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ORIGINAL ARTICLE

Validity expectancies shape the interplay of cueing and task demands during inhibitory control associated with right inferior frontal regions

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Abstract

The neural mechanisms of inhibitory control have extensively been studied, including the effects of demands to engage in inhibitory control and the effects of valid and invalid cueing. Theoretical considerations, however, suggest that the aforementioned factors exert joined effects on response inhibition processes that are further modulated by the subject's experience about the reliability of cue stimuli during response inhibition processes. To examine the underlying neurophysiological processes of these interactive effects we combined EEG signal decomposition with sLORETA source localization. We show that response inhibition performance is modulated by interactive effects between (1) cue information/validity, (2) demands on inhibitory control processes and (3) the subject's experience that cue information is valid/invalid during response inhibition processes. Only if demands on inhibitory control processes are high and when participants acquainted the experience that cue information is very likely to be valid, invalid cue information compromised response inhibition performance. The neurophysiological data show that processes in the N2 time window, likely reflecting braking processes, but not stimulus-related processes during response inhibition, are modulated. It seems that braking processes cannot be sufficiently deployed if cue information that has been experienced to be highly valid turns out to be invalid in situations placing high demands on inhibitory control. Source localization data reveals that the interactive effects of the examined factors specifically modulate processes in the right inferior frontal gyrus (BA47). This provides electrophysiological evidence that the rIFG is a hub region integrating different factors modulating inhibitory control.

Keywords Response inhibition · Inferior frontal gyrus · EEG · Source localization

Introduction

Neural mechanisms underlying response inhibition have extensively been studied. A well-established finding is that the frequency of occasions in which it is necessary to engage in inhibitory control (e.g. in Go/NoGo tasks) affects demands on response inhibition processes (Helton et al. 2005; Helton 2009; McVay and Kane 2009; Stevenson et al. 2011; Quetscher et al. 2015; Dippel et al. 2016; Wessel 2018). Response inhibition processes are more demanding when the probability to suppress a response is low (Dockree et al. 2004, 2006; Dippel et al. 2016, 2017). In such cases, the rate of false alarms increases (i.e. erroneous responding in NoGo trials). A possible reason is that a high probability that an upcoming stimulus is a Go stimulus (i.e. has a "Go-identity"), combined with a correspondingly low probability that an upcoming stimulus is a NoGo stimulus (i.e. has a "NoGo-identity"), facilitates uncontrolled, automated responding (Helton et al. 2005; Helton 2009; McVay and Kane 2009; Stevenson et al. 2011; Quetscher et al. 2015). However, the probability that an upcoming stimulus has a certain identity is not the only factor that may affect response inhibition performance. For example, the dual mechanisms of control framework dissociates proactive from reactive control situations (Braver 2012). Proactive control relies on the anticipation of an upcoming interference, or occasion to engage in high levels of cognitive control. Crucially, such anticipation of an upcoming event and the possibility to engage in proactive control strongly relies on the validity

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with which an upcoming event can be predicted, e.g. using a hint (cue). Indeed, several lines of research suggest that prior cueing can affect response inhibition and that invalid cues compromise performance (Smith et al. 2007; Randall and Smith 2011; Vuillier et al. 2016; Liebrand et al. 2017; Hong et al. 2017). Yet, effects related to the probability of "stimulus identity" and "cue validity" on response inhibition have so far only been considered independently from each other. Yu and Dayan (2005), however, suggested that uncertainty about the "identity" and the "validity" of a (cue) stimulus are processed via distinct neurobiological systems and may, therefore, jointly determine performance on a task. Taking all this together, it is conceivable that response inhibition performance is affected by *joint effects* of the probability/certainty about the identity of an upcoming stimulus (whether it is Go or NoGo) and the validity of a cue that an upcoming stimulus has a certain identity. To the best of our knowledge, the neurophysiological processes and associated functional neuroanatomical structures underlying these effects are elusive. Here, we close this gap analyzing EEG data with a focus on temporal EEG signal decomposition and source localization.

We hypothesize that changes in the validity of a cue stimulus should have different effects depending on the demands on response inhibition processes; i.e. depending on the probability whether an upcoming stimulus it is Go or NoGo stimulus. The reason is as follows: since response inhibition processes are more demanding when the frequency of inhibition of a response is low (Dockree et al. 2004, 2006; Dippel et al. 2016, 2017), valid cue information should be particularly helpful. Therefore, we hypothesize that valid and invalid cue information effects should mostly be evident when there is a low probability that an upcoming stimulus is a NoGo stimulus. Importantly, the effect of valid and invalid cue information should further differ depending on whether this cue information is supposed to be valid. If a cue stimulus is highly likely to be valid, a violation of this assumption should lead to severe problems in response inhibition. If a cue stimulus is highly improbable to be valid, it is likely that the subject learns not to take this information into account. As a consequence, no assumptions may be made whose violation could compromise response inhibition. Therefore, we hypothesize that declines in response inhibition performance are stronger when invalid cue information is presented in a context in which this cue information is supposed to be highly valid, compared to a context where this is not the case. This effect should then be strongest when response inhibition processes are more demanding; i.e. when the frequency of NoGo trials is low. Importantly, Yu and Dayan (2005) frame the effects of "identity" and "validity" on task performance within a Bayesian framework. In such frameworks, extracted regularities about the context (e.g. cue validity) shape further expectations and ultimately mediate learning effects (Friston et al. 2017). The prior learning experience of stimuli has already been shown to affect inhibitory control of actions and the probability that new stimulus information is integrated to inform inhibitory control (Stock et al. 2017a). It is, therefore, likely that it makes a difference whether people are first confronted with cues having a low, or a high probability of being valid. We assume that when people are initially confronted with a high probability of invalid cues, they learn not to take them into account. The opposite is likely when people are initially confronted with a high probability of valid cues. Therefore, we hypothesize that interactive effects between "identity" and "validity" are only evident when people are initially confronted with a high probability of valid cues.

