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A global single beta burst prior to stopping an ongoing movement suggests a causal role of beta in movement cancellation

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Abstract

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Abstract: The general question of this research is how beta oscillations are implicated in stopping an ongoing movement. Previous studies regarding movement cancellation have found a significant increase in beta activity in sensorimotor areas, especially in the form of transient increases in beta oscillations, called beta bursts. However, the functional role of beta band activity in stopping is still unclear, mainly because the behavioural tasks used cannot measure the exact timing when the subjects start the stopping process and therefore it is only possible to infer the stopping time. To resolve this, we used head-fixed rats running on a treadmill while performing a Go/NoGo task. In some NoGo trials, the rat starts to run, realizes the mistake and stops before crossing a fixed distance threshold. These are the events being analyzed, called near-mistake events (N=39,366). We found a single beta burst occurring prior to stopping in all five brain regions analyzed (from 44.2±20.1 ms to 55.8±16.0 ms) and positive correlations of beta burst number and power increase with movement speed before stopping. We also found a single alpha burst prior to and during stopping in all brain regions (from 45.9±20.1 ms to 57.1±19.3 ms), supporting previous studies' findings of alpha band involvement in inhibitory motor actions. Our findings on beta bursts suggest a causality role in stopping an ongoing movement while our results of alpha bursts need to be further analyzed to understand its functional role.

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List of Abbreviations

EEG	Electroencephalography
NME	Near-Mistake Event
CWT	Continuous Wavelet Transform
PETH	Peri-Event Time Histogram

Introduction

Studying the patterns of neural oscillations of the brain can provide a better understanding of different brain areas functioning and processing (Barone & Rossiter, 2021). Neural oscillations can be recorded with different methods, including via electroencephalography (EEG) (Barone & Rossiter, 2021). Within the range of 13 to 30 Hz, these neural oscillations are known as beta oscillations (Barone & Rossiter, 2021), and have been related to sensorimotor functions (Baker, 2007). It has been hypothesized that the overall role of beta activity in motor regions is maintaining the current motor state (Engel & Fries, 2010). The "normal" state is usually immobility and a strong beta band activity is necessary to maintain it (Engel & Fries, 2010). To change this state, for example by executing a movement, a prior decrease in beta band activity needs to occur (Engel & Fries, 2010). Our main objective for this project is to understand the amplitude and timing of beta oscillations when an ongoing movement needs to be stopped, not only in sensorymotor regions, but also looking into visual, hippocampus and frontal regions.

Evidence of beta playing a role in motor control is supported by studies involving transcranial alternating current stimulation and motor tasks. Using a 20Hz stimulation to entrain beta oscillations in the cortical activity of healthy humans resulted in slowing a voluntary movement (Pogosyan *et al.*, 2009). Moreover, 20Hz stimulation in the pre-supplementary motor area in humans performing a stop-signal task, a task which requires the subject to execute a movement in response to a Go signal and to cancel a movement in response to a Stop signal, served to enhance movement inhibition (Leunissen *et al.*, 2020). These findings indicate that beta is indeed involved in movement control and suggest a causal role in slowing and stopping voluntary movements. An interesting study in healthy subjects performing both slow and fast movements found a positive correlation between an increase in beta activity in motor cortex after a movement and an increase in movement speed (Zhang *et al.*, 2020). This finding supports the theory of maintaining the current

motor state (Engel & Fries, 2010) and suggests that the activity of beta band is dependent on how difficult it is to maintain and change the motor state.

Still, there are two questions regarding beta and motor control that do not have consensus answers. First, it is unclear whether the increased beta band activity seen in movement cancellation is due to transient increases in beta oscillations, called beta bursts, or an overall increase in beta power, or even if there is any beta power increase at all. In a human study using visual stimuli in a stop-signal task, beta bursts rate in frontal areas increased during movement cancellation, followed by a increase in beta bursts rate in bilateral sensorimotor areas (Wessel, 2020). However, in this study, there were successful stop trials without beta bursts prior to stopping and unsuccessful stop trials with beta bursts, so beta activity may not be causal in stopping at a single trial level (Wessel, 2020). Another human study using auditory stimuli in a stop-signal task showed an increase in beta bursts during successful stop trials as opposed to unsuccessful stop trials in the subthalamic nucleus and motor thalamus, supporting the causal role of beta at a single trial level (Diesburg *et al.*, 2021). A study in macaque monkeys using the stop-signal task showed similar beta bursts in medial frontal areas during stoppping that were found in the aforementioned human studies, even though they do not occur regularly enough to explain the movement cancellation (Errington *et al.*, 2020). Previously, a macaque monkey study used a reaching task to show that, at an individual trial level, there was an increase in beta bursts power in motor cortex and striatum after performing a movement (Feingold *et al.*, 2015), correlating as well with the role of beta in maintain a motor state (Engel & Fries, 2010). These studies show that beta bursts are observed in movement cancellation and after a movement, with contradictory findings in their role and significance to the action performed during the tasks. Adding to this, the type of stimuli the stop-signal tasks use might be another variable to consider when possibly explaining mixed findings.

In contrast to the notion that there is a relationship between the number of beta bursts and stopping movements, recordings from the human right inferior frontal gyrus during performance of a stopping task revealed that during stopping there was an increase in beta band power (Schaum *et al.*, 2021). Similar findings were reported in a stop-signal task in

humans, where successful stop trials showed an increase in beta band in the same region (Swann *et al.*, 2009). In contrast with these findings, a rat study using cued choice tasks had similar beta bursts decrease in basal ganglia in both Go and NoGo cues, suggesting that they are not related to maintaining the current motor state (Leventhal *et al.*, 2012). These results present contradictions in the beta band activity during stopping, which might be explained by the different variables in those studies, such as the research subjects, brain areas recorded and the design of the motor tasks.

