# Basal ganglia activation localized in MEG using a reward task

Linnea Sepe-Forrest, Frederick Carver, Romain Quentin, Tom Holroyd, Allison Nugent

Neuroimage: Reports 1 (2021) 100034

Contents lists available at ScienceDirect

Neuroimage: Reports

journal homepage: www.sciencedirect.com/journal/neuroimage-reports

# Basal ganglia activation localized in MEG using a reward task☆

Linnea Sepe-Forrest [a,*], Frederick W. Carver [a], Romain Quentin [b], Tom Holroyd [a], Allison C. Nugent [a]

[a] Magnetoencephalography Core, National Institute of Mental Health, Bethesda, MD, USA
[b] Human Cortical Physiology and Neurorehabilitation Section, National Institute of Neurological Disorders and Stroke, Bethesda, MD, USA

ARTICLE INFO

*Keywords:*
Magnetoencephalography
Basal ganglia
Reward
Cortico-striatal
Gambling

ABSTRACT

The basal ganglia are a crucial component of neural networks underlying reward response and many other behaviors. Magnetoencephalography (MEG) can be used to non-invasively study the spatiotemporal dynamics of activity in neural networks. However, challenges associated with detecting deep sources has caused many to doubt the ability of MEG to detect basal ganglia signals. In this study, we employed a gambling task to assess the feasibility of using MEG to investigate basal ganglia-cortical networks during reward processing. Participants gambled to win or lose 5 or 25 cents and received unexpected high-value rewards of 50 cents at random intervals. We contrasted activity between reward conditions in the beta (15–30 Hz), gamma (30–60 Hz), and high gamma (60–150 Hz) bands. We found differences in oscillatory power in the beta and high gamma bands while contrasting the large reward condition with both the small reward and large loss conditions. Basal ganglia activity was localized to the caudate, putamen, and globus pallidum, while cortical activity appeared primarily in parietal and temporal areas. Our results show robust basal ganglia power differences in response to reward and corroborate animal literature showing beta and high gamma activation in the striatum. This experiment demonstrates that it is possible to study basal ganglia activity using MEG and reveals specific characteristics of the normal reward response that will inform future research.

## 1. Introduction

The basal ganglia play a crucial role in many aspects of human behavior including reward processing and motor control. Altered functioning has been associated with a variety of psychiatric and neurological conditions such as addiction (Cooper et al., 2017; Volkow et al., 2019), Parkinson's disease (Heimer et al., 2006; Singh, 2018), and depression (Forbes, 2019; Luyten and Fonagy, 2018). In all of these conditions, aberrant striatal activity and reward functioning have been linked to greater impairments in functioning (Aarts et al., 2012; Macpherson and Hikida, 2019). Additionally, prior studies suggest differences in basal ganglia activity may also indicate risk and severity of neurological illnesses (Petersson et al., 2020). While differences in basal ganglia activation have been linked to reward-related deficits, it is not clear how these changes occur on an electrophysiological level. To determine how striatal dysfunction may differ in brain-based disorders, it is first necessary attain a detailed understanding of the neural activity underlying the basic reward response.

Many functional magnetic resonance imaging (fMRI) studies have examined basal ganglia response in reward processing (Schultz, 2016a, b; Wang et al., 2016; McClure et al., 2004; Wang et al., 2016). These studies have found blood oxygenation level dependent (BOLD) changes, indirect correlates of neural activation, in cortical-basal ganglia networks. While fMRI cannot directly measure fast temporal or spectral patterns of response, electroencephalography (EEG) can be used to study oscillatory power and evoked responses by directly measuring neuronal activity through electrical changes. EEG has therefore allowed the detection of aberrant event related oscillations in response to reward that differ in addiction, depression, and other conditions (Keren et al., 2018; Luking et al., 2016; Stewart & May 2016). While previous

☆ All data is available upon request from the authors. This research was funded by the NIMH Intramural Research Program. This protocol, **NCT00397111**, was approved by the NIH Institutional Review Board. All participants provided written informed consent before participating in this study. The authors have no conflicts of interest.

* Corresponding author. Indiana University Bloomington, Department of Psychology, Room A208C, USA.
*E-mail addresses:* lisepe@iu.edu (L. Sepe-Forrest), carverf@nih.gov, fredcarver@gmail.com (F.W. Carver), rom.quentin@gmail.com (R. Quentin), holroydt@nih.gov (T. Holroyd), nugenta@nih.gov (A.C. Nugent).

https://doi.org/10.1016/j.ynirp.2021.100034
Received 6 March 2021; Received in revised form 18 June 2021; Accepted 5 July 2021
Available online 28 July 2021

experiments suggest that oscillatory power differences measured in EEG may originate from the basal ganglia, the exact sources contributing to altered reward processing remain largely unknown (Glazer et al., 2018; Keren et al., 2018).

To fully understand the neural characteristics of these reward networks, it is important to simultaneously measure where and when activity in basal ganglia and cortical areas occurs throughout reward anticipation and processing. Similar to EEG, magnetoencephalography (MEG) allows measurement of direct neuronal activity through magnetic fields. While the electric potentials measured by EEG may be smeared or disrupted by surrounding tissue, magnetic fields are not distorted while leaving the brain, making it easier to locate the original source (Hari and Puce, 2017). Therefore, is it possible to non-invasively localize the sources of oscillatory activity as well as study the temporal, spectral, and spatial characteristics of event-related responses through magnetic fields (Hari and Puce, 2017). Although this would suggest that MEG is an ideal tool to study reward processing, there has been doubt about the ability to detect deep subcortical sources such as the striatum (Attal et al., 2007). Therefore, the number of reward studies analyzing cortico-basal ganglia networks using MEG has been limited (Attal and Schwartz, 2013).

While the greater depth of basal ganglia sources results in lower signal-to-noise ratios compared to cortical sources, technical advances in instrumentation and beamforming methods can be used to successfully localize subcortical activity (Vrba and Robinson, 2001). Notably, deep structures such as the amygdala and hippocampus have been localized using these improved methods (Pizzo et al., 2019; Salvadore et al., 2010; Cornwell et al., 2008). Whereas the MEG signal is primarily composed of activity from cortical pyramidal cells oriented in parallel, the striatum is made up of medium spiny neurons (Steiner and Tseng, 2017). These cells are organized in bundles in a “closed-field” orientation that may produce a more limited electromagnetic field (Attal et al., 2007). Nevertheless, there is evidence that striatal regions still generate an overall directional current (Krishnaswamy et al., 2017; Rektor et al., 2005; Steiner and Tseng, 2017).

This will be the first study exclusively focused on testing whether it is possible to measure these signals through MEG. While a previous study in Parkinson’s patients found activity that appears to overlap with basal ganglia structures, authors suggest that it was not clear that this activity was localized in the basal ganglia (Timmermann et al., 2003). To elicit striatal activity, our study employs a gambling task based off a paradigm that elicited strong basal ganglia activation in fMRI by Riba et al. (2008). Similar to Riba et al., (2008), our task incorporates both win and loss trials, with an occasional high reward condition, known as the ‘Boost’ condition, where participants received a high reward greater than the amount they bet on. This Boost condition is thus unexpected as the participants do not expect to receive this reward given their chosen number. Riba et al., (2008) found the greatest BOLD response in the Boost condition, indicating that the unexpected reward may be critical in eliciting maximum basal ganglia activation. Therefore, we predict to see the greatest differences in striatal signal while contrasting activity between the Boost condition and lower reward or loss conditions in MEG. To ensure that activation is localized in basal ganglia, rather than leakage from cortical regions, we will measure activity across the entire brain before conducting detailed ROI tests on striatal regions that were significant in the whole brain analysis. Our design also targets basal ganglia activation by specifically analyzing beta, gamma, and high gamma frequency bands that have been detected in this region through animal studies and deep brain stimulation. Activity in these bands has been detected in the striatum and other basal ganglia areas during reward processing (Berke, 2009; Leventhal et al., 2012). Beta and high gamma activity have appeared during dopamine administration and reward delivery (Brown et al., 2001; A. A. Kuhn, Kupsch, Schneider and Brown, 2006; J. Kuhn et al., 2017), while gamma has been implicated in differentiating between varying types of reward (Irving, 2016; Kalenscher et al., 2010).

Our main hypothesis is that basal ganglia activity during reward processing can be localized with MEG. Specifically, we expect that beta power in basal ganglia areas will increase in response to loss and low reward conditions, while gamma and high gamma activity would increase in response to large rewards (Berke, 2009; Brown et al., 2001; Lega et al., 2011). We predict to see strong activity from the nucleus accumbens as this has region has been commonly associated with reward functioning in both neuroimaging and animal reward studies (Floresco, 2015; Liu et al., 2011; Salgado and Kaplitt, 2015) Additionally, we predict activity in the caudate and putamen will be detected as both areas have been associated with reward processing, specifically in response to reward prediction errors (Asaad and Eskandar, 2011; Sommer and Pollmann, 2016). We also expect to see activation in the globus pallidus as it has been involved in value encoding (Fiore et al., 2018; Tachibana and Hikosaka, 2012). While the primary purpose of this study is a proof of concept, an additional goal is to elucidate the spatiotemporal dynamics of the response to reward in basal ganglia and other areas. Overall, our study tests the feasibility of using MEG to detect precise, measurable subcortical and cortical reward related activity and shows the potential of MEG as a powerful tool in the field of reward research.