Regarding cognitive-neurophysiological processes and associated functional neuroanatomical structures, it is crucial to consider that neural activity of the motor inhibition process overlaps with processes related to non-motor, stimulus-driven (attentional) processes (Mückschel et al. 2017b; Hong et al. 2017). Against the background of functional neuroanatomical data, there is an intense debate whether activations of the cortical inhibitory control network are due to stimulus-dependent attentional processes or due to (motor) response-inhibition processes per se (Simmonds et al. 2008; Sharp et al. 2010; Hampshire et al. 2010; Dodds et al. 2011; Hampshire 2015; Hampshire and Sharp 2015; Aron et al. 2015). Indeed, stimulus-related processes are important during response inhibition (Boehler et al. 2009; Chmielewski and Beste 2016a, b), and they are able to predict response inhibition performance (Boehler et al. 2009; Stock et al. 2016). The distinction between motor and non-motor processes is even more an issue considering that event-related potentials (ERPs) are composed of various amounts of signals from different sources (Nunez et al. 1997; Huster et al. 2015; Stock et al. 2017b). Moreover, they reflect a mixture of different codes related to stimulus processing ('stimulus codes') and response selection ('response selection codes') (Mückschel et al. 2017b; Chmielewski et al. 2018). While there are well-described ERP indices of response inhibition (i.e. NoGo-N2 and NoGo-P3) (Huster et al. 2013), it has been shown that these intermingle coding levels during the inhibition of responses, especially in the N2 time window (Mückschel et al. 2017a; Chmielewski et al. 2018). This has been delineated by means of a temporal EEG signal decomposition method—residue iteration decomposition (RIDE) (Ouyang et al. 2015a, b). Using RIDE, stimulus-related codes (perception/attention) are captured by the S-cluster (Ouyang et al. 2011, 2015a), and "response selection codes" are reflected by the RIDE C-cluster (Verleger et al. 2014, 2017; Bluschke et al. 2017; Ouyang et al. 2017; Mückschel et al. 2017a). During inhibitory control, the supplementary motor area processes stimulus codes (S-cluster) and response selection codes (C-cluster) (Mückschel et al.

2017a); whereas the inferior frontal cortex only processes response selection codes (Mückschel et al. 2017a). We hypothesize that especially the C-cluster should be modulated. The reason is that previous findings suggest that the effects of valid and invalid cue information mainly affect processes related to (motor) responding and response selection during response inhibition (Smith et al. 2007; Randall and Smith 2011; Vuillier et al. 2016; Liebrand et al. 2017; Hong et al. 2017). Moreover, such processes are modulated by the probability to engage in inhibitory control processes (Dockree et al. 2004, 2006; Dippel et al. 2016, 2017). It is, therefore, possible that the combination of these factors also modulates response coding processes reflected by the C-cluster. Since previous findings suggest that modulations in the C-cluster are associated with inferior frontal cortex activity, we hypothesize that inferior frontal cortical regions are modulated by interactive effects between (1) cue information/validity, (2) demands on inhibitory control processes and (3) the subject's experience that cue information is valid/invalid during response inhibition processes.

Materials and methods

Sample size calculation and participants

The study hypothesis states an interaction between "cue validity" (valid vs. invalid), "ratio" (1:6 vs. 4:5 NoGo/Go trials) and the "order of blocks". The latter factor is a between-subject factor, the others are within-subject factors. Previous own data has shown that the probability of an upcoming stimulus having a certain "ratio" has strong effects on inhibitory control ($\eta_p^2 = 0.35$) (Dippel et al. 2017). However, further interactive effects with the factors "cue validity" and the "order of blocks" may be much weaker. Therefore, we conservatively considered a smaller effect size between $\eta_p^2 = 0.07$ and $\eta_p^2 = 0.10$ to be detectable with a power of at least 90%. The power analysis revealed that a sample of $N = 30$ is required for that. Importantly, the actually observed effects size (see "Results") was larger.

