate that fewer neurons are needed for the production of practiced motor actions.<sup>19</sup> Our translational neuroscience laboratory has conducted a series of foundational MEG studies, which have revealed that individuals with CP have a stronger sensorimotor beta ERD throughout the motor planning and execution stages of a knee extension motor task.<sup>25,26</sup> Further, we have determined that these aberrant beta oscillations are tightly coupled with the slower reaction times, motor production errors, and altered mobility of individuals with CP.<sup>26,27</sup> Despite these breakthroughs in our understanding of the neurophysiology of CP, we still have a knowledge gap in regard to how physical therapy may affect the sensorimotor cortical oscillations in a beneficial way.

The primary aim of this investigation was to use MEG imaging to monitor for potential changes in the sensorimotor cortical oscillations of individuals with CP after 8 weeks of a leg high-velocity therapeutic power training protocol. Based on the current literature, we hypothesized that the sensorimotor beta ERD would be weaker after completion of the therapeutic power training protocol. Secondly, we hypothesized that these neurophysiological changes would be accompanied by improvements in the leg power production and mobility.

## Methods

### Subjects

Using the effect size (1.3) for the pre–post difference in the leg peak power seen in individuals with CP after



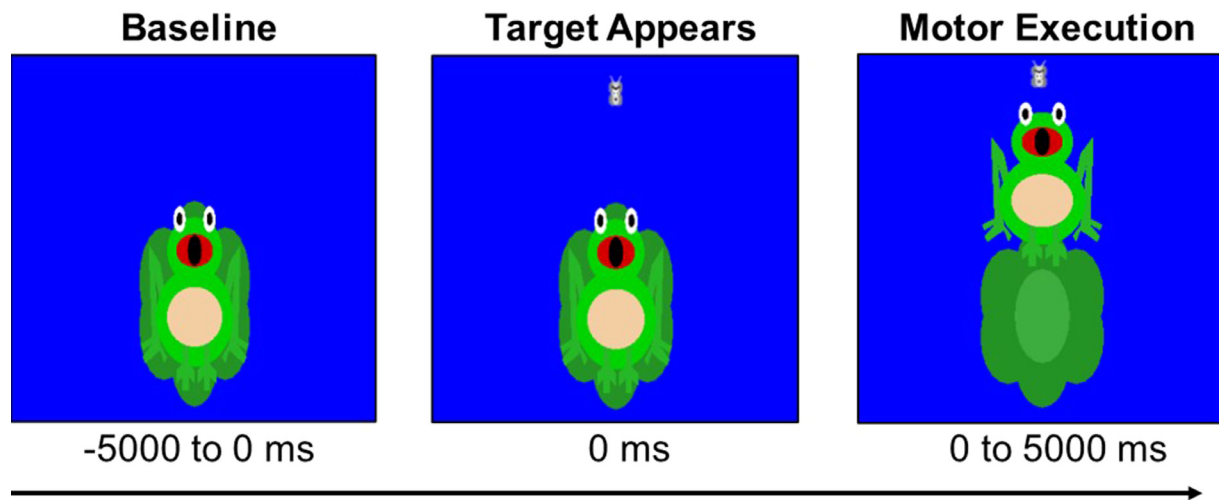
**Fig 1** Beta ERD. Conceptual representation of the change in spectral power relative to a baseline period (ie, no movement period) during leg motor actions. A greater reduction in the spectral power at the beta frequency relative to the baseline signifies a stronger beta ERD, whereas a weaker beta ERD indicates less of a reduction in the spectral power relative to the baseline. The brain images depict when the beta ERD is stronger (top panel) and weaker (bottom panel) while producing leg motor actions. The neural time courses extracted from the peak voxel of the respective brain images show that a stronger beta ERD is associated with a larger reduction in the beta power relative to the baseline period (more negative), whereas a weaker beta ERD is associated with less of a reduction in the beta power relative to the baseline period. In the neural time course, 0 milliseconds represents the onset of the leg motor action; changes in the relative power prior to movement onset represent changes in the cortical oscillations associated with motor planning and those after movement onset are associated with the execution of the leg motor action.