The second open question in this field of research is related to the functional role of beta in motor control, whether it has a causal or consequential role. One reason for not knowing for sure the functional role is due to the behavioural tasks used, such as the stop signal task. In stop trials the focus is on preventing the initation of a voluntary movement, meaning that in successful trials, the stop signal causes the subject to cancel a planned, but never-initiated movement. Thus, the stop signal reaction time reported in these tasks is an estimate of the time when the subject decides to stop, inferred through race models, and not a exact recorded time. Overall, this means that increases in beta band activity during these stop trials could just support the fact that beta is involved in maintaining the current motor state (Engel & Fries, 2010), rather than actually causing cessation of movement, since in the stop signal task there is no actual stopping of an ongoing movement.

To overcome these limitations our project relies on a novel paradigm for head-fixed rats running on a treadmill. Rats are trained for a behavioural task, called Go/NoGo task, where there are two visual stimuli, one that signals the rat to run and another that signals to stop, a Go stimulus and a NoGo stimulus, respectively. Sometimes during the NoGo trials, a near-mistake event can occur, where the rats incorrectly initiate a running response, but realizes its mistake and stops the in-progress movement before it crosses a fixed distance threshold. By measuring the treadmill velocity and calculating the time point at which the rat initiates the stopping of the ongoing movement, we gain an observable time point of overt stopping behavior that can be measured. Thus, beta activity can be precisely aligned relative to the initiation of the stopping movements on each trial.

Aims of the study

The main aim of this research was to understand the role of beta in stopping an ongoing movement, whether it is causal or consequential. This also includes understanding their activity and possible correlations in different brain regions as well as whether beta bursting or an overall increase in power are related to stopping.

Materials and Methods

Contributions

The behavioural data was collected prior to my arrival to the research group. The data analysis was done by me and another Master student, Reetta Ojala, both with equal contribution to the project.

Behavioural Task

The Go/NoGo task was done during the rats' active cycle. The room was dark with dim light (>590nm) and the screen displaying the visual stimuli was illuminated the entire time. This task involves a Go stimulus and a NoGo stimulus. Firstly, there are correct performances in this task, called a hit and a correct rejection. A hit happens when the Go stimulus appears; at first the rat is immobile and as the Go stimuli appears the rat starts running and crosses the fixed threshold. A correct rejection is when there is a NoGo stimuli; the rat is once again immobile at first, then the NoGo stimuli comes on and the rat remains immobile until the stimuli has disappeared. Then there are error trials, called a false alarm and a miss. A false alarm is when the NoGo stimuli appears, and the rat runs past the distance threshold. A miss is when the Go stimuli comes on and the rat remains immobile throughout the whole trial. A near-mistake movement happens during the NoGo trials, when the rat starts to run, realizes the mistake and stops prior to crossing the distance threshold. These near-mistake events (NMEs), marking the time when the rat initiates the stopping response, are the focus of this project.

Research subjects and EEG array

The data was collected from 14 rats, and each had multiple sessions on different days with multiple near-mistake movements per session. Given the novelty of the task, only male rats were acquired, specifically Lister-Hooded rats, approximately 11 months old when the behavioural task was taking place. The electrode array used contained 32 electrodes that were distributed across nearly the entire anterior-posterior and medial-lateral extent of the

brain. Thus, they covered the following brain regions: frontal cortex (N=4), motor cortex (N=6), somatosensory cortex (N=4), hippocampus (N=6) and visual cortex (N=12).

Data analysis using MATLAB

Since the focus was on analyzing the NMEs, only these events (N=39,366) were taken from the EEG recordings of the sessions (N=306). Throughout the work different time windows of the near-mistake movement were used, ranging from a 2 second time window to a 4 second time window. These time windows were chosen since the goal was to understand if the beta activity is the cause of stopping and a 2 second window included the entire near-mistake movement time course. Since the electrodes were organized according to their channel on the amplifier, the data was also organized so that the first electrode corresponded with electrode nr. 1 and so on. This was possible by using an index that was in the data, of the number of the electrode and their current position and sorting the data so that it went from electrode nr.1 to electrode nr.32. Afterwards the data also needed to be organized by brain areas so we could analyze brain area-specific beta activity during near-mistakes. For this, there was a region list where each brain region had multiple electrode numbers corresponding to a particular region. This allows us to group individual electrodes by brain region.

Focusing on analyzing possible beta bursts during NME, the continuous wavelet transform (CWT) was performed on the EEG data because it can resolve changes in power at a range of frequencies over time. The CWT requires a lot of memory, so the analysis was done in the computer cluster. In this type of plot, edge artifacts can arise because the wavelets might go beyond the edges of the time window, making the result returned by the wavelet transformation of the data unreliable. In order to prevent edge artifacts from influencing our power measurements during the 2 second time window around the NME, we used a 4 second time window and then the resulting data was cut into a 2 second time window, which removed the window edges with artifacts while leaving the critical window to our analysis (2 seconds around the near-mistake) intact. In terms of parameters used for this, the frequency range was 10-45 Hz. After observing the resulting data, normalization was done in two different ways, using two different z scores. One was by using the entire session

all together, and another was getting the z score for each individual near-mistake trial. The two normalized data produced similar results, with the data normalized by entire session being too smooth in comparison to the data normalized by near-mistake movements. Thus, the data normalized by near-mistake movements will be the data used for the next steps.

Different categories can be defined for NMEs based on the velocity the rat achieved right before it starts to stop. This division was done for each rat individually, since there is variance between rats, and the NMEs were divided into three categories, from a lower velocity to a higher velocity.

Afterwards, bursting activity was defined by establishing a threshold for each trial of data with the beta band only (beta = 15-25 Hz). Threshold calculations were based on the work done in a previous study (Shin *et al.*, 2017) and adapted for the data in this research. This threshold needs to be specific but not too high, so that it is able to distinguish between bursts and "normal" activity. Multiple thresholds were calculated, and one was chosen based on visual evaluation of raster plots. The beta band frequency was averaged for each trial individually. Once every trial of each session is averaged, the threshold is calculated for each channel and each trial, with this formula: mean(averaged data) + 2*std(averaged data); with std standing for standard deviation. This provides a threshold based on the data for each specific trial. After this, the maximum value of each consecutive period in which the data is higher than the threshold is chosen and considered a beta burst event.

