Intact Proactive Motor Inhibition after Unilateral Prefrontal Cortex or Basal Ganglia Lesions

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Abstract

Previous research provided evidence for the critical importance of the PFC and BG for reactive motor inhibition, that is, when actions are cancelled in response to external signals. Less is known about the role of the PFC and BG in proactive motor inhibition, referring to preparation for an upcoming stop signal. In this study, patients with unilateral lesions to the BG or lateral PFC performed in a cued go/no-go task, whereas their EEG was recorded. The paradigm called for cue-based preparation for upcoming, lateralized no-go signals. Based on previous findings, we focused on EEG indices of cognitive control (prefrontal beta), motor preparation (sensorimotor mu/beta, contingent negative variation [CNV]), and preparatory attention (occipital alpha, CNV). On a behavioral level, no differences between patients and controls were found, suggesting an intact ability to proactively prepare for motor inhibition. Patients showed an altered preparatory CNV effect, but no other differences in electrophysiological activity related to proactive and reactive motor inhibition. Our results suggest a context-dependent role of BG and PFC structures in motor inhibition, being critical in reactive, unpredictable contexts, but less so in situations where one can prepare for stopping on a short timescale.

INTRODUCTION

The ability to inhibit one’s actions is at the core of executive control functions and critical for goal-directed behavior (Bari & Robbins, 2013). Response inhibition (i.e., the inhibition of motor output) is not a unitary function but involves different subtypes, including reactive and proactive inhibition (Aron, 2011). Reactive inhibition is considered to be invoked by an imperative stimulus, such as a stop sign. Proactive inhibition, on the other hand, is prospective and prepares the system for the potential need to withhold one’s motor action. Both proactive and reactive inhibition are presumably enabled through frontal BG connections, although distinct loops might be relevant for either one (Di Caprio, Modugno, Mancini, Olivola, & Mirabella, 2020; Cirillo, Cowie, MacDonald, & Byblow, 2018; Hermans et al., 2018; Jahanshahi, Obeso, Rothwell, & Obeso, 2015; Jahfari et al., 2012). Importantly, both reactive and proactive inhibition are closely interrelated with attentional functions, as one needs to attend to relevant signals in the environment to adapt one’s behavior (Bari & Robbins, 2013). It is debated, though, how inhibition and attention work together and to what extent brain regions involved in inhibitory control are specific for inhibition or serve more generic, attentional functions (Aron, Robbins, & Poldrack, 2014; Erika-Florence, Leech, & Hampshire, 2014). In this work, we took a combined lesion and electrophysiology approach to test the critical impact of the PFC and BG on proactive inhibition and to examine the neurocognitive processes through which they influence response inhibition (Szczepanski & Knight, 2014; Floden & Stuss, 2006; Picton et al., 2007). We measured the EEG in patients with focal and unilateral PFC or BG lesions and healthy controls who performed in a cued go/no-go paradigm (Liebrand, Pein, Tzvi, & Krämer, 2017) calling for proactive and reactive inhibition.

Extensive research in humans and animals has linked response inhibition to a network of prefrontal regions, most prominently the inferior frontal gyrus (IFG), the pre-SMA, and BG nodes (Aron et al., 2014). Scalp and intracranial EEG recordings suggest that oscillatory activity, especially in the beta frequency range, might play an important role for inhibition-related communication within this network (Chen et al., 2020; Wessel, 2020; Fonken et al., 2016; Swann et al., 2009, 2011). Less agreement prevails as to which cognitive function can be ascribed to these regions and how critical and specific this network is for response inhibition. An influential account considers the so-called hyperdirect pathway from IFG to the subthalamic nucleus as the critical and specific pathway for fast reactive stopping (Aron et al., 2014). This notion has recently gained more support through multisite invasive brain recordings in humans (Chen et al., 2020). However,
in a previous EEG study in PFC lesion patients (Krämer et al., 2013), we did not find evidence for a critical role of the IFG for reactive inhibition, as patients were equally fast as controls in stopping in a stop signal task. Patients had an increased error rate in a go/no-go task and reduced parieto-occipital activity to stop signals, which was taken as evidence for impaired attentional control (Krämer et al., 2013). An increased error rate in a go/no-go task after dorsal frontal lesions is in accord with a large behavioral study in frontal lobe lesion patients by Picton et al. (2007). The pattern of an increased no-go error rate with concomitantly preserved inhibitory speed might indicate an inability to maintain inhibitory control and adequately prepare for upcoming no-go stimuli. In this work, we followed up on this notion and investigated proactive inhibition in a sample of patients with chronic, unilateral PFC or BG lesions.

In a recent study in young, healthy participants, we investigated the temporal dynamics of proactive and reactive motor inhibition using EEG in a novel cued go/no-go task (Liebrand et al., 2017). We focused on oscillatory activity over frontal regions, presumably reflecting cognitive control (Wessel, 2020; Rangel-Gomez, Knight, & Krämer, 2015; Huster, Enríquez-Geppert, Lavalle, Falkenstein, & Herrmann, 2013; Swann et al., 2011; Alegre et al., 2004); over sensorimotor regions as final gate for motor actions (Picazio et al., 2014; Krämer, Knight, & Münte, 2011; Neuper, Wotruba, & Pfurtscheller, 2006); and over occipital regions speaking for attentional modulation of visual processing (Lavalle, Meemken, Herrmann, & Huster, 2014; Klimesch, 2012; Hanslmayr, Gross, Klimesch, & Shapiro, 2011). During the cue–target interval, the anticipation of no-go signals led to increased visual attention, reflected in reduced occipital alpha power, and to a modulation of sensorimotor cortex activity, reflected in reduced beta power ipsilateral to the relevant response hand. Also the contingent negative variation (CNV), the most common electrophysiological index of preparatory cortical activity (Brunia & van Boxtel, 2001), was enhanced when anticipating a no-go signal. Increased prefrontal activity, indicated by higher frontal beta power, was observed after target presentation only, but not during the cue–target interval (Liebrand et al., 2017). These results stress the close interrelation between proactive inhibition and attentional control and suggest that different neurocognitive mechanisms are engaged when preparing for or reacting to no-go signals (Di Caprio et al., 2020; Hermans et al., 2018; Jahfar, Stinear, Claffey, Verbruggen, & Aron, 2010). The aim of the current study was to directly test the hypothesized impact of BG and prefrontal nodes on proactive and reactive inhibition and the associated cortical electrophysiological markers. As in the previous work, we investigated (i) beta power over prefrontal regions related to cognitive control; (ii) sensorimotor mu/beta oscillations and the CNV, indexing preparatory (motor) activity; and (iii) occipital alpha power associated with visual attention. We expected patients with lesions to the PFC or BG to show behavioral deficits (error rates and proactive slowing) in proactive inhibition and reduced neurophysiological markers of proactive and reactive motor inhibition.

