Title: Voluntary movement initiation is not coupled to optimal sensorimotor oscillatory phases

Running title: Oscillatory phase and movement initiation

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Key points summary

- Motor cortical activity alternates between periods of excitation and inhibition, but the influence of these brain states on fundamental motor behaviors like movement initiation is unknown.
- We examined whether self-paced voluntary movements are preferentially initiated during optimal sensorimotor oscillatory phases.
- Our results showed no evidence that voluntary movements were predominantly initiated during optimal phases across a range of frequencies.
- We conclude that initiation of voluntary, self-paced movements is not strongly coupled to optimal sensorimotor oscillatory phases in the healthy human brain.

Abstract

Successful initiation of a voluntary movement requires transmission of descending motor commands from the primary motor cortex (M1) to the spinal cord and effector muscles. M1 activity alternates between brief excitatory and inhibitory brain states in the form of oscillatory phases that correlate with single neuron spiking rates and population-level neuronal activity. The influence of these brief brain states on fundamental motor behaviors, like movement initiation, is not known. Here, we asked if voluntary movement initiation occurs during specific oscillatory phases of sensorimotor rhythms using a combination of transcranial magnetic stimulation (TMS), electroencephalography (EEG), and behavioral testing. To address this, we empirically determined the time point at which M1 released the motor command required to produce a simple finger movement during a self-paced movement initiation task. We then probabilistically modeled the oscillatory phase at this time point in the mu (8-12 Hz) and beta (13-30 Hz) ranges, and at each frequency between 8 and 50 Hz, determining each subject's preferred movement initiation phase for each frequency. After pooling the identified phases across subjects, we identified no significant clustering of preferred movement initiation phases within mu or beta frequencies, or at any other frequency between 8 and 50 Hz. These results demonstrate that movements were not preferentially initiated during optimal oscillatory phases at any frequency. Thus, we conclude that initiation of self-paced voluntary movements is not strongly coupled to optimal sensorimotor oscillatory phases in the healthy human brain. It remains to be determined if more complex aspects of motor behavior like action selection occur during optimal oscillatory phases.

Keywords: movement initiation, voluntary movement, brain oscillations, electroencephalography, sensorimotor rhythms

Introduction

The ability to initiate voluntary movements is central to human behavior, as it allows humans to effectively interact with their environment. Voluntary movements are initiated when the primary motor cortex (M1) issues descending motor commands that are transmitted to the spinal cord and connected muscles. However, M1 activity is not static but dynamically fluctuates, rapidly oscillating between periods of excitation and inhibition (Buszaki 2006; Murthy and Fetz 1992; Murthy and Fetz 1996a; Murthy and Fetz 1996b; Salmelin and Hari 1994; Pfurtscheller and Aranibar 1979; Pfurtscheller and Lopes da Silva 1999) that influence local neuronal firing rates (Haegens et al. 2011), population-level activity (Miller et al. 2012), and communication between M1 and the spinal cord (Khademi et al. 2018; Khademi et al. 2019; Zrenner et al. 2018; Hussain et al. 2019; Mima and Hallett 1999). While oscillatory brain states are known to shape non-motor sensory and cognitive functions like perception (VanRullen 2016; Busch et al. 2009; Dugué et al. 2009; Hanslmayr et al. 2014; Baumgarten et al. 2015;), attention (VanRullen 2018; Busch and VanRullen 2010), and decision-making (Wyart et al. 2012; Hamel-Thibault et al. 2016), their contribution to fundamental motor behaviors like movement initiation is not known.

Within M1, single-neuron spiking rates and descending output are both increased during excitatory sensorimotor mu (8-12 Hz) trough phases when mu power is also high (Haegens et al. 2011; Zrenner et al. 2018; Hussain et al. 2019). It has been proposed that oscillations in the membrane potential of layer V pyramidal neurons produce these phase- and power-dependent fluctuations in M1 activity and descending output (Hussain et al. 2019). Given that voluntary movements are only initiated when cortical activity reaches an excitatory threshold (Hanes and Schall 1995; Chen et al. 1998), we asked whether voluntary movements are most easily and thus predominantly initiated during excitatory oscillatory phases, when sensorimotor cortical pyramidal neurons are already partially depolarized and therefore closer to reaching this threshold.

We explored this possibility by quantifying the extent to which self-paced voluntary movement initiation was coupled to specific phases of sensorimotor oscillatory activity in

21 healthy humans. Our results revealed no evidence of coupling between sensorimotor oscillatory phase and voluntary movement initiation across a range of frequencies. We therefore conclude that voluntary movement initiation is not strongly coupled to optimal sensorimotor oscillatory phases in healthy humans.

Methods

Data acquisition

Subjects and experimental design. 21 healthy subjects participated in this study (11 F, 10 M, age = 27.66 ± 1.09 [SEM] years), which involved single-pulse transcranial magnetic stimulation (TMS), electroencephalography (EEG) and electromyography recordings (EMG), and a self-paced movement initiation task. All subjects underwent a complete neurological examination prior to participation. Inclusion criteria were: age > 18 years, clear hand dominance (either left- or right-handed), and willingness and ability to give consent. Exclusion criteria were: history of any severe or progressive neurological disorder or medical condition, history of seizures, use of anti-psychotic, anti-depressant, or prescription stimulant medications, previous diagnosis of drug dependence, and presence of TMS contraindications. This study was approved by the National Institutes of Health Combined Neuroscience Section Institutional Review Board. Prior to participation, all subjects provided their written informed consent. See Figure 1A for a depiction of the experimental timeline.