undergoing a high-velocity power training protocol,<sup>18</sup> 6 participants would provide greater than 85% power to detect a similar difference at a .05 alpha level. We enrolled 11 individuals with CP who had either a spastic diplegic or hemiplegic presentation (age=15.9±1.1 years; 8 males; Gross Motor Function Classification System [GMFCS] levels I-III; table 1). Participants were excluded if they had an orthopedic surgery or antispasticity

treatments within the last 6 months, dorsal rhizotomy, and/or clinical diagnosis of an arterial ischemic stroke or middle cerebral artery stroke. Sixteen neurotypical controls (age=14.6±0.8 years; 10 males) completed a single MEG scan as a comparison group (table 1). The Institutional Review Board reviewed and approved the protocol for this investigation (082-18-FB). All participants were recruited via community flyers and word of mouth. All

**Table 1** Demographic information for participants with cerebral palsy and neurotypical controls

Cerebral Palsy				Neurotypical	
Age (Years)	Sex	CP Type	GMFCS	Age (Years)	Sex
16.7	F	Spastic diplegia	I	16.8	M
16.7	M	Spastic diplegia	III	18.7	M
16.8	M	Spastic diplegia	II	9.1	F
11.6	M	Spastic diplegia	I	13.5	M
23.2	M	Spastic diplegia	II	17.3	M
15.9	M	Spastic diplegia	I	11.0	M
19.0	M	Spastic diplegia	II	17.2	M
12.8	F	Spastic diplegia	III	17.7	F
13.2	M	Hemiplegia	I	17.8	M
13.0	F	Hemiplegia	I	10.6	F
21.8	M	Hemiplegia	I	15.7	F
				16.6	F
				14.9	M
				12.9	M
				13.3	M
				9.9	F



**Fig 2** Depiction of the MEG experimental paradigm. Each trial was 10,000 milliseconds in length. Participants started the trial at rest for 5000 milliseconds. Then, a target moth appeared, prompting the participant to generate an isometric knee extension force that matched the force value. A successful match occurred when the moth that represented the target force was inside the frog's mouth for 300 milliseconds.

participants consented and provided assent for their participation.

### High-velocity therapeutic power training protocol

The leg power training protocol was performed on a Total Gym GTS,<sup>a</sup> and weights were added to the system for resistance.<sup>28</sup> Each training session consisted of unilateral and bilateral leg presses with the training load initially at 40% of the participant's 1-repetition maximum (1RM) and progressed toward 80%. The training consisted of the participant performing 6 sets of 5 repetitions for each leg separately and bilaterally. The therapeutic instructions for the concentric phase were to push as fast as possible, and for eccentric phase to lower the weight in a slow and controlled fashion over a 1- to 2-second time period. To minimize fatigue, 1-2 minutes of rest were given between sets. The power training protocol was conducted by licensed pediatric physical therapists (BC and HR) 3 times a week for 8 weeks (24 treatment sessions), with 1 day of rest between sessions. Each therapy session was 30 minutes in duration, beginning with a 5-minute warm-up that consisted of over-ground walking and performing leg presses that were less than what was performed during the training.

### Clinical outcomes

The Total Gym GTS was retrofitted with a linear cable sensor<sup>b</sup> (1000 Hz) and a custom LabView<sup>c</sup> program was created to quantify the bilateral leg press power production. Power was calculated as the product of the linear velocity and the amount of weight lifted. For peak power assessment, the participant performed 5 leg press trials where they attempted to move 50% of their baseline 1RM as fast as possible. 1RM was assessed on the Total Gym. Additional clinical outcomes measures included 1-minute walk and 10-m walk tests. Postintervention testing was completed on a separate day from the last training session but was completed within 1 week of finishing the protocol. Paired *t* tests at a .05 alpha

level were conducted to determine whether there were significant changes in the respective outcome variables.