## 2. Methods

### 2.1. Participants

Twenty-five healthy volunteers with no personal or family history of psychiatric disorders (first-degree) participated in this study. Individuals completed diagnostic assessment via the Structured Clinical Interview for DSM-V-TR Disorders (SCID-NP) and the Family Interview for Genetics Studies (FIGS). Participants were enrolled in clinical trial NCT00397111 and provided written informed consent. This study included 21 females and 4 males with a mean age of 32 (SD = 9.0). The age of inclusion was 18–65 years old. Participants additionally had no serious medical illnesses, were not pregnant or nursing, and had no MRI contraindications.

### 2.2. Gambling task

The task was written with PsychoPy software (https://www.psychopy.org). Participants viewed stimuli while sitting upright in the MEG scanner. They received instructions that they would have the opportunity to gamble in each trial for either 5 or 25 cents (Fig. 1). The task consisted of 180 trials, split into 6 blocks of 30 trials each. At the beginning of each trial, participants saw two cards, one labeled 5 and one labeled 25. For each trial, there was a 50% chance of the 5 or 25 appearing on the left or right side. Participants were instructed to choose whether to “bet” 5 or 25 cents by left or right button press. After the cards were displayed for 2 s, the chosen card turned green (Win), red (Loss), or yellow (Boost) for 1 s. In expected reward trials, the participants won or lost the amount of money that they bet on. In congruent ‘Win5’ and ‘Win25’ trials the participants won 5 or 25 cents, while participants lost 5 or 25 cents in ‘Lose5’ and ‘Lose25’ trials, respectively. In ‘Boost’ (unexpected reward) trials, the number changed to 50, regardless of whether participants chose 5 or 25, and the participant’s total winnings increased by 50 cents. Each trial was coded to be a ‘win-loss’ trial or a ‘Boost’. On each win-loss trial participants had a 50% chance of winning or losing. Participants started with $0 and were told that they would not have money deducted from their compensation if their ending value was negative. There was an equal probability of winning or losing on each win-loss trial, and additional 50 cents on the Boost trials. Therefore, it was highly improbable for a subject to end with a negative value as the total earnings. Timing in this study was based on a similar gambling paradigm by Gehring and Willoughby (2002). The running total was displayed during the inter-stimulus interval, which lasted 1 s. Out of the 180 trials, participants randomly

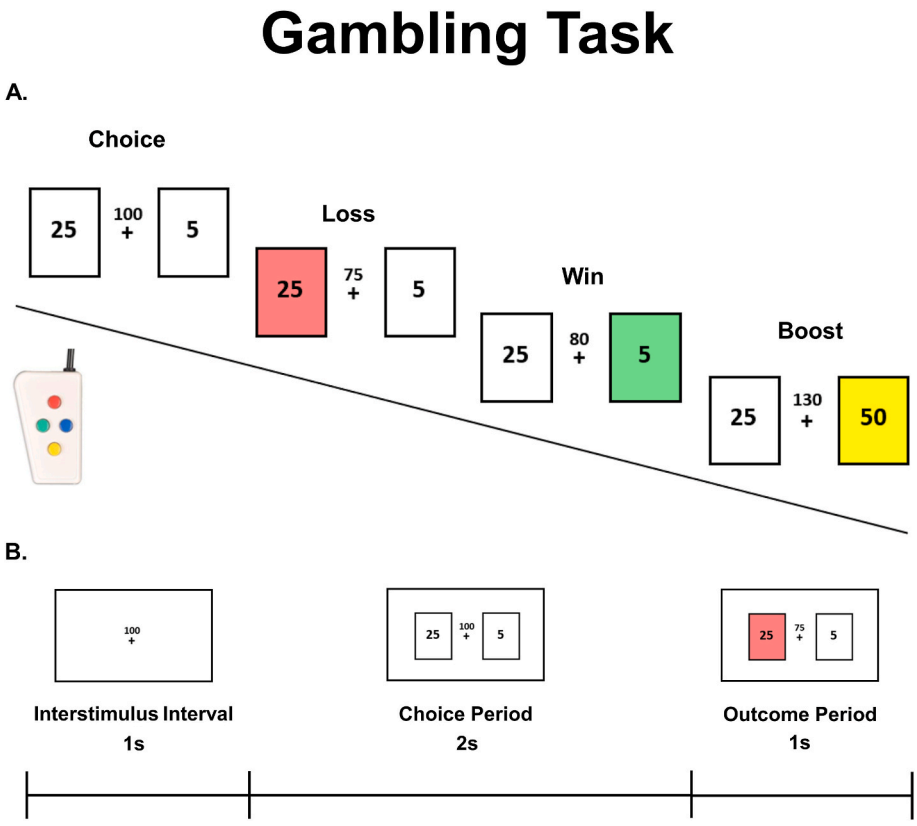


**Fig. 1.** Experimental design. A) Choice, Loss, Win, and Outcome trials. During the choice period, participants, 25 and 5. Within this period, participants 'bet' on a number by pressing the corresponding button (right button for the right number, left button for the left number). During the outcome period in trials without a Boost outcome, the box surrounding the chosen number would change colors with green indicating a win and red indicating a loss of the number of cents the subject chose to bet on. In trials with a Boost outcome, the box would turn yellow and the subject would gain 50 cents regardless of the chosen number. B). Timing information for each period within a single trial. The 1 s interstimulus trial consisted of fixation mark under the running total of monetary wins. Following the interstimulus trial was a 2 s choice period and 1 s outcome period.

received between 34 and 36 Boost trials. Because the participants could choose either 5 or 25, the numbers of trials for each quantity varied between participants. Participants with less than 20 of any trial type were excluded from analyses. The final number of participants in each contrast was 23 in Lose25 vs. Lose5, 22 in Win25 vs. Win5, 23 in Boost vs. Lose25, and 22 in Boost vs. Win5.

### 2.3. MEG and MRI data acquisition and preprocessing

MEG data were collected on a 275-channel CTF MEG system (CTF systems, Inc., Coquitlam, British Columbia, Canada) located in a magnetically shielded room. Fiducial coils were placed on participants' preauricular points and nasion to localize the position of the head inside the MEG system at the beginning and end of every recording. Data were digitally sampled at 1200 Hz after application of a quarter Nyquist low-pass filter at 300 Hz. Participants with excessive artifacts in their data or head movement exceeding .5 cm were excluded from the analysis. Line noise was removed from the data using a 3 Hz notch filter at 60 Hz and higher harmonics of 120, 180, and 240 Hz. Visual inspection in Data-Editor (CTF systems, Inc., Coquitlam, British Columbia, Canada) was used to mark eye blink, muscle, heartbeat and other common artifacts, and trials contained artifacts were removed from subsequent analysis. Additionally, remaining artifacts were reduced with SAM beamforming as these methods are designed to attenuate signal from outside the voxel (Sekihara & Nagarajan, 2008).

T1 weighted MRI images were acquired on a 3-T scanner (General Electric, Milwaukee, WI). Anatomical MRI images were co-registered with MEG data fiducial markers using Analysis of Functional Neuro-Images (AFNI) software (Cox, 1996). The MRI was used to generate a multisphere head model for source estimation (Huang et al., 1999). Data was transformed into common (MNI) space in AFNI and retained to apply to beamformed MEG images. Data preprocessing and analysis were conducted using the computational resources of the NIH HPC Biowulf cluster (http://hpc.nih.gov).

### 2.4. MEG data analysis

MEG data were localized to anatomical space using synthetic aperture magnetometry (SAM) beamforming on a 5 mm grid (Vrba and Robinson, 2001). SAM was used to estimate source power within the beta (15–30 Hz), gamma (30–60 Hz), and high gamma (60–150 Hz) frequency bands. Activity was analyzed during the 1 s reward feedback period in three overlapping time windows from 0 to 0.5s, 0.25–0.75s, and 0.5–1s. Power was estimated separately for each window and normalized by noise estimates based on signal covariance. The covariance matrix was summed across both conditions in each contrast, then inverted, to generate the beamformer. No regularization was used. To analyze power differences during the unexpected reward condition with the smallest reward and greatest loss conditions, contrasts were calculated for each participant as the log power ratio between pairs of conditions in the same time window. Activity in the Boost condition was separately contrasted with the Win5 and Lose25 conditions. To assess neural differences dependent on magnitude of reward valence (win or loss) the Lose5 condition was contrasted with activity in the Lose25 condition, and the Win25 condition was contrasted with the Win5 condition. Comparisons between the Boost condition and both Win25 and the Lose25 conditions were not performed as analyses were limited to contrasts with the largest differences in reward magnitude.

Results for each subject were transformed to common (MNI) space. We performed whole-brain group one-sample *t*-tests for each contrast using the AFNI program 3dttest++ to assess overall differences between reward trial types. We report results significant at $q < 0.05$, where q is the p-value corrected for false discovery rate (FDR). Our cluster defining threshold was set to $p = .001$ (uncorrected), and we report only clusters with a minimum size of 40 voxels. To report only the most salient regions we performed a cluster analysis using AFNI 3dclust with nearest neighbor (NN) of 2 (edges touching) and a minimum cluster size of 40. The identification of the anatomical area for each cluster was performed using the CA_ML_18_MNI atlas (Eickhoff et al., 2005). The full list of

regions showing statistically significant activity in these analyses can be found in supplementary materials (Supplementary Tables 1–4).

