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Research Article

Decoding human response inhibition: evidence from GPi and thalamic electrophysiology during a go/no-go task

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ABSTRACT

The globus pallidus internus (GPi), a critical output structure of the basal ganglia, plays a central role in motor control by facilitating or inhibiting cortical commands through its connections with the thalamus. This study investigates the involvement of the GPi and thalamus in inhibitory processes during a Go/No-Go task in six patients undergoing deep brain stimulation (DBS) for dystonia or Tourette syndrome. Local field potentials (LFPs) were recorded from externalized DBS electrodes prior to pulse generator implantation. In line with recent computational models of the basal ganglia, we hypothesized differential activity in the GPi for Go and No-Go stimuli, reflecting its role in inhibitory functions. Our findings revealed distinct averaged LFP patterns in the GPi and thalamus to Go and No-Go stimuli, and in addition pronounced differences in beta-band time–frequency activity. These findings provide direct electrophysiological evidence for the GPi's involvement in proactive inhibition which paves the way for more fine-grained analyses of inhibitory functions.

Introduction

The globus pallidus internus (GPi) is the final output relay of the basal ganglia (BG) for the control of movements (DeLong, 1971; Alexander and Crutcher, 1990; Wichmann et al., 2018) by sending an inhibitory connection to the thalamus which in turn excites the cortex. Elaborating on the "standard model" of the BG (Albin et al., 1989; Mink, 1996), which holds that two main loops from the striatum team up to facilitate the appropriate cortical motor command, Frank (2006) proposed an anatomically inspired computer model that assigns distinct roles to the different relays of these loops. The direct pathway involves striatal medium spiny neurons (commonly referred to as 'Go' neurons), which predominantly express dopamine D1 receptors (Gerfen, 2000), and inhibit the GPi and the substantia nigra pars reticulata (SNr). By inhibiting the GPi/SNr the inhibitory output of the GPi/SNr is weakened, leading to a disinhibition of the thalamus, which thus activates the cortex and facilitates motor action. Neurons of the indirect pathway (NoGo neurons) harbor D2 receptors and inhibit the globus pallidus externus (GPe). The activity of the inhibitory projection of the GPe to the GPi/SNr is thus weakened, leading to a disinhibition of the GPi/SNr, which in turn results in suppression of the thalamus. According to the model of Frank (2006), the modulations of the direct Go- and indirect NoGo-pathways are action specific. By contrast, the excitatory hyperdirect pathway from the cortex to the subthalamic nucleus (STN) and onwards to both GPe (excitatory) and GPi/SNr (excitatory) is thought to modulate all actions in a non-specific manner. This circuitry leads to the facilitation of the most appropriate action and the simultaneous suppression of other less appropriate actions. In the anatomically inspired

Abbreviations: BG, basal ganglia; CM-VOI, center median/ventralis oralis internus; CT, computed tomography; DBS, deep brain stimulation; FoG, freezing of gait; GPe, globus pallidus externus; GPi, globus pallidus internus; LFP, local field potential; SNr, substantia nigra pars reticulate; SSRT, stop-signal reaction time; STN, subthalamic nucleus; Vim, ventralis intermedius.

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computational model of Frank distributed Go and NoGo units represent the positive and negative evidence for the competing action plans. This model suggests that differential activity should be observable in the GPi as a function of whether a stimulus requires an action (Go) or not (NoGo).

In humans, the GPi has become accessible to recording of electrophysiological activity, because it has been shown to be an appropriate target structure for deep brain stimulation (DBS) in various disorders (Lozano et al., 2019) including Parkinson's disease (e.g., Odekerken et al., 2013; Au et al., 2021; Wong et al., 2019), dystonia (e.g., Volkmann et al., 2014; Yousef et al., 2024; Chudy et al., 2023), Tourette syndrome (Aydin et al., 2024; Wehmeyer et al., 2021; Coulombe et al., 2019; Müller-Vahl et al., 2021), and Huntington's disease (e.g., Gonzalez et al., 2014; Bonomo et al., 2021; Gruber et al., 2014). Placement of electrodes in the thalamus or combined electrode implantation is an alternative strategy in Tourette syndrome (Müller-Vahl et al., 2021) or in individual patients with dystonia (Altamirano and Salinas-Barboza, 2024; Cif et al., 2025). Local field potentials (LFPs) can be recorded from target structures during cognitive or motor tasks when DBS electrodes are externalized between implantation of the electrodes and pulse generator placement (e.g., Cole et al., 2022; Münte et al., 2017; Heldmann et al., 2017; Neumann et al., 2022; Runge et al., 2024) allowing to assess the role of these subcortical nuclei to task execution.