Beta bursting rates were then calculated for each rat individually and for the respective three different categories of NMEs. The MATLAB histcounts function was used to determine how many beta bursts were found every 50 ms (bin width = 50), for each rat, meaning in all trials and sessions of each rat. Then this result was divided by the total number of trials in each rat and consequently multiplied with the number of bins in one second to get the beta bursts per second of each rat, for each electrode and each category of NME.

Next, after calculating beta bursting rates, raster plots were done for each category of NMEs and for each brain region. Furthermore, peri-event time histograms (PETH) were calculated

and plotted in different ways. Once again with a sampling rate of 200 Hz, and the bin width chosen was 50 ms.

Variability was seen between different electrodes in each region and each rat when analyzing PETH, so we defined which electrodes are considered responsive electrodes by using the beta bursting rates calculated for the NME category with the highest velocity, called group 3 (G3). For this a baseline was established for each electrode, by z scoring the interval from -0.775 to -0.5 sec, with the zero moment being the NME. The baseline interval was chosen based on the low level of beta bursting activity observed there. Right before the NME, during the interval -0.525 to -0.025 sec, any electrode that exceeded a z score of 2.36 for a continuous period of 50 ms was considered a responsive electrode. This was done for all the electrodes of each rat. This data was used to analyze bursting activity of different brain regions, not only latency but also power of beta bursting throughout brain regions.

To compare and analyze the different NME categories, this last analysis to find the responsive electrodes was done not only in G3 but also in the other groups, G1 (lowest velocity) and G2 (mid velocity). With this, it is possible to analyze the number of responsive electrodes in each group and also the differences between brain regions.

While analyzing the data normalized by NMEs, the beta band was not the only one to show a high amplitude in bursting activity. Theta and alpha band also shown a high bursting activity before the NME. Thus, the analyzes that were done for beta bursting activity were also repeated for alpha band (alpha = 8-12 Hz). Unfortunately, theta is a too low oscillation for these analyzes to be performed.

Results

EEG data, in which the CWT was perfomed, was analyzed to study bursting activity, first by averaging the data of the three different categories of NME across trials, electrodes of each brain region and then rats. This will reveal if there are any meaningful changes in bursting activity during the NME, since a CWT plot shows differences in amplitude of bursting activity. Taking as an example Figure 1, panel A, the amplitude of bursting activity is distributed as colors, from the lowest amplitude in blue to the highest amplitude in yellow.

Because all five brain regions show a similar pattern in bursting activity through all the frequencies, from 1 to 45 Hz, as well as between categories of NMEs, Figure 1 only shows the averaged data for each NME category of two regions, motor and hippocampus. From Figure 1, it is possible to see the similarities in bursting activity as well slight differences in amplitude and timing between regions and NME categories, which is recurrent in all five regions. Firstly, G1 across five brain regions show a lower amplitude of bursting activity across frequencies compared to G2 and G3. There is an increase in theta (3-5 Hz), alpha (7-11 Hz) and beta (15-25 Hz) shown as a single burst before the stopping moment, marked as the zero moment, more noticeable in frontal and motor regions. There is also a clear increase in 1 to 3 Hz power in the interval of -1 to -0.5 sec before the stopping moment, especially high in the visual region. This bursting activity from 1 to 3 Hz is also seen in G2 but not as much in G3. The pattern of bursting activity before the stopping moment observed in G1 is also reflected in G2 and G3, with an increased in amplitude as the velocity achieved before stopping increases. In G2, alpha band activity has a higher amplitude in the hippocampus and visual regions as well as in G3. Moreover, this single burst is observed not only before the stopping moment but also after starting to stop until they fully stop. Regarding beta band activity in G2, it has a slightly lower amplitude in the frontal region and similar amplitudes in the other brain regions, while G3 shows higher amplitudes in the hippocampus and somatosensory regions compared to the other regions. Beta activity described above starts before stopping and ends prior to fully stopping. In both G2 and G3

theta band activity before the stopping moment is similar in all regions, with higher amplitudes in the visual region, especially in G3.

Given that G3 had the highest bursting activity, this group was selected to further analyze this activity at a single trial level. After the threshold for beta bursts was established, beta bursting rates were calculated during NME. Figure 2 represents data from one example rat, where panel A displays a raster plot of beta bursting activity in all trials of that rat for an electrode in the motor region and panels B,C, D, E and F show the PETH trial-averaged beta bursts per second, with each electrode in their respective brain region. The raster plot englobes all trials of that example rat, each trial is a line in the y axis and each dot represents a beta burst. In panel A, right before the NME located at 0 sec, all trials show an increase in beta bursts in the motor region, suggesting a single beta burst as a regular occurrence prior to stopping. Panels B to F show variability across electrodes but similar patterns within regions. Right before the zero moment, signaling the NME, visual reports the highest trial-averaged beta bursts per second, close to 1.4, while frontal shows the lowest values, less than 0.8.

To characterize what is a meaningful increase of beta bursts before NME, responsive electrodes were calculated via a z score for each region and rat of G3. Every electrode that exceeded a z score of 2.36 for 50 ms continuously was considered a responsive electrode. Out of the 14 rats, all rats had responsive electrodes in at least one brain region. Figure 3 shows the averaged PETH of responsive electrodes in G3 across all rats and representing the five brain regions. Across the time window shown in the figure, all the regions appear to have the same behavior of beta bursting activity, similarly increasing and decreasing at the same moments, with differences in their rate. Before the stopping moment, delimited by the vertical black dashed line, the somatosensory region has the highest beta bursting rate, followed by visual, frontal, motor and hippocampus region respectively. After the stopping moment, beta bursting activity starts to decrease in all regions.