In order to confirm and add detail to the basal ganglia findings we performed two secondary analyses. First, to provide unbiased estimates of the effect size, we extracted anatomical ROIs for the caudate, putamen, and globus pallidus using the CA_ML_18_MNI atlas. For contrasts that showed statistically significant differences in these regions after FDR correction over the entire brain, we calculated the mean power difference for each subject across the entire anatomically defined ROI. One sample *t*-tests were used to determine significance, with an FDR threshold of $q < 0.05$ across ROIs, contrasts, frequency bands, and time windows. We then calculated Cohen's *d* effect sizes and the level of significance for each ROI. Finally, in order to provide additional information on the time course of activation for statistically significant ROIs we conducted a 3 d+time analysis in AFNI. SAM contrasts were calculated from a t = 0 to t = 1s time window and the time course of the activation of each frequency band was estimated at .1s sliding windows with a time step of .05s. Finally, to determine if leakage from neighboring cortical regions significantly influenced basal ganglia results, we additionally conducted sliding window ROI analyses for the insula (Supplementary Methods).

## 3. Results

### 3.1. Whole brain analysis: boost vs. Win5

In the whole brain analysis of the beta power band, decreased beta power occurred in the Boost condition compared to the Win5 condition during the 0.25-0.75s ($t(21) = 3.7921$, $p < .001$, $q < 0.005$) and the 0.5-1s time windows ($t(21) = 3.7921$, $p < .001$, $q < 0.002$) (Fig. 2A). Within basal ganglia regions, power was reduced in the caudate and putamen. This response primarily occurred on the right side in the 0.25-0.75s time window and the left side in the 0.5-1s time window.

There were no statistically significant differences observed in gamma band power. However, there were increases in power in the high gamma band in the Boost condition compared to the Win5 condition for the 0–.5s ($t(21) = 3.7921$, $p < .001$, $q < 0.005$), 0.25-0.75s ($t(21) = 3.7921$, $p < .001$, $q < 0.001$), and 0.5-1s time windows ($t(21) = 3.7921$, $p < .001$, $q < 0.001$) (Fig. 2B). Increases in power in the basal ganglia were observed in all three time windows. High gamma differences in the basal ganglia were right lateralized in the 0.25-0.75s window with bilateral activation occurring in the 0.5-1s time window.

For comparison and detection of possible leakage, insula results are shown in Supplemental Results and Supplemental Figs. 1 and 2. Other cortical and subcortical findings that were significant during the 0–.5s, 0.25-0.75s, and 0.5-1s time windows can be found in Supplemental Tables 1 and 2

### 3.2. Whole brain analysis: boost vs. Lose25

Decreases in beta power were observed in the Boost condition compared to the Lose25 condition during the 0.25-0.75s ($t(22) = 3.8193$, $p < .001$, $q < 0.003$) and the 0.5-1s time windows ($t(22) = 3.8193$, $p < .001$, $q < 0.002$). Within the striatum, there was a reduction in the left caudate and left putamen in the 0.5-1s time window. Basal ganglia activation during these time windows appeared within a cluster that included the middle temporal gyrus, cerebellum, and parietal areas.

As in the Boost vs. Win5 analysis, there were no statistically significant findings in the gamma band analyses. However, in the high gamma band analyses, we observed increased power in the Boost condition compared to the Lose25 condition in the 0.25-0.75s ($t(22) = 3.8193$, $p < .001$, $q < 0.014$) and 0.5-1s ($t(22) = 3.8193$, $p < .001$, $q < 0.050$)

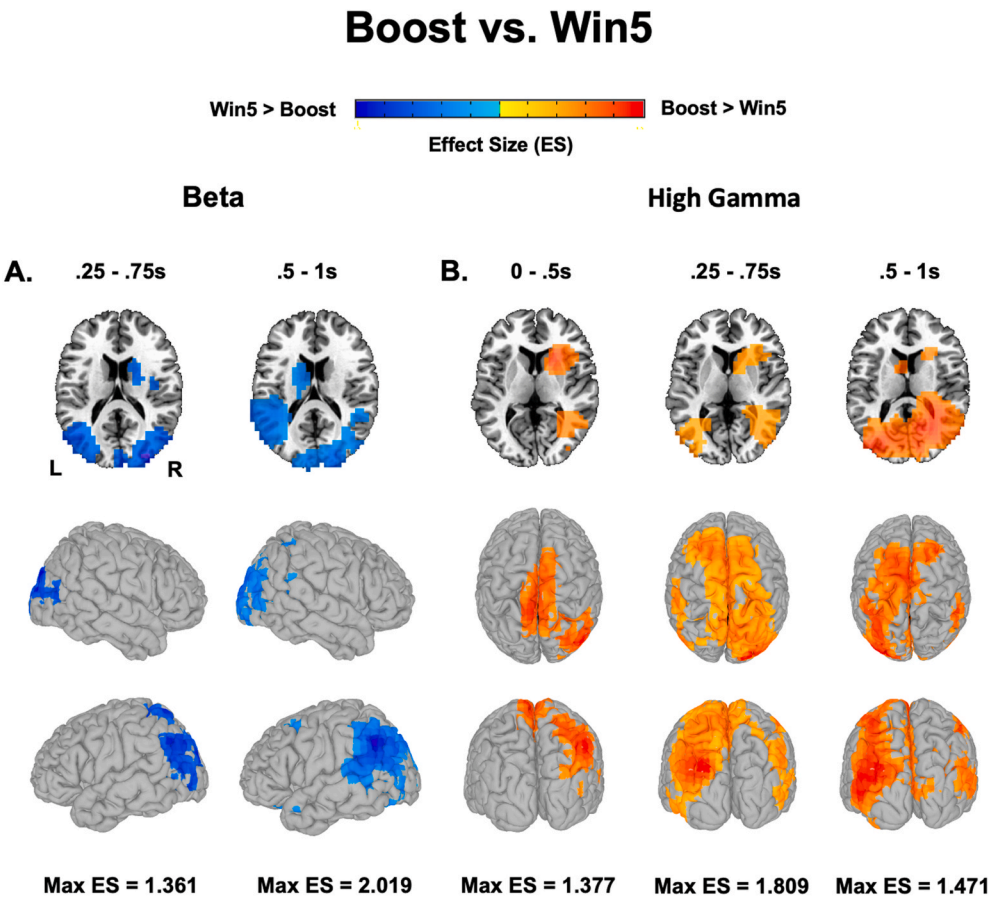


**Fig. 2.** Boost vs. Win5 subcortical and cortical activation differences between the two conditions thresholded at $p = .001$. Cortical data was displayed using SUMA-AFNI surface mapper (Saad and Reynolds, 2012). The activation color scale is the Cohen's *d* effect size of the difference. Max ES signifies the maximum effect size in each analysis, which was used to set the maximum and minimum of the color scale for each plot. Blue indicates greater activation in the Win5 condition, while red indicates greater activation in the Boost condition. A) Beta (15–30 Hz) band activation during the 0.25-0.75s and 0.5-1s time windows. B) High gamma (60–150 Hz) band activation during the 0–.5s, 0.25-0.75s, and 0.5-1s time windows.

analyses (Fig. 3B). While these effects did not meet our threshold for significance in basal ganglia regions, prominent increase in power were observed in the left superior parietal lobe, cuneus, inferior parietal lobe, angular gyrus, precuneus, and many other cortical areas.

Further discussion of possible leakage from cortical areas in the beta band assessed through the insula analysis is addressed in the Supplemental Results and Supplemental Fig. 1. All additional statistically significant cortical and subcortical results during the 0–.5s, 0.25-0.75s, and 0.5-1s time windows can be found in Supplemental Tables 3 and 4

### 3.3. Whole brain analysis: Other contrasts

We observed no statistically significant differences in power between the Win25 vs. Win5 or the Lose5 vs. Lose25 conditions for any frequency band or time window.

### 3.4. ROI analysis: boost vs. Win5

In the beta band, our post-hoc ROI analysis showed reduced power in the Boost condition compared to the Win5 condition during the 0.25-0.75s and 0.5-1s time windows (Table 1). These effects were found in the right caudate ($t(21) = -2.867$, $p = .009$, *Cohen's d* = 0.611) during the 0.25-0.75s time window, and the left caudate ($t(21) = -4.795$, $p < .001$, *Cohen's d* = 1.022) and left putamen ($t(21) = -2.788$, $p = .011$, *Cohen's d* = 0.594) during the 0.5-1s time window. Although the right putamen showed reduced power in the Boost condition in the whole brain analysis, but these effects were not statistically significant across the entire ROI ($t(21) = -1.934$, $p = .066$). Plotting the difference in beta power over smaller time windows to examine the temporal response (Fig. 4A) revealed the difference in power peaking sharply at 700 ms in the right caudate and putamen. For the left caudate and left putamen, differences in power between reward conditions showed a broader temporal distribution with the peak difference in both ROIs occurring at 800 ms post-feedback.