### MEG data acquisition, experimental paradigm, and source reconstruction

Neuromagnetic responses were sampled continuously at 1 kHz with a MEG system<sup>d</sup> as the participant generated an isometric knee extension force with their right leg to match target forces that varied between 15% and 30% of their baseline maximum isometric force.<sup>26,29</sup> All participants performed the task with the same leg to improve the image quality and source localization across all participants. One hundred target forces were visually displayed as a moth and the force generated by the participant was shown as a frog that was animated vertically, based on the isometric force generated. Each trial consisted of a 5000-millisecond rest period followed by a 5000-millisecond period when the participant attempted to position the frog's mouth over the moth's position (figure 2). See Kurz et al<sup>25</sup> for a more detailed description of our MEG experimental design.

Each MEG data set was individually corrected for head motion and was subjected to noise reduction using the signal space separation method with a temporal extension.<sup>30</sup> The continuous magnetic time series were divided into epochs of 4500 milliseconds in length and centered on movement onset (0 milliseconds) with the baseline defined as -2000 to -1500 milliseconds. The beta time frequency windows were imaged using a beamforming approach to calculate the source power across the entire brain volume.<sup>31</sup> Neural time courses (ie, virtual sensors) were extracted from the peak voxel identified in the images, and the maximum beta ERD reduction seen in the time window of interest was subsequently calculated. For a more detailed description of our imaging methodology, see Wiesman and Wilson.<sup>32</sup>

Separate *t* tests were used to assess the group differences seen in the relative oscillatory power of the sensorimotor cortical response and potential therapeutic changes between pre- and posttherapy time points. Lastly, Pearson's

correlations were performed to determine whether the change in the strength of the beta sensorimotor cortical oscillations was linked with the observed changes in the respective clinical outcome variables (ie, performance). All statistical analyses were performed at the .05 alpha level with JASP version 0.14.1.<sup>e</sup>

## Results

### Clinical outcomes

The 11 participants with CP had a 95% training compliance, completing on average 22.8 out of 24 physical therapy sessions. The participants increased their 1RM (pre=158.3±24.7 kg; 95% confidence interval [CI], 109.8-206.7; post=247.5±41.5 kg; 95% CI, 166.1-328.9;  $P<.01$ ), peak power production (pre=509.9±64.7 W; 95% CI, 383.1-636.7; post=677.1±113.3 W; 95% CI, 455.1-899.1;  $P=.02$ ), and 1-minute walk (pre=74.5±9.1 m; 95% CI, 56.7-92.3; post=80.9±8.0 m; 95% CI, 65.3-96.5;  $P=.03$ ) after 8 weeks of power training (figure 3). The 6.4-unit change in the 1-minute walk represented a medium minimum clinically important difference (5.6),<sup>33</sup> suggesting that the improvements were clinically discernable. However, there were no changes in the preferred 10-m walking speed (pre=1.1±0.1 m/s; 95% CI, 1.0-1.2; post=1.1±0.1 m/s; 95% CI, 1.0-1.2;  $P=.49$ ).

### Sensor-level and beamforming results

Four individuals with CP were removed from analysis due to major artifacts during their MEG scan. The spectrogram permutation tests revealed that there was a significant decrease in beta ERD (18-24 Hz) across a large number of sensors over the sensorimotor cortex ( $P<.0001$ , corrected), which began approximately 200 milliseconds prior to the onset of the isometric force and was sustained as the participants attempted