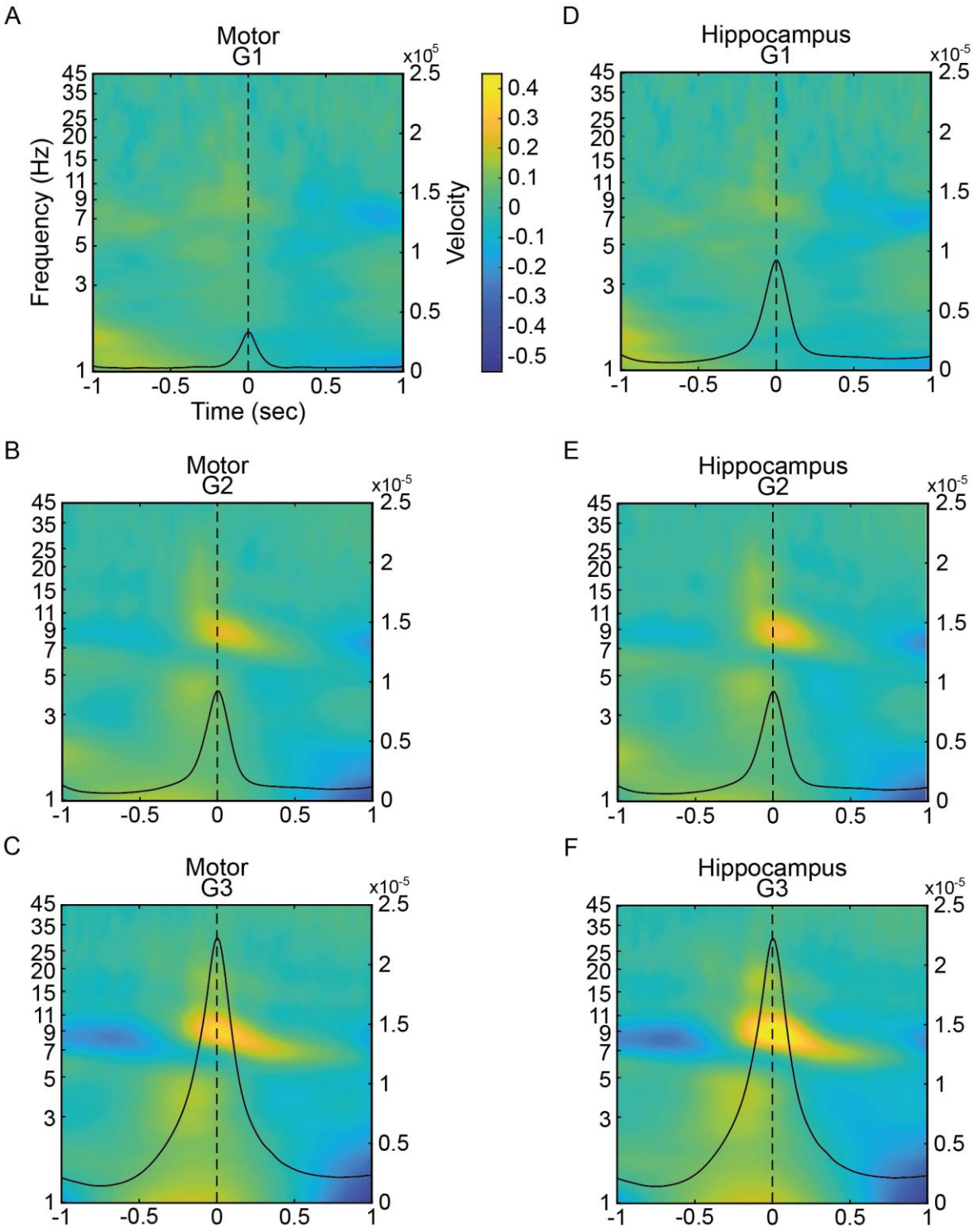

Figure 1 – Average bursting activity of each NME category in motor and hippocampus regions. The scalogram display the amplitude range of the bursting activity in the spectrograms. The x axis represents the time during the NME. The y axis on the left represents the frequencies, from 1 to 45 Hz, while the y axis on the right represents the velocity of each NME category.