We enrolled $N = 31$ healthy young right-handed volunteers (15 females) between 20 and 25 years of age (23.9 ± 2.6) in the experiment. Participants were randomly assigned to one of two groups, which did not differ in mean age ($t(29) = -0.25$, $p = 0.801$). None of the participants reported neurological and psychiatric illnesses. All participants had a normal or corrected-to-normal vision and were free of any medication. In addition, participants were required to abstain from drinking alcohol the day before the appointment and from caffeine intake on the day of the appointment. All individual participants included in the study gave informed consent and were reimbursed for their participation or received course credits. The two groups did not differ regarding the type of received reimbursement (i.e. money or course credit; $\chi^2(1) = 0.03$; $p = 0.857$). The Ethics committee of the Medical Faculty of the TU Dresden approved of the study. Written informed consent was obtained from all individual participants included in the study.

Task and study design

We created a modified version of a sustained attention to response (SART) task (please refer to Fig. 1 for a schematic overview of the experimental conditions).

During this task, white digits "1" to "9" were displayed on a 24″ TFT-monitor at a viewing distance of ~ 58.5 cm. The digits were presented for 250 ms above a central fixation cross (cross width: 1° viewing angle; center-to-center digit to cross distance: 2.6° viewing angle). The presentation of the digit was followed by a mask that randomly varied in presentation between 1100 and 1600 ms. Participants were required to respond to all numbers as fast as possible via pressing the space bar on a standard PC keyboard, except "3" which served as the "NoGo" stimulus. Importantly, the digits representing Go and NoGo trials were preceded by a cue stimulus (2.4° viewing angle). This cue stimulus was a red or green filled circle, which was presented at the same spatial position above the fixation cross as the target stimuli. A green dot indicated that a Go stimulus is to be expected, a red dot indicated that a NoGo stimulus is to be expected. Participants were informed that a colored circle (red or green) always preceded the digit, which may or may not reveal something about the type of the upcoming stimulus. Still, we pointed out that only the category of the digit (i.e., "3" or not "3") was relevant for responding. The cue stimulus was presented for 200 ms and followed by a black screen of 300 ms in which only the fixation cross was visible. The cue stimuli could either be valid or invalid. The experimental procedure was as follows: the experiment was divided into two main blocks varying whether the cue stimulus was valid in 80% of cases or has no validity (i.e. the cue was only valid in 50% of cases). This order of the blocks was counterbalanced across the sample of participants; i.e. one half of the participants ($N = 15$; 9 females) began the experiment with the block in which cues were valid in 80% of cases, the other half of the participants ($N = 16$; 7 females) began the experiment with 50% valid cues. Within each of these two blocks, 2 sub-blocks were included between which the ratio of NoGo and Go trials was varied. In one sub-block, the ratio was 1 (NoGo):6 (Go) trials, in the other sub-block the ratio was 4 (NoGo):5 (Go) trials. The order of these sub-blocks was counterbalanced within each group of participants beginning the experiment with either 80% valid cues or 50% valid cues. That is, for the half of the participants starting the experiment with 80% valid cues the first sub-block had

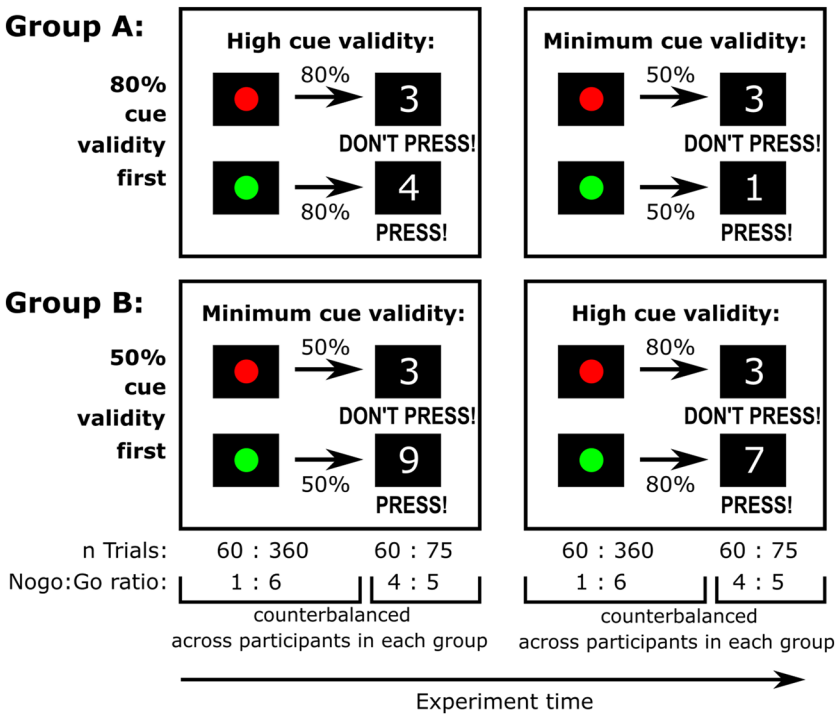

Fig. 1 Schematic overview of the task conditions used in this experiment, namely (1) cue validity order (displayed in rows), (2) cue validity block (displayed in the four framed cells) and (3) NoGo:Go ratio (displayed below the framed cells)