With regard to the GPi, Herrojo-Ruiz et al. (2014) simultaneously recorded local field potentials from the sensorimotor region of the GPI and scalp EEG over the posterior medial frontal cortex in patients with idiopathic dystonia (without hand involvement) performing a flanker task in the context of DBS surgery. During error trials, a distinct error-related potential emerged in the pallidum approximately 60 ms before the cortical error-related negativity (ERN). These results indicate that the human pallidum is involved in performance monitoring, a key component of executive functions, and is exerting a feedforward influence on cortical error monitoring mechanisms. Navid et al. (2022) assessed LFPs either from the GPi (in patients with dystonia) or from the subthalamic nucleus (in patients with Parkinson's disease) during a variant of the flanker task. Theta-band activity time-locked to the cue stimulus was similarly enhanced in both GPi and STN recordings and response-related beta power was more strongly expressed in the GPi group. These findings suggest that GPi activity plays an important role in cognitive processes related to action selection and conflict resolution. Our own group has investigated the role of the GPi in reward processing by recording LFPs from patients with various movement disorders (Münte et al., 2017). In eight such patients performing a lottery task involving gain and loss feedback, we observed a feedback-related negativity at frontal midline EEG electrodes and valence-dependent modulations of GPi activity, including polarity inversions consistent with a local generator. Wavelet analyses further revealed reward-related responses in the high beta to low gamma range. These findings suggest that the human GPi contributes to reward processing, potentially via its projections to the lateral habenula.

In parallel, the thalamus has emerged as a critical subcortical hub in cognitive processing (Fiebelkorn et al., 2019; Jaramillo et al., 2019), with specific thalamic nuclei orchestrating synchronized activity in visual cortical areas during attentional tasks and maintaining dynamic interactions with the BG (Lopes da Silva et al., 1980; Saalmann et al., 2012; Zhou et al., 2016; Halgren et al., 2019). Recently, Mazetti et al. (2019) employed MEG and structural MRI and suggested that the GPi and thalamus are part of a subcortical executive control network.

In the present study, we employed a Go/NoGo task in six patients with dystonia or Tourette syndrome receiving DBS of the GPi and recorded from the DBS electrodes while they were temporarily externalized prior to implantation of the pulse generator. In light of the model of Frank (2006), we hypothesized that we should see differential activity in the GPi for Go and NoGo stimuli. Five of the patients also had additional electrodes placed in the thalamus which allowed to assess the contribution of the thalamus in addition.

Method

All procedures were approved by the ethical review board of Hannover Medical School. The study was performed in compliance with the Declaration of Helsinki. Patients gave informed consent prior to participation in the study.

Patients, Surgery, and electrode localization

Six patients with either Tourette syndrome or dystonia were included in the study (mean age 41 = years, range, 28 – 65 years, 3 women). All patients were right-handed. The patient characteristics are given in Table 1.

DBS electrodes were implanted in all patients bilaterally in the posteroventral lateral GPi guided by CT-stereotactic surgery aided by magnetic resonance imaging and microelectrode recording as described in detail elsewhere (Runge et al., 2023). Patients were implanted either under general anaesthesia (patients with Tourette syndrome) or under local anaesthesia (patients with dystonia). The preliminary target in the GPi was 20–22 mm lateral to, 4 mm below the intercommissural line, and 2–3 mm anterior to the midcommissural point. The thalamic target was the intersection of the center median/ventralis oralis internus (CM-VOI) nuclei in patients with Tourette syndrome, 5–6 mm lateral to, at the level of the intercommissural line, and 3–4 mm posterior to the midcommissural point, and the ventralis intermedius (Vim) nucleusin one of the patients with dystonia, 12 mm lateral to, at the level of the intercommissural line, and 4 mm posterior to the midcommissural point.

In all patients the Medtronic model 3387 (Medtronic Neurological Division, MN, USA) DBS electrode was used which harbors 4 contacts of 1.27 mm diameter and 1.5 mm length spaced 1 mm apart. Postoperative stereotactic CT confirmed DBS electrode placement according to the initially planned targets (see Neumann et al., 2018; Baldermann et al., 2024). There were no intraoperative or postoperative adverse events.

Stimuli

Stimuli comprised 188 black and white line drawings of animals and 188 further drawings of tools. Each image appeared centered in the middle of a video monitor positioned approximately 1 m in front of the patients for a duration of 100 ms and subtended approximately 5 degrees of visual angle in width and 4 degrees in height. The interstimulus interval was jittered between 1100 and 1350 ms (rectangular distribution). In any given experimental run, the task of the participants was to press a button for one category of stimuli (Go stimuli) while withholding a response to the other category of stimuli (NoGo stimuli). The participants gave responses with their right index finger using a response device held in their right hand. An illustration of the task is given in Fig. 1.