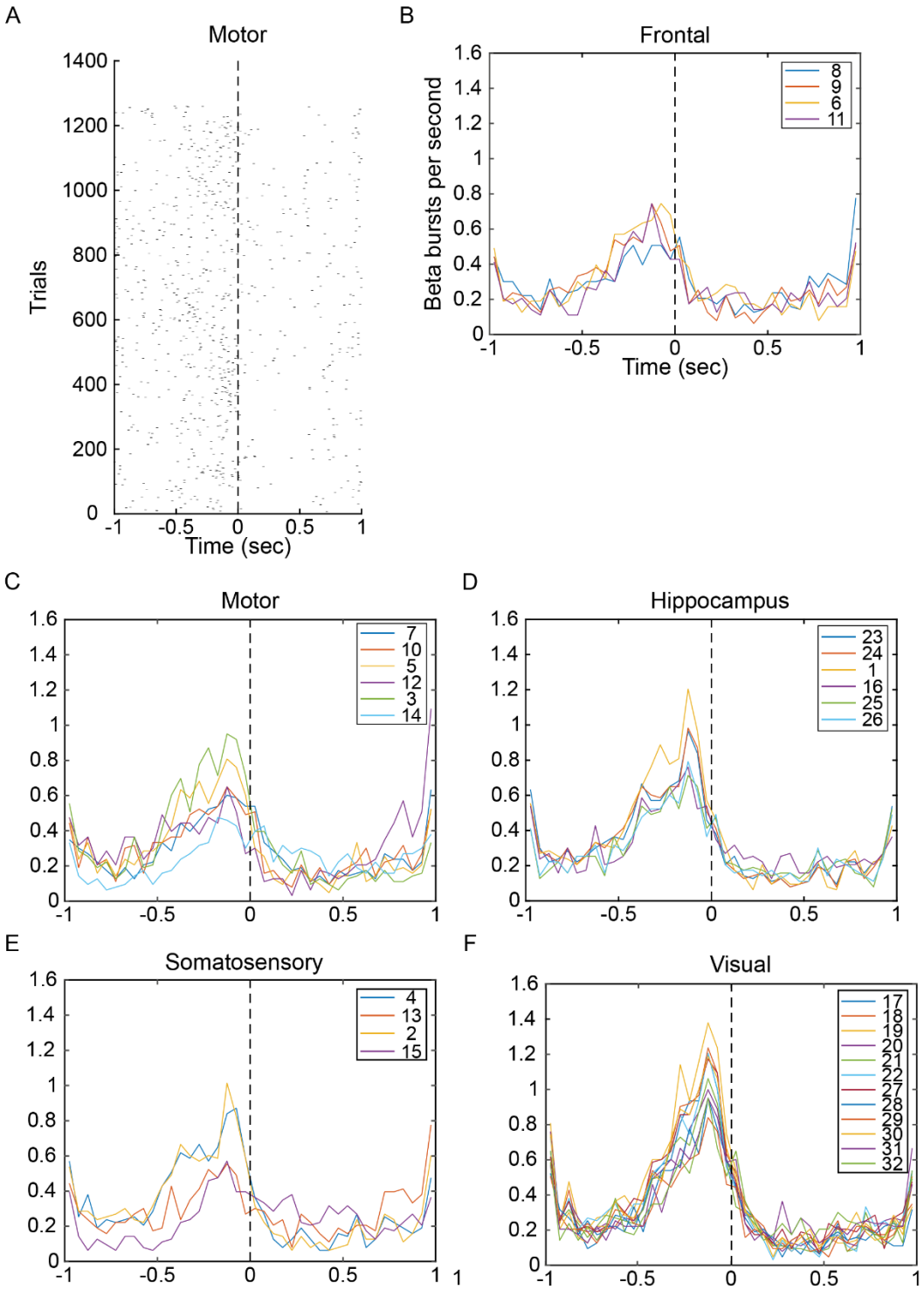

Figure 2 – An example rat's PETH trial-averaged of beta bursting rate for each region and example raster plot of individual trials beta bursts for one electrode in the motor. Panel A has the number of trials in that rat in the y axis, each line representing a trial. Panels B to F show the beta bursts per second in the y axis. The legend in each panel displays the numbers of the electrodes in the respective brain region. All panels show the timing in the x axis, with the NME displayed as a dashed vertical line at 0 sec.

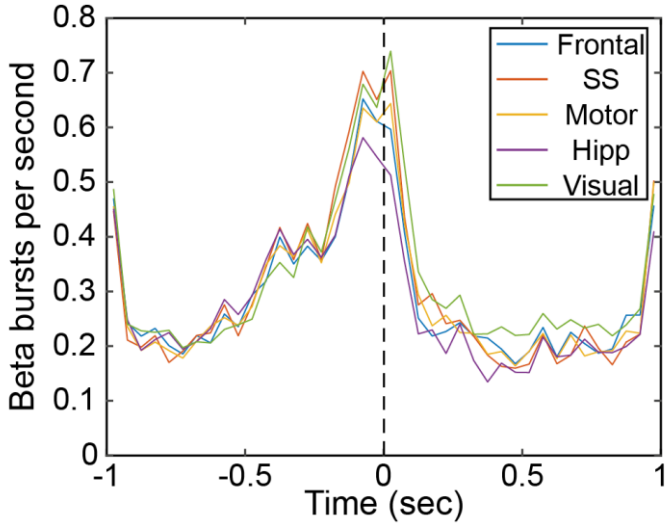

Figure 3 – Brain regions average of beta PETH of responsive electrodes in G3. The x axis represents the timing during the NME, with the dashed line showing the NME, while the y axis represents beta bursts per second. The legend displays each brain region and respective plot line. SS, Somatosensory; Hipp, Hippocampus.

To distinguish the single timed beta burst prior to NME seen in Figure 3, we focused on the interval from -0.175 to 0.075 sec from that figure. From this interval the maximum beta burst value and their latency to the NME of all rats were chosen, and the average across rats for all regions of beta burst and latency are shown in Figure 4, panels A and B, respectively. In panel A, it is noticeable that the averaged beta bursting rate right before the stopping moment is between 0.4 and 0.6 in all brain regions. The somatosensory region has a moderately lower average (0.4553±0.0320) and visual has a slightly higher average (0.5075±0.0410) in comparison to the other regions. Regarding panel B, the latencies within regions are quite variable, ranging from 0.02 sec to 0.08 sec, with the visual region reporting the lower latency (44.2308±20.0731 ms) while hippocampus region (55.7692±15.9412 ms) register the highest averaged latency. Moreover, both beta bursts and respective latencies show variability across rats in all regions, especially their latencies.

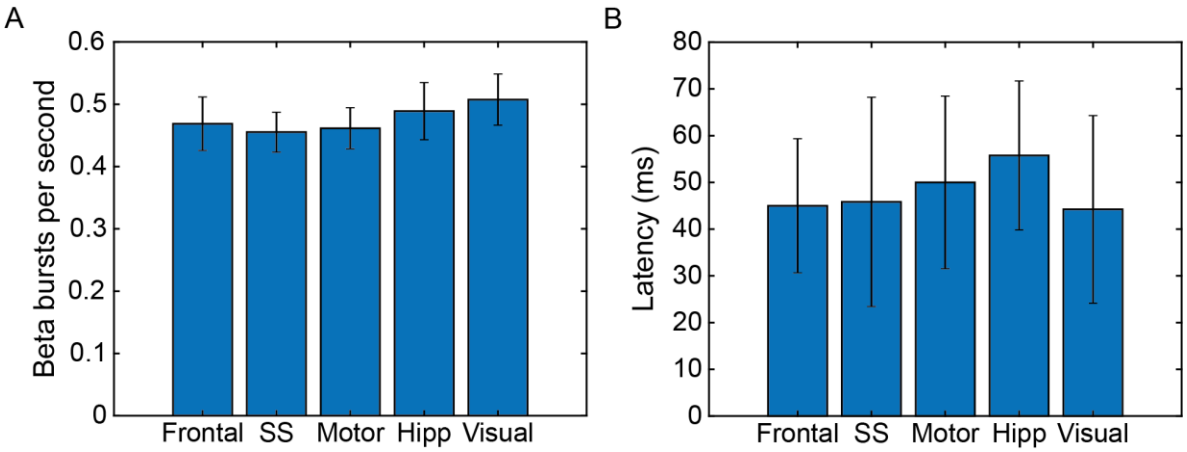

Figure 4 – Beta bursts activity and latencies in the interval right before and after the NME ([-0.175 0.075] sec). Panel A shows an average beta bursting rate for each region with standard error across rats. The y axis represents the beta bursts per second. Panel B shows an average latency for each brain region with standard error across rats. The y axis represents the latency of beta bursts related to NME. SS, Somatosensory; Hipp, Hippocampus.