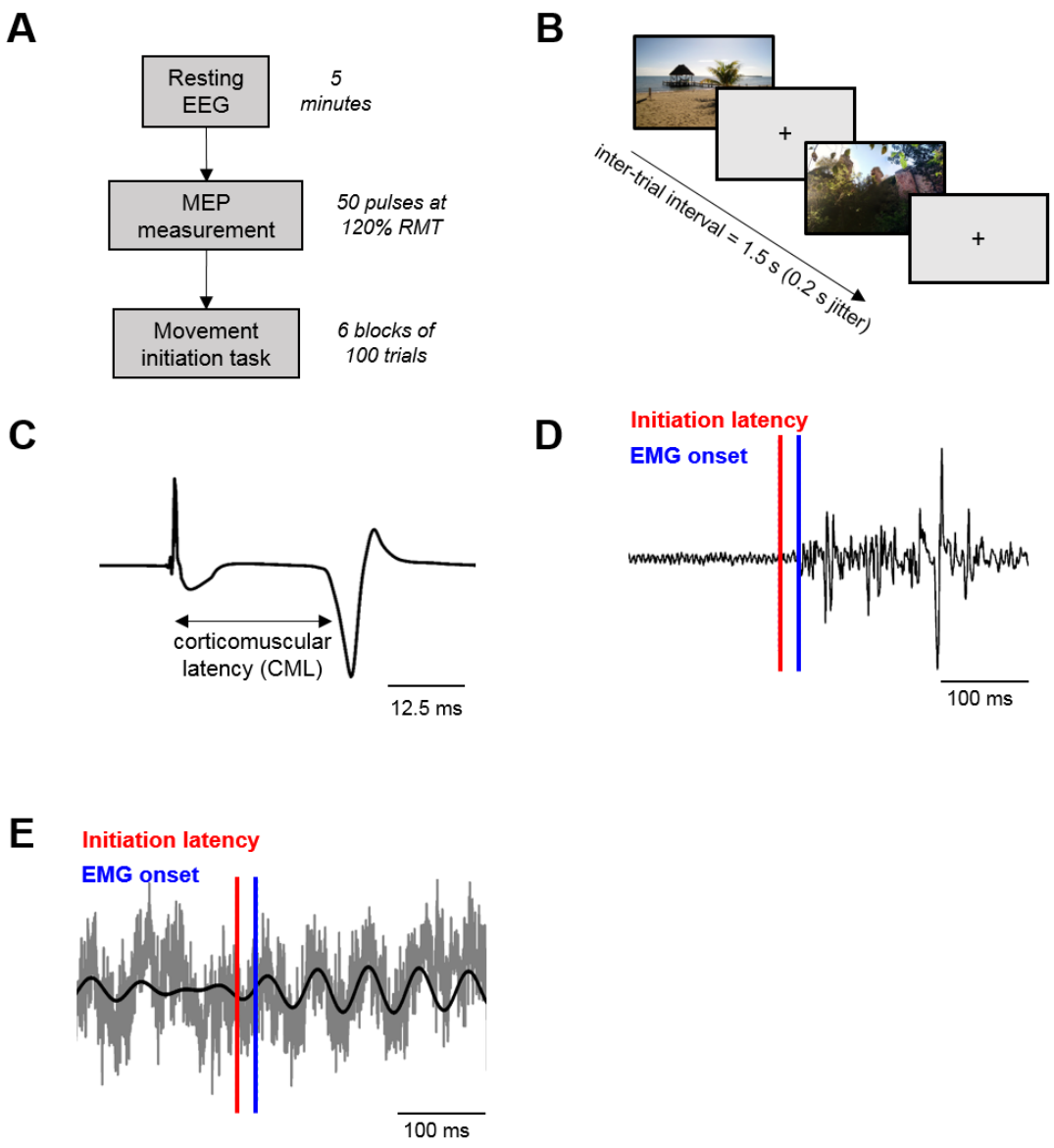

Figure 1. Depiction of experimental approach. A) Experimental timeline. First, resting EEG was recorded, followed by measurement of motor-evoked potentials (MEPs) as needed to determine individual corticomuscular latencies (CMLs). Then, subjects completed a movement initiation task. B) During this task, subjects viewed a series of pictures on a computer screen. Subjects were instructed to press a button using their left index finger whenever they wished to move to the next picture. EMG from the left first dorsal interosseous muscle (L. FDI) and EEG were recorded during the task. C) MEPs were used to calculate each subject's individual corticomuscular latency, defined as the time from TMS delivery to MEP onset. D) The initiation latency for each trial and subject was determined by first identifying the time of motor command release from M1, defined as that trial's EMG onset minus that subject's CML. E) The oscillatory phase at the desired frequency was then probabilistically modeled at each trial's initiation latency (see *Methods* for additional details). The grey and black traces reflect raw and filtered EEG data, respectively.