Data acquisition and analysis

Bipolar recordings were collected between various contacts of the DBS electrodes. Data was obtained from the recordings of the different contacts of the DBS electrodes. An important consideration when recording bipolar LFPs, as discussed by Herrojo-Ruiz et al. (2014), is that

Table 1
Patient characteristics.

Patient	Diagnosis	YGTSS, total	YGTSS, motor	Medication
#1, w, 28 y	Tourette	32	12	Neuroleptics
#2, m, 33 y	Tourette	38	20	Neuroleptics
#3, m, 35 y	Tourette	44	23	–-
#4, m, 37 y	Tourette	39	21	Neuroleptics
#5, w, 65 y	Dystonia	–-	–-	Benzod., L-Dopa
#6, m, 47 y	Dystonia	–-	–-	Baclofen

m, w, y = man, woman, years; YGTSS = Yale Global Tic Severity Scale; Benzod. = Benzodiazepines.

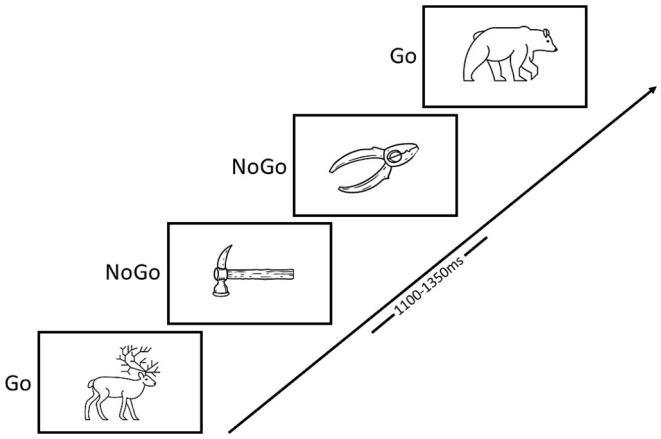

Fig. 1. Illustration of the Go / NoGo task. The designation of a stimulus category as Go or NoGo stimulus was changed for different experimental runs.

polarity cannot be definitively determined because the "differential" electrical potential is measured between two contacts located in or near the target structure. Additionally, depending on the exact placement of the recording sites, polarity may reverse between subjects. In the current data set, we carefully reviewed the individual patient data and found that, despite this potential issue, very similar results were obtained across patients, thanks to the highly consistent contact localization. We report and illustrate the derivations showing the greatest differences between conditions (always involving contact 3 in the GPi and contact 2 in the thalamus).

LFPs were computed by extracting for a time-window −100 ms to 1000 ms relative to the Go and NoGo stimuli. Only trials in which a correct response was given in the Go trials or response was withheld in the NoGo were included in the averages. Surface EEG was recorded in all patients from Fz electrodes referenced to the average activity at the two mastoid processes. Surface recordings were analyzed as described for the LFP. Data was low-pass filtered to 12 Hz and trials with amplitudes exceeding + 100 or −100 μV were rejected to address movement artifacts. The EEG was further visually inspected to catch remaining artifacts.

For the computation of time–frequency data, epochs extending from −2000 ms to 2000 ms with respect to the onset of the Go and NoGo trials were extracted. Again, only trials with correct responses were included in the analysis. Single trials were convoluted with a 7 cycle complex Morlet wavelet for frequencies ranging from 1 Hz to 40 Hz (linear increase). Changes in time varying energy (square of the convolution between wavelet and signal) were computed for each trial and averaged for each condition. Power increase or decrease was computed with respect to baseline (−200 to 0 ms before the stimulus).

As in Münte et al. (2017), to reduce the inter-individual variability in the amplitude of the electrical activity, we normalized the activity of the two conditions for each participant by subtracting the mean and dividing by the standard deviation of the time series of the merged two conditions. In the time frequency data, this was done independently for each frequency. In addition, in the time–frequency data, in order to address the multiple-comparison problem, we used the nonparametric permutation test proposed by Maris and Oostenveld (2007), clustering sensor-time–frequency points. We performed all possible permutations between the two conditions ($27 = 128$) and only those clusters with $p < 0.05$ (corrected) will be reported. Data were sampled at 1000 points/s and data analysis was performed using EEG-Lab (Delorme and Makeig, 2004) and ERP-Lab (Lopez-Calderon and Luck, 2014) software.

Results

Behavioral data

Percentage of correct Go responses was 84.2 + -8.8 % and the percentage of correct NoGo trials was 83.3 + -14.9 %. There were no significant differences between the percentage of correct Go and NoGo responses (signed-rank = 12, $p = 0.81$). The d-prime of participants was 2.58 + -1.20, indicating a good performance in the task. The reaction time to correct go responses was 507 + -111 ms.