**METHODS**

**Participants**

Nine patients (39–63 years, mean = 53 years, six women) with focal unilateral prefrontal lesions due to tumor resection or stroke took part in the study (five left hemisphere, four right hemisphere). Five of those patients were recorded in Oslo, three in Berkeley, and one in Lübeck. One other PFC patient had to be excluded because of excessive movement artifacts in the EEG. Nine patients (37–67 years, mean = 58 years, one woman) with focal unilateral lesions in the BG caused by stroke participated in the experiment (five left hemisphere, four right hemisphere), all recorded in Lübeck. Data of another three BG patients had to be discarded because of poor behavioral performance (below 70% correct go trials), suggesting they did not understand or were unable to implement the task instructions. Further characteristics of the two patient groups can be found in Table 1. Exclusion criteria for patients were a comorbid neurological/psychiatric disease or medication, noncorrectable visual impairments, hand motor deficits, nonfocal or secondary lesions, and drug or alcohol abuse. Patients were included based on a structural MRI scan before the experiment. A neuroradiologist confirmed the lesion and that no other signs of pathology were present in the brain. The experiment took place at least 6 months after stroke or surgery. Note that PFC patients were measured, on average, considerably longer after their injury compared with the BG patients (Table 1).

Twenty-two healthy participants served as controls and were matched as close as possible to the patients in terms of age, sex, and education (36–68 years, mean = 55 years, eight women; Lübeck: n = 21, Oslo: n = 1). Another six controls had to be excluded from analyses because of extensive EEG artifacts (n = 4) or poor behavioral performance (n = 2). All controls were by self-report free of neurological or psychiatric disorders. All participants reported normal or corrected-to-normal vision. The study was performed in agreement with the Declaration of Helsinki. All participants gave informed consent. The study was approved by the University of California, Berkeley Committee for Protection of Human Subjects and the Department of Veterans Affairs Northern California Health Care System Human Research Protection Program; by the Norwegian Regional Committee for Medical Research Ethics, Region South; and by the Ethics Committee of the University of Lübeck. Patients and controls received monetary compensation for participation, or travel and accommodation expenses were covered.

**Lesion Reconstruction**

Lesion reconstructions were based on structural MRIs obtained after study inclusion. Lesions were outlined by

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**Table 1**

<table>
<thead>
<tr>
<th>Patient Groups</th>
<th>Characteristics</th>
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<tr>
<td>PFC Patients</td>
<td>39–63 years, mean = 53 years, six women</td>
</tr>
<tr>
<td>BG Patients</td>
<td>37–67 years, mean = 58 years, one woman</td>
</tr>
<tr>
<td>Controls</td>
<td>22 healthy participants</td>
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**Downloaded from** https://direct.mit.edu/jocn/article-pdf/33/9/1862/1956191/jocn_a_01691.pdf by guest on 20 August 2021
drawing manually on fluid attenuated inversion recovery images of each participant’s brain using the software ITK-Snap (Yushkevich et al., 2006; www.itksnap.org) or MRIcron (Rorden, Karnath, & Bonilha, 2007; people.cas.sc.edu/rorden/mricron/). T1- and T2-weighted images provided additional information to determine the borders of the lesions. The resulting lesion masks were transferred to normalized space using the SPM software (SPM12; www.fil.ion.ucl.ac.uk/spm/). We followed two different procedures for normalization. For BG lesions, best results were obtained with the Clinical Toolbox for SPM (Rorden, Bonilha, Fridriksson, Bender, & Karnath, 2012). For PFC lesion reconstructions, superior results were achieved using a procedure suggested by Ripollés et al. (2012). In PFC patients, T1 images were segmented and normalized at the same time using the “unified segmentation” approach. Then, the normalization transformation was applied to fluid attenuated inversion recovery and lesion reconstruction images. Involved lesion volumes were calculated using MRcron. Overlay of lesion reconstructions of BG and PFC patients are presented in Figure 1C. Note that, in the overlay, left-sided lesions were flipped for visualization.

### Experimental Paradigm

The participants performed a cued go/no-go task (see Figure 1A), very similar to the paradigm used by Liebrand et al. (2017). A centrally presented square or circle served as cue, and the following target stimulus was always a triangle. The two cues were randomly presented with 50% each. Over all trials, the following triangle was presented in the center of the screen in 75% of the trials. In the remaining 25% of the trials, the triangle following the circle was lateralized to the left side (5°), and the triangle following the square was lateralized to the right side (5°). Participants had to press the right mouse button to targets following the square and the left button to targets following the circle. Before each block, participants were told to refrain from pressing the button either when the triangle appeared on the right side (block: stop right) or on the left side (block: stop left). The instruction about which stimulus and response side had to be attended to and potentially be inhibited alternated from block to block.

This design results in two different trial conditions, which we refer to as “Maybe Stop” and “Certain Go” (see Figure 1A). In the Maybe Stop condition, the cue indicated
that the participant might have to stop afterward, whereas in a Certain Go condition, participants knew that no stop was required in any case. In the Maybe Stop condition, participants had to stop in 25% of the trials (Stop trials) but were instructed to press the button in the remaining 75% (No Stop trials). In the Certain Go condition, participants had to press the button both in response to central targets (75%, Frequent Go trials) and to lateralized targets (25%, Rare Go trials). For instance, if the current block was a Stop-left block and a circle was presented followed by a triangle on the left side, the response had to be withheld (Maybe Stop condition, Stop trial). If in the same block a square was presented followed by a triangle on the right side, the participant had to press the right button (Certain Go condition, Rare Go trial). In summary, cues comprised either square or circle, while the target always being a triangle. All centrally presented targets signaled a go reaction, whereas half of the lateralized presented targets demanded a no-go, depending on the stopping rule in the respective block. In the following, we refer to trials with 75% probability as frequent trials (No Stop and Frequent Go) and to those with 25% probability as rare trials (Stop, Rare Go).

Cue and target stimuli were presented for 100 msec. The target followed the cue after an ISI of 1000 msec. The time between two subsequent trials was jittered (1300–1600 msec). The experiment was divided into six blocks with 160 trials each, resulting in 960 trials. The attended side of the first block was counterbalanced among participants. Throughout the whole experiment, a fixation line was presented underneath the stimuli, which participants were instructed to fixate. Participants were told to respond as fast and accurately as possible and not to press the button until the triangle had appeared. Patients used the hand ipsilateral to the lesions’ side to avoid general motor slowing because of the lesion. Controls used the same hand as the patient whom they were matched to. Participants used their index and middle fingers to respond.