EEG and EMG recording. 64-channel EEG signals (ground: O10; reference: AFz) and bipolar EMG signals (ground: dorsum of left wrist) were recorded at 5 kHz (low-pass hardware filtering: 1250 Hz [cutoff frequency], 0.001 µV resolution) during single-pulse TMS delivery and the self-paced movement initiation task using TMS-compatible amplifiers (NeurOne Tesla, Bittium, Finland). EEG impedances were maintained below 10 kΩ and EMG was recorded from the left first dorsal interosseous muscle (L. FDI) using disposable adhesive electrodes arranged in a belly-tendon montage.

Single-pulse TMS delivery. The scalp hotspot for the L. FDI muscle was first identified over the hand representation of the right M1 as the site that elicited both the largest motor-evoked potential and a visible, focal twitch in the L. FDI muscle following suprathreshold single-pulse TMS. Afterwards, the resting motor threshold (RMT) was determined using an automatic threshold-tracking algorithm (Adaptive PEST Procedure; Awiszus and Borckhardt 2011). Then, 50 single TMS pulses were delivered to the L. FDI hotspot at 120% RMT (inter-pulse interval: 6 s with 0.9 s jitter). This intensity was chosen to elicit a motor-evoked potential (MEP) on each of the 50 trials, so that each subject's mean MEP could be used to calculate that subject's corticomuscular latency. The corticomuscular latency was defined as the amount of time needed for an action potential to travel from the cortex to the L. FDI muscle (see *EMG processing* below). All TMS procedures were performed using a figure-of-eight coil held at approximately 45° relative to the mid-sagittal line (MagStim Rapid^2, biphasic pulse shape; MagStim Co Ltd., UK). RMT was on average 61.62 ± 2.68 % of maximum stimulator output.

Self-paced movement initiation task. Subjects performed a self-paced voluntary movement initiation task during which they viewed a series of unique pictures on a computer screen (Places Scene Recognition Database; Zhou et al. 2017; see Figure 1B). Subjects were instructed to view each picture for as long as they desired and then press a button on a standard keyboard (left CTRL button) using their left index finger when they wished to view the next picture in the series. Between pictures, a fixation cross was presented (inter-trial interval: 1.5 s with 0.2 s jitter). In this way, the task was

designed to ensure that subjects produced a simple, self-paced voluntary movement whenever they desired. During task performance, the left arm was supported on a soft surface to ensure that subjects kept their arm as relaxed as possible while preventing any extraneous muscle activation or movement. EEG and EMG recordings were obtained during task performance. The movement initiation task was divided into 6 blocks, with each block containing 100 unique pictures. After completing 3 blocks, subjects were given a short break before resuming the task. To ensure that subjects were not merely reacting as fast as possible to the presentation of each picture and that movements were truly self-paced, trials in which reaction times were faster than 400 ms were excluded from analysis.

Data analysis

EMG processing. EMG signal processing was performed using FieldTrip (Oostenveld et al. 2011) and custom-written scripts (MATLAB; TheMathWorks, Natick MA). EMG data were used for two purposes: 1) to measure individual corticomuscular latencies from the mean MEP signal obtained using single-pulse TMS, and 2) to evaluate the timing of movement onset during the self-paced movement initiation task.

For MEP analysis, data were divided into segments (-0.25 to -0.100 s relative to TMS pulse), demeaned, and linearly detrended. For each subject, MEP signals were averaged to generate a mean MEP signal, and the MEP onset latency was visually identified as the inflection point between post-stimulus baseline EMG activity and the beginning of the MEP waveform. In two subjects, MEP onset latencies could not be reliably identified, so the onset latencies for these subjects were set equal to the mean onset latency obtained in all other subjects (23.5 ms). MEP onset latencies were then used to define each subject's individual corticomuscular latency (CML, see Figure 1C), indicating the amount of time needed for neural activity within sensorimotor regions to reach the target FDI muscle. On average, individual corticomuscular latencies were 23.5 ± 0.36 ms.

For movement onset analysis, EMG data were divided into segments (-1.2 to +1.2 s relative to the button press during the movement initiation task), demeaned, and linearly detrended. A two-pass band-stop filter (zero-phase shift Butterworth filter; 58-60 Hz) was applied to remove line noise. Then, a two-pass high-pass filter (zero-phase shift Butterworth filter; cutoff frequency: 20 Hz) was applied. The onset of voluntary EMG activity for each button press was visually identified using a combination of spectral and time-series EMG signal analysis. Specifically, the EMG signal was spectrally decomposed over time using the multi-taper method with Hanning tapers (20-500 Hz; time window=0.100 s, 0.2 ms resolution). Time-frequency representations were then plotted alongside time-series EMG signals to visually identify the time point reflecting voluntary EMG onset preceding each button press. Visual identification was used for EMG onset detection as it is still the gold standard (Tenan et al. 2017; see Figure 1D). Trials in which EMG onsets could not be reliably identified (e.g., inadequate relaxation of FDI muscle prior to button press) were excluded from analysis.