to match the presented force targets. We imaged the beta ERD from -200 to 300 milliseconds in each participant using a baseline period of equal duration and bandwidth (-2000 to -1500 milliseconds) to identify the underlying cortical regions generating the response. The resulting images indicated that the beta ERD was centered on the leg region of the contralateral sensorimotor cortices (figure 4), with additional clusters seen in bilateral superior parietal lobules and bilaterally in the occipital cortices. The local maxima of these responses in the grand-averaged images were subsequently used as seeds for extracting virtual sensors in each participant (figure 5). Inspection of the neural time courses derived from the sensorimotor cortices showed that the strength of the beta ERD in the sensorimotor cortices was greater in participants with CP prior to power training compared to controls (CP<sub>Pre</sub>=-25.9±1.8%; 95% CI, -36.4 to -15.4; neurotypical controls [NTs] = -17.2±3.6%; 95% CI, -21.9 to -12.5;  $P=.04$ ). After undergoing power training, the participants with CP had a reduction in the strength of the beta ERD (CP<sub>Post</sub>=-14.8±3.6%; 95% CI, -18.4 to -11.2;  $P=.02$ ), and the strength of the oscillations approximated to the controls ( $P=.64$ ). In contrast to the cluster in the sensorimotor cortices, statistical analyses of the parietal and occipital clusters indicated that there were no pre- to posttraining differences in individuals with CP or differences from the controls in these respective areas ( $P>.05$ ).

### Correlation analysis

The leg peak power production after the therapeutic power training was tightly linked with the change in the strength of the sensorimotor beta ERD after training ( $r=0.79$ ,  $P=.03$ ; figure 6). This suggests that the participants with CP who had greater leg power production posttraining also tended to have weaker beta ERD in the sensorimotor cortices (ie, responses more similar to controls). None of the respective measured variables were correlated with age ( $P>.05$ ), hence

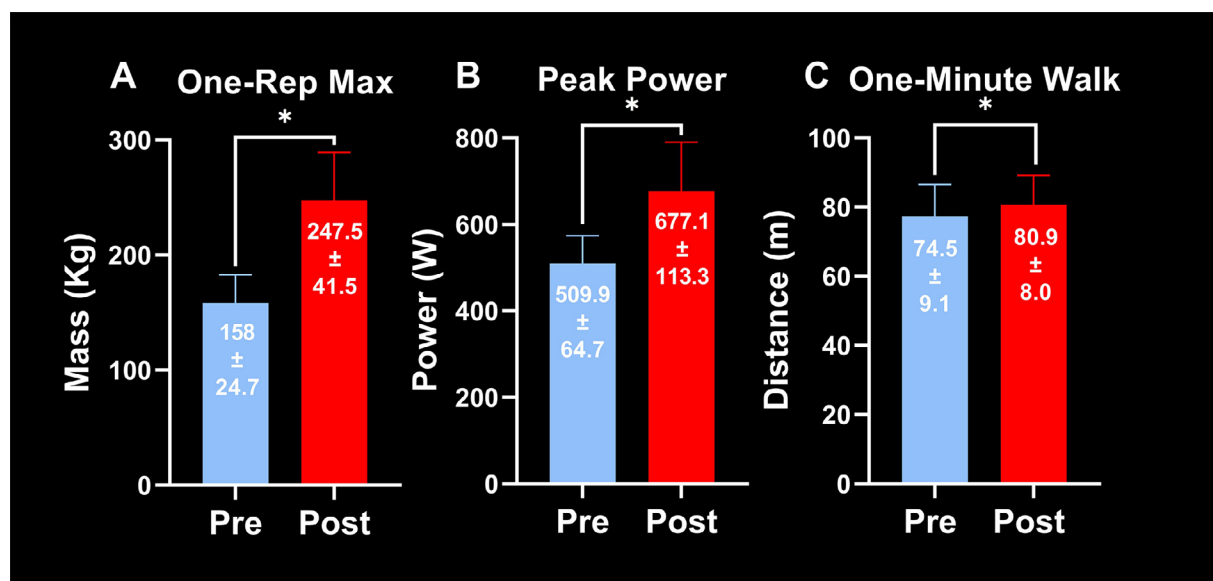
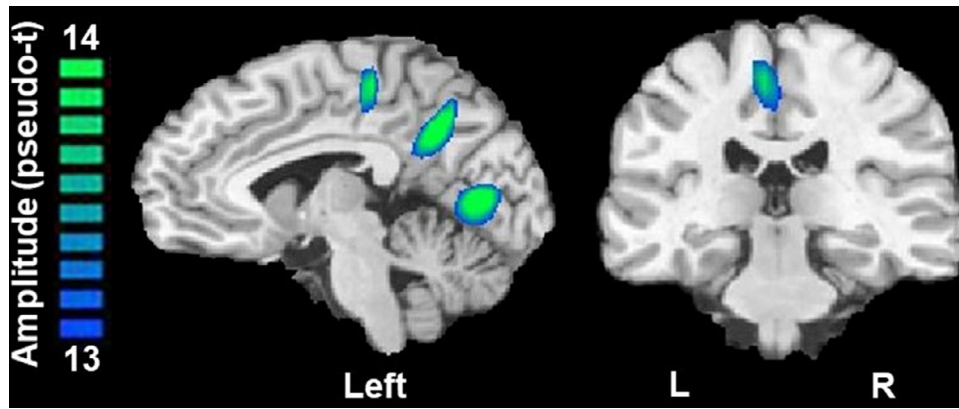