a ratio of 1:6 Nogo/Go trials and the second a ratio of 4:5 Nogo/Go trials. In the following second block (i.e. with 50% cue validity), the ordering of the sub-blocks was the same. For the other half of that participants, the first sub-block had a ratio of 4:5 Nogo/Go trials and the second a ratio of 1:6 Nogo/Go trials. Again, in the following second block (i.e. with 50% cue validity), the ordering of the sub-blocks was the same. For the participants in the group starting the experiment with 50% valid cues, the same procedure was applied. Within each sub-block, the presentation sequence of Go and NoGo trials was pseudorandomized ensuring that all Go digits were presented equally frequent. Importantly, and as done in previous studies (Dippel et al. 2016), the number of NoGo trials was set to $N = 60$ and held constant over the experimental condition to achieve a comparable signal-to-noise ratio (SNR) in the neurophysiological data on NoGo trials. Therefore, the number of Go trials was varied to achieve the different ratios of NoGo and Go trials. Before the start of the experiment, the participants practiced 50 trials. After the initial practice trials, participants carried out the experiment consisting of $N = 1110$ trials in total within approximately 40 min. This time includes short breaks after every 185 trials.

It may be argued that the study design varied different NoGo ratio blocks within the cue validity blocks but not the other way around. We chose this procedure for several reasons: first, the most important experimental manipulation beyond classical Go-NoGo paradigms was the effect of the preceding cue, which varied in validity. Therefore, we treated the cue validity blocks as the main blocks of the experiment, subsuming variations in NoGo ratios. Second, adding another hierarchical order (i.e., varying cue validity within a block of a fixed NoGo ratio) would have doubled the duration of the experimental session; i.e. to $N = 2220$ and 80 min duration. This makes it likely that fatigue effects occur during the experiment, which are difficult to control since these strongly vary between subjects. Related to that, another order factor would then be needed to account for the hierarchical order that is presented first, which would have further complicated the study design. Therefore, we decided to use the study/task design outlined above.

EEG recording and processing

EEG activity was recorded at a sampling rate of 500 Hz using 60 Ag/AgCl electrodes mounted in an elastic cap in equidistant positions using a QuickAmp amplifier (BrainProducts, Inc.). The reference was placed at electrode position Fpz and the ground electrode at coordinate $\theta = 58$, $\phi = 78$. Electrode impedances were below 5 kΩ. Offline EEG data processing was performed using the Brain Products' Brain Vision Analyzer 2 software package. After an initial visual trial-by-trial raw data inspection step to remove gross technical artifacts (i.e. "offsets" in the EEG), a band-pass IIR filter was applied (0.5–25 Hz, 48 db/oct). EEG channels that showed technical problems during acquisition (e.g. flatlines due to oversaturation) were discarded prior to the next analysis step (on average 0.77 channels; maximum 4 channels). Then, an independent component analysis (ICA, infomax

algorithm) was run to detect and discard periodically occurring artifacts (i.e. blinks, vertical and horizontal eye movements, pulse artifacts). After the ICA, the discarded EEG channels were topographically interpolated. The EEG data were then segmented to the onset of the Go and NoGo stimuli. Segments started − 200 ms before the target and ended 1500 ms thereafter. Separate segments were built for different possible cue-target combinations: i.e. high cue validity high NoGo stimulus probability, low cue validity low NoGo stimulus probability, high cue validity low NoGo stimulus probability, low cue validity high NoGo stimulus probability. The same was done for Go trials. Only trials with correct responses were included in the analysis, i.e. Go trials with responses and NoGo trials without responses. This was done to ensure that task-irrelevant confounding processes, leading to erroneous responses, do not explain the results. On the basis of the segmented data, an automatic artifact rejection procedure was applied with an amplitude criterion (maximal amplitude + 200 µV, minimal amplitude − 200 µV) and a maximal value difference criterion of 200 µV in a 200 ms interval as well as an activity below 0.5 µV in a 100 ms period as rejection criteria. 84.7% of trials (± 7.6%; smallest number of trials in one condition of one participant: 29) remained after the artifact rejection steps. Then, a current source density (CSD) transformation was conducted, which allows a reference-free evaluation of the EEG data (Nunez and Pilgreen 1991). Importantly, the spatial filter properties of the CSD-transformation (Kayser and Tenke 2015) do not violate assumptions of RIDE, because the temporal decomposition is conducted for each single electrode channel separately (Ouyang et al. 2015a). After the CSD transformation, the data were baseline corrected to a time interval from − 200 ms to 0 ms (i.e. Go/NoGo stimulus presentation). This single-trial data was used for temporal EEG data decomposition (Ouyang et al. 2015b; Chmielewski et al. 2018).