EEG processing. EEG signal processing was performed using FieldTrip (Oostenveld et al. 2011) and custom-written MATLAB scripts (TheMathWorks, Natick MA). Both resting-state and task-related EEG were recorded and analyzed.

Resting EEG data were divided into 1 s segments and task-related EEG data were divided into 2.4 s segments (±1.2 s relative to the button press). Segmented data were re-referenced to the average reference, demeaned, and linearly detrended. Afterwards, the portion of the scalp EEG signal reflecting right sensorimotor cortical activity was extracted using the Hjorth transformation (Hjorth 1975; Zrenner et al. 2018; Hussain et al. 2019; central=C4, surround=FC2, FC6, CP2, CP6). Trials containing artifacts within this signal were automatically identified (kurtosis > 4, maximum amplitude > 50 μV) and excluded from further analysis.

EEG power was calculated for frequencies between 8 and 50 Hz within a 150 ms window using an autoregressive modeling approach (Burg's method, data downsampled to 500 Hz, model order=26, frequency resolution=1 Hz; Krusienski et al.

2006; McFarland and Wolpaw 2008; Hussain et al. 2019). For resting data, this window was centered on the midpoint of each 1 s segment. For task-related data, this window was centered on the initiation latency of each trial (see *Identification of initiation latency*). Power spectra were averaged across all available trials for both rest and task-related EEG data. For resting and task-related data, 244.76 ± 15.77 and 87.9 ± 12.68 segments were available for power calculations, respectively.

Identification of initiation latency. To quantify the extent to which movement initiation is coupled to specific oscillatory phases, we first identified the time point at which the motor command producing the button press for each trial was released from M1. This time point was defined as the initiation latency and was calculated by subtracting the individual corticomuscular latency from each trial's EMG onset time point (see Figure 1D and 1E). This approach identified a single time point for each button press that corresponded to the moment of movement initiation, defined here as the time at which M1 released the motor command required to generate the voluntary button press.

Probabilistic phase modeling. To account for any noise in EMG onset detection and subsequent initiation latency calculations caused by the generally noisy nature of EMG signals, we used a probabilistic phase modeling approach. First, Hjorth-transformed task-related EEG data were band-pass filtered into the desired frequency ranges (see below for details on filtering) and phase angles were extracted using the Hilbert transform. For each resulting frequency, we used Monte Carlo simulations (N=20,000) to probabilistically model a distribution of possible phases corresponding to each trial's empirically-determined initiation latency (see *Identification of initiation latency*). This distribution was modeled with a noise of zero mean (centered on each trial's initiation latency) and a frequency-dependent standard deviation equal to $1/8^{th}$ of a single cycle per frequency (e.g., standard deviation = 12.5 ms for a 10 Hz oscillation). These simulations generated a gaussian distribution of possible phases at the time of movement initiation for each trial and frequency. Based on previous work demonstrating that oscillatory phase and power can interact to shape brain function (Ai and Ro 2014; Mathewson et al. 2009; Hussain et al. 2019), trials were then sorted into power quartiles

for each frequency, with power quartiles defined across all available trials within each subject. Phase distributions obtained for each trial were combined within power quartiles, generating four phase distributions reflecting different power levels for each frequency and subject. Each of these distributions was further binned according to phase (100 bins), converted to a probability distribution and smoothed with a median filter (order: 18). Then, the center phase angle of the bin during which movements were most likely to occur was identified for each subject and defined as that subject's preferred initiation phase for that frequency and power level. Preferred phases were pooled across subjects and used for statistical analysis. Trials in which too little data either before or after the initiation latency were available for probabilistic phase modeling were excluded from analysis. On average, probabilistic phase modeling was performed on 87.9 ±12.68 trials.

Coupling between movement initiation and sensorimotor oscillatory phases at mu and beta frequencies. Using the approach described above, we first tested whether voluntary movements were preferentially initiated during optimal sensorimotor mu (8-12 Hz) and beta (13-30 Hz) oscillatory phases, as activity in these frequency ranges is most prominent in sensorimotor cortex and has previously been shown to covary with several aspects of M1 activity (Haegens et al. 2011; Murthy and Fetz 1992; Murthy and Fetz 1996a; Murthy and Fetz 1996b; Miller et al. 2012; Hussain et al. 2019; Zrenner et al. 2018; Khademi et al. 2018; Khademi et al. 2019). Hjorth-transformed task-related EEG data were band-pass filtered into mu and beta frequency ranges (zero-phase FIR filter, Blackman-Harris window, order=2500, mu passband=8-12 Hz, beta passband=13-30 Hz; Hussain et al. 2019) and the Hilbert transform was used to extract phase angles from these filtered data. Probabilistic phase modeling was performed using standard deviations equal to 1/8th of a single mu or beta cycle, defined based on the mean of the mu and beta frequency ranges (i.e., 10 Hz and 21.5 Hz, respectively).