Averaged LFP

Event-Related potentials showed an increased P300 for the NoGo cue response compared to the Go one at the Fz electrodes (Fig. 2). The two conditions were significantly different at around 410 ms until 450 ms and then between 600 ms and 800 ms.

Contacts at the GPi showed significant differences between the two conditions in both right and left locations. In the right GPi, the bipolar montage between contacts 3 and 1 revealed significant differences between 330 ms and 420 ms, while the difference between electrodes 3 and 2 showed significant differences between 250 ms to 316 ms and between 467 and 494 ms. Notably, the earlier activity exhibited an inverted polarity pattern between the two electrode pairs. In the left GPi, the montage between contact 3 and 0 showed significant differences between the two conditions between 363 ms and 390 ms. Interestingly, an earlier activity was found in the differential activity between contacts 3 and 1 in the 146–180 ms time range (Fig. 2).

In the five patients who had also electrodes to record from the thalamus (Fig. 3), LFPs revealed significantly higher activity for Go compared to the NoGo condition in the time ranges 147–186 ms, 270–360 ms and 708–848 ms after cue presentation between contacts 2 and 1 in the right thalamus. In contrast, the montage between electrodes 3 and 2 revealed significant differences between 304 and 360 ms. In the left thalamus, significant differences were found between 260–485 ms (montage between electrodes 2 and 0), and between 600 and 658 ms as well as between 701 and 754 ms for the montage between electrodes 3 and 2. Importantly, Go activity was larger (either positive or negative) than NoGo activity.

Time-frequency data

Both left and right GPi presented significant differences in power (Fig. 4) among conditions in the low beta band (12–15 Hz), 500–600 ms after the stimulus. This activity lasted longer in the right GPi, starting around 400 ms in the difference between electrodes 1–0, and with additional activity in the beta band (15–25 Hz). In addition, the electrode contacts 3–2 presented a very early significant difference among conditions at 10 Hz in the first 200 ms after stimulus presentation.

We did not find task-related modulations of thalamic time frequency data.

Discussion

Our findings demonstrate differential involvement of the GPi and thalamus in Go/No-Go task execution, aligning with Frank's (2006) computational model that situates the GPi within both the direct and indirect pathways. Consistent with our hypothesis, distinct patterns of averaged LFP activity were observed in the GPi for Go and NoGo stimuli. Specifically, in the GPi, significant differences between conditions were evident in both hemispheres, with temporal dynamics and polarity variations between electrode pairs. These findings align with the notion that the GPi integrates and modulates action-specific signals in accordance with the direct (Go) and indirect (NoGo) pathways of the BG. Moreover, the GPi exhibited condition-specific activity patterns in the time–frequency analysis, with Go stimuli eliciting higher activity across