Fig 3 One-repetition maximum, leg peak power, and 1-minute walk. Pretraining and posttraining differences in (A) strength, (B) leg peak power, and (C) 1-minute walk. All values are mean±standard error. \* $P<.05$ .





**Fig 4** Beamformer images. A strong beta band (18-24 Hz) ERD was detected in MEG sensors near the sensorimotor cortex. This response started about 200 milliseconds before the initiation of the isometric force and was sustained throughout the duration of the contraction (2500 milliseconds). We applied a beamformer source reconstruction approach to the peak response (–200 to 300 milliseconds; 18-24 Hz). The resulting images were grand-averaged across all participants and visits and revealed activity in the leg region of the sensorimotor cortices, with additional islands of activity in the parietal and occipital cortices.

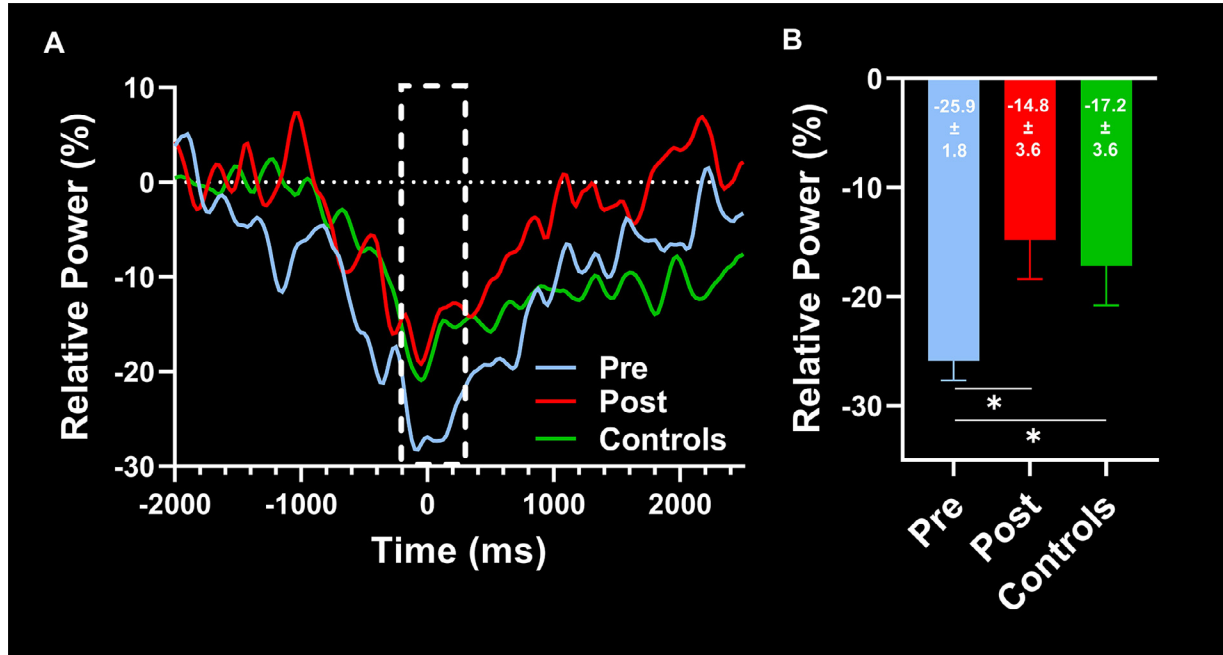
suggesting that age likely did not influence the study outcomes.