### Figure 1

Experimental design, behavioral results, and lesion reconstruction. (A) Design of the cued go/no-go task. A cue (square or circle) indicated the relevant response side for the upcoming target stimulus (triangle). On the top row, a block is illustrated where participants had to refrain from pressing the button when the target (triangle) appeared on the right side of the screen. Similarly, in the block displayed in the bottom row, a stop was required when the target appeared on the left side. In the Maybe Stop condition (dark gray), the cue indicated that the response might have to be stopped later on, whereas in the Certain Go condition (light gray), participants always had to execute the motor action to the target. (B) Behavioral results showing mean RTs in No Stop, Frequent Go, and Rare Go trials. The SEMs are displayed as error bars. Patients and controls responded faster in Frequent Go than No Stop and Rare Go trials. (C) Overlay of lesion reconstructions of BG and PFC patients, normalized into the Montreal Neurological Institute space. The color coding indicates the number of patients with damaged tissue in that area. In the top row, left-sided lesions were flipped; in the bottom row, we display nonflipped lesions.
perform the task. Before the start of the experiment, participants practiced the task in three short blocks with 16 trials each. This practice session could be repeated until participants affirmed to fully understand the instructions.

The experiment was controlled using Presentation software (Neurobehavioral Systems). Stimuli were presented on a computer screen, about 1 m away from the participant. Participants sat in a comfortable chair, and after each of the experimental blocks, they had a short break of 20 sec to relax. The total duration of the experiment was about 50 min.

**EEG Recordings and Data Preprocessing**

In Lübeck, the EEG was recorded with a 64-channel BrainAmp MR plus amplifier, with a sampling rate of 250 Hz. Electrodes were placed according to an extension of the international 10–20 system (Nuwer et al., 1998). Vertical and horizontal eye movements (vertical EOG [vEOG] and horizontal EOG [hEOG]) were recorded. For vEOG, we used an electrode placed below the right eye and a frontopolar electrode, and for hEOG, two electrodes located on the outer canthus of each eye were used. The ground was placed at Fpz, and the EEG was recorded against a reference electrode placed on the right earlobe. In Oslo and Berkeley, the EEG was recorded with a 64 + 8 channel BioSemi Active-Two amplifier, with a sampling rate of 1024 Hz in Oslo and 256 Hz in Berkeley. The hEOG was recorded as in Lübeck, and for the vEOG, one electrode was placed below and one above the right eye. The CMS (common mode sense) and DRL (driven right leg) electrodes of the BioSemi systems were placed at two locations close to Pz in Oslo and Berkeley.

**Behavioral Data Analysis**

We first tested whether patients and control participants differed in basic sociodemographic variables, using independent-samples t tests. BG and PFC patients did not differ from the control group, including all matched control participants in terms of age and years of education: BG versus all controls: age, $t(29) = 0.8, p = .425$; education, $t(29) = 0.5, p = .643$; PFC versus all controls: age, $t(29) = -0.8, p = .434$; education, $t(29) = 0.2, p = .850$; PFC versus BG: age, $t(16) = 1.3, p = .205$; education, $t(16) = 0.2, p = .862$. We then performed ANOVAs comparing all patients against all controls (see Voytek & Knight, 2010, for a similar approach).

Our general data analysis rationale was the following: As a measure for proactive inhibition in the cue–target interval, we contrasted trials in which the later motor response might (Maybe Stop) or might not have to be inhibited (Certain Go). To assess how proactive inhibition modulated response execution after target signals, we compared trials following cues that signaled that responses might (No Stop) or might not have to be stopped (Frequent Go; Swann, Tandon, Pieters, & Aron, 2013; Swann et al., 2012). Finally, to assess actual reactive motor inhibition after target presentation, we contrasted trials in which the response had to be inhibited (Stop) with matched go trials still requiring a response (Rare Go).

Mean RTs, failed inhibitions, commission, omission, and premature error rates (button presses before the target stimulus had appeared) were computed for each participant and submitted to mixed-design ANOVAs with the within-subject factor Condition (depending on the comparison) and the between-subject factor Group (prefrontal lesion patients: PFC group, BG lesion patients: BG group, and controls: CTR group). The behavioral measure of proactive inhibition was the RT difference between No Stop compared with Frequent Go and Rare Go trials (Liebrand et al., 2017).

**EEG Data Analyses**

EEG data analysis was performed with EEGLAB (Delorme & Makeig, 2004), ERPLAB (Lopez-Calderon & Luck, 2014), and custom written MATLAB (The MathWorks) scripts. EEG data were rereferenced off-line to the average of the signal from the two earlobe electrodes. The data were high-pass filtered with 0.5 Hz (Butterworth filter, second order) and subsequently low-pass filtered with 40 Hz (Butterworth filter, sixth order). Data from Oslo were down-sampled to 256 Hz. The data were segmented into epochs for the different conditions. Epochs included 1000 msec before and 2000 msec after the stimulus. The baseline was defined as the 100 msec preceding the stimulus. An independent components analysis, as implemented in EEGLAB (Infomax extended), was performed on the averaged data including all conditions. Independent components accounting for blink artifacts and horizontal eye movements were identified and removed from the data (Jung et al., 2000). Trials affected by other artifacts caused, for example, by muscle tension were rejected from further analysis, with a threshold for rejection of ±80 μV. Noisy channels identified by visual inspection and artifact rejection thresholds were interpolated (range: 0–7 channels; mean = 1.5 channels per participant). If more than 30% of the data of one participant were rejected, this participant was excluded from analysis (one PFC patient, four controls). Current source density interpolation of the data was estimated through Laplacian computation based on a spherical spline interpolation (with a spline order of 4; Kayser & Tenke, 2006). Specifically, we used the CSD Toolbox for MATLAB (Kayser, 2009). We took advantage of the Laplace transformation, as it accentuates local effects while filtering out distant effects because of volume conduction, which increases the signal-to-noise ratio, especially in combination with higher density recordings (256 electrodes; Babiloni et al., 1995).

In terms of ERPs, we analyzed the amplitude of the CNV in the cue–target interval. As measure of the amplitude, we chose the area under the curve in a given time window, zeroing negative values in positive waveforms and vice
versa. The analyzed time window (1000–1100 msec) and the selection of electrodes (Cz, FCz) were based on visual inspection of the data and on a previous study (Liebrand et al., 2017). The CNV was analyzed with an ANOVA including the within-subject factors Condition (Maybe Stop vs. Certain Go) and Electrode (FCz, Cz) and the between-subject factor Group. For visualization only, data were low-pass filtered at 15 Hz.