Coupling between voluntary movements and oscillatory phases at all frequencies. After evaluating coupling between voluntary movements and oscillatory phases in the mu and beta ranges, we performed an additional exploratory analysis at all frequencies between

8 and 50 Hz. Hjorth-transformed task-related EEG data were band-pass filtered with the pass band centered on each frequency between 8 and 50 Hz (1 Hz resolution; zero-phase FIR filter, Blackman-Harris window, order=2500, bandwidth=2 Hz), and the Hilbert transform was again used to extract phase angles. Probabilistic phase modeling was performed using frequency-dependent standard deviations equal to 1/8th of a single cycle per frequency, defined as the center of each passband.

Statistical analysis

All statistical analysis was performed in MATLAB using custom-written scripts combined with the CircStat Toolbox (Berens et al. 2009). Alpha was equal to 0.05 for all analyses.

We first tested whether sensorimotor power differed significantly between rest and movement initiation for all individual frequencies between 8-50 Hz. Because power values at several frequencies diverged from normality, spectral power for each frequency was compared between conditions using the Wilcoxon Signed-Rank test, followed by FDR-correction for multiple comparisons (Benjamini and Hochberg 1995).

We then determined if self-paced voluntary movements occurred predominantly at optimal sensorimotor oscillatory phases that were consistent across subjects by pooling the preferred initiation phases for each frequency and power quartile across subjects. Deviations from circular uniformity at the group level were tested using the Rayleigh test, followed by FDR-correction for multiple comparisons. Here, any substantial clustering of preferred initiation phases would be detected as significant deviations from circular uniformity, supporting the hypothesis that voluntary movements are preferentially initiated during optimal oscillatory phases.

Results

Power spectral densities were calculated between 8 and 50 Hz during quiet rest and movement initiation. Although spectral power was on average weaker during movement initiation in the mu and beta ranges, these differences were not significant (Wilcoxon Signed-Rank test with FDR-correction, $p>0.22$ for all, see Figure 2C).

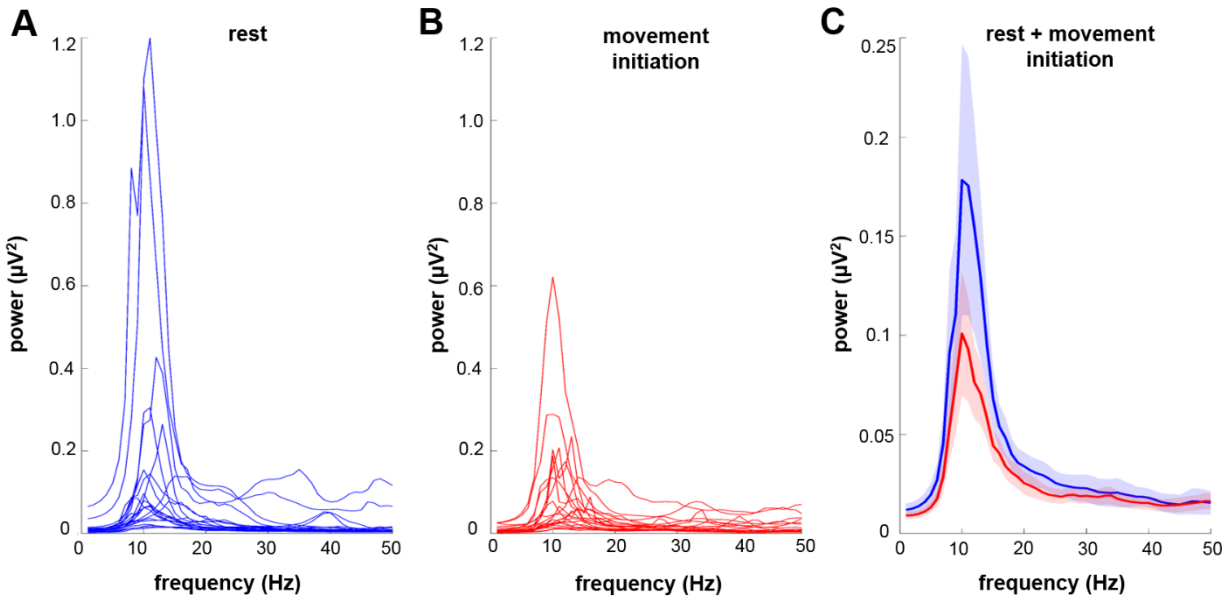

Figure 2. Sensorimotor oscillation strength during rest and movement initiation. A and B) Individual subject sensorimotor spectral power during rest (A, blue lines) and movement initiation (B, red lines). C) Group mean sensorimotor spectral power during rest (blue line) and movement initiation (red line). After FDR-correction, spectral power did not differ significantly between rest and movement initiation at any frequency between 8 and 50. Note that spectral power between 1-6 Hz is near zero due to poor frequency resolution below 6 Hz (window size for PSD calculation=150ms). Shading, ± 1 SEM.

Coupling between voluntary movement initiation and sensorimotor oscillatory mu and beta phases was evaluated by identifying each subject's preferred movement initiation phase in these frequency ranges. After pooling preferred phases across subjects, group-level distributions did not significantly deviate from uniformity for any power quartile for either frequency band (Rayleigh test for circular uniformity with FDR-correction: $p>0.46$ for all, see Figure 3).

