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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

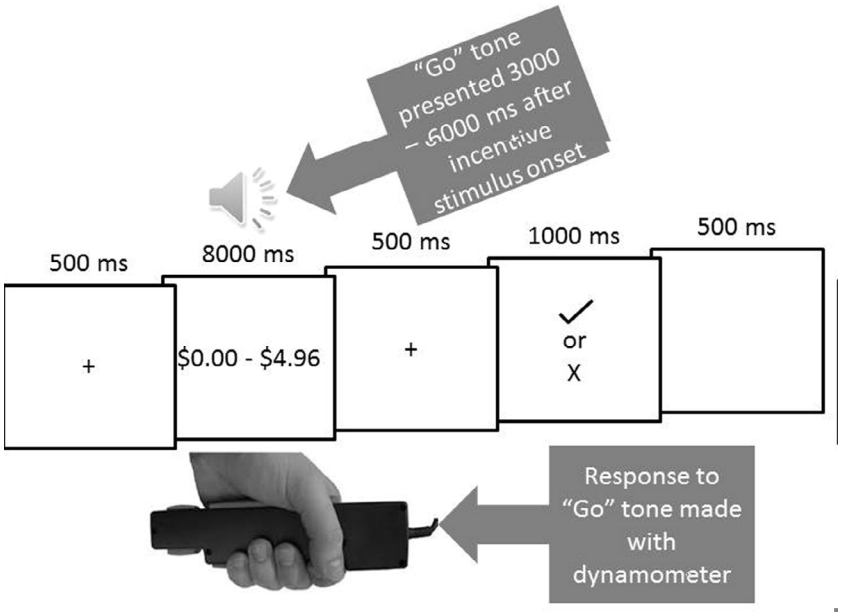

Fig. 1. Pictorial description of stimulus presentation.

standard deviation of the RMSE-EMG in this window. The first visible peak was 3.6–7.0 standard deviations above the background mean, suggesting these peaks were truly muscle onsets and not background noise.

Statistical analysis

Because of the nested nature of the data in the mediation analysis, we were less concerned with the number of participants and more so with the number of trials per participant for the task. This is due to the fact that trials are essentially the unit of observation for within-subject mediation and power for within-subject effects is primarily influenced by the number of observations at this level (power for between-subject differences or Within × Between interactions would be much lower [Snijders, 2005]). Therefore, each participant completed 168 trials of the task (which was estimated to give approximately 80% power to detect a mediation; based on PowMed.R: http://davidakenny.net/cm/mediate).

All analyses were conducted in R (R Core Team, 2015), using ''dplyr'' and ''lme4'', and ''ggplot2'' for visualizations (Bates et al., 2015; Wickham and Francois, 2015; Wickham, 2009). Linear-mixed effect regression (LMER) was used to control for the nested nature of data. LMER conveys several advantages over other approaches to longitudinal data (e.g., repeated measures ANOVA), such that the LMER approach allows for missing data points from individual participants, data points collected at different times, and greater control over the number of parameters tested (e.g., specific control over linear or quadratic predictors). To control for the nested structure of our data, we added random-effects of participant for both the intercept and slope to all of our regression models. These random-effects account for the between-subject variability in the data, allowing the fixed-effects to be interpreted like regression coefficients in a traditional linear model.

Prior to analysis, the variables of incentive and natural log beta-power were mean-centered to ease the interpretation of the statistical models and reduce potential collinearity. Incentive was centered around the grand mean of $1.80 and the natural log of beta power was centered around the grand mean of -2.35 ln(μV^2). A series of LMERs were tested using a four-step series of regressions (Baron and Kenny, 1986; see https://github.com/keithlohse/reward_mediation for data and R code). The following mixed-effect models were tested:

