Circadian modulation of motor-related beta oscillatory responses

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ABSTRACT

Previous electrophysiological investigations have evaluated movement-related beta (14–28 Hz) oscillatory activity in healthy participants. These studies have described an abrupt decrease in beta activity that starts before movement onset, and a sharp increase in beta power that peaks after movement termination. These neural responses have been respectively termed the event-related beta desynchronization or pre-movement beta ERD, and the post-movement beta rebound (PMBR). Previous studies have shown that a variety of movement parameters and demographic factors (e.g., age) modulate the amplitude of these oscillatory responses, and in the current study we evaluated whether the amplitudes follow a biological temporal rhythm (e.g., circadian), as it is known that spontaneous beta levels increase from morning to afternoon in some brain areas. To this end, we used magnetoencephalography (MEG) to evaluate oscillatory activity during a right hand finger-tapping task in four participants who were recorded at three different times (09:00, 12:00, 16:00) on three consecutive days (i.e., 36 total MEG sessions). All MEG data were corrected for head motion and examined in the time–frequency domain using beamforming methods. We found a significant linear increase in beta ERD amplitude from 09:00 to 16:00 h in the left precentral gyrus, left premotor cortices, left supplementary motor area (SMA), and right precentral and postcentral gyri. In contrast, the amplitude of the PMBR was very steady across the day in all brain regions except the left SMA, which exhibited a linear increase from morning to afternoon. Finally, beta levels during the baseline period also increased from 09:00 to 16:00 in most regions of the cortical sensorimotor network. These data show that both the pre-movement beta ERD and spontaneous beta levels strongly increase from morning to afternoon in the motor cortices, which may indicate that the amplitude of the beta ERD response is determined by the spontaneous beta level during the motor planning period.

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Introduction

Over the past decade, numerous magnetoencephalography (MEG) and electroencephalography (EEG) studies have examined movement-related oscillatory responses in healthy participants (Cheyne et al., 2008; Gaetz et al., 2010, 2011; Hall et al., 2011; Jurkiewicz et al., 2006; Muthukumaraswamy, 2010; Tzagarakis et al., 2010; Wilson et al., 2010, 2011a). These electrophysiological methods have excellent spatiotemporal resolution, which has allowed neural activity serving individual movements to be decomposed into planning, execution, and termination stages, and these three stages of movement have been tentatively linked to three distinct oscillatory responses (Cheyne et al., 2008; Gaetz et al., 2010, 2011; Hall et al., 2011; Jurkiewicz et al., 2006; Muthukumaraswamy, 2010; Tzagarakis et al., 2010; Wilson et al., 2010, 2011a). Briefly, there is an event-related desynchronization

(ERD) in the beta-frequency range (14–28 Hz) that peaks before movement onset and continues slightly after movement execution, which is termed the pre-movement beta ERD response. There is an event-related synchronization (ERS) in the gamma-frequency range that coincides with the onset of movement and is relatively brief (i.e., ~200 ms), and finally there is a post-movement beta ERS that reaches a maximum amplitude slightly after the termination of movement (Cheyne et al., 2008; Gaetz et al., 2010, 2011; Hall et al., 2011; Heinrichs-Graham et al., in press; Jurkiewicz et al., 2006; Tzagarakis et al., 2010; Wilson et al., 2010, 2011a). This latter response is generally termed the post-movement beta rebound (PMBR). For clarity, the precise frequency range (e.g., 14–28 Hz) and time window vary slightly between studies, but this variance likely reflects demographic factors and/or small differences in the MEG data analysis approach or task design (e.g., see Cheyne et al., 2008).

In addition to characterizing the oscillatory dynamics, many studies to date have evaluated how specific task parameters, diseases, therapies, and/or demographic variables (e.g., age) modulate the amplitude of beta responses. For example, a recent MEG study demonstrated that the amplitude of the beta ERD linearly scaled with the directional

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uncertainty of the movement (Tzagarakis et al., 2010), which is in agreement with a previous study showing that the beta ERD was more strongly lateralized when the upcoming movement was cued to one side with certainty versus ambiguous cueing (Doyle et al., 2005). There are also data showing that diazepam, a GABA-A modulator, accentuates the pre-movement beta ERD, but not the PMBR or the movement-related gamma response (Hall et al., 2011). Studies in patient populations have shown that the amplitude of the beta ERD is reduced in Parkinson's disease (Heinrichs-Graham et al., in press), and that all three oscillatory responses are abnormal in adolescents with psychosis (Wilson et al., 2011a). MEG studies of stroke patients have reported that effective motor-related therapies are associated with a decrease in PMBR amplitude following treatment (Wilson et al., 2011b). Other studies have shown that the amplitude of the PMBR increases as a function of age, with little-to-no synchronization occurring in younger children (Gaetz et al., 2011). Age also modulates the beta ERD, but this effect varies by motor region (Wilson et al., 2010). Finally, limb-dependent differences in the location and peak frequency of the response have been shown with the beta ERD and PMBR (Kurz et al., 2014; Wilson et al., 2010).