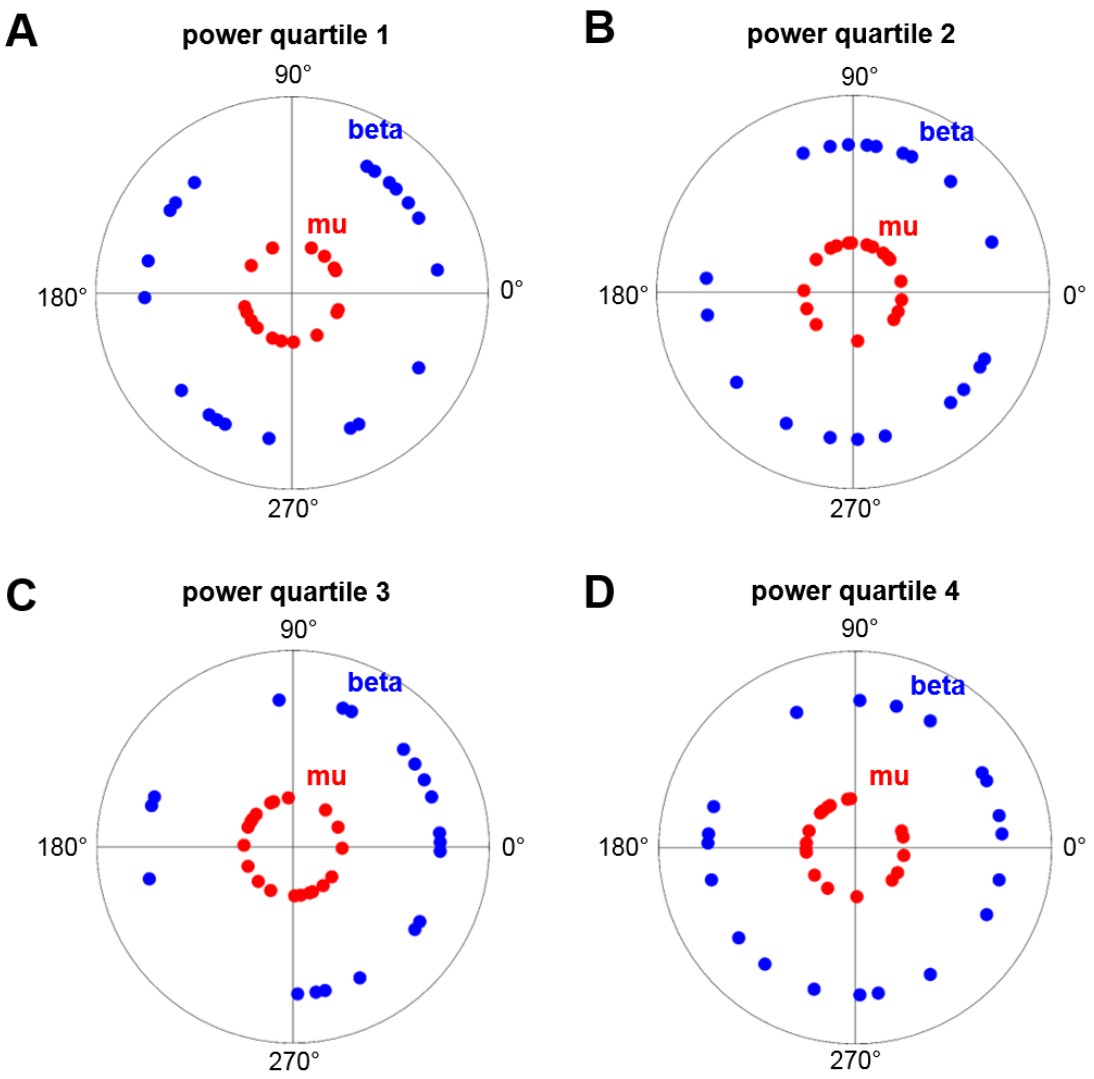

Figure 3. Voluntary movement initiation is not coupled to optimal sensorimotor mu or beta oscillatory phases. Each dot reflects the preferred movement initiation phase for an individual subject in the mu (red dots), and beta (blue dots) ranges for each power quartile (quartiles 1-4, panels A-D). Note that group-level distributions of preferred phases were not clustered near any optimal phase for either frequency band, regardless of power quartile, indicating that voluntary movements were not preferentially initiated during optimal oscillatory phases. 0°=rising phase, 90°=peak phase, 180°=falling phase, 270°=trough phase.

We further evaluated whether significant coupling between voluntary movement initiation and oscillatory phases was present at any individual frequency between 8 and 50 Hz. This was again done by identifying each subject's preferred movement initiation phase for each power quartile and frequency and pooling preferred phases across subjects. However, group-level distributions again did not significantly deviate from uniformity for any frequency or power quartile (Rayleigh test for circular uniformity with FDR-correction: $p>0.38$ for all, see Figure 4).