To investigate if, besides the beta bursts amplitude, there was also an increase in responsive electrodes as the near-mistake movement velocity increased, the number of responsive electrodes were calculated for G1, G2 and G3 data and those values were averaged across rats for each NME category and respective brain region. Figure 5 shows the percentage of the average number of responsive electrodes for each NME category and brain region. Across all brain regions, G1 has the lowest percentage of responsive electrodes, below 25%, while G3 and G2 present similar percentages, all above 40%, across all regions, with visual being highest in G3 and the rest of the brain regions higher in G2. Furthermore, G1 shows more responsive electrodes in somatosensory (23.2143±2.7544 %) and less in visual (17.2619±2.0416 %); just as G1, G2 has a higher number of responsive electrodes in somatosensory (55.3571±2.6347 %) and lower in visual (43.4524±2.5381 %); G3 exhibits a higher percentage of responsive electrodes in visual (57.7381±2.3633 %) and lower in frontal (41.0714±2.6769 %).

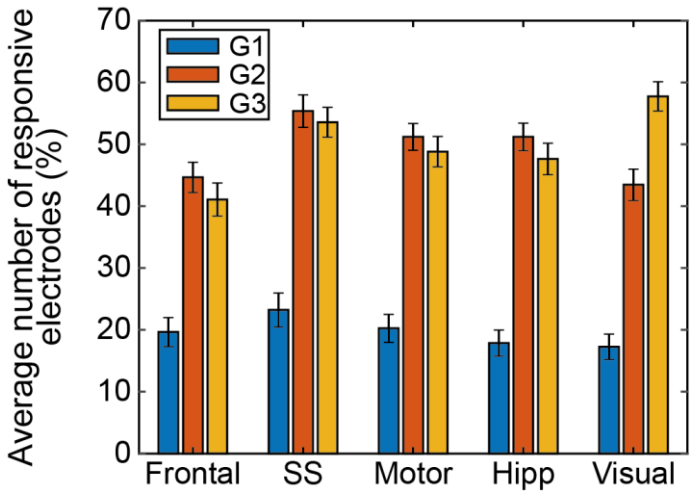

Figure 5 – Percentage of beta responsive electrodes in each NME category and respective brain region. The y axis represents the average number of responsive electrodes. The legend displays the NME category respective bar plot colors. Error bars show standard error across rats. SS, Somatosensory; Hipp, Hippocampus.

Surprinsingly, we also observed a bursting event in alpha band prior to stopping. Therefore, we characterize it in a similar matter to beta. As seen in Figure 1, alpha band has a significant increase in bursting activity during the stopping moment, especially in G3. Figure 6 shows an example of a rat's G3 data, where panel A displays a raster plot of alpha bursting activity in all trials for an electrode in the motor region and panels B, C, D, E, and F show the PETH trial-averaged alpha bursts per second, with each electrode in their respective brain region. The raster plot englobes all trials of that example rat, each trial trial is a line in the y axis and each dot represents an alpha burst. In panel A, right before the NME located at 0 sec, all trials show an increase in alpha bursts in the motor region, suggesting a single alpha burst as a regular occurrence prior to stopping. Panels B to F show variability across electrodes but similar patterns within regions. Frontal region records the lowest values right before NME, close to 1, comparing to the other regions, while visual reports the highest bursts per second, close to 1.5.