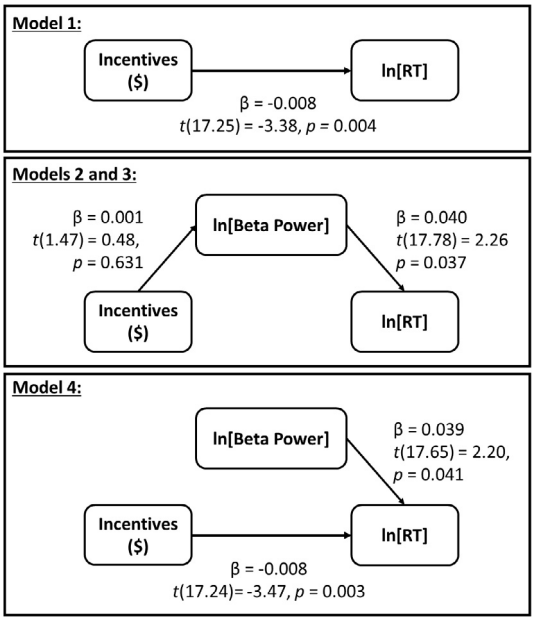

Fig. 2. Pathways and inferential statistics for the four main mixed-effect regression models. The model of a priori interest, Model 4, showed independent effects of incentive and beta power in predicting premotor reaction times. Note that degrees of freedom and *p*-values are based on the Satterthwaite approximation.

Table 1. Summary statistics for the linear mixed-effect regression models

Model	Fit statistics			Random-effects variance components (*SD*)		
	AIC	BIC	Deviance	Subjects	Incentive	lnBeta
M1	−1497.5	−1461.6	−1509.5	0.145	0.006	*na*
M2	992.4	1028.3	980.4	0.452	0.001	*na*
M3	−1503.1	−1467.2	−1515.1	0.144	*na*	0.056
M4	−1514.1	−1454.2	−1534.1	0.145	0.005	0.056
M5	−1513.0	−1447.1	−1535.0	0.145	0.006	0.055

Note that all models are based on 2966 observations from 19 individuals. Only Models 1, 3, 4, and 5 can be compared as the outcome is the same in these models (i.e., natural log premotor reaction time) whereas in Model 2 the outcome is different (i.e., natural log beta power). Random-effects variance components are shown as standard deviation (*SD*). All models were fit using maximum likelihood estimation. AIC = Akaike Information Criterion, BIC = Bayesian Information Criterion, na = not applicable because the given model did not contain this variable as a predictor.

