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Repetitive transcranial magnetic stimulation interrupts phase synchronization during rhythmic motor entrainment

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Abstract

Rhythmic stimuli delivered through the auditory system can facilitate improved motor control following a motor impairment. The synchronization of movement to rhythmic auditory cues is characterized by quick, stable coupling of motor responses to rhythmic auditory cues. The exact neural sites responsible for this transformation of auditory input into timed rhythmic motor output are not clear. Neuroimaging studies have identified left ventral premotor cortex (vPMC) and left superior temporal–parietal (STP) activation during rhythmic auditory–motor synchronization. To investigate brain areas necessary for different types of rhythmic auditory–motor synchronization, we delivered repetitive transcranial magnetic stimulation (rTMS) to 15 healthy individuals prior to a rhythmic–auditory tapping task. Subthreshold rTMS was administered separately to the left vPMC and STP at a frequency of 0.9 Hz for 15 min. Phase synchronization error (difference between auditory stimulus and response onsets) significantly increased after rTMS to STP as compared to baseline. Synchronization error also increased after rTMS to vPMC as compared to baseline, but not significantly. Absolute period error, (absolute difference between metronome interval and response interval) was not affected by rTMS. The significant effect of rTMS at the STP expands upon previous imaging research, suggesting that this area is part of the network responsible for rhythmic auditory–motor synchronization. The effect of rTMS on phase synchronization, but not period synchronization suggests these are separate neural processes controlled by different neural networks.

Keywords

Transcranial magnetic stimulation; Rhythmic entrainment; Auditory–motor; Synchronization

The use of rhythmic entrainment of motor skills represents an effective therapeutic variable to improve motor impairments. Specifically, rhythm-based interventions utilize the individual's ability to synchronize their movements to rhythmic auditory cues in order to improve temporal stability and motor control. Gains in motor function have been demonstrated in stroke, Parkinson's, and traumatic brain injury populations [8,14,24,27]; suggesting that rhythmic auditory stimuli facilitate the planning and execution of motor behaviors, and ultimately improve motor control following central nervous system damage. The mechanism by which

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temporal information from rhythmic auditory networks entrains motor responses, however, is largely unknown.

There exist multiple explanations for how and where auditory–motor synchronization occurs in the central nervous system. Some models of rhythmic movement argue that internal timing mechanisms located subcortically are responsible for repetitive rhythmic movements [22]. While subcortical areas may be part of the network underlying auditory–motor synchronization, individuals with cerebellar and basal ganglia pathologies are able to achieve auditory–motor entrainment. These findings suggest that other neural areas are available to synchronize movements to rhythmic auditory stimuli [4,15,25].

Warren et al. [31] proposed a model that identified auditory–motor transformation as a function of the dorsal auditory pathway, transforming musical as well as speech stimuli into motor acts. Recent imaging research points toward these direct connections between auditory and motor cortices along the dorsal auditory pathway as part of a nonlinear network responsible for motor entrainment of rhythmic auditory stimuli. Two cortical areas primarily involved in auditory–motor entrainment include ventral premotor cortex (vPMC) and left posterior superior temporal parietal junction (STP) [2,7,10,23,25].

The premotor cortex plans voluntary movements guided by stimuli from both visual and auditory senses [17]. Human imaging studies have revealed activation vPMC during rhythmic motor synchronization to auditory stimuli [10,19,25]. Other imaging investigations implicate the STP as an active brain region during the entrainment of movements to rhythmic auditory stimuli [2,7,10]. In speech research, the posterior STP has been proposed to act as an auditory–motor interface linking auditory representations and motor movements such as gestures during repetitive actions [31]. Chen et al. [2] expanded upon these results and found activation at the posterior STP during synchronized finger tapping, showing that the motor properties in this area are not limited to speech rehearsal.

Previous work has used auditory–motor synchronization tasks to analyze interactions between the auditory and motor systems, but has not shed light on what specific synchronization mechanism (period or phase synchronization) if any, is mediated at locations active during functional imaging. Studies investigating rhythmic motor synchronization have provided evidence for differential and partially independent nonlinear control strategies in regard to phase and period synchronization, depending on the magnitude of perturbation in the rhythmic stimulus frequency [11,26,28]. The present investigation employed slow frequency repetitive transcranial magnetic stimulation (rTMS) to produce a temporary inhibition of neural processing in the left vPMC and STP that would influence performance of rhythmic auditory–motor synchronization task. We predicted that rTMS to vPMC and STP would have differential effects on phase synchronization and period error.