To study inhibition-related power changes in the alpha (9–14 Hz) and beta (15–25 Hz) band, single trial data were convolved with a complex Morlet wavelet, as implemented in MATLAB (function cwt with parameter specification emor1-1.5),

\[ w(t) = (\eta f_0)^{-0.5} e^{-2\eta f_c - \eta^2 f^2} \]

where \( f_0 = 1 \) was the bandwidth parameter and \( f_c = 1.5 \) was the wavelet center frequency (Teolis, 1998). Specifically, we computed changes in time-varying energy (square of the convolution between wavelet and signal) in the studied frequencies (1–40 Hz, linear increase) and averaged these values for each participant. Data were computed as percent power change with respect to a pre-stimulus baseline (−250 to −50 msec before the stimulus). The selection of the analyzed alpha/mu (9–14 Hz) and beta (15–25 Hz) frequencies was based on previous literature (Liebrand et al., 2017; Solbakk & Løvstad, 2014; Krämer et al., 2011). To reduce the number of statistical comparisons and to increase the signal-to-noise ratio, we clustered the electrodes into ROIs: left prefrontal (F3, F5, FC3, FC5), right prefrontal (F4, F6, FC4, FC6), left central (C3, C5, CP3, CP5), and right central (C4, C6, CP4, CP6), based on previous studies (Liebrand et al., 2017; Krämer et al., 2013). Clustering was performed after single-channel time frequency values were computed. Similar to ERP data, electrodes of patients with left-sided lesions were flipped along the midline. Because the performing hand also depended on the lesion side, we also accounted for sensorimotor activity with this procedure. Contralateral corresponds to the contralateral hemisphere (in relation to the responding hand), and ipsilateral corresponds to the ipsilateral hemisphere. Mean time–frequency power in a given time window (see below) was subjected to mixed-design ANOVAs. The time windows were based on our previous study (Liebrand et al., 2017; Krämer et al., 2013). As measurement window for effects before target onset, we chose 700–1100 msec when the cue-related activity was distant and target onset (1100 msec) was closest. For target-related effects, we investigated the window between 200 and 500 msec, focusing on the period when participants were executing the motor response.

For sensorimotor effects between cue and target, mean mu and beta activity over sensorimotor clusters (700–1100 msec) was subjected to ANOVAs with the within-subject factors Condition (Maybe Stop vs. Certain Go) and Hemisphere (contra- vs. ipsilateral to the upcoming motor response) and the between-subject factor Group (BG, PFC, CTR). The same analysis was performed for beta power over frontal sites. For attention-related effects between cue and target, we subjected mean alpha power over occipital clusters (700–1100 msec) to an ANOVA with within-subject factors Condition (Maybe Stop vs. Certain Go), Stimulus (contra- vs. ipsilesional side of upcoming possibly lateralized target stimulus), and Hemisphere (contra- vs. ipsilesional) and the between-subject factor Group. Finally, to test for a modulation of beta power after target signals, we subjected mean beta power over prefrontal electrodes (200–500 msec) to ANOVAs with the factors Condition and Hemisphere (contra- vs. ipsilesional) and the between-subject factor Group. As conditions, Rare (Stop- vs. Rare Go-) and Frequent (No Stop- vs. Frequent Go-) Trials were separately analyzed.

Bayesian Statistics

In addition to classical statistics (t test, ANOVA, etc.), we performed Bayesian statistics, which has the advantage of being able to make statements not only for the alternative hypothesis but also for the null hypothesis. Bayes factor (BF) values indicate how many times more likely the observations are under the alternative relative to the null hypothesis. According to common guidelines, BF values > 5 provide “moderate” support for the alternative hypothesis, and BF values > 10 indicate “strong” support. On the other hand, BF values < 0.33 provide “moderate,” and BF values < 0.1 provide “strong” support for the null hypothesis (van Doorn et al., 2020). We used the open-source statistics software JASP Version 0.12.2.0 to compute BF s using default priors (Love et al., 2019). In JASP, BF s are computed using the Savage Dickey density ratio method, which can be interpreted as the weight of evidence for one hypothesis over another (Wagenmakers et al., 2018; Wagenmakers, Lodewyckx, Kuriyal, & Grasman, 2010). We report the results of the Bayesian model averaging (BF mac ) implemented in JASP as analysis of effects across matched models.

RESULTS

Behavioral Results

On the behavioral level, we first tested whether participants were slower in No Stop trials as index of proactive inhibition. Indeed, participants were faster in Frequent Go (398 ± 99 msec) compared with No Stop trials (461 ± 75 msec), but also in Rare Go trials (441 ± 119 msec; Figure 1B). This was reflected in a significant main effect of Condition, \( F(2, 74) = 19.182, p < .001, BF = 1.261e + 6 \) (Frequent Go vs. No Stop trials, \( F(1, 37) = 44.164, p < .001, BF = 1.633e + 6 \); Frequent Go vs. Rare Go trials, \( F(1, 37) = 37.777, p < .001, BF = 87350.249 \); No Stop vs. Rare Go trials, \( F(1, 37) = 1.303, p = .261, BF = 0.706 \). This replicates previously observed effects of proactive inhibition, reflected in slower responding in No Stop trials (Liebrand et al., 2017). In contrast to our previous study,
the older participants in the current study also slowed down in Rare Go trials. Importantly, there was no effect of Group, meaning that patients were neither slower than controls nor did they show altered proactive inhibition: Group, \( F(2, 37) = 0.416, p = .663, BF = 0.507; \) Group × Condition, \( F(4, 74) = 0.985, p = .421, BF = 0.209. \) This was also true on the single-subject level, as no individual BG patient (all \( t > -1.4, p > .178 \)) nor any PFC patient (all \( t > 1.3, p > .211 \)) showed a significant RT slowing in Frequent Go in contrast to No Stop trials. The moderate evidence for the null effect speaks for behaviorally intact proactive inhibitory control in the patient groups.

Figure 2. Sensorimotor activity during cue–target interval. (A) Activity in the cue–target interval in PFC patients. Beta (15–25 Hz) power at sensorimotor clusters contra- and ipsilateral to the standard motor response is displayed. The analyzed time window (700–1100 msec) is indicated as dotted box, and the SEMs are shown as shaded area. The cue appeared at 0 msec, and the target stimulus was at 1100 msec. In PFC patients, beta power was lower on the contra- than on the ipsilateral side but did not differ between Maybe Stop and Certain Go trials. The topographic plot to the right shows the scalp distribution of the beta band in the Certain Go condition (lesion side indicated with black oval). (B) Mean mu and beta activity (700–1100 msec) in the cue–target interval in all three groups. Displayed is activity at the contra- and ipsilateral side to the upcoming standard motor response. The SEMs are displayed as error bars. In the beta band, PFC patients and controls showed increased activity on the contra- compared with the ipsilateral side. In the mu band, this effect was present in PFC patients only. (C) Mean mu activity (700–1100 msec) related to the conditions Maybe Stop and Certain Go in the three groups with no significant condition effects or Condition × Group interactions.