**Table 1**
ROI analyses of basal ganglia regions in Boost vs. Win5 contrast in the beta band.

| Beta Boost vs. Win5 | | | |
|---|---|---|---|
| ROI | Time | *p* | *d* |
| Right Caudate | .25-.75s | .009 | .611 |
| Left Caudate | .5-1s | <.001 | 1.022 |
| Right Putamen | .25-.75s | .066 | .412 |
| Left Putamen | .5-1s | .011 | .594 |

In the high gamma band, the ROI analysis confirmed increases in power in the Boost conditions during all three time windows (Table 2). In the 0–.5s window, we observed increased power in the right caudate ($t(21) = 3.628$, $p = .002$, *ES* = 0.774), right globus pallidus ($t(21) = 2.865$, $p = .009$, *Cohen's d* = 0.611), and right putamen ($t(21) = 3.608$, $p = .002$, *Cohen's d* = 0.769). Likewise, in the 0.25-0.75s window, power differences were detected in the right caudate ($t(21) = 4.605$, $p < .001$, *ES* = 0.981), left caudate ($t(21) = 2.674$, $p = .014$, *Cohen's d* = 0.570), right globus pallidus ($t(21) = 3.348$, $p = .003$, *Cohen's d* = 0.714), and the right putamen ($t(21) = 3.686$, $p = .001$, *Cohen's d* = 0.786). Finally, in the 0.5-1s window we found increased power in the right putamen ($t(21) = 2.955$, $p = .008$., *Cohen's d* = 0.630), left caudate ($t(21) = 2.818$, $p = .010$, *Cohen's d* = 0.601), and right caudate ($t(21) = 3.843$, $p < .001$, *Cohen's d* = 0.819). In the temporally resolved analysis (Fig. 5), we observed sustained power differences in the left caudate in the Boost vs.

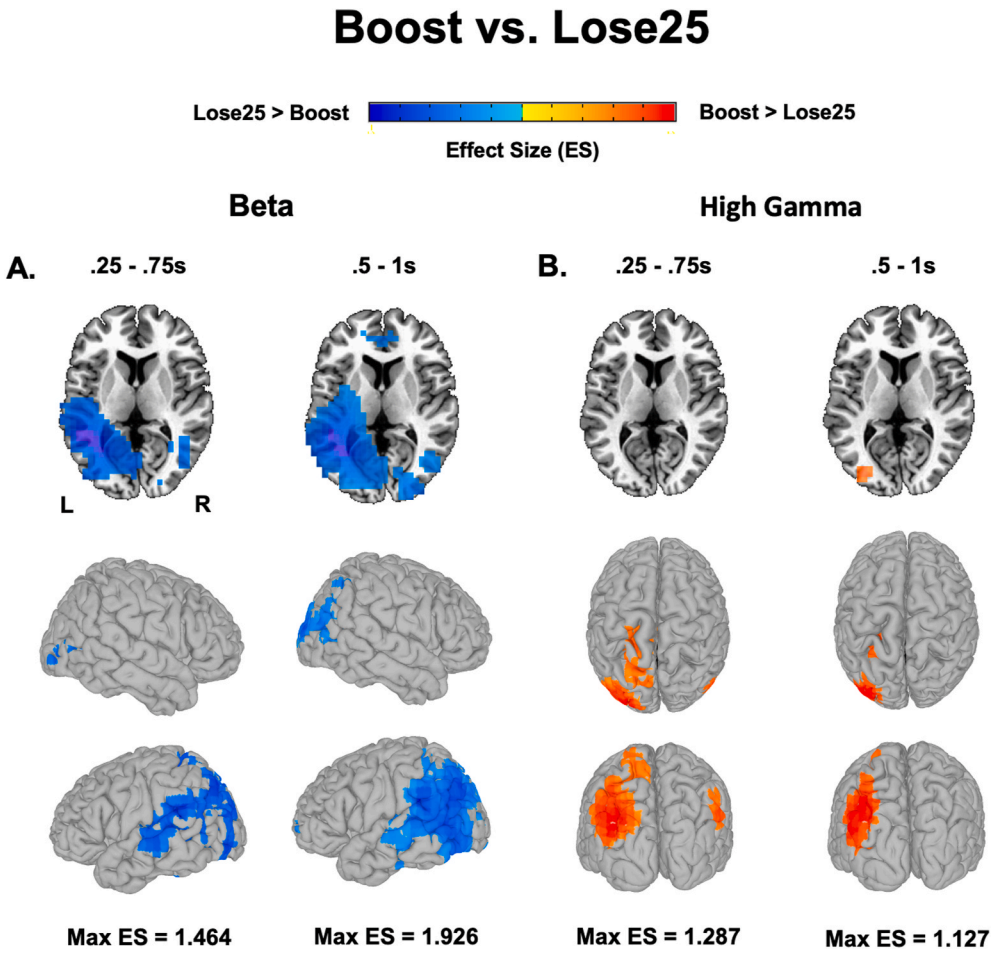


**Fig. 3.** Boost vs. Lose25 subcortical and cortical activation differences between the two conditions thresholded at $p = .001$. The activation color scale is the Cohen's *d* effect size of the difference. Max ES signifies the maximum effect size in each analysis, which was used to set the maximum and minimum of the color scale for each plot. Blue indicates greater activation in the Lose25 condition, while red indicates greater activation in the Boost condition. A) Beta (15–30 Hz) band activation during the 0.25-0.75s and 0.5-1s time windows. B) High gamma (60–150 Hz) band activation during the 0.25-0.75s, and 0.5-1s time windows.

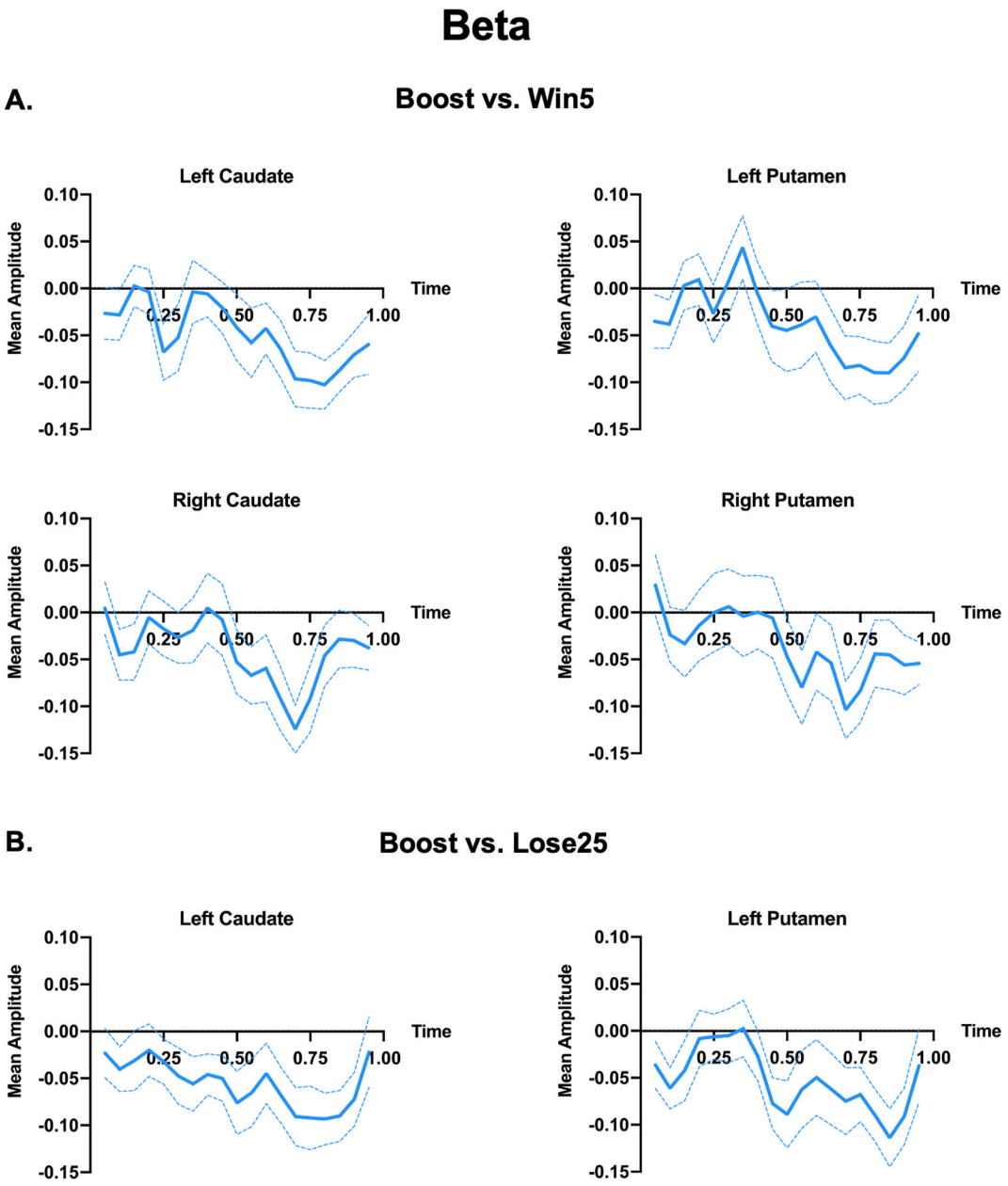


**Fig. 4.** Fractional change in log beta power in basal ganglia regions throughout the 1 s reward outcome period. Time series analysis shows mean amplitude over .1s intervals, plotted at the center of the interval. A) Boost vs. Win5 activation differences in left caudate, left putamen, right caudate, and right putamen. B) Boost vs. Lose25 activation differences in left caudate and left putamen.