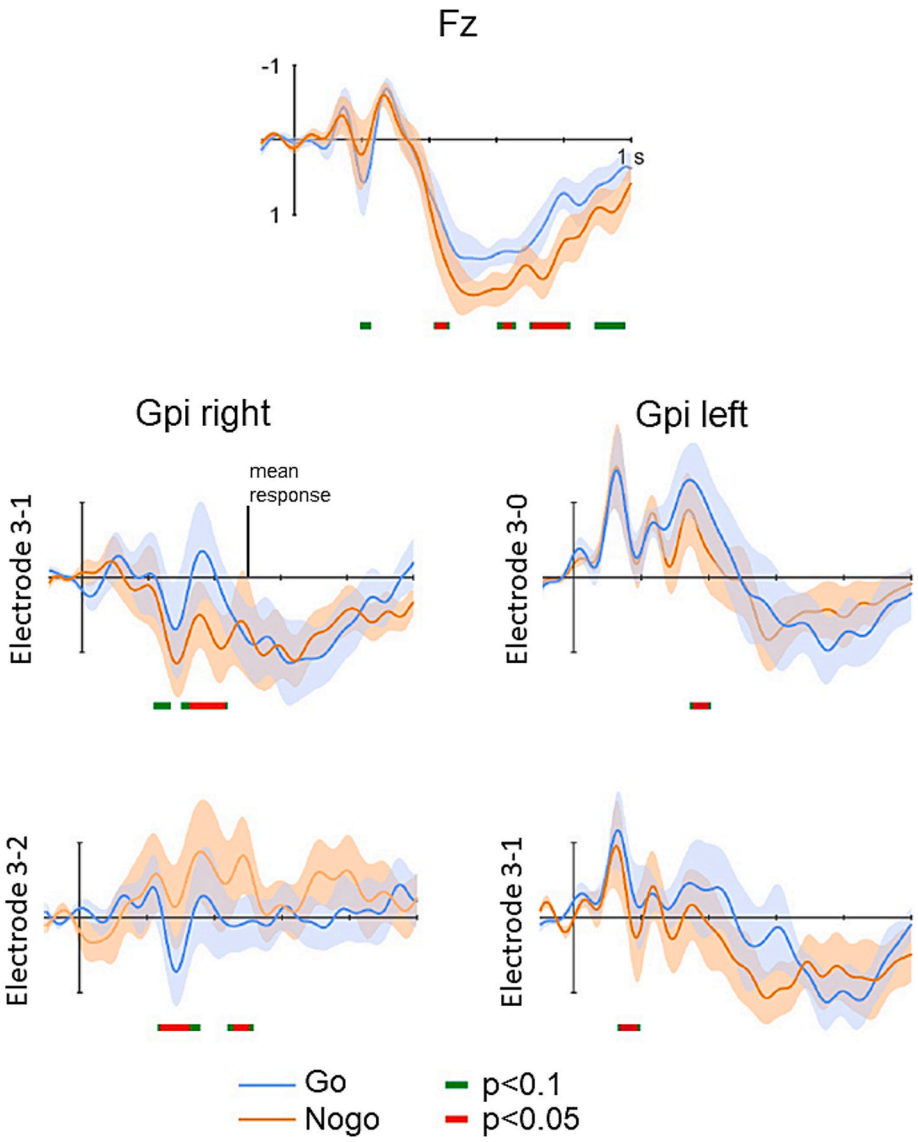

Fig. 2. Time domain averages of surface activity (Fz referenced to the mean activity at the two mastoid processes) and local field potentials from the GPI. To account for inter-individual amplitude differences data was normalized as described in Münte et al. (2017). Time 0 marks the stimulus onset. Event-Related potentials of the Cue Go (blue) and NoGo (orange) responses at Fz (top) and LFPs in the right and left GPi. Shaded areas indicate the standard deviation of the mean in each time point. Red line: $p < 0.05$; Green line: $p < 0.1$.

multiple time windows and frequency bands. Importantly, early beta-band differences in the GPi suggest complementary roles for these structures in the facilitation of motor actions and the suppression of competing responses.

Paradigm

The Go/NoGo paradigm and the Stop-Signal paradigm are widely used to study inhibitory control, yet they differ in their design and cognitive processes. The Go/NoGo paradigm used in the current study requires participants to respond quickly to "Go" stimuli while withholding responses to "NoGo" stimuli, emphasizing ***proactive*** inhibition. In contrast, the Stop-Signal paradigm presents a "Go" task interrupted by a stop signal, requiring participants to cancel an already-initiated response, thus engaging ***reactive*** inhibition (Verbruggen and Logan, 2008). While the Go/NoGo task primarily assesses sustained attention and response inhibition, the Stop-Signal task focuses on the ability to abort prepotent responses, often yielding longer reaction times due to post-error slowing. EEG/ERP studies using surface recordings in normal participants have revealed distinct neural mechanisms underlying these tasks. For instance, NoGo-N2 and NoGo-P3 components have been consistently observed in Go/NoGo tasks, reflecting conflict monitoring and inhibition processes (Falkenstein et al., 1999). In the Stop-Signal paradigm, the stop-signal reaction time (SSRT) and associated ERP components, such as the stop-P3, provide a measure of the efficiency of the inhibitory process (Kok et al., 2004). Go/NoGo and stop-signal paradigms thus complement each other in understanding the dynamics of cognitive control and its neural correlates. The surface ERP (Fig. 1, upper panel) clearly shows a NoGo-P3 in the patients, whereas there was only a hint of a NoGo-N2 in the surface ERP. The invasive data from the GPi show a clear differentiation of Go and NoGo responses as early as 200 ms in the LFP and a clear differential response in the beta-band in the time frequency analysis starting around 400 ms. This shows an involvement of the GPi in proactive inhibition. In addition, the recordings from the thalamus reveal differential activity for Go and NoGo responses which is in line with the circuitry outlined in basal ganglia models. In this regard, the question arises as to the relationship of the LFP activity and the motor responses. We (Münte et al., 2008) and others