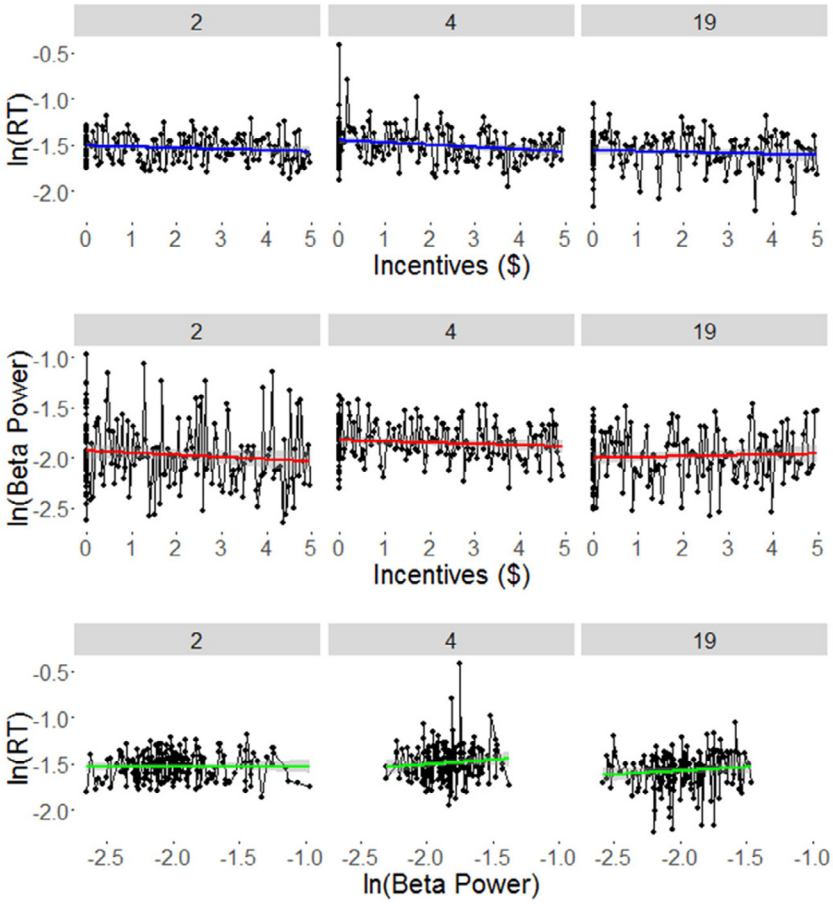

Fig. 3. Individual data are shown for three randomly selected participants (2, 4, and 19). The different rows show the bivariate relationships between natural log transformed premotor reaction time, incentives (in dollars), and natural log transformed beta power. The linear model of best-fit for each participant is overlaid on the raw data.

- *Model 1:* ln(Premotor RT) ~ Constant + Incentive.
- *Model 2:* ln(Beta Power) ~ Constant + Incentive.
- *Model 3:* ln(Premotor RT) ~ Constant + ln(Beta Power).
- *Model 4:* ln(Premotor RT) ~ Constant + Incentive + ln(Beta Power).
  - The comparison of Model 4 to Model 1 served as the statistical test of the mediation effect.
- *Model 5:* ln(Premotor RT) ~ Constant + Incentive + ln(Beta Power) + Incentive*ln(Beta Power).
  - This model tested for potential moderating influences of incentives and beta power.

Comparisons between models were based on the Akaike Information Criterion (AIC). Within each model, the statistical significance of individual coefficients was calculated based on the Satterthwaite approximation (Satterthwaite, 1946; which approximates the *t*-distribution for mixed effect models) implemented in the "lmerTest" package (Kuznetsova et al., 2015).

RESULTS

Inferential statistics and fixed-effects for the different regression models are shown in Fig. 2, model fit statistics and random-effects are provided in Table 1. Testing Model 1 of the mediation analysis revealed a significant relationship between incentives and RT, with greater incentives predicting quicker RTs, $t(17.25) = -3.38$, $p = .004$. This suggests motivation improved motor performance. Fig. 3 displays the relationship between incentive and premotor RT for three randomly selected participants.

Testing Model 2 of the mediation analysis revealed the relationship between incentives and beta-suppression was not significant, $t(1.47) = 0.48$, $p = .631$. This suggests motivation did not alter motor cortical activity. The second row of Fig. 3 depicts the relationship between incentive and beta-suppression for three randomly selected participants.

Testing Model 3 of the mediation analysis revealed a significant relationship between beta-suppression and RT, greater beta-suppression was associated with faster premotor RT, $t(17.78) = 2.26$, $p = .037$. This suggests motor cortical activity improved motor performance. The third row of Fig. 3 depicts the relationship between beta-suppression and premotor RT for three randomly selected participants.

As shown in Fig. 2, Model 4 of the mediation analysis revealed higher incentives predicted quicker premotor RT even after accounting for beta-suppression, $t(17.24) = -3.47$, $p = .003$. This test also showed greater beta-suppression predicted quicker premotor RT after accounting for incentives, $t(17.65) = 2.20$, $p = .041$. As shown in Table 1, Model 4 was also a better fit for the data, AIC = −1514.1, than either Model 1, AIC = −1497.5, or Model 3, AIC = −1503.1. Sobel's test of mediation was also applied (using the *t*-test statistics from Models 2 and 3, which yielded a nonsignificant result for beta-suppression mediating the relationship between incentives and premotor RT, Sobel test statistic = 0.480, $p = .631$.Thus, results suggest motivation and motor cortical activity can *independently* predict motor performance. The predictions of this model are shown in Fig. 4.

Finally, Model 5 tested the interaction of incentives with beta-suppression, but this interaction was not statistically significant, $t(35.53) = -0.99$, $p = .324$, nor was the model a statistically better fit than the simpler Model 4, AIC = −1513.0.