It is important to note that the choice of the baseline did not affect the pattern of results in the EEG data; i.e. when the baseline was set to the period prior the presentation of the cue, the results remained the same. That is, there was no effect of the "baseline position" when modeling this factor as an additional variable in the ANOVAs (all $F < 0.99$; $p > 0.4$). This is further supported by Bayesian analysis, for which we used the method of Masson (2011). With this method, the probability of the null hypothesis being true given the obtained data $p(H_0|D)$ can be calculated. It is shown that for all the respective interactions and main effects of the factor "baseline position" $p(H_0|D)$ was > 0.85. According to Raftery (1995), the results of the Bayesian analysis provide strong evidence for the null hypothesis.

Temporal EEG signal decomposition and data quantification

Full mathematical details of the residue iteration decomposition (RIDE) can be found elsewhere (Ouyang et al. 2011, 2015a). The RIDE toolbox and manual are available at http://cns.hkbu.edu.hk/RIDE.htm. Briefly, RIDE decomposes the EEG single-trial data into three clusters. The S-cluster is correlated to the stimulus onset, the R-cluster to the response. The third C-cluster has a variable latency, which is estimated by the algorithm and iteratively improved. Since the R-cluster cannot reliably be estimated in NoGo trials due to a low frequency of responding in these trials (Ouyang et al. 2013), only the S-cluster and the C-cluster were calculated (Chmielewski et al. 2018). To estimate the C-cluster latency, RIDE uses a nested iteration scheme. During this procedure, the initial latency of the C-cluster is estimated using a time window function. The S-cluster is iteratively removed, and the latency of the C-cluster is re-estimated in every iteration step using a template matching approach. The initial time window for the estimation of the C-cluster was set to 200–700 ms after stimulus onset because that window has to cover the time interval where the components are supposed to occur (Ouyang et al. 2015b). The time window for the S-cluster was set to − 200 to 400 ms around stimulus onset. The RIDE clusters are then given for every single subject and data quantification (mean amplitudes) were conducted at this level in a defined time interval. This time interval and relevant electrodes were defined after a visual inspection of the data (please refer to Supplementary Table 1). Afterwards, the choice of electrodes and time windows was validated using a statistical procedure described in Mückschel et al. (2014): within each of the defined search intervals, the mean amplitude is estimated at all electrodes. Then, each electrode was compared against the average of all other electrodes using Bonferroni-correction for multiple comparisons. Only electrodes showing significantly larger amplitudes (i.e., negative for N-potentials and positive for the P-potentials) than the remaining electrodes were selected. This validation procedure revealed the time windows and electrodes as identified by visual inspection.

Source localization analyses

To examine which functional neuroanatomical structures are associated with modulations in the different RIDE cluster, source localization was carried out applying sLORETA (Chmielewski et al. 2018). It has been shown that source localization results based on ERPs and RIDE data are highly similar (Chmielewski et al. 2018). sLORETA provides a single linear solution to the inverse problem without a localization bias (Pascual-Marqui 2002; Marco-Pallarés et al. 2005; Sekihara et al. 2005). The reliability of sLORETA sources

has been corroborated by EEG/fMRI and EEG/TMS studies (Sekihara et al. 2005; Dippel and Beste 2015). The software partitions the intracerebral volume into 6239 voxels at 5 mm spatial resolution before the standardized current density at each voxel is calculated. sLORETA uses a realistic head model based on the MNI152 template. Details about the precise sLORETA contrasts calculated are given in "Results". Yet, for the contrasts, we calculated *t*-statistics on log-transformed data with no smoothing of the variances. Comparisons were based on statistical non-parametric mapping (SnPM). This procedure uses the sLORETA-built-in voxel-wise randomization tests with 3000 permutations (Nichols and Holmes 2002). Voxels with significant differences ($p < 0.05$, corrected for multiple comparisons) between contrasted groups/conditions were located in the MNI-brain http://www.unizh.ch/keyinst/NewLORETA/sLORETA/sLORETA.htm.

The main anatomical region affected by the task factors was determined based on the voxel position with maximum activation difference and using the MNI template as provided by the sLORETA software package. As shown in "Results" (see below), especially the C-cluster was affected by the joined effects of the probability/certainty about the identity of an upcoming stimulus (i.e. is Go or NoGo) and by the validity of a cue that an upcoming stimulus has a certain identity.

Statistics

The behavioral data were analyzed separately for Go and NoGo conditions using mixed effects ANOVAs. These included the within-subject factor "cue validity" (valid vs. invalid), "ratio" (1:6 vs. 4:5 NoGo/Go trials) and the between-subject factor "order of blocks" (i.e. participants started the experiment with a low vs. high probability that the presented cues are valid). For the analysis of the neurophysiological data, the factors "Go/NoGo" and "electrode" were also included in the ANOVAs as additional within-subject factors. A Greenhouse–Geisser correction was applied and all post hoc tests (unpaired *t*-tests if including the between-subject factor "order of blocks", else paired *t*-tests) were Bonferroni-corrected. All variables were normal distributed as indicated by Kolmogorov–Smirnov tests (all $z < 0.74$; $p > 0.4$).

