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MOTIVATION AND MOTOR CORTICAL ACTIVITY CAN INDEPENDENTLY AFFECT MOTOR PERFORMANCE

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Abstract—The present study explored the relationship between motor-preparatory electroencephalographic (EEG) activity, motivation, and motor performance (specifically premotor reaction time [RT]). Participants performed a RT task by squeezing a hand dynamometer in response to an auditory ''go'' signal. We recorded EEG and electromyography to index beta-suppression and premotor RT, respectively. Participants' motivation on each trial was modulated by offering monetary incentives at different magnitudes. Mixed-effect linear regression models showed that monetary incentive predicted premotor RT when controlling for beta-suppression, and beta-suppression independently predicted premotor RT. Thus, it appears motivation and beta-suppression can facilitate motor performance independent of one another. A plausible explanation of this effect is that motivation can affect motor performance independent of the motor cortex by influencing subcortical motor circuitry.

Key words: incentives, electroencephalography, beta-suppression, electromyography, premotor reaction time.

INTRODUCTION

Motivation and motor cortical activity are both known to affect motor performance (e.g., Doyle et al., 2005; Johnson, 1922; van Wijk et al., 2009). However, the interrelationships among these variables are less well-known. One possibility is that motivation affects motor performance by preparing the motor cortex for action, which in turn elicits quicker activation of the muscles required for action. Accordingly, it would be predicted that the relationship between motivation and muscle activation speed would be mediated by motor cortical activity. Alternatively, motivation and motor cortical activity could have independent effects on motor performance. For example, motivation could modulate reward-sensitive subcortical motor circuitry (e.g., ventral tegmental area [VTA] and reticular formation [RF]) connected to musculature via the reticulospinal tract, while motor cortical activity influences performance via the corticospinal tract (Butler and Hodos, 2005). The present study tested models investigating the independent and interdependent (i.e., motor cortical activity mediates motivational effects) relationships of motivation and motor cortical activity to motor performance.

BETA-SUPPRESSION AND MOTOR PERFORMANCE

Activity in the beta frequency bandwidth (13–30 Hz) of the electroencephalogram (EEG) recorded over contralateral motor cortex decreases prior to movement (Pfurtscheller and Lopes Da Silva, 1999). This 'beta-suppression' is often accompanied by faster reaction times (RT) (Doyle et al., 2005; van Wijk et al., 2009) and has been interpreted as a preparatory state of the motor system (Neuper and Pfurtscheller, 2001; for a review, see van Wijk et al., 2012). Specifically, beta activity may play a 'gating role' whereby it inhibits motor output, thus beta-suppression would 'unlock the gate,' facilitating output to the corticospinal tract and ultimately the motoneurons responsible for innervating muscles required for an action (Engel and Fries, 2010).

BETA-SUPPRESSION AND MOTIVATION

Beta-suppression is modulated by dopamine levels in basal ganglia, with higher levels of dopamine eliciting greater beta-suppression (for a review, see Jenkinson and Brown, 2011; Kühn et al., 2008). Through this relationship, motivation may influence beta-suppression and, thus, motor performance. Specifically, motivation increases dopamine levels (Tobler et al., 2005), and therefore should enhance beta-suppression. Beyond this neurobiological rationale, a practical reason for motivation to be associated with beta-suppression exists. Specifically, when one is pursuing a goal, they must prepare to act toward the goal, and this action preparation likely involves motor cortical activity. Based on this reasoning, Gable et al. (2016) conducted a study contrasting beta-suppression on trials where a goal (reward) was being

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Abbreviations: AIC, Akaike Information Criterion; EEG, electroencephalographic; LMER, linear-mixed effect regression; RF, reticular formation; RMSE, root-mean squared error; RT, reaction time; VTA, ventral tegmental area.

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pursued with beta-suppression on trials where no reward was possible. The authors observed greater beta-suppression on trials with a reward at stake, suggesting a link between motivation and beta-suppression.