Subjects were 15 neurologically intact right-handed volunteers (11 females, mean age 24.6 ± 3 years). Exclusion criteria were: history of seizures or epilepsy and/or currently taking medications known to increase the risk of seizures, known neurological disorders, pregnancy, or professional and/or amateur musician. All study procedures were approved by the local institutional review board and all participants gave written informed consent.

Silver–silver chloride surface EMG electrodes were applied to the right first dorsal interosseous (FDI) muscle. Participants were seated in a dental chair and a swim cap was placed over their head in order to mark TMS coil locations. The International 10–20 EEG System was used to position the coil over the desired locations. To determine the two desired stimulation areas we used Talairach coordinates from previous fMRI studies that investigated rhythmic motor entrainment by auditory stimuli. The Talairach coordinates were then translated into 10–20

scalp coordinates using the Münster T2T-Converter (www.neuro03.uni-muenster.de/ger/t2tconv/). See Fig. 1 for stimulation locations.

With the stimulator set at 75% of maximum output, TMS pulses were delivered through an air-cooled 70 mm mean loop Figure 8-shaped coil using a Magstim Super Rapid Magnetic Stimulator (Magstim Ltd., UK). Pulses were delivered to the scalp location approximating the left primary motor cortex (M1) to identify the location that produced the largest motor evoked potentials (MEPs) in the relaxed contralateral FDI. These MEPs were recorded (bandpass 2–10 kHz), rectified and amplified with a Viking II Electromyograph (Nicolet BioMedical; Madison, WI). Once this optimal stimulating point was identified, we determined resting TMS motor threshold; defined as the lowest stimulation intensity that elicited a discernable MEP in at least three of six stimulations using an oscilloscope gain of 50 $\mu\text{V}/\text{cm}$ [32]. In a relaxed muscle, motor threshold reflects global excitability of the corticospinal pathway, including large pyramidal cells, excitatory/inhibitory interneurons, and spinal motoneurons [13]. In the present study, the threshold measurement provided the reference for TMS intensity during stimulation to the vPMC and STP. Each participant received rTMS to both locations (vPMC and STP), and the order of stimulation was counterbalanced among the participants. For each location, rTMS was delivered at a frequency of 0.9 Hz at an intensity of 90% motor threshold for 15 min. These parameters are in agreement with established safety guidelines for rTMS [32] and are known to induce cortical inhibition in areas outside the motor cortex [20]. rTMS periods were separated by approximately 15 min. Prior to rTMS of the first location, each participant completed a baseline (control) tapping block, consisting of 10 trials. Immediately after receiving each stimulation condition, participants again completed one block of 10 tapping trials. Motor threshold was rechecked after each set of 10 trials.

During each tapping trial, the participant held the tapping probe in the right hand and tapped in synchrony to a 10-stimulus auditory sequence lasting 5 s. Stimuli were delivered by a metronome, generated by a Grass Instruments Click-Tone Generator (Grass Technologies, West Warwick, RI), with an effective click duration of 5 ms through headphones, at a rate of 2 Hz. Synchronized stylus tapping was measured by two commonly used variables: synchronization error and period error [29]. See Fig. 2 for description of these variables. Phase synchronization error is an indicator of beat or pulse tracking of the rhythmic cue. An individual's response generally precedes the stimulus by 20–80 ms, creating a negative phase asynchrony [1]. This well-established phenomenon is hypothesized to occur because of central anticipatory mechanisms that attempt to overcome sensorimotor delays in nerve conduction time between stimulus onset and the tap, or to overcome delays in higher level sensorimotor processing [1]. Absolute period error indicates variability or stability of period or interval entrainment between rhythmic cue and motor response. During tapping trials, participants held an insulated metal probe in their right hand. When the participant tapped, the metal probe made contact with a metal plate, completing the 9-V battery circuit. The battery voltage from the contact of the probe with the plate was sampled by a Measurement Computing Data Acquisition Unit (Measurement Computing Corporation, Norton, MA) at a rate of 1000 Hz. The resulting data file was processed and analyzed by Matlab (The Mathworks, Natick, MA) custom software. The voltage from the Grass Click-Tone Generator was similarly processed. The signal onsets were used to establish stimulus and response times.