Participants did not commit more errors in Frequent Go than in No Stop trials (condition: \( F(1, 37) = 1.9, p = .180, BF = 0.386; \) and this did not differ between groups (group: \( F(2, 37) = 1.874, p = .168, BF = 0.774; \) Group × Condition: \( F(2, 37) = 0.468, p = .630, BF = 0.268. \) However, participants committed more premature errors in Certain Go than
Maybe Stop trials (condition: $F(1, 37) = 15.997$, $p < .001$, $BF = 467.187$), but these impulsive errors did not differ between groups (Group: $F(2, 37) = 0.674$, $p = .516$, $BF = 0.568$; Group × Condition: $F(2, 37) = 1.777$, $p = .183$, $BF = 0.629$). Patients did not differ from controls in the percentage of failed inhibitions (Group: $F(2, 74) = 1.3$, $p = .273$, $BF = 0.5$). This was also true when comparing single patients to the overall control group. No BG patient ($t < 1.3$, $p > .204$) and only two PFC patients ($t < 2.8$, $p > .010$) showed an increased rate of failed inhibitions compared with the control group, speaking for preserved ability to execute motor inhibition. Finally, we assessed in patients whether RTs differed if target stimuli were presented on the ipsi- or contralesional side. In Rare Go trials, RTs were similar for ipsilesional (440 ± 125 msec) and contralesional (442 ± 122 msec) stimuli and did not differ between patient groups: Laterality, $F(1, 37) = 0.0$, $p = .892$, $BF = 0.163$; Group, $F(2, 74) = 0.5$, $p = .634$, $BF = 0.550$; Group × Laterality, $F(2, 74) = 0.1$, $p = .945$, $BF = 0.194$.

In summary, we find evidence for a successful experimental manipulation of proactive inhibition with slower responses in trials when participants had to be prepared to stop and with more premature errors in trials when they were certain to respond. However, we find no effect of BG or PFC lesions on behavioral indices of proactive inhibition, and Bayesian statistics indicate moderate evidence against group differences in RTs.

**EEG Results**

The EEG was recorded with different systems and amplifiers at Lübeck (BrainAmp MR plus) and Oslo/Berkeley (BioSemi Active-Two). To ensure that this did not affect our results, we compared baseline corrected broadband (1–40 Hz) and alpha/beta power between the sites using independent-samples t tests. There was no difference in oscillatory power: broadband power, $t(38) = -0.7$, $p = .459$, $BF_{10} = 0.4$; alpha power, $t(38) = -1.0$, $p = .330$, $BF_{10} = 0.5$; beta power, $t(38) = -1.6$, $p = .109$, $BF_{10} = 1.0$. Moreover, there were no differences between recordings in Oslo and Berkeley: broadband power, $t(38) = 0.6$, $p = .582$, $BF_{10} = 0.6$; alpha power, $t(38) = -0.1$, $p = .909$, $BF_{10} = 0.5$; beta power, $t(38) = 0.7$, $p = .506$, $BF_{10} = 0.6$.

**Proactive Sensorimotor Effects**

First, we were interested in the modulation of mu and beta activity over the sensorimotor cortex during the cue–target interval (Figure 2). With regard to beta power, there was no effect of Condition, $F(1, 37) = 0.34$, $p = .854$, $BF = 0.163$, and no interaction of Condition × Group,

![Figure 3](https://example.com/figure3.png)

**Figure 3.** Effects of attentional modulation. (A) Alpha (9–14 Hz) power in PFC patients in the cue–target interval for the Maybe Stop (red) and Certain Go condition (black) at occipital clusters. The analyzed time-window (700–1100 msec) is displayed as dotted box. The cue appeared at 0 msec, and the target stimulus was at 1100 msec. Alpha power was lower in Maybe Stop than Certain Go trials at both sides. The SEM is displayed as shaded area. The topographic plot to the right shows the scalp distribution of the alpha band as difference between Maybe Stop and Certain Go trials. (B) Mean occipital alpha power between 700 and 1100 msec for all three groups. The SEMs are displayed as error bars. In both controls and patients, alpha power was decreased in Maybe Stop compared with Certain Go trials.
F(2, 37) = 0.219, p = .804, BF = 0.151. In PFC patients and controls, beta power was reduced on the contra- compared with the ipsilateral side: Group × Hemisphere, F(2, 37) = 5.3, p = .009, BF = 74.074. Across all groups, there was marginal evidence for decreased mu power in Maybe Stop compared with Certain Go trials (condition: F(1, 37) = 3.748, p = .061, BF = 1.285; Figure 2C), but this did not differ between groups (Group: F(2, 37) = 0.4, p = .690, BF = 0.256; Group × Condition: F(2, 37) = 0.5, p = .620, BF = 0.181). There was strong evidence for group differences in the lateralization of mu power: Group × Hemisphere, F(2, 37) = 4.8, p = .014, BF = 110.042 (Figure 2B). Only in PFC patients, mu was decreased on the contra- compared with the ipsilateral side: Hemisphere in PFC, F(1, 8) = 12.0, p = .009, BF = 517.568; Hemisphere in BG, F(1, 8) = 1.6, p = .243, BF = 1.428; Hemisphere in CTR: F(1, 21) = 3.0, p = .098, BF = 1.620. Summarizing, we find no evidence for condition effects on preparatory mu and beta power. However, mu and beta power reductions were more lateralized to the intact hemisphere after PFC damage, indicating a reorganization of function.

Preparatory Attentional Effects

To investigate the role of visual attention in proactive motor control, we examined condition differences in alpha power over occipital sites in the cue–target interval. After cue onset in both Maybe Stop and Certain Go trials, alpha decreased until about 400 msec and then increased again. Whereas alpha in Certain Go trials increased toward the baseline level, this rebound was dampened in Maybe Stop trials, resulting in reduced alpha power (Figure 3). We subjected mean alpha power between 700 and 1100 msec to a repeated-measures ANOVA with within-subject factors Condition, Stimulus (attended contra- vs. ipsilesional side), and Hemisphere (contra- vs. ipsilesional) and the between-subject factor Group. We found strong evidence for reduced alpha power in Maybe Stop compared with Certain Go trials, F(1, 37) = 33.8, p < .001, BF = 3.363e + 18. At the same time, the data provided anecdotal evidence against an interaction of this effect with the factor Group: Group × Condition, F(2, 37) = 0.7, p = .502, BF = 0.345. Importantly, in each of the three groups, we found strong evidence (BF > 100) for a condition effect on alpha (Figure 3).

As expected, the alpha power decrease was lateralized dependent on the attended stimulus side: Hemisphere × Stimulus, F(1, 37) = 25.008, p < .001, BF = 3481.667. In all three groups, alpha decreased more in the contra- than the ipsilesional hemisphere when preparing for ipsilesional target stimuli, F(1, 37) = 9.116, p = .005, BF = 10.007, and for contralesional target stimuli more in the
ipsi- than contralesional hemisphere, $F(1, 37) = 8.329$, $p = .006$, BF = 43.164. This effect did not differ between groups: Group × Stimulus × Hemisphere, $F(2, 37) = 0.110$, $p = .896$, BF = 0.126. Visual inspection suggested a reduced alpha suppression in PFC patients compared with BG patients and controls, but the factor group was not significant, $F(2, 37) = 2.174$, $p = .128$, BF = 0.812.