**Table 2**
ROI analyses of basal ganglia regions in Boost vs. Win5 contrast in the high gamma band.

| High Gamma Boost vs. Win5 | | | |
|---|---|---|---|
| ROI | Time | *p* | *d* |
| Right Caudate | 0–.5s | .002 | .774 |
| | .25-.75s | <.001 | .981 |
| | .5-1s | <.001 | .819 |
| Right Putamen | 0–.5s | .002 | .769 |
| | .25-.75s | .001 | .786 |
| | .5-1s | .008 | .630 |
| Right Globus Pallidum | 0–.5s | .009 | .611 |
| | .25-.75s | .003 | .714 |
| Left Caudate | .25-.75s | .014 | .570 |
| | .5-1s | .010 | .601 |

Win5 contrast from 300 ms to 1s post-feedback. The right caudate, right putamen, and right globus pallidus showed similar time courses, with the strongest increases in power appearing between 300 and 600 ms.

### 3.5. ROI analysis: boost vs. Lose25 contrast

In our ROI analysis of the Boost vs. Lose25 condition, decreased power was observed in the beta band (Table 3). These effects were seen in the left caudate ($t(22) = -2.717$, $p = .0126$, *Cohen's d* = 0.566) and left putamen ($t(22) = -2.908$, $p = .008$, *Cohen's d* = 0.606). In the temporal analysis, the power differences in the left caudate and left putamen were greatest between 700 and 850 ms (Fig. 4B).

## 4. Discussion

This experiment demonstrated that it is possible to detect basal

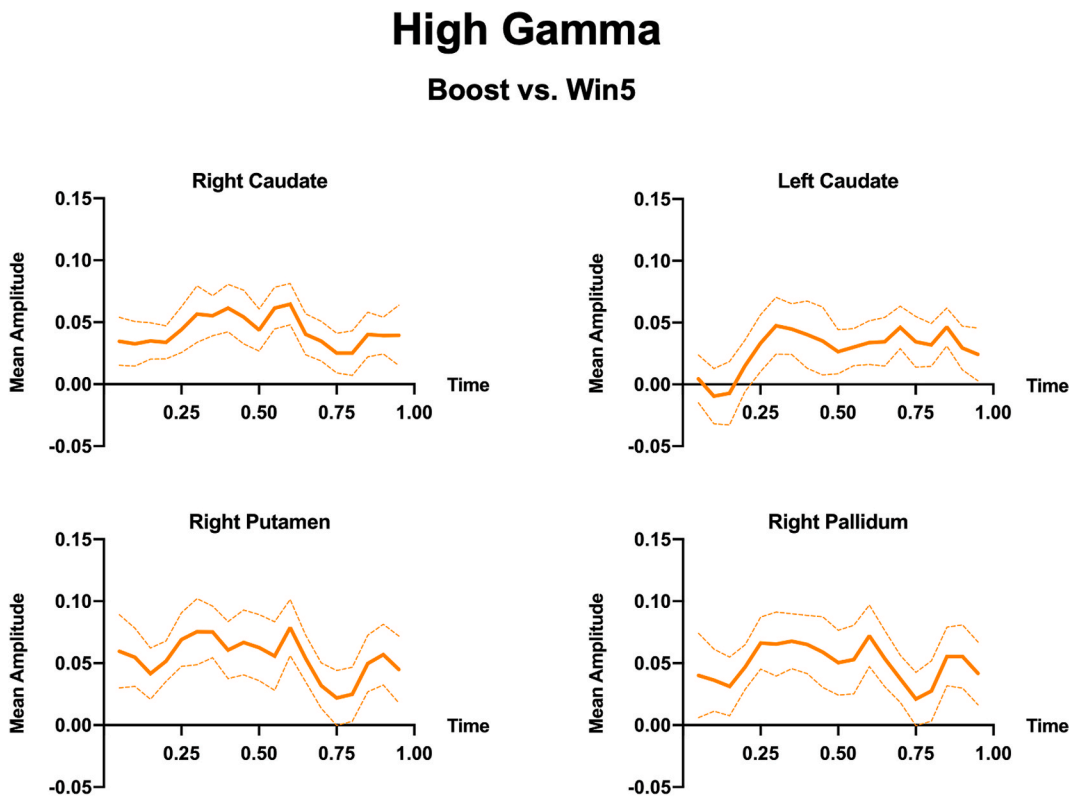


**Fig. 5.** Fractional change in log high gamma power in basal ganglia regions throughout the 1 s reward outcome period. Time series analysis shows mean amplitude over .1s intervals, plotted at the center of the interval. Boost vs. Win5 activation differences shown in left caudate, right caudate, right putamen, and right globus pallidum.

**Table 3**
ROI analyses of basal ganglia regions in Boost vs. Lose25 contrast in the beta band.

| Beta Boost vs. Lose25 | | | |
|---|---|---|---|
| ROI | Time | *p* | *d* |
| Left Caudate | .5-1s | .012 | .566 |
| Left Putamen | .5-1s | .008 | .606 |

**Cohen's D.*

ganglia responses during reward processing using MEG. There are several reasons why our experimental task was well suited to study basal ganglia sources. First, we based our MEG paradigm on an fMRI task that elicited strong basal ganglia activation in an unexpected reward, or Boost condition (Riba et al., 2008). In our study, only analyses with the Boost condition showed statistically significant basal ganglia activation differences, thereby demonstrating the importance of the unexpected reward condition. The importance of the boost condition in inducing measurable basal ganglia signal is underscored by the fact that we did not observe significant changes in power for contrasts between our highest expected win (Win25) or loss (Lose25) conditions and the lowest win (Win5) or loss (Lose5) conditions. A second strength of this task was the simple design that allowed us to focus on neural responses in contrasts with the only difference being the monetary reward. In addition to demonstrating the feasibility of detecting basal ganglia signals, this study revealed specific activation differences in response to the Boost condition compared to either a smaller reward (Win5) or a large loss (the Lose25). Surprisingly, this activity only appeared in the dorsal striatum and globus pallidus. Although the purpose of this paper was a proof of concept, rather than an investigation of reward behavior, our findings were nonetheless consistent with prior studies on reward behavior with basal ganglia activity shifting from beta to high gamma following reward receipt (see Berke, 2009).

We investigated power in frequency bands that have been directly recorded from basal ganglia regions in animal models during reward receipt (Berke, 2009; Kalenscher et al., 2010) and dopamine administration (J. Kuhn et al., 2017; West et al., 2018), specifically in the beta (15–30 Hz), gamma (30–60 Hz), and high gamma (60–150 Hz) bands. We did not see results in the gamma band which was unexpected, as we predicted to see a decrease activity based on prior findings in rodents showing a decrease in both beta and gamma activity after reward delivery (Berke, 2009). However, prior studies on gamma activation in response to reward have found conflicting results. While human studies using EEG have found increased beta-gamma (25–35 Hz) activity in cortico-striatal areas in response to reward, direct recording in animals suggest that gamma activation in higher frequencies (45–55 Hz and 70–85 Hz) are involved in positive reward response (HajiHosseini et al., 2012; Marco-Pallares et al., 2015; van der Meer and Redish, 2009). Therefore, the exact gamma response to reward across different frequency ranges remains unclear. While it is possible that gamma activity did not differ between reward conditions in this study, there also are possible reasons why these differences were not detected. Gamma is an intermediate band between beta and high gamma, thus larger effects of decreased beta and increased high gamma activation may obscure activity in the gamma band, leading to no detectable differences. Additionally, as cited above, the range of frequencies in the gamma band in other studies have been variable and it is possible that the frequency band chosen in our study (30–60 Hz) measures a different response than those observed in previous studies. Although gamma results did not align with our hypotheses, we did see decreased power in the beta band and increased power in the high gamma band in response to the Boost condition in basal ganglia areas.

Basal ganglia regions with statistically significant activation differences in these analyses include the caudate, putamen, and globus pallidus. These findings were expected as previous studies have indicated

these regions may play an important role in processing reward prediction errors (Asaad and Eskandar, 2011; Schultz, 2016a; Sommer and Pollmann, 2016). However, contrary to our hypotheses, we did not detect activity in the nucleus accumbens. This was surprising as the nucleus accumbens has been commonly associated with reward processing across numerous meta-analyses (Floresco, 2015; Liu et al., 2011; Salgado and Kaplitt, 2015). It is unclear why activation was not seen in this region. While is possible that the nucleus accumbens may not have been involved in the task, a similar paradigm in fMRI elicited strong activity from this area (Riba et al., 2008). Therefore, it is possible that this response was detectable through MRI, but not MEG, due to the characteristics of this structure and analysis methods in this study. Specifically, its small volume and greater depth from the MEG sensors relative to other basal ganglia regions may mean its signal is especially difficult to detect using MEG. Detecting signal from the nucleus accumbens in MEG may require a greater number of trials to increase signal to noise ratio, or a task exclusively focused on activating the nucleus accumbens. Importantly, nucleus accumbens volume is less than one tenth that of the caudate and putamen, which were detected in all contrasts with statistically significant results (Ahsan et al., 2007). Additionally, previous MEG studies indicate that it may be relatively more difficult to differentiate deep sources compared to areas closer to the surface, suggesting the present study may be limited in its ability to discriminate specific signal from the nucleus accumbens (Tierney et al., 2020).