## Discussion

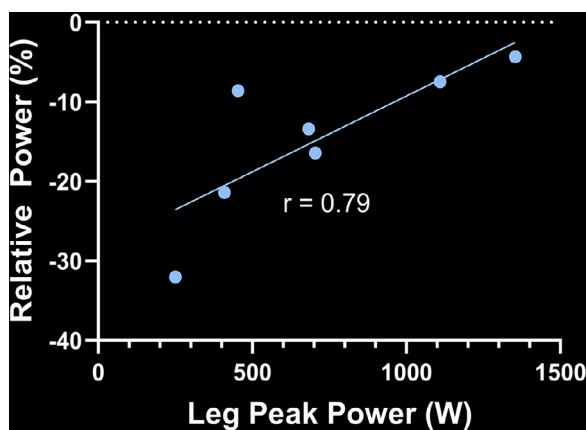
We identified that the strength of the sensorimotor beta ERD was stronger for the participants with CP in comparison with

the controls prior to undergoing the therapeutic power training protocol. This result aligns with our prior studies that have also shown the beta ERD to be stronger in individuals with CP while performing a leg target matching task.<sup>25-27</sup>

The differences in the strength of the sensorimotor cortical oscillations seen in the participants with CP could be dependent on several possible neurophysiological mechanisms. First, the stronger beta ERD might be related to fewer



**Fig 5** Neural time course of the beta sensorimotor cortical oscillations. **(A)** Neural time courses of the beta oscillations extracted from the peak voxel of the sensorimotor cortices are shown, where the percentage change in power relative to the baseline is shown on the y-axis and time is shown on the x-axis. The onset of the isometric knee extension force is at time 0 milliseconds and the white dashed box represents the time window that was imaged. As shown, the beta sensorimotor cortical activity was stronger before therapeutic power training and became weaker after power training and similar to that seen in the NT controls. **(B)** Our statistical analyses confirmed that individuals with CP had a stronger beta ERD compared to NTs prior to undergoing power training. However, after the individuals underwent the power training the beta ERD became weaker and was not significantly different from what was seen in the controls. \* $P < .05$ .



**Fig 6** Correlation between post peak power and post relative power of the beta ERD. This correlation implies that participants with CP who demonstrated greater leg power production post training also tended to demonstrate a weaker beta ERD.

neurons oscillating at the beta frequency during movement relative to the baseline. In other words, more pyramidal neurons were needed during the execution of the leg motor action in those with CP. Alternatively, it is possible that the stronger beta oscillations seen in this investigation might be related to alterations in the  $\gamma$ -aminobutyric acid (GABA) interneurons. Pharmacology-MEG studies with NTs have provided supporting evidence that an increased concentration of the inhibitory GABA neurotransmitter within the sensorimotor cortices results in a stronger motor related beta ERD.<sup>34,35</sup> Based on this scenario, the stronger beta ERD seen for the individuals with CP in this study might be reflective of heightened GABAergic activity. This impression seems to align with the prior positron emission tomography studies that have shown that individuals with CP tend to have increased GABA<sub>A</sub> receptor binding potential within the motor cortices.<sup>36,37</sup>