The CNV, measured in the cue–target interval, had its maximum at FCz/Cz (Figure 4) and increased toward the onset of the target. Visual inspection suggested that the BG group showed a sustained negativity in the cue–target interval with no continuous increase toward the target (Figure 4A). We subjected the area under the curve between 1000 and 1100 msec to an ANOVA with the within-subject factors Condition (Maybe Stop vs. Certain Go) and Electrode (FCz, Cz) and the between-subject factor Group (Figure 4B). Only in controls, but not in patients, an increased CNV in Maybe Stop compared with Certain Go was observed: condition for CTR, $F(1, 21) = 13.994$, $p = .001$, BF = 19.455; condition for BG, $F(1, 8) = 0.156$, $p = .703$, BF = 0.331; condition for PFC, $F(1, 8) = 0.040$, $p = .846$, BF = 0.324. Despite the moderate evidence against a condition effect in PFC and BG patients, the interaction with the factor Group was only marginally significant. The Bayesian analysis indicated equivocal evidence for and against the interaction: Condition × Group, $F(2, 37) = 2.977$, $p = .063$, BF = 1.082. There was no main effect of Group, $F(2, 37) = 1.801$, $p = .179$, BF = 0.803.

Summing up the results for the cue–target interval, proactive inhibition resulted especially in attentional modulation of occipital alpha activity and a modulation of the preparatory activity reflected in the CNV. Although neither PFC nor BG lesions impacted the occipital alpha modulation, both lesion groups were lacking the CNV modulation.

**Proactive Prefrontal Activity**

First, we analyzed whether beta power over prefrontal electrodes was modulated in the cue–target interval. We subjected mean beta power at prefrontal sites between 700 and 1100 msec to an ANOVA with the within-subject factors Condition (Maybe Stop vs. Certain Go) and Electrode (FCz, Cz) and the between-subject factor Group. There was trend level or anecdotal evidence for increased prefrontal beta in the Certain Go compared with the Maybe Stop condition, and no evidence for differences between groups regarding this effect: Condition, $F(1, 37) = 3.516$, $p = .069$, BF = 2.069; Group, $F(1, 37) = 1.638$, $p = .208$, BF = 0.722; Condition × Group, $F(2, 37) = 0.827$, $p = .445$, BF = 0.190 (data not shown). As in our previous study, we found no convincing evidence of a prefrontal beta effect when preparing to stop.

![Figure 5](image.png)

**Figure 5.** Target related prefrontal activity. Mean prefrontal beta power (200–500 msec) in all three groups. The SEMs are displayed as error bars. (A) Activity in frequent trials is shown. In patients and controls, beta power was increased when they prepared to inhibit the button press (No Stop) compared with trials where a certain motor action could be anticipated (Frequent Go). Also, beta power was increased in BG compared with PFC patients and controls. (B) Beta power over prefrontal sites in rare trials is displayed. Beta power both in controls and patients was increased when the motor action had to be inhibited (Stop) compared with when a button press was realized (Rare Go). Data are displayed without one BG patient, who was an outlier in terms of frontal beta power. (C) Topographic map of differential mean beta power (Stop minus Rare Go) between 200 and 500 msec after target signal appearance in control participants.
To test for a modulation of beta power after target signals, we subjected mean beta power over prefrontal electrodes between 200 and 500 msec to repeated-measures ANOVAs with factors Condition and Hemisphere (contra- vs. ipsilesional) and the between-subject factor Group (Figure 5). This was done separately for rare (Stop vs. Rare Go) and frequent (No Stop vs. Frequent Go) trials. The first comparison (Stop vs. Rare Go; Figure 5B) tests the modulation of beta when actually stopping an action as compared with trials with an equally infrequent go target stimulus. The second comparison (No Stop vs. Frequent Go; Figure 5A) tests to what extent the slower responses in No Stop trials, when participants had been prepared to stop, was also associated with increased frontal beta power.

Beta power over prefrontal electrodes was higher in Stop than Rare Go trials, $F(1, 36) = 7.271, p = .011, BF = 242.442$, with no difference between groups: Group × Condition, $F(2, 36) = 0.646, p = .530, BF = 0.278$. Note that one BG patient showed extreme beta power compared with other participants ($>5$ SDs above mean value in one condition) and was removed from the statistics for this comparison. Beta power was also increased in No Stop compared with Frequent Go trials, $F(1, 37) = 8.163, p = .007, BF = 1.639$, although Bayesian statistics indicated only anecdotal evidence. The effect did not differ between groups: Group × Condition, $F(2, 37) = 1.437, p = .251, BF = 0.278$. In No Stop and Frequent Go trials, prefrontal beta was increased in BG compared with controls, whereas PFC patients did not differ from controls, but the evidence for a group effect was only weak: group, $F(2, 37) = 3.072, p = .058, BF = 1.526$ (BG vs. CTR, $F(1, 29) = 5.994, p = .021, BF = 3.181$; PFC vs. CTR, $F(1, 29) = 0.140, p = .711, BF = 0.441$).

To summarize, actual stopping was associated with a relative increase of beta power over frontal electrodes across all three groups, whereas we found only weak evidence for a beta increase when only slowing down because of proactive inhibition (Table 2).

### DISCUSSION

We aimed to investigate the role of PFC and BG for proactive and reactive motor inhibition. To this end, patients with unilateral lesions to the BG or lateral PFC performed in a cued go/no-go task. We predicted both patient groups to show impairments in behavioral and neural indices of both proactive and reactive motor inhibition. Importantly, we did not find differences between patients and controls in behavior, reflected in comparable RTs and performance accuracy. In line with behavioral findings, apart from the CNV, patients did not show altered prefrontal, sensorimotor, or occipital oscillatory activity related to experimental
manipulation of proactive or reactive inhibition. These results support a preserved ability in both patient groups to employ preparatory and reactive inhibitory processes. However, in contrast to control participants, neither BG nor PFC patients showed a modulation of the CNV when anticipating stopping, although the evidence for a group by condition interaction was not strong. This indicates altered attentional and/or sensorimotor preparation, which did not result in performance deficits. Our results suggest that inhibitory functions in predictable proactive settings do not crucially rely on BG and PFC. Caution is warranted though as reorganization of function since the injury and the spared hemisphere might have helped to compensate for lesion-induced impairments (Krämer et al., 2013; Voytek et al., 2010; Levine et al., 2002; Stuss et al., 2000).