Changes in motor threshold, synchronization error and absolute period error were analyzed using a repeated-measures ANOVA with Green-House Geisser corrections and with post hoc comparisons via the least significant difference (LSD). The first tap was excluded from analysis because it is simply a reaction to the first cue.

We found a significant difference for motor threshold between conditions ($F_{2, 28} = 20.797$, $p < .01$, Partial Eta Squared = .598), with significant increases following rTMS to STP (mean =

65 ± 10.6 , $p < .01$) and vPMC (mean = 66 ± 11.4 , $p < .01$) compared to baseline motor threshold (mean = 62 ± 9.8). There was no significant difference in post-stimulation motor thresholds between the STP and vPMC conditions ($p = .061$). See Fig. 3.

A negative asynchrony occurred in all three conditions, meaning that in each condition the participant's tap came before the onset of the stimulus. We found a significant between-condition difference in synchronization error ($F_{2, 26} = 4.58$, $p = .041$, Partial Eta Squared = .25). When compared to the baseline condition (mean = $-.034 \pm .016$), synchronization error was greater after rTMS to both STP (mean = $-.051 \pm .036$) and vPMC (mean = $-.042 \pm .024$). Post hoc comparison revealed that the synchronization error after rTMS to the STP junction was significantly greater than the synchronization error in the baseline condition ($p = .041$). The difference in synchronization errors between baseline and vPMC conditions were not significant ($p = .062$). There was no significant difference between the left STP and vPMC conditions ($p = .078$). See Fig. 4.

Absolute period error was slightly lower following STP stimulation (mean = $.020 \pm .004$) than at baseline (mean = $.022 \pm .006$) and after vPMC stimulation (mean = $.020 \pm .007$), which were essentially equivalent. These small differences did not reach significance ($F_{2, 26} = .230$, $p = .80$).

We used low frequency rTMS to disrupt function of the left STP and vPMC to investigate the role of these areas in rhythmic auditory–motor entrainment. Synchronization error after rTMS to the STP was significantly greater than at baseline. We also found an increase in synchronization error after rTMS to the vPMC as compared to baseline, but this increase was not significant. Examination of these results suggests that participants' ability to synchronize the onset of the tap response with the onset of the auditory stimuli significantly decreased after rTMS to the left STP area. Essentially, participants tapped farther ahead of the stimulus after rTMS. Absolute period error, as stability of rhythmic interval tracking, was not affected by either rTMS condition.

These results provide several insights into the process of auditory–motor synchronization. First, our results imply the left posterior STP junction is part of a neural network responsible for phase synchronization. Increased phase synchronization error after rTMS to the left STP is consistent with neuroimaging studies that have shown activation of this area during auditory–motor tasks [2,10]. The disruption in phase synchronization after rTMS to the STP extends these neuroimaging findings, showing that this area is not only active during auditory–motor synchronization, but may play a role in the transformation of auditory input into motor output. Hickok et al.'s [7] finding of activation during the motor phase of an auditory–motor task suggests this area is not limited to perceptual processing of the stimulus, but also involves motor networks as well. Accordingly, we postulate that stimulation to the left STP disrupted the temporal matching of the motor output with the auditory input.

Interestingly, we found increased negative asynchrony post-STP stimulation, i.e., compared to the control condition, tap onset occurred even earlier than the auditory cue. This is not an entirely surprising result based upon the hypothesized physiological effects of slow rTMS on cortical excitation and inhibition. For example, Rustuccia et al. [20] found that specific phases of somatosensory evoked potentials were inhibited following 0.9 Hz rTMS to the somatosensory cortex. Similar inhibition of MEPs have been demonstrated following slow rTMS [12]. Slow rTMS delivered to motor and non-motor cortical regions is presumed to increase activity of cortical inhibitory interneurons. Our finding of increased negative asynchrony may reflect inhibition of neural inputs to central anticipatory processing, resulting in a greater anticipatory response. Conversely, this result may reflect a rapid adaptation by the

rhythmic–auditory network to overcome an rTMS-induced disruption of central auditory–motor processing. Certainly, these hypotheses warrant further study.