Results

Behavioral data

The false alarm (FA) rate is the most crucial behavioral parameter in Go/NoGo paradigms. For that parameter the ANOVA revealed a main effect of "ratio" ($F(1,29) = 174.30$; $p < 0.001$; $\eta_p^2 = 0.857$) showing more FAs in the 1:6 than the 4:5 condition. This corroborates previous findings (Helton 2009; Stevenson et al. 2011; Dippel et al. 2016). As expected, there was also a main effect "cue validity" ($F(1,29) = 8.57$; $p = 0.007$; $\eta_p^2 = 0.228$) showing that FA rates were higher for trials with invalid cues, compared to trials with valid cues. Importantly, there was an interaction "ratio × cue validity × order of blocks" ($F(1,29) = 4.11$; $p = 0.045$; $\eta_p^2 = 0.130$). To examine this interaction in more detail, we first calculated the validity effect in NoGo trials by calculating the difference "FA invalid cue minus FA valid cues". This was done for each "ratio" of NoGo/Go trials and "order of blocks" separately. In this score, positive values denote that more false alarms were committed after invalid, compared to valid cue information. The repeated-measures ANOVA using that score/index revealed an interaction "ratio × order of blocks" ($F(1,29) = 6.28$; $p = 0.021$; $\eta_p^2 = 0.189$). No other main or interaction effects were significant (all $F < 1.93$; $p > 0.2$). This interaction "ratio × order of blocks" is shown in Fig. 2.

Post-hoc tests revealed that there was no difference in the FA score between the 1:6 and 4:5 NoGo/Go trial ratio when participants started the experiments with a low probability that the presented cues are valid (i.e. 50% valid cues) ($t(14) = -0.11$; $p > 0.9$). This lack of an effect is corroborated by Bayesian analysis (refer Table 1). According to Raftery (1995), the results of the Bayesian analysis provide very strong evidence for the null hypothesis.

When participants started the experiment with a high probability that the presented cues are valid (i.e. 80% valid

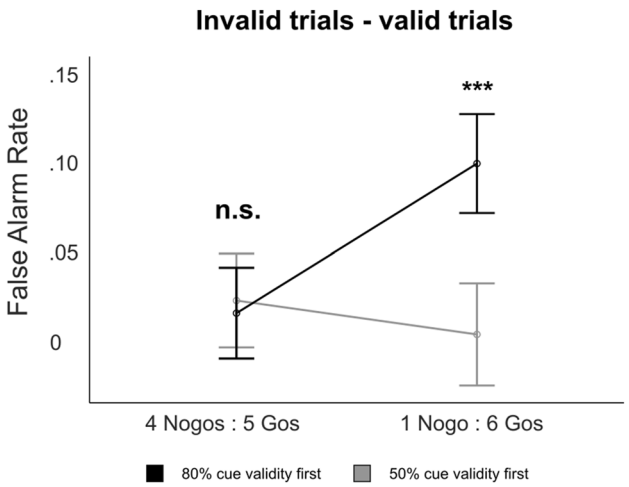

Fig. 2 The validity effect in NoGo trials (difference: FA invalid cue minus FA valid cues). In this score, positive values denote that more false alarms were committed after invalid compared to valid cue information. The mean and standard error of the mean is shown. Red lines denote the group with 80% valid cue information in the first block. Blue lines denote the group with 50% valid cue information in the first block. The ratio of Go and NoGo is shown on the *x*-axis

Table 1 Bayesian probabilities for the null hypothesis that there is no interaction effect between Nogo:Go ratio and cue validity given the data ($p(H_0|D)$) and the alternative hypothesis that there is such an effect ($p(H_1|D)$)

Order of blocks	Dependent variable	$p(H_0\|D)$	$p(H_1\|D)$
50% validity first	False alarm rate	0.761	0.239
	C cluster N2 amplitude	0.099	0.901
80% validity first	False alarm rate	0.261	0.739
	C cluster N2 amplitude	0.731	0.269

The probabilities suggest that there is no interaction effect in neurophysiological data (C cluster N2 amplitude) when the first half of the experiment involved a useful cue (80% validity). Likewise, an interaction effect regarding false alarm rates is unlikely when participants were initially confronted with an uninformative cue (chance level validity)

cues), it is shown that the FA score was higher in the 1:6 than in the 4:5 condition ($t(15) = -2.21$; $p = 0.018$). It is also shown that only in the 1:6 condition, and when participants started the experiment with a high probability that the presented cues are valid (i.e. 80% valid cues), the FA score was significantly different from zero ($t(16) = 3.13$; $p = 0.007$). The FA score did not differ from zero in all other conditions (all $t < 0.57$; $p > 0.5$). This shows that more false alarms are evident after invalid cue information whenever NoGo trials were presented rarely and when subjects started the experiment with a high probability that the cue information is valid. The FA data thus shows that response inhibition performance is modulated by interactive effects between (1) cue information/validity, (2) demands on inhibitory control processes and (3) the subject's experience that cue information is valid/invalid during response inhibition processes.