**Behavioral Results**

Patients were able to engage in proactive motor inhibition, as reflected in preserved RT slowing when movement cancellation was anticipated. Moreover, compared with controls, patients did not show an increased rate of failed inhibitions, speaking against an impairment in reactive motor inhibition. These results are in contrast to previous findings reporting behavioral deficits in motor inhibition in patients with lesions to PFC and BG (Krämer et al., 2013; Swick, Ashley, & Turken, 2008; Picton et al., 2007; Rieger, Gauggel, & Burmeister, 2003). How can these observations be reconciled? Whereas BG and PFC seem to be key regions in unpredictable situations calling for stopping (Aron, Herz, Brown, Forstmann, & Zaghoul, 2016), they might be of less importance in proactive settings where inhibition can be prepared for on a short timescale. In the present paradigm, participants were able to form reliable temporal predictions about any upcoming need for action inhibition using the cues and fixed cue–target intervals. In contrast, previous research focused on unpredictable, reactive inhibition as in go/no-go or stop signal tasks, or on cued inhibition with longer, variable cue–target intervals (Di Caprio et al., 2020; van Belle, Vink, Durston, & Zandbelt, 2014; Zandbelt, Bloemendaal, Negers, Kahn, & Vink, 2013; Jahfari et al., 2010). Single-interval predictions, as called for in this study, might rely more on the cerebellum, as suggested by a recent behavioral lesion study (Breska & Ivry, 2018). Intact behavioral performance despite altered preparatory CNV effects in the patients would be compatible with a cerebellar interpretation of the current findings. As we did not compare performance in different contexts, this remains to be investigated in future studies.

Results of lesion studies are also notoriously challenging to compare and interpret as exact lesion locations, size, etiology, and time since injury vary, rendering definite conclusions difficult. Also, because of the small sample size, we refrained from analyzing the effect of lesion side, although it might be that right- more than left-sided lesions impact response inhibition. However, behavioral results across patients were consistent, even on a single-subject level. Furthermore, we might have introduced a bias, with patients performing the task exclusively with their ipsilesional hand, that is, unaffected hemisphere. Indeed, there is some debate as to whether motor action execution and inhibition rely on overlapping (pre)motor circuits (Mirabella, 2014; Mattia et al., 2012). Asking patients to use the ipsilesional hand might have masked impairments in response inhibition resulting from the damaged hemisphere. However, several reviews report bihemispheric systems underlying response inhibition (Zhang, Geng, & Lee, 2017; Swick, Ashley, & Turken, 2011). Within these limitations, we suggest that frontal BG inhibitory control is context dependent, being strongest in unpredictable reactive situations and of less importance in proactive settings in which a demand for inhibition can be reliably predicted.

**Proactive Inhibition and Attentional Functions**

We previously found occipital alpha to be reduced in trials requiring proactive inhibition, speaking for an increased engagement of visual attention (Liebrand, Kristek, Tzvi, & Krämer, 2018; Liebrand et al., 2017). Here, we replicate this effect in controls and patients, indicating intact attentional modulation in both patient groups. This was unexpected, in particular for the PFC group, as PFC lesions have been associated with attentional deficits (Solbakk & Lovstad, 2014; Szczepanski & Knight, 2014; Barcelo, Suwazono, & Knight, 2000). Also, when performing in a stop signal task, patients with unilateral lesions to the PFC showed a reduced parietal N2 in response to stop signals, suggesting limited attentional capacity (Krämer et al., 2013). In contrast to these early visual responses, preparatory, lateralized visual alpha does not seem to be affected by prefrontal lesions. Alternatively, and as the lesions were unilateral, the intact hemisphere might have been sufficient to compensate for the damage (Voytek et al., 2010).

As in our previous study (Liebrand et al., 2017), controls had an increased CNV in trials calling for proactive inhibition (Maybe Stop) compared with trials without (Certain Go). This modulation was absent for BG and PFC patients, although it should be noted that the interaction with group was only marginally significant and the Bayesian analysis indicated equivocal evidence for and against an interaction. The CNV is the most prominent neural component implicated in both attentional and sensorimotor preparatory processes (Brunia & van Boxtel, 2001; Tecce, 1972; Walter, Cooper, Aldridge, McCallum, & Winter, 1964). Our results thus suggest an atypical attentional and/or motor preparation in patients. In line with our findings, previous work suggested both BG (Purzner et al., 2007; Bares & Rektor, 2001; Ikeda et al., 1997; Pulvermüller et al., 1996; Amabile et al., 1986) and PFC (Funderud et al., 2013; Rosahl & Knight, 1995) as sources of the CNV signal. However, there does not seem to be a
direct, linear relationship between CNV and behavior (Kononowicz & Penney, 2016), and in our study group, differences in the CNV did not result in behavioral impairments.

**Sensorimotor Activity**

In controls and both patient groups, no difference in sensorimotor beta between Certain Go and Maybe Stop trials was found, in contrast to the previously observed ipsilateral beta decrease in Maybe Stop compared with Certain Go trials (Liebrand et al., 2017). This might indicate an age-related effect, given that participants in our previous study were healthy young students. Here, in contrast, we recruited older participants to match the patient sample. Inhibitory control is a cognitive function, which is known to decline with age (Anguera & Gazzaley, 2012; Gazzaley & D’esposito, 2007), often explained by prefrontal changes (Coxon, Van Impe, Wenderoth, & Swinnen, 2012; Kramer, Humphrey, Larish, Logan, & Strayer, 1994). For instance, longer stop signal RTs have been observed in older adults, speaking for slowing of inhibitory processes (Coxon et al., 2012; Bedard et al., 2002; Williams, Ponesse, Schachar, Logan, & Tannock, 1999). However, also motor regions seem to be affected, as sensorimotor alpha decreases during sustained inhibition in older adults (Bönstrup, Hagemann, Gerloff, Sauseng, & Hummel, 2015). Moreover, in a go/no-go task, older participants showed a smaller decrease of sensorimotor beta during preparation and execution of movements and increased mu following response inhibition (Schmiedt-Fehr, Mathes, Kedilaya, Krauss, & Basar-Eroglu, 2016). Overall, attentional modulation of visual activity and an increased CNV turn out to be the most robust indicators of proactive control in this cued go/no-go paradigm, whereas modulation of actual sensorimotor activity reflected in mu and beta power seems to be less consistent (Liebrand et al., 2017, 2018).

Although we did not observe any condition effects on sensorimotor beta and mu, we found a much more pronounced lateralization of beta and mu in PFC patients compared with controls and BG patients. As motor-related changes in mu and beta power are supposedly generated in pericentral gyri (Ritter, Moosmann, & Villinger, 2009; Crone et al., 1998), which were spared in the lesion patients, these differences are likely due to altered prefrontal control over ipsilesional motor activity. As patients did the task with their ipsilesional hand, this altered sensorimotor activity did not affect task performance though. It would be interesting to test in future studies whether not only patients’ motor abilities but also their inhibitory performance decline when using the contralesional hand (Mattia et al., 2012).

**Prefrontal Beta Effects**

In previous studies with the cued go/no-go paradigm, prefrontal beta was increased in response to a target stimulus indicating to withhold or adapt a motor action or when motor inhibition had been anticipated (Liebrand et al., 2017, 2018). We interpreted these findings as increased engagement of prefrontal cognitive control when motor behavior might need to be adapted (Swann et al., 2009, 2011). Here, we replicated these results in control participants and in both patient groups. However, the beta increase in No Stop trials, when inhibition had been anticipated but did not have to be implemented, was less robust and Bayesian analyses indicated only weak evidence for the effect.