While these motor-related oscillatory responses have been widely studied and appear to be quite robust, little is known about their test–retest reliability or whether factors like muscle fatigue, mental fatigue, participant satiety, sleepiness/arousal level, or circadian clock modulate the responses. There is reasonable evidence that resting-state power in the beta frequency band is weakest in the morning hours and that it increases during the late afternoon in healthy adults (Cacot et al., 1995; Gundel and Hilbig, 1983; Toth et al., 2007). There is also abundant evidence that motor performance is influenced by the biological clock (Carrier and Monk, 2000; Drust et al., 2005; Gueugneau and Papaxanthis, 2010; Jasper et al., 2009), and a recent fMRI study of finger-tapping showed that time-of-day effects could be discerned in multiple motor regions (Peres et al., 2011). Unfortunately, the relationship between fMRI activation and neural oscillatory activity in electrophysiology is only partially understood, which prevents any strong conclusions regarding circadian effects on motor-related oscillations. In the current study, we used MEG to evaluate neural oscillatory activity during a finger-tapping task in four male participants who were recorded at three different times, 09:00, 12:00, and 16:00, on three consecutive days (i.e., nine MEG sessions per participant). As a control condition, we also examined resting-state beta activity levels in these participants at the same time points. Our primary goal was to determine whether the amplitude of motor-related beta oscillatory activity varied depending on the circadian clock, and if so to identify the critical brain regions that exhibit a chronologically-dependent response. Based on previous findings of increased resting-state beta during the later afternoon, we hypothesized that the amplitude of the beta ERD and PMBR responses would be stronger at 16:00 relative to 09:00, and that day-to-day variability would be minimal indicating a strongly reliable response.

Materials and methods

Participant selection

Four healthy males who were right handed participated in the study. The mean age was 31.5 years-old (range: 24–39) and all participants had at least 18 years of formal education. Exclusionary criteria included any pre-existing major psychiatric or neurological disorder, active brain infection, presence of brain neoplasm or space-occupying lesion, history of head trauma, current or history of substance abuse, and the MEG Laboratory's standard exclusion criteria (e.g., orthodontic braces, extensive dental work, ferromagnetic implants, or pacemakers). Written informed consent was obtained following the guidelines of the University of Nebraska Medical Center's Institutional Review Board, who reviewed and approved the study protocol.

Experimental paradigm

Participants were instructed to abstain from alcohol and to sleep normally throughout the study and for several days prior to study initiation. Normal sleep was defined as obtaining one's personal average amount of sleep each night (not more or less), and we expect compliance was high as all participants were laboratory personnel. Participants underwent three MEG recordings per day (09:00, 12:00, and 16:00) for three consecutive days, and each of the nine sessions included the same resting-state and finger tapping experiments. Throughout these experiments, participants were seated in a custom chair within the magnetically-shielded room with their head positioned in the helmet-shaped MEG sensor array. During the movement task, participants were instructed to fixate on a centrally-presented cross hair and to perform a single flexion–extension of the second metacarpus phalangeal (i.e., index finger) of the right hand each time a dot reached the 12 o'clock position. This dot completed one full rotation around a clock-like circle without tick marks or numbers every 6 s, and was meant to serve as a pacing device (Heinrichs-Graham et al., in press; Wilson et al., 2013a). The precise timing of movement onset was determined by a fiber optic switch, whereby a signal was emitted from one side of a groove that functioned as the finger rest area. Initiation of movement allowed the signal beam to contact a sensor on the opposite side of the groove and this event generated a trigger pulse that was recorded with the MEG data. Each participant performed approximately 120 trials. Participants also completed a six minute block of eyes-open rest (i.e., fixation on a crosshair) during each MEG session. Total MEG recording time was ~17 min per session (including both tasks).

Structural magnetic resonance imaging (sMRI)

High-resolution neuroanatomic images were acquired using a Philips Achieva 3T X-series scanner. The T1-weighted sagittal images were obtained with an eight channel head coil using a 3D fast field echo sequence with the following parameters: TR = 8.1 ms; TE = 3.7 ms; field of view: 24 cm; matrix: 256 × 256; slice thickness: 1 mm with no gap; in-plane resolution: 0.9375 × 0.9375 mm; and sense factor: 1.5. The structural volumes were used for MEG coregistration and spatial normalization.

MEG data acquisition & MRI coregistration

All recordings were conducted in a one-layer magnetically-shielded room (MSR) with active shielding engaged. With an acquisition bandwidth of 0.1–330 Hz, neuromagnetic responses were sampled continuously at 1 kHz using an Elekta Neuromag system with 306 magnetic sensors, including 204 planar gradiometers and 102 magnetometers (Elekta, Helsinki, Finland). Using MaxFilter (v2.1.15; Elekta), MEG data from each participant were individually corrected for head motion and subjected to noise reduction using the signal space separation method with a temporal extension (tSSS; Taulu et al., 2005; Taulu and Simola, 2006).

Prior to MEG measurement, four coils were attached to the participant's head and the locations of these coils, together with the three fiducial points and scalp surface, were determined with a 3-D digitizer (Fastrak 3SF0002, Polhemus Navigator Sciences, Colchester, VT, USA). Once the participant was positioned for MEG recording, an electric current with a unique frequency label (e.g., 322 Hz) was fed to each of the coils. This induced a measurable magnetic field and allowed each coil to be localized in reference to the sensors throughout the recording session. Since coil locations were also known in head coordinates, all MEG measurements could be transformed into a common coordinate system. With this coordinate system, including the scalp surface points, each participant's MEG data were coregistered with their T1-weighted MRI data prior to source space analyses