Comparing results across different contrasts, both the Boost vs. Win5 and Boost vs. Lose25 contrasts revealed significant basal ganglia and cortical differences. However, only the contrast with the Win5 condition revealed significant basal ganglia differences in both the beta and high gamma band. In the Boost vs. Win5 analysis, these effects specifically appeared in the caudate and putamen, corroborating prior literature implicating these region's involvement in reward response (Arsalidou et al., 2020; Kasanova et al., 2017; Munte et al., 2017; Tricomi and Fiez, 2012). We found increased beta band power in the Win5 condition compared to Boost, which aligns with prior findings reporting increased basal ganglia beta power during lower arousal and negative feedback conditions in reward paradigms (Amemori et al., 2020; Lega et al., 2011; Yaple et al., 2018). Alternatively, these results may instead reflect the strong effect of reduced beta power in response to the Boost condition. Decreased beta activity may be indicative of a desynchronization event occurring due to increased neuronal activity (Brookes et al., 2011; Damodaran et al., 2015; Donner and Siegel, 2011; A. A. Kuhn et al., 2004). Neurons in the striatum have been shown to engage and disengage in synchronous beta oscillations during reward seeking, suggesting this dynamic activity may play an important role in modulating neural responses to feedback (Courtemanche et al., 2003). In addition to decreased beta power, we also observed increased high gamma activity in the Boost condition compared to the Win5 condition. Taken together, these results mirror observations of the reward response in rodents, with activity shifting from beta to high gamma during the reward outcome period (Berke, 2009).

Similar to the Boost vs. Win 5 contrast, increased beta and decreased high gamma activity occurred in the Boost condition compared to the Lose25 condition in cortical regions. Within basal ganglia, significant effects only occurred in the beta band as opposed to in both beta and high gamma bands in the previous contrast. Thus, only beta activity in these regions appeared to differ based on the reward valence. This finding is consistent with a recent study conducted by Amemori et al. (2020) that found beta activity in the basal ganglia differentiated 'high-punishment' and 'high reward conditions'. Alternatively, this activity may reflect beta desynchronization in response to the Boost condition as discussed prior. The basal ganglia activation in this contrast appeared in the latest time window within a large cluster peaking in the left middle temporal lobe. However, visual inspection (Fig. 3) suggests these beta power effects may not be differentiable from a large cortical cluster peaking in the left middle temporal cortex. While activity in the earlier time window is primarily centered on the temporal cortex, the later time window shows activation spreading broadly to surrounding subcortical, cerebellar, parietal, and occipital areas. Prior studies indicate that the left middle temporal cortex may act as a 'cortical hub' and play an important role in processing the meaning of context-dependent words or objects (Binder and Desai, 2011; Mollo et al., 2018; Noonan et al., 2013; Whitney et al., 2011). It is possible that in our study this region shows the greatest sensitivity to differences between reward and loss conditions because this area is involved in interpreting the value of the outcome. Our results in the beta band may reflect an early temporal response assessing the meaning of the outcome before transmitting this information to basal ganglia and higher-level processing areas.

Contrary to our prediction, we did not see differences in basal ganglia signals in the high gamma band when contrasting Boost condition with the Lose25 condition. It is possible that these high frequency responses are sensitive to differences between outcome magnitudes, but not between positive and negative outcomes. Notably, a study conducted by Cohen et al. (2009) saw no differences in high frequency activity (50–80 Hz) between win and loss conditions while directly recording striatal response. These findings may indicate that certain areas in the basal ganglia respond similarly in high reward and loss conditions, as the striatum has been shown to be also involved in processing losses (Palminteri and Pessiglione, 2017; Pessiglione et al., 2006; Seymour et al., 2007). While basal ganglia activation was not detected, many cortical areas did show response differences between Boost and Lose25 conditions in the high gamma band. Activation differences were mostly localized to parietal and occipital regions, both of which have been involved in visually processing reward outcomes and interpreting their meaning (Hawellek et al., 2016; Wilson et al., 2018). Taken with our Boost vs. Win5 results, these results suggest that beta and high gamma power changes in cortical areas may differ based on both the outcome magnitude and type of outcome (reward or loss), while striatal regions may specifically show differences in the beta band based on outcome magnitude. In future studies, it would be beneficial to compare a 5 cent loss condition with a 50 cent loss condition to clarify whether high gamma differences did not occur due to a strong striatal response in loss, or less basal ganglia activation in both conditions.

This study shows that it is possible to use MEG to detect striatal reward response and detailed characteristics of this activity. Understanding the potential of MEG to measure these responses opens the door for future studies to utilize this technology to non-invasively detect differences in oscillatory activity in basal ganglia - cortical networks. While the main goal of this study was to detect basal ganglia signal, our results detected striatal responses that were consistent with previous reward studies. These results suggest that, in order to study striatal response to reward, it is important to measure high frequency activity in addition to beta and other lower frequency bands. Additionally, this experiment demonstrates that this task paradigm, which has not been used in electrophysiological experiments before, elicits strong basal ganglia activation that can be measured with MEG. While further research is still needed to understand the details of basal ganglia - cortical reward processing, this study demonstrated the feasibility of detecting basal ganglia signal.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Acknowledgements

We would like to thank Dr. Joyce Chung and members of the NIMH Healthy Research Volunteer Study, as well as Dr. Carlos Zarate and the Experimental Therapeutics and Pathophysiology Branch for assisting with the recruitment of participants. We would also like to thank all of

the healthy volunteers who participated in this experiment.

This project was supported by the National Institute of Mental Health Intramural Research Program.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ynirp.2021.100034.

## References


Aarts, E., Helmich, R.C., Janssen, M.J., Oyen, W.J., Bloem, B.R., Cools, R., 2012. Aberrant reward processing in Parkinson's disease is associated with dopamine cell loss. Neuroimage 59 (4), 3339–3346. https://doi.org/10.1016/j.neuroimage.2011.11.073.

Amemori, K.I., Amemori, S., Gibson, D.J., Graybiel, A.M., 2020. Striatal beta oscillation and neuronal activity in the primate caudate nucleus differentially represent valence and arousal under approach-avoidance conflict. Front. Neurosci. 14, 89. https://doi.org/10.3389/fnins.2020.00089.

Ahsan, R.L., Allom, R., Gousias, I.S., Habib, H., Turkheimer, F.E., Free, S., Hammers, A., 2007. Volumes, spatial extents and a probabilistic atlas of the human basal ganglia and thalamus. Neuroimage 38 (2), 261–270. https://doi.org/10.1016/j.neuroimage.2007.06.004.

Arsalidou, M., Vijayarajah, S., Sharaev, M., 2020. Basal ganglia lateralization in different types of reward. Brain Imaging Behav. https://doi.org/10.1007/s11682-019-00215-3.

Asaad, W.F., Eskandar, E.N., 2011. Encoding of both positive and negative reward prediction errors by neurons of the primate lateral prefrontal cortex and caudate nucleus. J. Neurosci. 31 (49), 17772–17787. https://doi.org/10.1523/JNEUROSCI.3793-11.2011.

Attal, Y., Bhattacharjee, M., Yelnik, J., Cottereau, B., Lefevre, J., Okada, Y., Baillet, S., 2007. Modeling and detecting deep brain activity with MEG & EEG. Annu Int Conf IEEE Eng Med Biol Soc 2007, 4937–4940. https://doi.org/10.1109/IEMBS.2007.4353448.

Attal, Y., Schwartz, D., 2013. Assessment of subcortical source localization using deep brain activity imaging model with minimum norm operators: a MEG study. PLoS One 8 (3), e59856. https://doi.org/10.1371/journal.pone.0059856.

Berke, J.D., 2009. Fast oscillations in cortical-striatal networks switch frequency following rewarding events and stimulant drugs. Eur. J. Neurosci. 30 (5), 848–859. https://doi.org/10.1111/j.1460-9568.2009.06843.x.

Binder, J.R., Desai, R.H., 2011. The neurobiology of semantic memory. Trends Cognit. Sci. 15 (11), 527–536. https://doi.org/10.1016/j.tics.2011.10.001.

Brookes, M.J., Wood, J.R., Stevenson, C.M., Zumer, J.M., White, T.P., Liddle, P.F., Morris, P.G., 2011. Changes in brain network activity during working memory tasks: a magnetoencephalography study. Neuroimage 55 (4), 1804–1815. https://doi.org/10.1016/j.neuroimage.2010.10.074.

Brown, P., Oliviero, A., Mazzone, P., Insola, A., Tonali, P., Di Lazzaro, V., 2001. Dopamine dependency of oscillations between subthalamic nucleus and pallidum in Parkinson's disease. J. Neurosci. 21 (3), 1033–1038. Retrieved from. https://www.ncbi.nlm.nih.gov/pubmed/11157088.