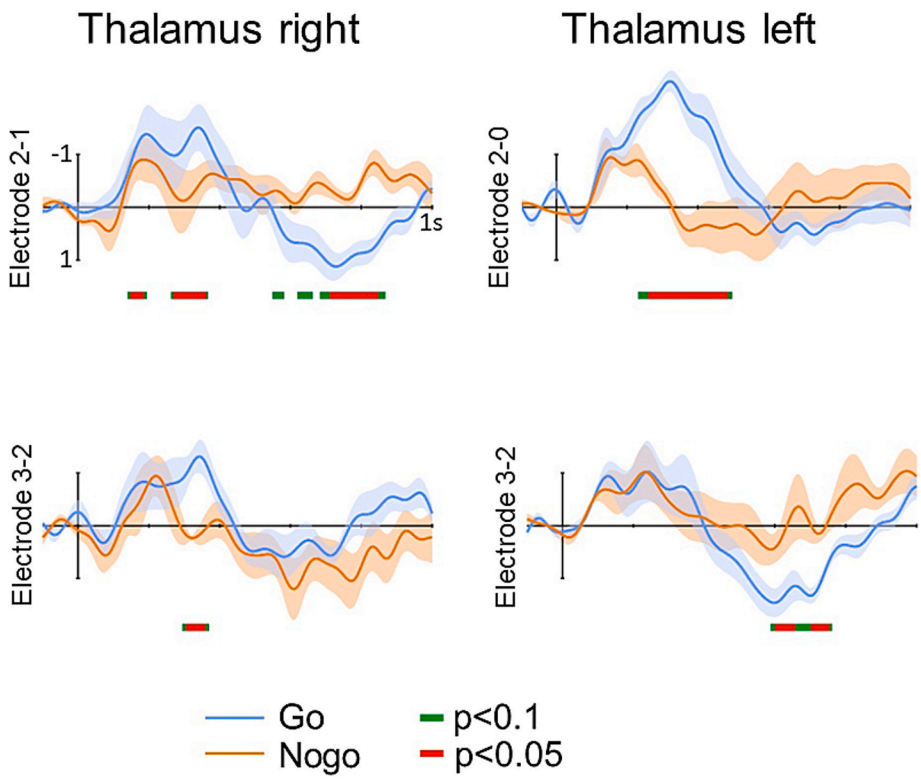

Fig. 3. LFPs from the right and left thalamus. Shaded areas indicate the standard deviation of the mean in each time point. Red line: $p < 0.05$; Green line: $p < 0.1$. To account for inter-individual amplitude differences data was normalized.

(e.g., Sildatke et al., 2021; Schüller et al., 2021) have reported intracranial recordings using choice reaction paradigms, which typically are evaluated by time-locking the neural responses to the response. A response-locked evaluation is less informative in a Go / Nogo paradigm, as used in the current study, because NoGo stimuli do not entail a response. As the mean reaction time was 507 ms in the current group of patients, the later effects (from 500 ms onward) might well be related to the motor response (c.f., Tran et al., 2024).

Previous evidence for inhibitory processes from invasive recordings in humans

While the current study appears to be the first to examine inhibitory processes in the GPi, previous studies have investigated another key-structure in the Frank (2006) model, i.e. the STN. For example, Ray et al. (2012) demonstrated that LFP recordings in the STN showed increased beta-band activity during tasks requiring response inhibition, suggesting its role in suppressing motor responses. Alegre et al. (2013) corroborated these findings by linking successful response inhibition in the stop-signal task with beta oscillations in the STN, particularly in patients with Parkinson's disease. Benis et al. (2014) further dissociated proactive and reactive inhibition, observing distinct STN activity patterns, with proactive control linked to preparatory neural activity and reactive inhibition marked by beta oscillations. Similarly, Bastin et al. (2014) identified STN neurons as critical for both inhibitory control and error monitoring, highlighting their dual role in suppression and performance evaluation. Wessel et al. (2016) found that stop-related beta activity in the STN reflects global motor suppression, further emphasizing its importance in reactive inhibition. Additionally, van Wijk et al. (2017) noted that decreased cortico-pallidal coherence, particularly in the beta band, correlates with faster reaction times, underscoring the functional connectivity of the STN in movement regulation. Finally, Fischer et al. (2017) showed that gamma-band activity in the STN increases not only during motor execution but also during movement inhibition, illustrating its involvement in both initiating and halting motor actions. These studies collectively underscore the pivotal role of the STN in the neural circuitry underlying inhibitory control.

With regard to the GPi and the thalamus there is less previous evidence for an involvement in inhibitory control from human invasive data. The studies of Navid et al. (2022) [primed flanker task], Herrojo-Ruiz et al. (2014) [flanker task], Münte et al. (2017) [gambling task] and Bockova et al. (2011) [three stimulsus novelty oddball task] did not involve paradigms specifically addressing inhibitory processes. Herrojo-Ruiz et al. (2014), however, hypothesized that pallidal activity emerging around 200 ms before a button press might reflect increased inhibition needed to enable correct response selection in the flanker paradigm. However, this interpretation is constrained by their experimental design, as the observed activity may also involve overlapping processes such as general or lateralized inhibition, response selection, and reward prediction. Bocková et al. (2011), on the basis of a single patient with GPi electrodes, suggested that early STN activation may suppress responses by inhibiting further cognitive processing and motor output, while concurrent GPi activity could contribute to thalamo-cortical inhibition of the motor program, consistent with earlier models (Mink, 1996; Coxon et al., 2006; Aron et al., 2007).

