

Reward-dependent Graded Suppression of Sensorimotor Beta-band Local Field Potentials During an Arm Reaching Task in NHP

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Abstract— A better understanding of reward signaling in the sensorimotor cortices can aid in developing Reinforcement Learning-based Brain-Computer Interfaces (RLBCI) for restoration of movement functions with fewer implants. Brain-computer interfaces (BCIs) using local field potentials (LFPs) have recently achieved performance comparable to spike-BCIs [1]. With superior stability over time, LFPs may be the preferred signal for BCIs. We show that sensorimotor LFPs can provide reward level information (R1 – R3) like spikes[2]. We used a cued reward-level reaching task in which reward information was temporally dissociated from movement information. This allowed the study of reward- and movement-related modulations in LFPs. We recorded simultaneously from contralateral primary -somatosensory (S1), -motor (M1), and the dorsal premotor (PMd) cortices in a female Macaca Mulatta. We found that all three cortices' average beta band (14-30 Hz) amplitude showed robust modulation with reward levels during the cue presentation period. Such modulation was consistently observed after controlling for cue color, differences in behavioral variables, and electromyogram (EMG) activity. Statistical amplitude analysis showed that reward level could be extracted from the simple LFP feature of beta band amplitude, even before a reaching target appeared, and no specific reach plan could be developed.

Clinical Relevance— The availability of reward-related signals in the sensorimotor cortical (S1, M1, and PMd) LFPs' prior to movement planning opens new avenues to build RLBCIs with fewer implants recording fewer sites among different cortices. Reward and motivational representations derived from LFPs, compared to spikes, allow the development of long-term clinical applications, given LFP's stability and ease of recording over long periods.

I. INTRODUCTION

The response of dopaminergic neurons in the deep brain structures such as the Ventral Tegmental Area and Substantia Nigra to a rewarding stimulus is broadcasted to several cortical and sub-cortical regions via the dopaminergic projections. Among others, projections received by the sensorimotor areas provide access to reward-related processing that invigorate goal-directed movements [3]. Previous studies in monkeys have demonstrated the role of S1, M1, and PMd cortices in representing reward expectation [4], [5], reward value, and motivational signals [2], [6], [7] during different motor tasks using spike data. However, whether LFPs from these regions contribute to processing reward information is not completely known. Our previous work [8], [9] along these lines found that features derived

from M1 LFPs and spikes show consistent modulation with reward expectation across different motor tasks and modes of task execution. As the next step, this work aims to characterize the response of S1, M1, and PMd LFPs to multilevel reward information strictly during the cue presentation period devoid of any movement-related information and attempted movement.

The results of this study will further our knowledge of sensorimotor systems beyond motor control and lay a foundation for developing long-term clinical applications such as RLBCIs utilizing more stable neural signals, LFPs.

II. METHODS

A. Experimental task and data acquisition

One nonhuman primate (Macaca Mulatta, female) performed a planar center-out reaching task (Figure 1) with her right arm using a robotic exoskeleton (KINARM lab, BKIN Technologies Ltd.) to earn a variable amount (1, 2, or 3 drops, 0.24ml per drop) of juice reward. Each trial began with the presentation of a center hold position (gray, 0.8cm). To proceed, the NHP reached to and held on the center until its color changed, cueing the reward level (R1, R2, or R3) of the trial. Next, the monkey maintained a hold until the cue disappeared, and a peripheral target (Go-cue) of the same color was presented in one of four possible directions (0°-Right, 90°- Up, 180°- Left, or 270°- Down). The monkey had

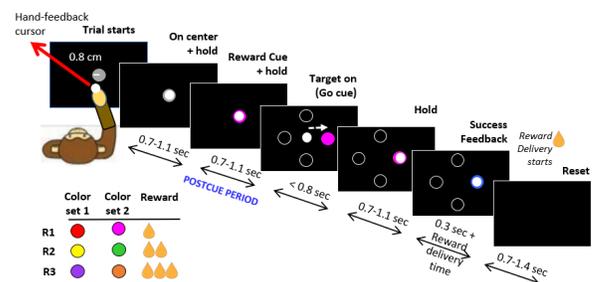


Figure 1. Typical trial progression in the arm reaching task with variable reward levels (R1, R2, and R3) cued by color.

<800ms to finish the movement towards the target. Once reached and held on target, feedback (300ms) was presented, indicating successful completion of the trial. Consequently, the cued number of juice drops were delivered with a 500ms delay between subsequent drops. The trial was reset (black screen) before the beginning the next trial. All hold times and inter-trial intervals were normally distributed with mean

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900ms (SD 50ms) and 1000ms (SD 100ms), respectively. Trials were considered failed when the NHP's hand velocity exceeded 1.5cm/s during the hold, or the reach wasn't complete in 800ms. Failed trials were not rewarded and repeated until completed successfully. The reward level and target direction were varied pseudo-randomly from trial-to-trial. The same experiment was conducted for two different color sets (color set 1, CS1, and color set 2, CS2).