Regarding the accuracy on Go trials, there were no main or interaction effects (all $F < 0.95$; $p > 0.338$). Concerning the reaction times (RTs) on Go trials, there was a main effect "ratio" ($F(1,29) = 102.47$; $p < 0.001$; $\eta_p^2 = 0.779$) showing that RTs were faster in the condition where 6-times more Go than NoGo trials were evident (i.e. the 1:6 NoGo/Go condition) (310 ms ± 5), compared to the condition where the frequency of Go and NoGo trials was almost equal (i.e. in the 4:5 NoGo/Go condition) (347 ms ± 7). This is an expected finding due to the automatic response tendency when there are 6-times more Go than NoGo trials. Lastly, there was a main effect "validity" ($F(1,29) = 7.06$; $p = 0.013$; $\eta_p^2 = 0.196$) showing that RTs were longer (331 ms ± 6) on invalid than valid trials (326 ms ± 5). No other main or interaction effects were significant (all $F < 1.43$; $p > 0.310$).

Neurophysiological data

The neurophysiological data, i.e. the S-cluster and the C-cluster data are shown in Figs. 3 and 4, respectively.

For the S-cluster data in the P1 and the N1 time window (Fig. 3), no main or interaction effects were evident (all $F < 0.73$; $p > 0.399$). Also in the N2 time window, no interaction effects explaining the behavioral data pattern was evident (all $F < 0.92$; $p > 0.336$). Using the amplitude values, there was only a main effect Go/NoGo ($F(1,29) = 34.87$; $p < 0.001$; $\eta_p^2 = 0.555$) showing the common effect that the amplitudes were more negative on NoGo (− 13.76 µV/m² ± 2.03), compared to Go trials (− 4.23 µV/m² ± 1.15).

The C-cluster is shown in Fig. 4a. Importantly, in the N2 time window, there was an interaction "Go/NoGo × ratio × order of blocks × validity" ($F(1,29) = 4.13$; $p = 0.035$; $\eta_p^2 = 0.138$). No other main or interaction effects were significant (all $F < 1.01$; $p > 0.4$). To analyze the interaction in more detail, ANOVAs were calculated for Go and NoGo trials separately. For the Go trials, there was no interaction "ratio × order of blocks × validity" ($F(1,29) = 0.15$; $p > 0.85$), but such an interaction was evident in NoGo trials ($F(1,29) = 9.40$; $p = 0.009$; $\eta_p^2 = 0.245$). To better capture the interactive effects, we calculated the validity effect in NoGo trials by calculating the difference "invalid cues minus valid cues" (as done for the FA rate data). This was done for each "ratio" of NoGo/Go trials and "order of blocks" separately. For amplitudes with negative polarities (as it is the case in the C-cluster N2 time window), this validity parameter is negative when amplitudes are larger in invalid than valid trials; the parameter is positive when amplitudes are smaller in invalid than valid trials. The descriptive values of the validity parameter showing the interaction "ratio × order of blocks" is also given in Fig. 4b. Post-hoc tests revealed that there was no difference in the validity parameter between the 1:6 and 4:5 NoGo/Go trial ratio when participants started the experiments with a high probability that the presented cues are valid (i.e. 80% valid cues) ($t(15) = 0.60$; $p > 0.5$). This lack of an effect is corroborated by Bayesian probabilities (see Table 1). Yet, when participants started the experiment with a low validity of the cues (i.e. 50%), the ratio differed between the 1:6 and 4:5 condition ($t(14) = 1.84$; $p = 0.031$). Since the calculated validity score for amplitudes with negative polarities is negative when amplitudes are larger in invalid than valid trials, the results show that the C-cluster in the N2 time window is more negative during invalid than valid trials when the NoGo/Go trial ratio is 4:5 and the opposite is the case when the ratio is 1:6. An sLORETA source estimation, calculated for this contrast, revealed activation differences mainly in the right inferior frontal cortex (BA47) (refer Fig. 4c).

Opposed to the N2 time window, no interaction "Go/NoGo × ratio × order of blocks × validity" was evident in the P3 time window ($F(1,29) = 0.89$; $p > 0.7$). There were only the main effects commonly observed in Go/NoGo tasks. This means that there was a main effect "Go/NoGo" ($F(1,29) = 85.09$; $p < 0.001$; $\eta_p^2 = 0.746$) showing that the