The lack of group differences stands in contrast to previous findings of impaired cognitive control in patients with lesions to the PFC. In a large voxel-based lesion symptom mapping study (n = 344), lesions in PFC were associated with behavioral deficits in cognitive control across several paradigms, with ACC and dorsolateral PFC as key regions (Gläscher et al., 2012). However, within the specific context of motor inhibition, a study using the stop signal task showed that patients with lesions to the lateral PFC displayed increased, possibly compensatory activity over intact prefrontal areas, indexed by an enhanced N2, together with unimpaired performance (Krämer et al., 2013). In this study, we did not observe any lateralization of the frontal beta effect, which might indicate that spared parts of the PFC were sufficient to generate the stopping-related beta increase. Note that this is in stark contrast to the clearly lateralized sensorimotor beta activity in PFC patients with a stronger beta decrease over the intact hemisphere (Figure 2). The remaining beta response over the damaged PFC might be explained with observations of widespread and heterogeneous stopping-related beta power increase over the PFC (Fonken et al., 2016).

Whereas numerous studies reported a stopping-related beta power increase over the PFC (Wessel, 2020; Liebrand et al., 2017, 2018; Picazio et al., 2014; Swann et al., 2009, 2011), a recent study employing multisite intracranial recordings from human subthalamic nucleus and PFC called into question the critical relevance of this effect for actual stopping (Chen et al., 2020). Importantly, although the authors replicate the finding of increased beta power after stop signals, this effect occurred after the estimated stopping latency and was unrelated to stopping latency or success (Chen et al., 2020). To conclude, beta power changes over prefrontal sites did not play a role when preparing to stop an action, that is, during the cue–target interval, in the present or previous studies with this cued go/no-go paradigm (Liebrand et al., 2017, 2018), and the beta modulation during action inhibition was not affected by the focal and unilateral PFC or BG lesions.

**Limitations**

Here, we report mostly null behavioral and electrophysiological findings, which are difficult to interpret. This study had a small sample size, which is typical for research in patient groups with focal lesions. Null results in chronic
and unilateral lesion patient samples may be due to activity in the intact hemisphere, and reorganization might have compensated for the functions compromised by the lesion. The exclusion of three BG patients due to poor behavioral performance might have bias toward null results, especially in the BG group, where lesions were heterogeneous.

However, other studies reported significant group effects in patient studies with comparable sample size (Krämer et al., 2013; Voytek et al., 2010; Swick et al., 2008; Ullsperger, von Cramon, & Muller, 2002). Also, behavioral results among included BG and PFC participants were consistent across participants. Moreover, whereas classical statistics (t tests and ANOVAs) are limited in drawing conclusions about null effects, Bayesian statistics can quantify evidence for the null hypothesis (Wagenmakers et al., 2018). Results from Bayesian analysis of our data support the absence of group differences both on a behavioral and neurophysiological level.

Conclusion

Our results show that unilateral BG and PFC lesions do not result in deficits in proactive, predictable situations calling for response inhibition. We observed that patients with lesions to BG and PFC showed intact behavioral markers of preparatory and reactive cancellation of actions. Apart from the CNV, no differential neural processing related to inhibitory processes was found. Within the discussed limitations of the present lesion study, we conclude that the involvement of BG and PFC in motor inhibition is context dependent. Whereas BG and PFC are key structures in demanding situations in which unpredictable, reactive stopping is required, they seem to be less important when inhibition can be reliably anticipated.

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Diversity in Citation Practices

A retrospective analysis of the citations in every article published in this journal from 2010 to 2020 has revealed a persistent pattern of gender imbalance: Although the proportions of authorship teams (categorized by estimated gender identification of first author/last author) publishing in the Journal of Cognitive Neuroscience (JoCN) during this period were M(an)/M = .408, W(oman)/M = .355, M/W = .108, and W/W = .149, the comparable proportions for the articles that these authorship teams cited were M/M = .579, W/M = .243, M/W = .102, and W/W = .076 (Fulvio et al., JoCN, 33:1, pp. 3–7). Consequently, JoCN encourages all authors to consider gender balance explicitly when selecting which articles to cite and gives them the opportunity to report their article’s gender citation balance. The authors of this article report its proportions of citations by gender category to be: M/M = .605; W/M = .25; M/W = .066; W/W = .079.

REFERENCES


Liebrand et al. 1875
Klimesch, W. (2012). Alpha-band oscillations, attention, and

Krämer, U. M., Solbakk, A. K., Funderud, I., Løvstad, M.,


control. PLoS One, 13, e0196855. DOI: https://doi.org/10

dynamics of proactive and reactive motor inhibition. Frontiers in Human

PMC5955690

toolbox for the analysis of event-related potentials. Frontiers in Human

PMC5406465

Love, J., Selker, R., Marsman, M., Jamil, T., Dropmann, D.,

Verhagen, J., et al. (2019). JASP: Graphical statistical software

for common statistical designs. Journal of Statistical Software, 88, 1–17. DOI: https://doi.org/10.18637/jss.v088.i02

Mattia, M., Spadacenta, S., Pavone, L., Quarto, P., Esposito, V.,


electrodes reveal a key role of premotor and motor cortices in stopping ongoing movements. Frontiers in Neuroscience, 5, 12. DOI: https://doi.org/10.3389/fnins.2012.00012, 22754525, PMCID: PMC3386527

Mirabella, G. (2014). Should I stay or should I go? Conceptual

underpinnings of goal-directed actions. Frontiers in Systems Neuroscience, 8, 206. DOI: https://doi.org/10.3389/fnsys.2014.00206, 25404988, PMCID: PMC4217496


Nuwer, M. R., Comi, G., Emerson, R., Fuglsang-Frederiksen, A.,


Purzner, J., Paradiso, G. O., Cunic, D., Saint-Cyr, J. A., Hoque, T.,


Rieger, M., Guggel, S., & Burmeister, K. (2003). Inhibition of ongoing responses following frontal, nonfrontal, and basal

control. Pla$$ One, 13, e0196855. DOI: https://doi.org/10

1.7131. Journal of Neurology, 2012.09.007,

https://doi.org/10.3389/fnhum.2014.00994, 28496405, PMCID: PMC5406465

PMC5955690

Jahfari, S., Verbruggen, F., Frank, M. J., Waldorp, L. J., Colzato, L.,


Jung, T. P., Makeig, S., Westerfield, M., Townsend, J., Courchesne,


Lavalle, C. F., Meeink, M. T., Herrmann, C. S., & Huster, R. J. (2014). When holding your horses meets the deer in the headlights: Time–frequency characteristics of global and selective stopping under conditions of proactive and reactive control. Frontier in Human Neuroscience, 8, 994. DOI: https://doi.org/10.3389/fnhum.2014.00994, 25540615, PMCID: PMC4262052


control. PLoS One, 13, e0196855. DOI: https://doi.org/10