LFPs were simultaneously recorded from chronically implanted 96-channel Utah arrays in the arm region of S1, M1, and PMd and sampled at 2KHz. All surgical procedures followed in this study were approved by the Institutional Animal Care and Use Committee, University of Houston. Surface EMG signals recorded from six muscles (Right (R.) Biceps Brachii, R. Triceps Brachii, R. Pectoralis Major, R. Brachioradialis, R. Zygomaticus major and Left (L.) Biceps Brachii) using gold cup electrodes (10mm) and hand velocity were sampled at 2KHz. Among 32 LFP channels per cortical region, bad channels were rejected based on visual inspection leading to 30, 31, and 28 channels in S1, M1, and PMd cortices, respectively. All analyses were conducted on successful trials combined across multiple sessions (17 sessions in CS1 and 5 sessions in CS2). Signal preprocessing and statistical analyses were performed in MATLAB 2016a.

B. Signal preprocessing

Hand velocities were smoothed using a Savitzky-Golay filter (3rd order, window length 100ms) downsampled to 1KHz. All other filtering operations used Butterworth filter and was applied in the forward and reverse directions to eliminate phase shift. EMG signals were amplified, online low-pass filtered 1KHz, and processed in the following manner [10]. Each EMG channel was filtered 40-500Hz (4th order) to remove muscle movement artifacts and dc noise. IIR notch (2nd order) filters were applied to remove electrical noise (60Hz) and its harmonics. Next, the filtered signal was down-sampled to 1KHz, rectified and smoothed, low-pass filtered 5Hz (5th order) to get the EMG envelope. EMG envelope was normalized with respect to its maximum value during the movement periods across all trials of each session.

Local detrending was done to remove low-frequency fluctuations from the recorded LFPs (function *locdetrend*, 500ms window with 250ms step size, Chronux Toolbox [11]). Next, LFPs were filtered 1-250Hz (3rd order) and notch filtered to remove the electrical noise (60Hz and harmonics). The filtered LFP signals corresponding to only successful trials were extracted and downsampled to 1KHz for further analysis.

C. Analysis of behavioral variables

The effect of reward cues on motor behavior was quantified using three variables, reaction time (the time from target presentation to the time when the hand speed was $\geq 15\%$ of the peak speed), peak speed (maximum hand speed achieved by the monkey during the reaching movement) and time to reward (the total time elapsed from cue presentation to the beginning of reward delivery). Trials with outlier RTs (rejection criterion: $RT > (\text{Median RT} + 4.45 * \text{Median Absolute Deviation (MAD)})$ or $RT < (\text{Median RT} -$

$4.45 * \text{MAD})$ were removed from further analysis. The non-parametric Kruskal-Wallis (KW) test was applied for comparing behavioral differences across reward levels. For statistical significance ($p < 0.05$), the Wilcoxon ranksum test performed pairwise comparisons. P-values were corrected for multiple comparisons using the False Discovery Rate (Benjamini-Hochberg method, FDR-BH).

To control for behavioral differences between the reward levels, kinematic matching of trials was performed. A sub-set of successful trials was considered matched when their median reaction time, peak speed, and time to reward didn't differ significantly (KW test, $p > 0.05$) with reward. The process started with considering all successful trials for a chosen reference reward level among R1, R2, or R3. For the selected level, the median value (M_{ref}) of a behavioral variable was computed for comparison with each trial of other reward levels. Next, the KW test was performed to identify if the variable varied across reward levels. If p -value < 0.05 , some trials (irrespective of their reward level) were truncated based on the criterion ($x > (M_{\text{ref}} + n * \text{MAD})$ or $x < (M_{\text{ref}} - n * \text{MAD})$) in each iteration where n is a real number > 0 . This process was repeated for multiple iterations with a gradual reduction in the value of n until the trials in different reward categories had similar reaction times, peak speed, and time to reward.

D. Extraction and analysis of beta-band amplitude

Preprocessed LFPs were filtered to extract beta (14-30Hz) band oscillations, we then applied the Hilbert transform. The magnitude of the transformed signal represented the amplitude of filtered oscillations. For every LFP channel, the beta amplitude was normalized (z-scored) to their mean and standard deviation during each session before the combined analysis. Next, the amplitude of each trial was extracted and aligned to four trial events; cue presentation (CUE), target presentation (TARG), target hold (HOLD), and feedback presentation (FB). Amplitude during a trial was baseline corrected by subtracting the mean amplitude across all trials during the precue period (0.7 sec before CUE). Based on the Nyquist criterion, the trial-wise amplitude was binned into 150ms non-overlapping bins.