Cohen, M.X., Axmacher, N., Lenartz, D., Elger, C.E., Sturm, V., Schlaepfer, T.E., 2009. Good vibrations: cross-frequency coupling in the human nucleus accumbens during reward processing. J. Cognit. Neurosci. 21 (5), 875–889. https://doi.org/10.1162/jocn.2009.21062.

Cooper, S., Robison, A.J., Mazei-Robison, M.S., 2017. Reward circuitry in addiction. Neurotherapeutics 14 (3), 687–697. https://doi.org/10.1007/s13311-017-0525-z.

Cornwell, B.R., Carver, F.W., Coppola, R., Johnson, L., Alvarez, R., Grillon, C., 2008. Evoked amygdala responses to negative faces revealed by adaptive MEG beamformers. Brain Res. 1244, 103–112. https://doi.org/10.1016/j.brainres.2008.09.068.

Courtemanche, R., Fujii, N., Graybiel, A.M., 2003. Synchronous, focally modulated beta-band oscillations characterize local field potential activity in the striatum of awake behaving monkeys. J. Neurosci. 23 (37), 11741–11752. Retrieved from. https://www.ncbi.nlm.nih.gov/pubmed/14684876.

Cox, R.W., 1996. AFNI: software for analysis and visualization of functional magnetic resonance neuroimages. Comput. Biomed. Res. 29 (3), 162–173. https://doi.org/10.1006/cbmr.1996.0014.

Damodaran, S., Cressman, J.R., Jedrzejewski-Szmek, Z., Blackwell, K.T., 2015. Desynchronization of fast-spiking interneurons reduces beta-band oscillations and imbalance in firing in the dopamine-depleted striatum. J. Neurosci. 35 (3), 1149–1159. https://doi.org/10.1523/JNEUROSCI.3490-14.2015.

Donner, T.H., Siegel, M., 2011. A framework for local cortical oscillation patterns. Trends Cognit. Sci. 15 (5), 191–199. https://doi.org/10.1016/j.tics.2011.03.007.

Eickhoff, S.B., Stephan, K.E., Mohlberg, H., Grefkes, C., Fink, G.R., Amunts, K., Zilles, K., 2005. A new SPM toolbox for combining probabilistic cytoarchitectonic maps and functional imaging data. Neuroimage 25 (4), 1325–1335. https://doi.org/10.1016/j.neuroimage.2004.12.034.

Floresco, S.B., 2015. The nucleus accumbens: an interface between cognition, emotion, and action. Annu. Rev. Psychol. 66, 25–52. https://doi.org/10.1146/annurev-psych-010213-115159.

Fiore, V.G., Nolte, T., Rigoli, F., Smittenaar, P., Gu, X., Dolan, R.J., 2018. Value encoding in the globus pallidus: fMRI reveals an interaction effect between reward and dopamine drive. Neuroimage 173, 249–257. https://doi.org/10.1016/j.neuroimage.2018.02.048.

Forbes, E.E., 2019. Editorial: missing the Pull of Life's rewards? Attention shifting and the development of depression. J. Am. Acad. Child Adolesc. Psychiatry 58 (3), 313–314. https://doi.org/10.1016/j.jaac.2018.07.907.

Gehring, W.J., Willoughby, A.R., 2002. The medial frontal cortex and the rapid processing of monetary gains and losses. Science 295 (5563), 2279–2282. https://doi.org/10.1126/science.1066893.

Glazer, J.E., Kelley, N.J., Pornpattananangkul, N., Mittal, V.A., Nusslock, R., 2018. Beyond the FRN: broadening the time-course of EEG and ERP components implicated in reward processing. Int. J. Psychophysiol. 132 (Pt B), 184–202. https://doi.org/10.1016/j.ijpsycho.2018.02.002.

HajiHosseini, A., Rodriguez-Fornells, A., Marco-Pallares, J., 2012. The role of beta-gamma oscillations in unexpected rewards processing. Neuroimage 60 (3), 1678–1685. https://doi.org/10.1016/j.neuroimage.2012.01.125.

Hari, R., Puce, A., 2017. MEG-EEG Primer. Oxford University Press, New York, NY.

Hawellek, D.J., Wong, Y.T., Pesaran, B., 2016. Temporal coding of reward-guided choice in the posterior parietal cortex. Proc. Natl. Acad. Sci. U. S. A. 113 (47), 13492–13497. https://doi.org/10.1073/pnas.1606479113.

Heimer, G., Rivlin, M., Israel, Z., Bergman, H., 2006. Synchronizing activity of basal ganglia and pathophysiology of Parkinson's disease. J. Neural. Transm. Suppl (70), 17–20. https://doi.org/10.1007/978-3-211-45295-0_4.

Huang, M.X., Mosher, J.C., Leahy, R.M., 1999. A sensor-weighted overlapping-sphere head model and exhaustive head model comparison for MEG. Phys. Med. Biol. 44 (2), 423–440. https://doi.org/10.1088/0031-9155/44/2/010.

Irving, J.M., 2016. The Role of Local Activity of the Nucleus Accumbens in Reward : Interneurons and Gamma Oscillations (Dissertation). University of Maryland Medical School, UMB Digital Archive database.

Kalenscher, T., Lansink, C.S., Lankelma, J.V., Pennartz, C.M., 2010. Reward-associated gamma oscillations in ventral striatum are regionally differentiated and modulate local firing activity. J. Neurophysiol. 103 (3), 1658–1672. https://doi.org/10.1152/jn.00432.2009.

Kasanova, Z., Ceccarini, J., Frank, M.J., Amelsvoort, T.V., Booij, J., Heinzel, A., Myin-Germeys, I., 2017. Striatal dopaminergic modulation of reinforcement learning predicts reward-oriented behavior in daily life. Biol. Psychol. 127, 1–9. https://doi.org/10.1016/j.biopsycho.2017.04.014.

Keren, H., O'Callaghan, G., Vidal-Ribas, P., Buzzell, G.A., Brotman, M.A., Leibenluft, E., Stringaris, A., 2018. Reward processing in depression: a conceptual and meta-analytic Review across fMRI and EEG studies. Am. J. Psychiatr. 175 (11), 1111–1120. https://doi.org/10.1176/appi.ajp.2018.17101124.

Krishnaswamy, P., Obregon-Henao, G., Ahveninen, J., Khan, S., Babadi, B., Iglesias, J.E., Purdon, P.L., 2017. Sparsity enables estimation of both subcortical and cortical activity from MEG and EEG. Proc. Natl. Acad. Sci. U. S. A. 114 (48), E10465–E10474. https://doi.org/10.1073/pnas.1705414114.

Kuhn, A.A., Kupsch, A., Schneider, G.H., Brown, P., 2006. Reduction in subthalamic 8-35 Hz oscillatory activity correlates with clinical improvement in Parkinson's disease. Eur. J. Neurosci. 23 (7), 1956–1960. https://doi.org/10.1111/j.1460-9568.2006.04717.x.

Kuhn, A.A., Williams, D., Kupsch, A., Limousin, P., Hariz, M., Schneider, G.H., Brown, P., 2004. Event-related beta desynchronization in human subthalamic nucleus correlates with motor performance. Brain 127 (Pt 4), 735–746. https://doi.org/10.1093/brain/awh106.

Kuhn, J., Haumesser, J.K., Beck, M.H., Altschuler, J., Kuhn, A.A., Nikulin, V.V., van Riesen, C., 2017. Differential effects of levodopa and apomorphine on neuronal population oscillations in the cortico-basal ganglia loop circuit in vivo in experimental parkinsonism. Exp. Neurol. 298 (Pt A), 122–133. https://doi.org/10.1016/j.expneurol.2017.09.005.

Lega, B.C., Kahana, M.J., Jaggi, J., Baltuch, G.H., Zaghloul, K., 2011. Neuronal and oscillatory activity during reward processing in the human ventral striatum. Neuroreport 22 (16), 795–800. https://doi.org/10.1097/WNR.0b013e32834b2975.

Leventhal, D.K., Gage, G.J., Schmidt, R., Pettibone, J.R., Case, A.C., Berke, J.D., 2012. Basal ganglia beta oscillations accompany cue utilization. Neuron 73 (3), 523–536. https://doi.org/10.1016/j.neuron.2011.11.032.

Liu, X., Hairston, J., Schrier, M., Fan, J., 2011. Common and distinct networks underlying reward valence and processing stages: a meta-analysis of functional neuroimaging studies. Neurosci. Biobehav. Rev. 35 (5), 1219–1236. https://doi.org/10.1016/j.neubiorev.2010.12.012.

Luking, K.R., Pagliaccio, D., Luby, J.L., Barch, D.M., 2016. Reward processing and risk for depression across development. Trends Cognit. Sci. 20 (6), 456–468. https://doi.org/10.1016/j.tics.2016.04.002.

Luyten, P., Fonagy, P., 2018. The stress-reward-mentalizing model of depression: an integrative developmental cascade approach to child and adolescent depressive disorder based on the Research Domain Criteria (RDoC) approach. Clin. Psychol. Rev. 64, 87–98. https://doi.org/10.1016/j.cpr.2017.09.008.

Marco-Pallares, J., Munte, T.F., Rodriguez-Fornells, A., 2015. The role of high-frequency oscillatory activity in reward processing and learning. Neurosci. Biobehav. Rev. 49, 1–7. https://doi.org/10.1016/j.neubiorev.2014.11.014.