The increase in synchronization error after rTMS to the left vPMC was not statistically significant, however, the possibility still exists that vPMC is involved in the rhythmic auditory–motor entrainment, as imaging studies reveal vPMC activation during auditory–motor entrainment tasks [25]. Further, Halsband et al. [6] found that individuals with a left premotor cortex lesion showed a disturbance in the reproduction of rhythm from memory, but not in the production of rhythmic movements guided by auditory stimuli; suggesting that PMC may be involved in the reproduction of rhythmic movements from memory but not in the temporal matching of movement to auditory cues. Clearly, the existing literature is inconsistent regarding the role of the premotor cortex in auditory–motor synchronization and further investigation is warranted.

We found no significant interruption of period synchronization post-rTMS and postulate that this process is mediated less by vPMC and STP function. Whereas phase synchronization may depend on cortical systems involving conscious tracking strategies, period matching may be an internal neural entrainment process relying on oscillatory subcortical networks, possibly involving direct projections from the auditory pathway into motor tissue in midbrain structures, e.g., via inferior colliculus or tectal longitudinal column nuclei [21]. Period entrainment may also be mediated by cerebellum or basal ganglia functions [16]. Previous work has demonstrated that rTMS to the cerebellum produced an increased variability in period matching during self-paced finger tapping [3]. Additionally, individuals with Parkinson's disease exhibit decreased ability to synchronize period after the removal of external cues compared to healthy controls, implicating the basal ganglia in the internal cueing of movements to rhythmic stimuli [4]. Our results and those of others demonstrate that period synchronization is not dependent on vPMC and/or STP and may be instead mediated by subcortical circuits.

We found a significant increase in motor threshold following rTMS to both the vPMC and STP compared to baseline. While the increase in motor threshold, which illustrates a decrease in motor cortex excitability, was significantly greater after both rTMS conditions, the increase was largest after rTMS to the vPMC. Although the underlying mechanisms of motor threshold changes are incompletely understood, such alterations likely result from rTMS inhibitory influences on interneurons with input to the corticospinal system. Gerschlag et al. [5] also found a significant decrease in motor cortex excitability after subthreshold, 1 Hz rTMS over the left premotor cortex. This decrease in motor cortex excitability could be due to a spread of stimulation to the primary motor cortex during vPMC stimulation. Direct connections between the premotor and primary motor cortices could facilitate such spread of stimulation [18]. No studies have reported the effect of temporal cortex rTMS on motor threshold, although TMS to visual [9] and prefrontal [30] cortices has been shown to alter motor cortex excitability.

A few limitations should be noted in the present study. First, we used the accepted 10–20 EEG system to reference the cortical areas for stimulation. While this method is commonly used in brain research, it does not account for neuroanatomical differences between individuals. As such, the possibility exists that rTMS was not entirely focused to STP or vPMC regions. As advances in MRI spatial resolution and localization improve; and are demonstrated to improve test–retest reliability for TMS, future investigations may include MRI-referenced stimulating positions. A second limitation is that the control tapping condition always occurred at the beginning of the session before any rTMS was administered. Rather than having a ‘no stimulation’ control condition, future studies should implement a sham rTMS condition. Third, we used motor threshold to determine the intensity of stimulation to vPMC and STP. The possibility exists that motor cortex excitability is not a good indicator of the excitability in non-

motor areas of the brain. However, this is standard practice in TMS studies, and improved methods for establishing TMS intensity have not been developed [32].

In conclusion, our finding that rTMS to the left STP increased synchronization error but did not affect period error implies that this area is involved in phase synchronization during rhythmic auditory–motor synchronization. Our results also suggest that different aspects of auditory–motor entrainment are mediated by different cortical networks.

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Fig. 1. (A) Figure-8 coil orientation, (B) approximate location of the left superior posterior temporal parietal (STP) site, and (C) the left ventral premotor cortex (vPMC) site.

Fig. 2.

Phase synchronization error and absolute period error. Phase sync error is the difference between onset of metronome click and participant tap. Period error is the absolute difference in the participant's inter-tap interval and the fixed interval (500 ms) between metronome clicks.

Fig. 3.

Mean motor threshold at baseline and immediately following 0.9 Hz rTMS to left superior temporal parietal (STP) and left ventral premotor cortex (vPMC). A significant between-condition difference was found (a^* , $p < .01$), and post hoc analysis revealed significantly greater motor threshold post-rTMS to STP ($p < .01$) and vPMC ($p < .01$).

Fig. 4.

Phase synchronization error during tapping at baseline (control) and following rTMS to STP and vPMC. A significant between-condition difference was found (a^* , $p < .05$), and post hoc analysis revealed significantly greater synchronization error post-rTMS to STP ($p = .04$).