The current data set thus presents more direct evidence for an involvement of GPi in inhibition which is consistent with the model of Nambu et al. (2012). These authors suggested a central role for the cortico–subthalamo–pallidal hyperdirect pathway in a dynamic center-surround model of basal ganglia function during voluntary limb movements. According to this model, an initial corollary signal via this pathway broadly suppresses activity in the thalamus and cortex, thereby inhibiting both the intended and competing motor programs. This is followed by a more selective disinhibition via the direct pathway, allowing the release of the chosen motor program. A third signal, potentially routed through the indirect pathway, reinforces suppression of competing actions. This sequential processing ensures precise initiation and execution of the selected movement while canceling

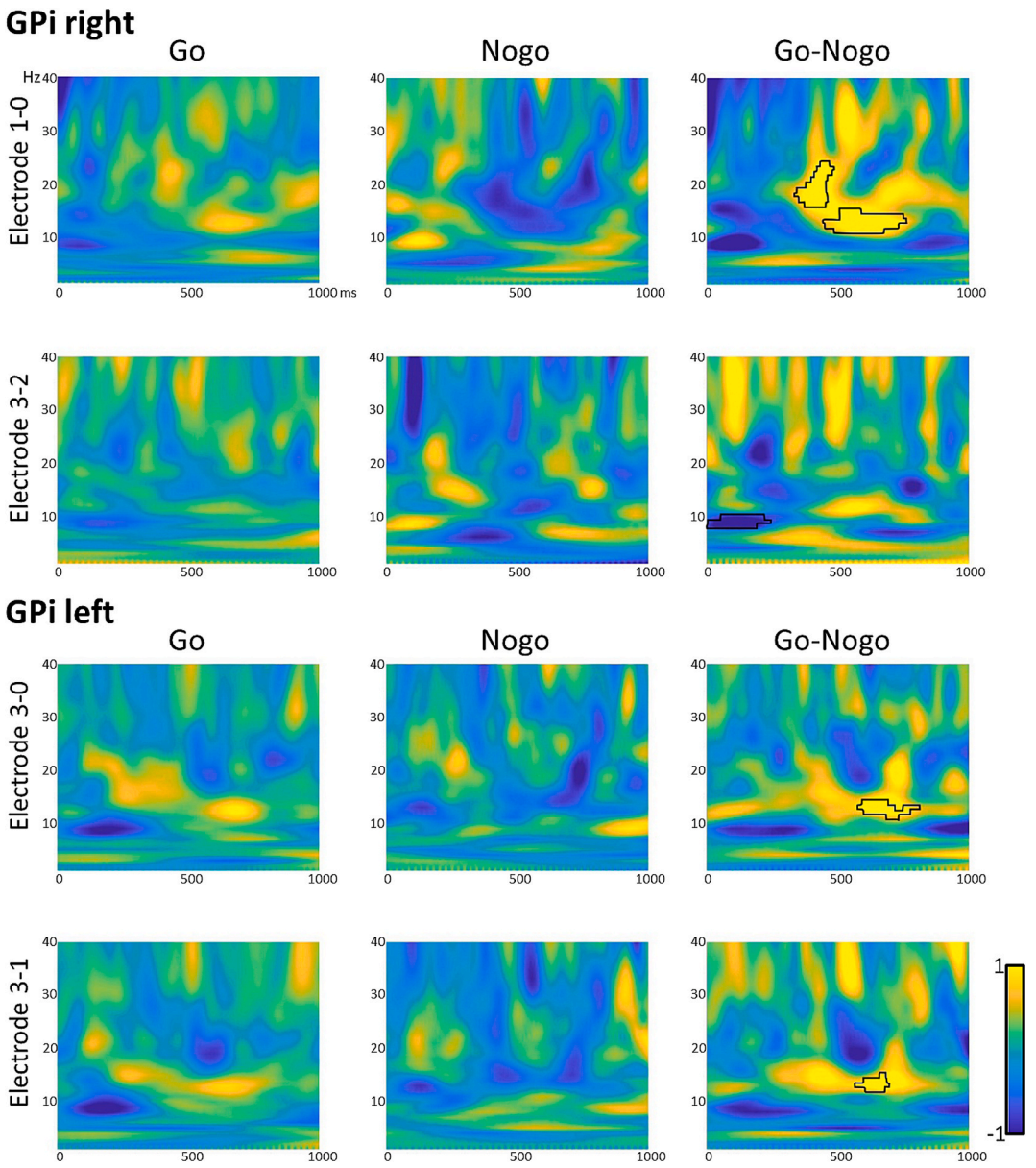

Fig. 4. Time frequency analysis of the GPI data (bipolar derivation, contacts indicated). The third column shows the difference in power for gain minus loss trials. Time 0 marks the stimulus onset. Power change in the right and left GPi related to the GO and NoGo cues, and its difference. Solid black line indicates the significant difference between the conditions corrected for multiple comparisons using a cluster permutation test ($p < 0.05$).

alternatives. The model thus places the GPi in the context of inhibitory functions.