McClure, S.M., York, M.K., Montague, P.R., 2004. The neural substrates of reward processing in humans: the modern role of FMRI. Neuroscientist 10 (3), 260–268. https://doi.org/10.1177/1073858404263526.

Macpherson, T., Hikida, T., 2019. Role of basal ganglia neurocircuitry in the pathology of psychiatric disorders. Psychiatr. Clin. Neurosci. 73 (6), 289–301. https://doi.org/10.1111/pcn.12830.

Mollo, G., Jefferies, E., Cornelissen, P., Gennari, S.P., 2018. Context-dependent lexical ambiguity resolution: MEG evidence for the time-course of activity in left inferior

frontal gyrus and posterior middle temporal gyrus. Brain Lang. 177–178, 23–36. https://doi.org/10.1016/j.bandl.2018.01.001.
Munte, T.F., Marco-Pallares, J., Bolat, S., Heldmann, M., Lutjens, G., Nager, W., Krauss, J.K., 2017. The human globus pallidus internus is sensitive to rewards - evidence from intracerebral recordings. Brain Stimul 10 (3), 657–663. https://doi.org/10.1016/j.brs.2017.01.004.
Noonan, K.A., Jefferies, E., Visser, M., Lambon Ralph, M.A., 2013. Going beyond inferior prefrontal involvement in semantic control: evidence for the additional contribution of dorsal angular gyrus and posterior middle temporal cortex. J. Cognit. Neurosci. 25 (11), 1824–1850. https://doi.org/10.1162/jocn_a_00442.
Palminteri, S., Pessiglione, M., 2017. Opponent brain systems for reward adn punishment learning: causal evidence from drug and lesion studies in humans. In: Dreher, J.-C., Tremblay, L.o. (Eds.), Decision Neuroscience : an Integrative Perspective. Elsevier/AP, Academic Press is an imprint of Elsevier, Amsterdam, pp. 291–303.
Pessiglione, M., Seymour, B., Flandin, G., Dolan, R.J., Frith, C.D., 2006. Dopamine-dependent prediction errors underpin reward-seeking behaviour in humans. Nature 442 (7106), 1042–1045. https://doi.org/10.1038/nature05051.
Petersson, P., Kuhn, A.A., Neumann, W.J., Fuentes, R., 2020. Basal ganglia oscillations as biomarkers for targeting circuit dysfunction in Parkinson's disease. Prog. Brain Res. 252, 525–557. https://doi.org/10.1016/bs.pbr.2020.02.002.
Pizzo, F., Roehri, N., Medina Villalon, S., Trebuchon, A., Chen, S., Lagarde, S., Benar, C. G., 2019. Deep brain activities can be detected with magnetoencephalography. Nat. Commun. 10 (1), 971. https://doi.org/10.1038/s41467-019-08665-5.
Rektor, I., Bares, M., Brazdil, M., Kanovsky, P., Rektorova, I., Sochurkova, D., Daniel, P., 2005. Cognitive- and movement-related potentials recorded in the human basal ganglia. Mov. Disord. 20 (5), 562–568. https://doi.org/10.1002/mds.20368.
Riba, J., Kramer, U.M., Heldmann, M., Richter, S., Munte, T.F., 2008. Dopamine agonist increases risk taking but blunts reward-related brain activity. PLoS One 3 (6), e2479. https://doi.org/10.1371/journal.pone.0002479.
Saad, Z.S., Reynolds, R.C., 2012. Suma. Neuroimage 62 (2), 768–773. https://doi.org/10.1016/j.neuroimage.2011.09.016.
Salgado, S., Kaplitt, M.G., 2015. The nucleus accumbens: a comprehensive Review. Stereotact. Funct. Neurosurg. 93 (2), 75–93. https://doi.org/10.1159/000368279.
Salvadore, G., Cornwell, B.R., Sambataro, F., Latov, D., Colon-Rosario, V., Carver, F., Zarate Jr., C.A., 2010. Anterior cingulate desynchronization and functional connectivity with the amygdala during a working memory task predict rapid antidepressant response to ketamine. Neuropsychopharmacology 35 (7), 1415–1422. https://doi.org/10.1038/npp.2010.24.
Schultz, W., 2016a. Dopamine reward prediction error coding. Dialogues Clin. Neurosci. 18 (1), 23–32.
Schultz, W., 2016b. Reward functions of the basal ganglia. J. Neural. Transm. 123 (7), 679–693. https://doi.org/10.1007/s00702-016-1510-0.
Sekihara, K., Nagarajan, S.S., 2008. Adaptive spatial filters. In: Adaptive Spatial Filters for Electromagnetic Brain Imaging. Springer Berlin Heidelberg, Berlin, Heidelberg, pp. 37–63.
Seymour, B., Daw, N., Dayan, P., Singer, T., Dolan, R., 2007. Differential encoding of losses and gains in the human striatum. J. Neurosci. 27 (18), 4826–4831. https://doi.org/10.1523/JNEUROSCI.0400-07.2007.
Singh, A., 2018. Oscillatory activity in the cortico-basal ganglia-thalamic neural circuits in Parkinson's disease. Eur. J. Neurosci. 48 (8), 2869–2878. https://doi.org/10.1111/ejn.13853.
Sommer, S., Pollmann, S., 2016. Putamen activation represents an intrinsic positive prediction error signal for visual search in repeated configurations. Open Neuroimaging J. 10, 126–138. https://doi.org/10.2174/1874440001610010126.
Steiner, H., Tseng, K.-Y., 2017. Handbook of Basal Ganglia Structure and Function, second ed. Elsevier/Academic Press, Amsterdam ; Boston.
Stewart, J.L., May, A.C., 2016. Electrophysiology for addiction medicine: from methodology to conceptualization of reward deficits. Prog. Brain Res. 224, 67–84. https://doi.org/10.1016/bs.pbr.2015.07.013.
Tachibana, Y., Hikosaka, O., 2012. The primate ventral pallidum encodes expected reward value and regulates motor action. Neuron 76 (4), 826–837. https://doi.org/10.1016/j.neuron.2012.09.030.
Tierney, T.M., Mellor, S., O'Neill, G.C., Holmes, N., Boto, E., Roberts, G., Barnes, G.R., 2020. Pragmatic spatial sampling for wearable MEG arrays. Sci. Rep. 10 (1), 21609. https://doi.org/10.1038/s41598-020-77589-8.
Timmermann, L., Gross, J., Dirks, M., Volkmann, J., Freund, H.J., Schnitzler, A., 2003. The cerebral oscillatory network of parkinsonian resting tremor. Brain 126 (Pt 1), 199–212. https://doi.org/10.1093/brain/awg022.
Tricomi, E., Fiez, J.A., 2012. Information content and reward processing in the human striatum during performance of a declarative memory task. Cognit. Affect Behav. Neurosci. 12 (2), 361–372. https://doi.org/10.3758/s13415-011-0077-3.
van der Meer, M.A., Redish, A.D., 2009. Low and high gamma oscillations in rat ventral striatum have distinct relationships to behavior, reward, and spiking activity on a Learned spatial decision task. Front. Integr. Neurosci. 3, 9. https://doi.org/10.3389/neuro.07.009.2009.
Volkow, N.D., Michaelides, M., Baler, R., 2019. The neuroscience of drug reward and addiction. Physiol. Rev. 99 (4), 2115–2140. https://doi.org/10.1152/physrev.00014.2018.
Vrba, J., Robinson, S.E., 2001. Signal processing in magnetoencephalography. Methods 25 (2), 249–271. https://doi.org/10.1006/meth.2001.1238.
Wang, K.S., Smith, D.V., Delgado, M.R., 2016. Using fMRI to study reward processing in humans: past, present, and future. J. Neurophysiol. 115 (3), 1664–1678. https://doi.org/10.1152/jn.00333.2015.
West, T.O., Berthouze, L., Halliday, D.M., Litvak, V., Sharott, A., Magill, P.J., Farmer, S. F., 2018. Propagation of beta/gamma rhythms in the cortico-basal ganglia circuits of the parkinsonian rat. J. Neurophysiol. 119 (5), 1608–1628. https://doi.org/10.1152/jn.00629.2017.
Whitney, C., Jefferies, E., Kircher, T., 2011. Heterogeneity of the left temporal lobe in semantic representation and control: priming multiple versus single meanings of ambiguous words. Cerebr. Cortex 21 (4), 831–844. https://doi.org/10.1093/cercor/bhq148.
Wilson, R.P., Colizzi, M., Bossong, M.G., Allen, P., Kempton, M., Mtac, Bhattacharyya, S., 2018. The neural substrate of reward anticipation in Health: a meta-analysis of fMRI findings in the monetary incentive delay task. Neuropsychol. Rev. 28 (4), 496–506. https://doi.org/10.1007/s11065-018-9385-5.
Yaple, Z., Martinez-Saito, M., Novikov, N., Altukhov, D., Shestakova, A., Klucharev, V., 2018. Power of feedback-induced beta oscillations reflect omission of rewards: evidence from an EEG gambling study. Front. Neurosci. 12, 776. https://doi.org/10.3389/fnins.2018.00776.