Statistical analyses were only performed for the postcue period (Figure 1). For each LFP channel, trial-to-trial binned beta amplitude was fit to independent predictors, reward level (R_i) and time bin (Bin_t), and their interaction ($R_i * \text{Bin}_t$) using a linear model described as

$$\text{Amp}_{i,t} \sim 1 + R_i + \text{Bin}_t + R_i * \text{Bin}_t \quad (1)$$

where i represents trial and t represents bin number (1 to 7) in a trial. Significant ($p < 0.05$) model coefficients for reward and/or reward-bin interaction reflected an effect of reward level on the postcue amplitude and were further analyzed using Wilcoxon rank-sum test. P-values from pair-wise tests (R1 vs. R2, R2 vs. R3, and R1 vs. R3) were corrected for multiple comparisons using the FDR-BH method.

III. RESULTS

A. Postcue Beta amplitude attenuates with reward level

We found a significant effect of time-bin and reward-bin interaction on postcue beta amplitude of all LFP channels in

S1, M1, and PMd cortices (Figure 2, unmatched). Compared to baseline, mean beta amplitude was suppressed after cue presentation based on the cued reward level. Suppression was highest for maximum reward (R3, red) and lowest for minimum reward (R1, green). Additionally, in line with the previous reports [12], beta oscillations were suppressed during the movement period in all three cortices. Figure 3 (color set 1, unmatched) shows the channel statistics. The y-axis represents the number of LFP channels with significant pairwise reward differences in a time bin (x-axis) during the postcue period.

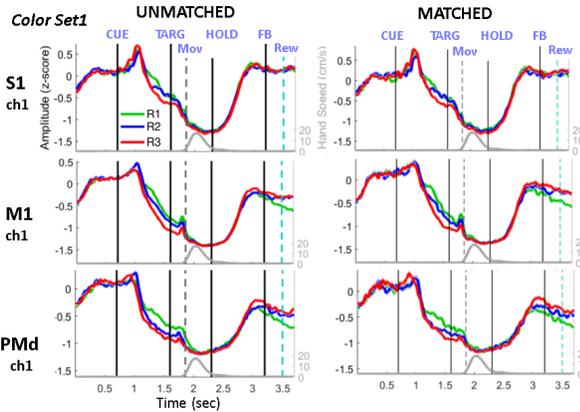


Figure 2. Mean beta amplitude for sample channels in S1, M1, and PMd cortices for different reward levels for unmatched and matched trials (CS1).

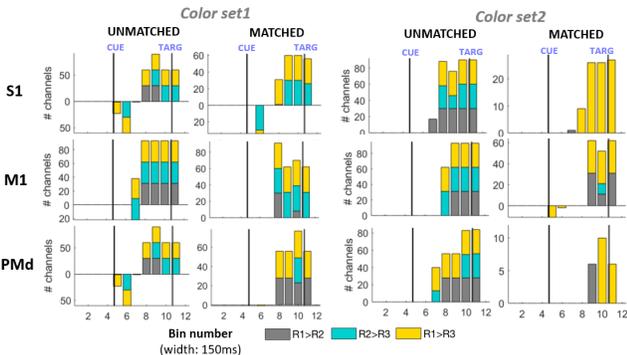


Figure 3. Channel statistics for color sets 1 and 2 for unmatched and matched cases. Each plot indicates the number of LFP channels that had significant differences in beta amplitude for a reward pair in a time bin.

B. Arm kinematics didn't contribute to LFP amplitude differences

We observed a significant effect ($p < 0.05$) of reward level on behavior variables (Figure 4, black plots) consistent with previous studies on human ([13]) and animal subjects ([14]). Both reaction time and time to reward are reduced with an increase in reward with no significant effect on peak speed. Multiple studies have previously shown that premovement beta power in the motor and premotor regions can predict behavioral performance and reaction times of upcoming movement [15], [16]. Accordingly, we controlled for the possible contribution of behavioral changes to reward-related beta suppression by performing kinematic matching of trials. (Figure 4, green plots). The analysis of beta amplitude for only matched trials revealed a similar reward-related suppression as observed with all trials shown in Figure 2

(matched) with statistics in Figure 3 (color set 1, matched).