Motivation has been linked to motor performance and beta-suppression, which has also been associated with motor performance. However, whether beta-suppression mediates the relationship between motivation and motor performance is still unclear. Only recently has evidence supporting this relationship been revealed. Specifically, Meyniel and Pessiglione (2014) had participants squeeze a hand dynamometer with the objective of spending as much time as possible above their target force in a trial. Participants were allowed to spontaneously take rest breaks within a trial, and each trial involved a monetary incentive. All the while, participants' magnetoencephalography (MEG) signals were recorded. Results indicated that participants' took shorter rest breaks during trials with high monetary incentives, and this relationship was mediated by increased beta-suppression during the highly incentivized trials. Accordingly, Meyniel and Pessiglione provide evidence that beta-suppression may mediate a relationship between motivation and motor performance.

PRESENT STUDY

One possibility is that motivation affects motor performance by preparing the motor cortex for action, which in turn elicits quicker activation of the muscles required for action. Accordingly, it would be predicted that the relationship between motivation and muscle activation speed would be mediated by motor cortical activity. Alternatively, motivation and motor cortical activity could have independent effects on motor performance. For example, motivation could modulate reward-sensitive subcortical motor circuitry (e.g., VTA and RF) connected to musculature via the reticulospinal tract, while motor cortical activity influences performance via the corticospinal tract (Butler and Hodos, 2005).

The present study aimed to examine whether motivation affects motor performance through beta suppression (motor cortical activity), or whether motivation and motor cortical activity influence motor performance independently. To test this, the present study investigated whether motor cortical activity mediates the relationship between motivation and motor performance, and also tested competing models wherein motivation and motor cortical activity independently affect motor performance. Results suggest motivation and motor cortical activity can have unique effects on motor performance.

EXPERIMENTAL PROCEDURES

Participants

Twenty right-handed young adults (five females, M_{age} = 22.3, SD = 3.56 years) participated in this experiment, but one participant's data were discarded due to excessive artifact in the EEG. Further information about participants can be found in Meadows et al. (2016).

Task

Participants completed four blocks of 42 trials of a RT task by squeezing a hand dynamometer in response to an auditory "go" signal. We attempted to modulate participants' motivation on each task trial by offering a particular monetary incentive. For further details about the task, see Fig. 1 and Meadows et al. (2016).

EEG recording and processing

EEG was recorded from 32 channels using a BrainVision actiCAP system (Brain Products GmbH, Munich, Germany; see Meadows et al. (2016) for further information about recording). Signal processing was conducted with BrainVision Analyzer 2.1 software (BrainProducts GmbH, Munich, Germany). Data were re-referenced to an averaged ears montage, band-passed filtered between 0.1 and 50 Hz with 24-dB rolloffs with a 60-Hz notch employing a zero-phase shift Butterworth filter. Next, eye-blinks were reduced employing the ICA-based ocular artifact rejection function within the BrainVision Analyzer software (electrode FP2 served as the VEOG channel; BrainProducts, 2013). This function searches for an ocular artifact template in channel FP2, and then finds ICA-derived components that account for a user specified (70%) amount of variance in the template matched portion of the signal from FP2. These components were removed from the EEG signal, which was then reconstructed for further processing. Next, data were segmented into epochs of the 3000 ms prior to the "go" signal. Then, we rejected segments wherein there was more than a 100-μV change in a moving 200-ms time window at any contralateral motor cortex electrode of interest: FC1, FC3, FC5, C1, C3, C5, CP1, CP3, and CP5. This resulted in the loss of an average of 17.6 (SD = 27.3) trials per participant. Next, a fast Fourier transformation was employed using 0.244-Hz bins and a Hamming window (50% taper). Spectral power was then averaged across the beta frequency bandwidth (13–30 Hz) for the previously noted electrodes of interest. Next, beta power at each of these electrodes was natural log transformed to approximate a normal distribution, and then the transformed beta power was averaged across the electrodes. This average served as our measure of beta-suppression (lower values indicating greater suppression).

EMG recording and processing

A BioPac BioNomadix wireless EMG system (Goleta, CA) was used to collect EMG activity at 1000 Hz from the flexor carpi radialis and extensor carpi ulnaris. Next, data were bandpass filtered between 5 and 250 Hz and rectified as root-mean squared error (RMSE). From the RMSE transformed EMG, we extracted premotor RT as the time from the go signal (which was indexed by a digital trigger) to the first visible peak in the RMSE-EMG. Premotor RT was natural log transformed to approximate a normal distribution. Premotor RT served as our measure of motor performance. As a validity check, we extracted the average, maximum, and