A key finding of this study was that the sensorimotor beta ERD was weaker after the participants with CP completed the therapeutic power training protocol. Furthermore, the strength of the beta ERD after therapy approximated that seen in controls. Together these results imply that the sensorimotor neural computations that are involved in the planning and execution of the leg motor actions were optimized in the participants with CP after undergoing the power training. This conjecture is supported by the clinical outcomes that show there were parallel improvements in the respective clinical outcomes of strength, power and some measures of mobility. Furthermore, our results show that the change in the strength of the beta ERD was tightly linked with the peak muscular power production of the participants with CP after undergoing therapy. This suggests participants with a larger reduction in the strength of the beta ERD also tended to be able to produce a greater amount of muscular power with their legs after therapy. As previously mentioned, we interpret the weaker beta ERD seen after the therapeutic power training to indicate that fewer pyramidal neurons were needed for the execution of the leg motor action, and/or a reduction in the activity of the GABA inhibitory interneurons.

Our results also demonstrated parallel improvements in leg strength (56.4% change), leg peak power production (32.8% change), and 1-minute walk (4.4% change). Overall,

these clinical outcomes are consistent with prior power training studies performed with individuals with CP.<sup>17,18</sup> Moreau et al noted significant improvements in the 10-m preferred walking speed after performing a knee extension power training protocol on the Biodex,<sup>18</sup> whereas the outcomes of the current study did not. We speculate that these differences might be partially because the Biodex protocol directly targets the quadriceps musculature that has been shown to be linked with the mobility challenges seen in individuals with CP.<sup>38,39</sup> A prior power training protocol that employed ecologically valid motor tasks (ie, walking, running, and stairs) also demonstrated a greater improvement in the 1-minute walk test (13% change) than what was seen here.<sup>17</sup> We suspect that these discrepancies might be explained by the principle of specificity. In other words, our therapeutic protocol directly targeted leg musculature power production and not mobility per se.

### Study limitations

Although our power analysis indicated that our sample size was adequate, the outcomes from this investigation may not be fully generalizable to wider populations of individuals with CP. In other words, there might be relevant differences in the cortical and muscular performance changes seen in patient populations with different GMFCS levels and presentation types (eg, hemiplegic vs diplegic). Upon completion of this investigation, it was also apparent that the tenets of producing high-velocity movements were unfamiliar to many of our participants. Furthermore, there were some challenges for the therapist to gauge whether the leg press was performed with enough speed. Potentially providing feedback to the patient as well as the physical therapist during the power training might augment greater cortical and clinically relevant changes in mobility. Lastly, the question remains as to whether the changes noted in our laboratory assessments translate to tangible improvements in the community mobility and participation of individuals with CP. Future studies should address these limitations by including a larger sample size with greater representation of all GMFCS levels and presentation types, providing feedback to physical therapists and patients, and including outcome measures that assess community participation. Future randomized controlled trials could employ a longer-term follow-up comparing therapeutic power training to usual care to understand the maintenance of these treatment effects.

### Conclusions

The overall outcomes of this investigation imply that beneficial changes in the strength of the beta sensorimotor cortical oscillations likely occur after individuals with CP complete therapeutic power training. Furthermore, these cortical changes appear to be tightly linked with leg peak power production. Together these results suggest that therapeutic power training may play a significant role in promoting beneficial neuroplastic and muscular plasticity changes. Ultimately, these changes have the potential to improve the mobility of individuals with CP.

## Suppliers

- a. Total Gym GTS; Total Gym Fitness, LLC.
- b. SGD-120-3, TE Connectivity.
- c. LabView, National Instruments.
- d. MEG system, MEGIN.
- e. JASP version 0.14.1; University of Amsterdam.

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