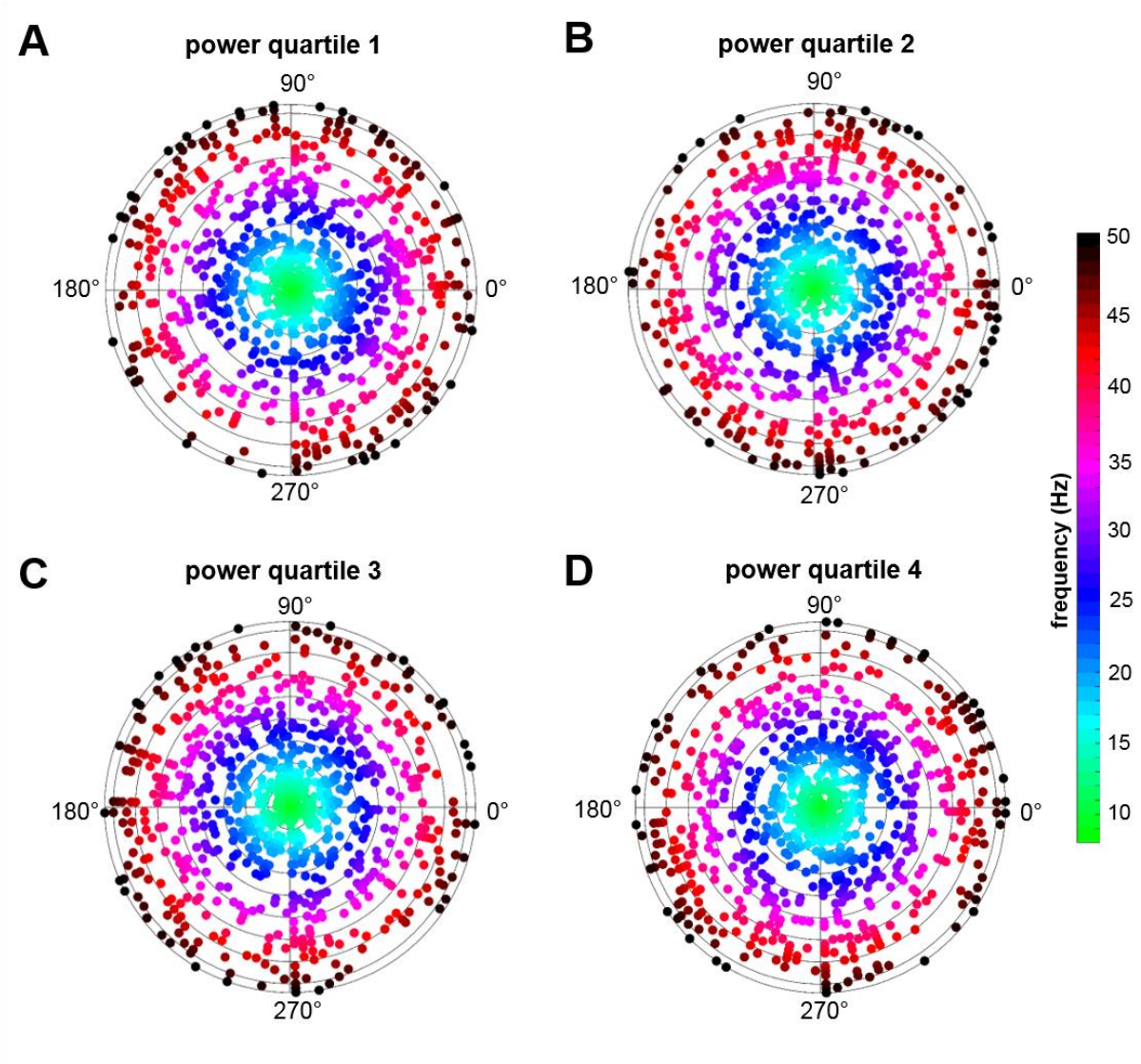

Figure 4. Voluntary movement initiation is not coupled to optimal sensorimotor oscillatory phases at any frequency between 8 and 50 Hz. Each dot reflects the preferred movement initiation phase for each subject. Each color represents a different frequency between 8-50 Hz (see color bar) for each power quartile (quartiles 1-4, panels A-D), and each radial division represents 5 Hz. Note that group-level distributions of preferred phases did not cluster near any optimal phase for any frequency, regardless of power quartile, indicating that voluntary movements were not preferentially initiated during optimal oscillatory phases. 0°=rising phase, 90°=peak phase, 180°=falling phase, 270°=trough phase.

Discussion

In this study, we evaluated whether voluntary movements are preferentially initiated during optimal sensorimotor oscillatory phases in healthy humans by quantifying coupling between self-paced voluntary movements and sensorimotor oscillatory phases. We found no evidence for such coupling, suggesting that the initiation of voluntary movements is not strongly constrained by excitatory oscillatory phases in the human brain.