Outlook and clinical implications

The current study as well as the work cited in the previous paragraph highlights the role of the STN, GPi and thalamus in inhibitory processes supported by basal ganglia cortical networks as proposed by Frank (2006). Yet, the emerging picture remains sketchy. In particular, it is not possible to relate neural effects to the model of Frank (2006) or other models of BG function (e.g., Schroll and Hamker, 2016). Importantly, the proportion of Go and NoGo stimuli in Go/NoGo tasks influences how participants predict upcoming stimuli and regulate their responses, ultimately shaping cognitive control and proactive inhibition (Li et al., 2016; Young et al., 2018; Wessel, 2018; Zhang et al., 2024; Georgiev et al., 2016). For instance, when the paradigm features a higher prevalence of Go stimuli, participants are more inclined to anticipate Go stimuli, facilitating quicker and more accurate responses. On the other hand, a greater proportion of NoGo stimuli leads participants to expect these stimuli more often, making it easier to proactive inhibition (Hester et al., 2004; Wessel, 2018). Investigating brain activity from patients with DBS of the GPi and the STN (a) by manipulating the ratio of Go and NoGo stimuli to assess different degrees proactive inhibition and (b) by contrasting experimental runs with and without stop-signals to delineate brain signatures of proactive and reactive inhibition (c.f., Soh et al., 2021) will provide a clearer picture of the role of STN and GPi in different aspects of inhibition. Such an approach might also enable a more direct relation of neural data to computational accounts.

We further speculate that in Parkinson's disease the individual pattern of neural effects obtained by a comprehensive mapping of inhibitory functions might be related to clinical manifestations of aberrant motor inhibition in this condition, e.g. freezing. Initial but purely behavioral evidence in this regard was provided by Cohen et al. (2014) who found that patients with freezing of gait (FoG) showed deficits in tasks associated with inhibitory control compared with patients without FoG. FoG scores as determined by experienced clinicians

were correlated with failures to respond on Go trials as well as with an inability to inhibit responses in NoGo trials in the Go-NoGo task. Neurophysiological markers obtained in the aforementioned fashion might also be used to predict response to DBS treatment (see Bahners et al., 2024, for a similar approach using DBS evoked potentials).

Limitations

As is often the case with invasive recordings in humans, the current study was performed in a limited group of participants. Moreover, patients are available only for a limited time between the first and second surgery, precluding a more fine grained investigation of proactive and reactive inhibition processes. While centrally active medication might also influence subcortical activity, the present data set does not allow a formal assessment of medication effects. Advancements in sensing devices (e.g., the Percept® system) could help overcome these limitations by enabling wireless, real-time recordings from patients across multiple sessions post-surgery (e.g., Provenza et al., 2024). Moreover, the target structures are selected for stimulation because of their known or suspected dysfunction—such as the STN in Parkinson's disease or the GPi in dystonia. Yet, experiments aim to understand the normal cognitive role of these structures, which creates a certain disaccord. Given these constraints, it may be more practical to include a diverse group of patients who share the target structure but have different underlying pathophysiology, as was done in this study. As it will never be possible to obtain invasive data from healthy individuals, a triangulation approach supplementing invasive recordings from patients with surface electrophysiology and functional imaging in normal participants and patients is needed for a fuller picture.

CRediT authorship contribution statement

Thomas F. Münte: Writing – original draft, Resources, Conceptualization. **Josep Marco-Pallares:** Writing – review & editing, Software, Methodology, Formal analysis. **Marcus Heldmann:** Writing – review & editing, Investigation, Formal analysis. **Seza Bolat:** Writing – review & editing, Methodology, Investigation. **Assel Saryyeva:** Writing – review & editing, Validation, Resources, Project administration. **Kirsten Müller-Vahl:** Writing – review & editing, Supervision, Resources. **Joachim K. Krauss:** Writing – review & editing, Validation, Supervision, Methodology.

Declaration of competing interest

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

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Data availability

The data supporting the findings of this study are available from the corresponding author on reasonable request.

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