DISCUSSION

The present study tested the interdependent and independent effects of beta-suppression and motivation (monetary incentives) on premotor RT. Results indicate motivation and beta-suppression can independently improve premotor RT. It appears that the relationship between motivation and muscle activation speed are not mediated by motor cortical activity. This is the first study to reveal that when motivation and beta-suppression are considered together, they can have unique effects on motor performance.

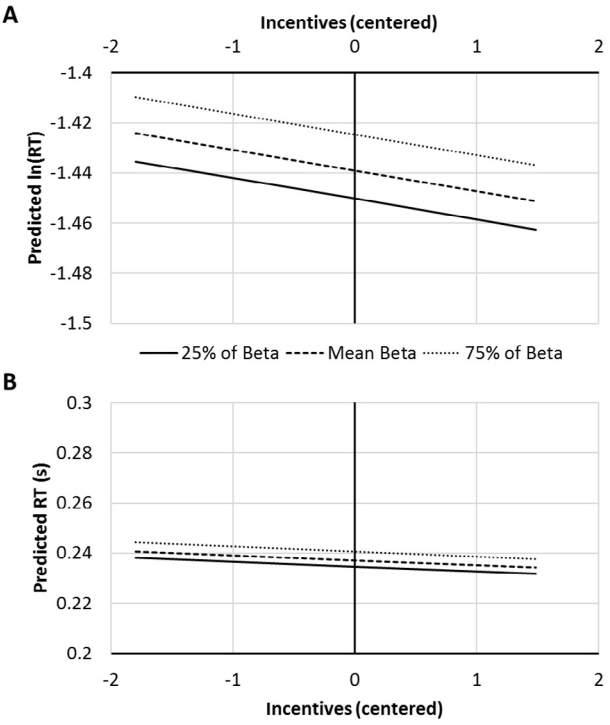

Fig. 4. Predictions from the best-fitting linear mixed-effect regression model are shown (i.e., Model 4 predicting premotor RT as a function of beta power and incentive). Panel A shows the predicted natural log transformed premotor reaction time as a function of incentive (centered around the grand mean of $1.80) and three different levels of beta power (the 25th percentile, the mean, and the 75th percentile). Panel B shows the same relationships, but the model predictions have been exponentiated to show premotor RT in seconds.

Results are in accord with Meyniel and Pessiglione (2014), who observed that motivation and beta-suppression influenced motor performance, but do not concur with the observation that beta-suppression mediated the relationship between motivation and motor performance. However, there are numerous differences between the way in which the variables/constructs were derived, operationalized, and submitted to statistical analysis in the two studies. Perhaps the most important difference is that Meyniel and Pessiglione measured motor performance in terms of the length of rest time participants spontaneously took within task trials, whereas the present study operationalized motor performance as premotor RT. It follows then that beta-suppression may explain the effect of motivation on some forms of motor performance but not others.

The question then arises as to how beta-suppression and motivation independently affect motor performance. The extant literature provides reasonable theories as to how beta-suppression may affect motor performance. For example, a common hypothesis suggests that beta oscillations prevent the motor cortex from initiating voluntary movements, and that suppression of the oscillations allows movement initiation and ultimately production (Engel and Fries, 2010). This theory is similar to the beta oscillations' 'gating role' described by van Wijk et al. (2012).

Motivation may be able to affect motor performance independent of motor cortex by influencing subcortical motor circuitry. For example, it is possible motivation may activate the VTA, which is the origin of the reward-sensitive mesolimbic dopamine system (Butler and Hodos, 2005). VTA in turn could activate the RF, which influences muscle activation via the reticulospinal tract. Therefore, the effects of motivation on motor performance in the present study may have been mediated by subcortical, rather than cortical, motor activity, precluding the activity's detection with EEG. Future research could test this theory by imaging subcortical structures like VTA and RF with functional magnetic resonance imaging while employing a paradigm similar to that of the present study.

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