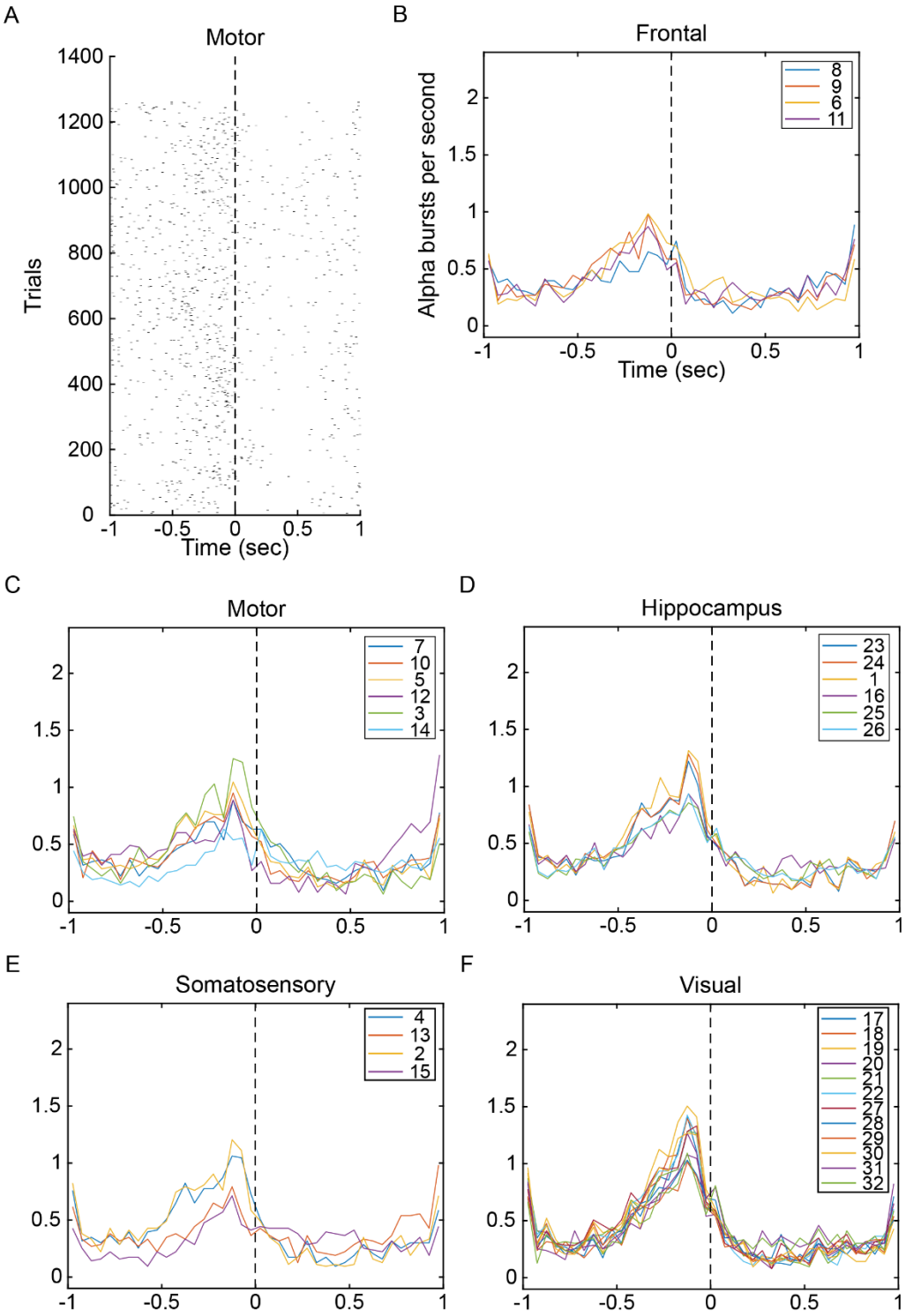

Figure 6 – An example rat's PETH trial-averaged of alpha bursting rate for each region and example raster plot of individual trials alpha bursts for one electrode in the motor. Panel A has the number of trials in that rat in the y axis, each line representing a trial. Panels B to F show the alpha bursts per second in the y axis. The legend in each panel displays the numbers of the electrodes in the respective brain region. All panels show the timing in the x axis, with the NME displayed as a dashed vertical line at 0 sec.

Next, we calculated and chose the responsive electrodes in G3 of all rats to distinguish the meaningful increases in alpha bursts prior to stopping. In terms of contribution of responsive electrodes, all the rats had responsive electrodes in at least one brain region. Figure 7 represents the averaged PETH of responsive electrodes in G3 for each brain region. Motor, somatosensory and visual regions have similar decreases and increases in alpha bursting rates throughout the NME, while frontal and hippocampus regions show more variance in their alpha bursting rates. Before the NME moment, located at 0 sec and delimited by the black dashed line, hippocampus has the highest alpha bursting activity while frontal region has the lowest.

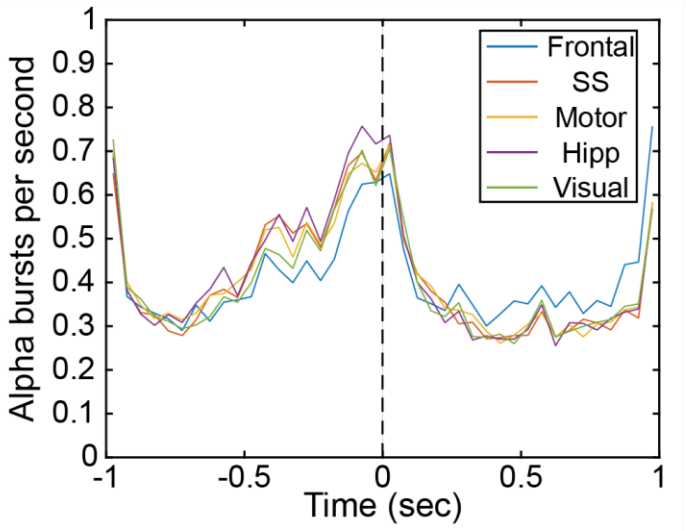

Figure 7 – Brain regions average of alpha PETH of responsive electrodes in G3. The x axis represents the timing during the NME, with the dashed line showing the NME, while the y axis represents alpha bursts per second. The legend displays each brain region and respective plot line. SS, Somatosensory; Hipp, Hippocampus.

To distinguish the single timed alpha burst prior to NME seen in Figure 7, we focused on the interval from -0.175 to 0.075 sec from that figure. From this interval the maximum alpha burst value and respective timing was found in every PETH of responsive electrodes in G3 for every rat. Figure 8 shows the averaged maximum alpha bursts (panel A) and latencies to the NME (panel B) across rats in all brain regions. In panel A, all regions show values within 0.5 and 0.75 alpha bursts per second. Frontal region has the lowest average (0.5656±0.0539) while hippocampus has the highest (0.6668±0.0399). Then, panel B shows

the averaged latencies of alpha bursts for each region. There is high variability in every region in terms of the averaged latency value, with latencies ranging from 0.02 to 0.08 sec. Motor reports to lowest average (45.8333±20.1120 ms) while visual region has the highest average (57.1429±19.3345 ms).