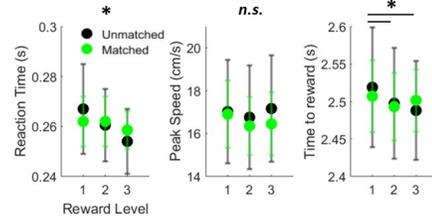


Figure 4. Change in median behavioral variables with reward level (unmatched trials, CS2). Black asterisk (*) without horizontal bars indicates significant differences between all reward pairs and n.s. indicates no significant differences. Green plots show median values after matching, which have no significant differences.

C. Choice of cue color doesn't affect reward-dependent beta amplitude modulation

To investigate whether the choice of cue color led to observed differences in beta amplitude with reward levels, we conducted the same experiment with another set of isoluminant colors (CS2). We performed the LFP analysis in the unmatched and matched cases (Figure 5). We observed a similar reward to beta amplitude relationship (amplitude decreases with higher reward) during the postcue period as in CS1. Channel statistics in Figure 3 (CS 2) show the specific reward pairs at each time bin (0.15s period) where beta amplitude differed significantly.

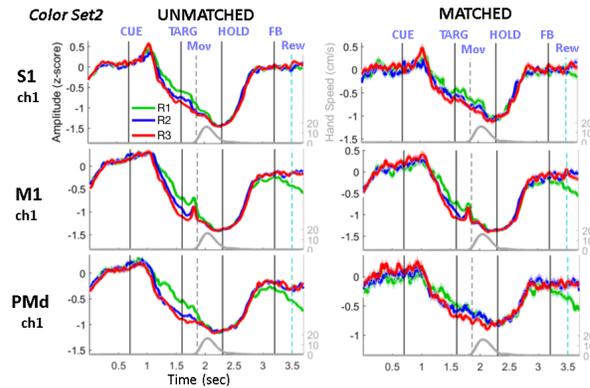


Figure 5. Modulation in beta amplitude with reward level (CS2) for unmatched and matched trials.

D. Graded suppression in beta amplitude not related to arm kinetics (EMG)

After controlling for the effects of differences in behavioral variables and choice of cue color, we analyzed EMG activity during the postcue period shown by the double arrow in Figure 6. Although the monkeys were not allowed to move their hand (> 1.5 cm/s) during the hold cue, residual EMG activations could have contributed to the observed beta amplitude. To address this possibility, we fit a similar linear model to trial-to-trial EMG amplitude as for beta amplitude and asked if the independent predictors explained the signal variance during the postcue period using only kinematically matched trials (CS2). No significant main effect of reward or reward-bin interaction on binned EMG amplitude was seen for any channel, indicating postcue EMG didn't vary with reward level and thus not related to the observed differences in beta amplitude.

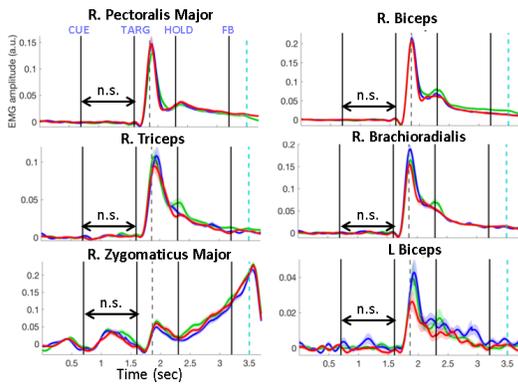


Figure 6 Mean EMG amplitude of different muscles for R1, R2, and R3 trials in the matched case (CS2).

IV. DISCUSSION AND CONCLUSION

We designed an arm-reaching task with variable reward levels (R1, R2 and R3) to study the role of S1, M1, and PMd cortical LFPs in reward processing. Beta band amplitude in all three cortices showed reward-dependent modulations beginning ~ 300 ms after cue presentation and continued until the movement onset. A higher expected reward level was associated with more postcue amplitude suppression compared to baseline. Amplitude differences were robustly detected from M1 and S1 cortices compared to the PMd region, as indicated by the number of channels exhibiting reward differences. It is unlikely that such beta amplitude modulation with reward was due to differences in behavioral variables, as beta differences continued to exist even after the variables were matched. We also showed that neither EMG activity nor choice of cue color changes the response of beta amplitude to reward level. However, prior to movement, beta reduction in our case could represent a preparatory response initiated by the presentation of reward predicting cue, in agreement with the previous report [17]. A decrease in beta power with an increase in reward level may also be related to gating of sensorimotor processes leading to increased cortical [18] and corticospinal excitability [19]. It could also represent differences in the level of motor preparation [20] and attention. Overall, we demonstrate that a simple LFP feature such as S1, M1, and PMd beta amplitude can consistently differentiate between reward levels even before any movement information is available. The presence of this signal in multiple cortices presents the potential of building RLBCI using fewer recording sites and implants.

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