The possibility that voluntary movements are predominantly initiated during optimal sensorimotor oscillatory phases is supported by two lines of evidence. First, sensorimotor oscillations correlate with M1 spiking (Haegens et al. 2011), M1 population-level activity (Miller et al. 2012), and descending motor output (Zrenner et al. 2018; Hussain et al. 2019; Khademi et al. 2018; Khademi et al. 2019; Keil et al. 2013), all of which are required for voluntary movement. Specifically, work combining TMS with EEG recordings demonstrated that descending output from M1 to the spinal cord is increased during sensorimotor mu troughs compared to peaks when mu power is high. This difference in output diminishes at mid-range power levels and reverses when mu power is low (Hussain et al. 2019). Similarly, invasive recordings in non-human primates have shown that single-neuron spiking rates in sensorimotor regions are highest during mu troughs and lowest during mu peaks when monkeys are at rest (Haegens et al. 2011) and sensorimotor mu power is high (Pfurtscheller and Aranibar 1979; Pfurtscheller and da Silva 1999; Pfurtscheller et al. 2006). However, this phase-dependency disappears when monkeys are engaged in a perceptual task that likely desynchronizes and thus weakens the mu oscillation. Based on these previous findings, one possible explanation for the lack of coupling between movement initiation and sensorimotor oscillatory phases initiation observed in the current study is that at the time of movement initiation, sensorimotor oscillatory activity may have been desynchronized or weakened (Pfurtscheller and Aranibar 1979; Pfurtscheller and da Silva 1999; Pfurtscheller et al. 2006). However, this scenario is unlikely to be the source of our findings, as we did not observe any significant modulation of coupling between movement initiation and oscillatory phases across different power levels (see Figures 4

and 5) nor did we identify any significant decreases in sensorimotor power between rest and movement initiation periods (likely due to the small amplitude movement needed to successfully perform the task; see Figure 3C). We therefore suggest that our results cannot be explained by desynchronized sensorimotor oscillations eliminating our ability to detect phase-dependency in movement initiation.

Second, multiple previous reports have demonstrated that cognitive and perceptual function is enhanced during optimal oscillatory phases recorded from task-relevant brain regions (Busch et al. 2009; Dugué et al. 2009; Hanslmayr et al. 2014; Baumgarten et al. 2015; Busch and VanRullen 2010; Wyart et al. 2012; Hamel-Thibault et al. 2016; but see also Ruzzoli et al. 2019). These findings are most numerous in the perceptual domain, leading to the proposal that human perception is not continuous but occurs through discrete, rhythmic sampling of the environment, with each sample corresponding to an excitatory phase of oscillatory activity (VanRullen 2016). In the motor domain, it is thought that descending drive from M1 to the spinal cord during voluntary movement is also rhythmic in nature, occurring through phase-synchronization between cortical and spinal oscillatory activity across a range of frequencies (i.e., corticomuscular coherence, Mima and Hallett 1999). Specifically, it has been proposed that descending motor commands are issued rhythmically (Wessberg and Kakuda 1999; Vallbo and Wessberg 1993), producing rhythmic motor neuronal firing patterns that are detectable through spectral analysis of surface EMG signals (Mima and Hallett 1999; Halliday et al. 1995). However, it is important to note that corticomuscular coherence cannot be quantified during the transition from rest to voluntary movement that occurs during movement initiation, as estimating spinal motor neuron firing patterns requires sustained EMG activity which is only present during continuous motor output (Halliday et al. 1995). Evaluating whether descending motor commands required for movement initiation are issued rhythmically thus requires a behavioral approach. Using such an approach, we identified no clear phase-dependency of voluntary movement initiation. We therefore suggest that any rhythmicity in descending drive to spinal motor neurons is restricted to periods of sustained muscle activity and does not strongly

influence the timing of transitions between rest and active movement necessary for movement initiation.

Some limitations to this study exist. We used a simple, self-paced movement initiation task to evaluate coupling between movement and sensorimotor oscillatory phases that evaluated perhaps the most basic metric of motor behavior: the transition from rest to movement. Although we identified no coupling between movement initiation and sensorimotor oscillatory phases using this task, it is possible that other fundamental aspects of motor behavior show phase coupling. For example, movements initiated during optimal oscillatory phases may be faster, more accurate, and/or smoother than movements initiated during non-optimal phases. It is also possible that more complex aspects of motor behavior, like action selection or motor preparation, could show phase coupling. Another limitation to this study is that quantifying coupling of movement initiation and oscillatory phases necessarily depends on accurately identifying the onset of voluntary muscle activity from EMG recordings, which are typically noisy (Tenan et al. 2017). To mitigate this issue, we selected only those trials in which EMG activity onsets were easily visually identifiable, and in these trials, we probabilistically modeled the oscillatory phase at the time of motor command release from M1 to account for any potential inaccuracies in EMG onset detection. Despite these methodological advantages, it is still possible that more precise approaches are needed to identify coupling between voluntary movement initiation and oscillatory phase, such as invasive recordings from spinal motor neurons in animal models.

In conclusion, this study is the first to evaluate the influence of sensorimotor oscillatory phase on self-paced voluntary movement initiation. Yet, we identified no significant coupling between self-paced voluntary movements and sensorimotor oscillatory phases at any of the tested frequencies. We therefore conclude that voluntary movement initiation is not strongly coupled to optimal sensorimotor oscillatory phases in the healthy human brain.

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