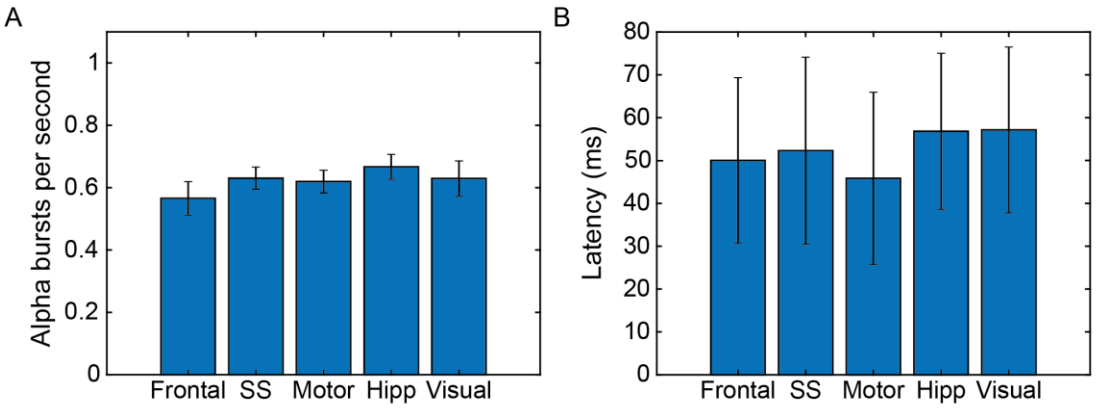

Figure 8 – Alpha bursts and latencies in the interval right before and after the NME ([-0.175 0.075] sec). Panel A shows the average alpha bursting rate with standard error across rats for each region. The y axis represents the alpha bursts per second. Panel B shows the average latency for each brain region with standard error across rats. The y axis represents the latency of alpha bursts related to NME. SS, Somatosensory; Hipp, Hippocampus.

To determine whether there is a positive correlation between an increase in responsive electrodes within brain regions and the movement velocity prior to stopping, the number of responsive electrodes in each NME category was calculated. Figure 9 shows the percentage of the averaged number of responsive electrodes across rats for each NME category and brain region. Across all regions, G1 presents the least responsive electrodes in every region; G2 has the highest responsive electrodes in somatosensory (64.2857±2.1830 %) and hippocampus (48.8095±2.3563 %); G3 has the highest responsive electrodes in motor (55.9524± 2.4534 %) and visual (62.5000±2.1493 %); the frontal region has the same average value for G2 (50±2.3230) and G3 (50±2.5254 %). Within each NME category, visual has the lowest responsive electrodes average for G1 (13.6805±1.8620 %) while hippocampus is the lowest for both G2 and G3 (45.2381±2.7793 %); somatosensory

presents the highest value of responsive electrodes for G1 (28.5714±2.1990 %) and G2 while visual is the highest in G3.

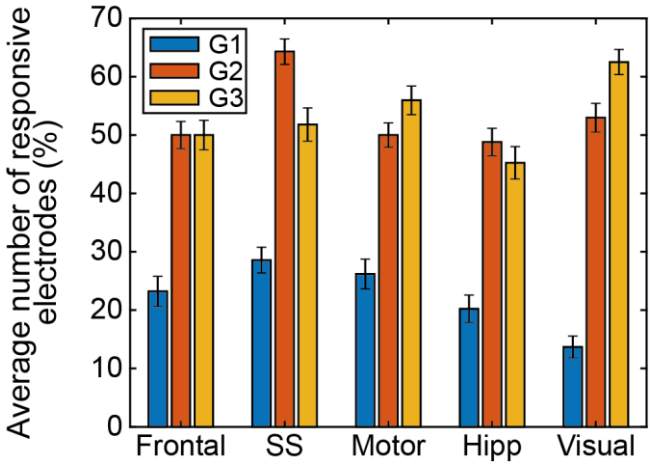

Figure 9 – Percentage of alpha responsive electrodes in each NME category and respective brain region. The y axis represents the average number of responsive electrodes. The legend displays the NME category respective bar plot colors. Error bars show standard error across rats. SS, Somatosensory; Hipp, Hippocampus.

Discussion

Stopping an ongoing movement is associated with a prior transient beta burst. Furthermore, this bursting event is seen in all five brain regions analyzised and its amplitude and number increases as the velocity before stopping is increased. A single alpha burst is also seen in all regions prior to stopping, and continuining during the stopping process. The amplitude of the alpha burst and number of alpha bursts in each region also positively correlates with the velocity of the movement prior to stopping.

Firstly, the behavior of beta band activity during the NME supports the hypothesis of beta's role in maintaining the status quo (Engel & Fries, 2010). Meaning that the changes seen in bursting activity are related to changes in movement, in initiating and cancelling a movement.

Besides a single beta burst occurring regularly prior to stopping in all NME categories, there was also a clear increase in that single beta burst amplitude as the near-mistake movement velocity increase, indicating a positive correlation between beta bursting amplitude and velocity of the ongoing movement before stopping, also seen in a human study (Zhang *et al.*, 2020). Furthermore, there was a positive correlation between the near-mistake movement velocity increase and the number of responsive electrodes in all brain regions. These findings support a causality role of beta bursts in stopping an ongoing movement.

There is also an interesting increase in beta bursts per second close to -1 sec and 1 sec, with the NME at 0 sec. This might possible be related to preparation of a movement and performance monitoring, respectively (Little *et al.*, 2019; Errington *et al.*, 2020).

A surprising result was the bursting activity seen in alpha, not only before stopping but also during the stopping process. The same analysis done for beta band was done for this frequency and results were highly similar to the beta band activity, with an increase in amplitude and differences in which brain regions reported higher and lower values. The amplitude of the single alpha burst seen during the NME, in all NME categories, showed a positive correlation with a movement velocity increase prior to stopping. Regarding the

increase seen in alpha bursts, there has been shown that alpha power is positively correlated with motor inhibition in sensorimotor areas (Bönstrup *et al.*, 2015). There is also a significant decrease in alpha bursts from -1 to -0.5sec before the NME. Given that the visual stimuli appeared around that time, this supports the hypothesis that alpha activity is linked with cued attention (Jones *et al.*, 2010). Our findings support the idea that alpha band is involved in inhibitory motor actions, such as stopping an ongoing movement, but as a global phenomenon and as a burst instead of frequency power.

To conclude, we show that both beta and alpha bursts are involved in stopping an ongoing movement. A single beta burst occurs regularly and globally prior to stopping which supports the causal role of beta bursts in movement cancellation. Furthermore, velocity of the movement prior to stopping has a positive correlation with both beta bursts number and amplitude in all regions. The alpha bursts observed support its relation to inhibitory actions in sensorimotor regions, but further studies need to be done to understand its role in stopping and possible correlation with beta bursting activity.

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