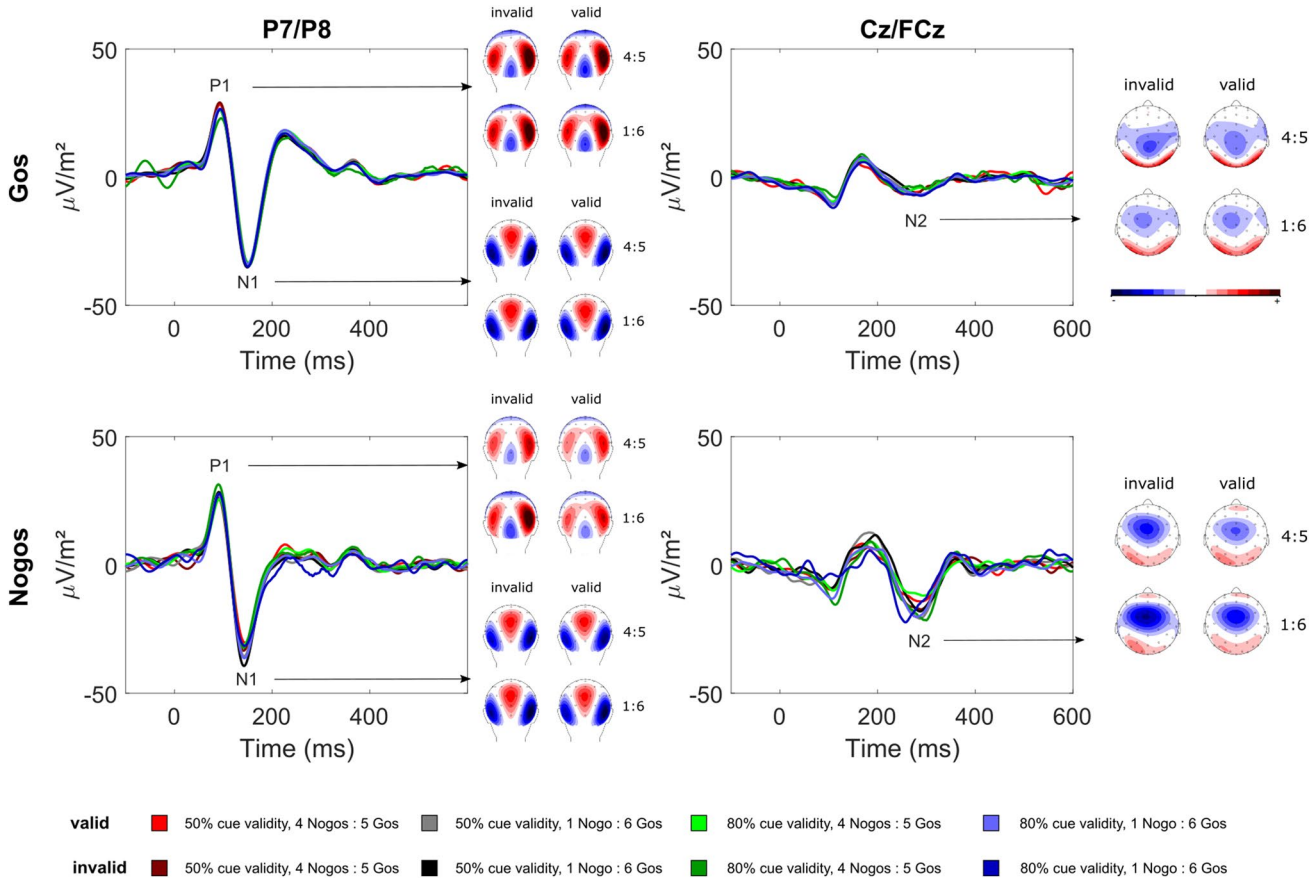

Fig. 3 The S-cluster is shown for the P1, N1 (left column) and the N2 time window (right column) at electrodes P7/P8 and Cz/FCz for Go trials (top row) and NoGo trials (bottom row). The different colors of the curves denote the different conditions. Note that the "order of blocks" factor is not shown, because there was no effect of this factor in the S-cluster. The scalp topography plots denote the distribution of the potential at the peak of each component. Red colors denote positive values, blue colors denote negative values

amplitudes were larger in NoGo (33.82 $\mu V/m^2 \pm 4.25$) than Go trials (8.43 $\mu V/m^2 \pm 3.33$). There was also a main effect "ratio" ($F(1,29) = 15.04$; $p = 0.001$; $\eta_p^2 = 0.342$) showing that the amplitudes were larger when the NoGo/Go trial ratio was 1:6 (22.32 $\mu V/m^2 \pm 3.67$) than 4:5 (18.14 $\mu V/m^2 \pm 3.47$). This replicates other recent data (Mückschel et al. 2017b; Chmielewski et al. 2018).

Taken together, only the C-cluster data in the N2 time window revealed interactive effects that are able to explain the behavioral effects. These modulations were associated with the right inferior frontal gyrus.

Discussion

The current study focused on interactive effects of (1) cue information/validity, (2) demands on response inhibition processes and (3) the subject's experience that cue information is highly likely to be valid/invalid during response inhibition processes. Neurophysiological processes underlying these interactive effects were examined combining EEG recordings with temporal signal decomposition methods and source localization (sLORETA).

The behavioral data show that false alarms rates were higher when there was a ratio of 1:6 (NoGo vs. Go) trials, compared to a ratio of 4:5. This corroborates previous findings showing demands on inhibitory control processes are higher and erroneous responding on NoGo trials is more frequent when the probability to engage in inhibitory control processes is low (Dockree et al. 2004, 2006; Dippel et al. 2016, 2017; Wessel 2018). However, only when demands on inhibitory control processes were high (i.e. in the 1:6 condition), invalid cue information induced an increase in the rate of false alarms, compared to valid cue information. Importantly, these differences between valid and invalid cue information were furthermore only evident when participants started the experiment with a high probability that the cue information was valid. This was shown by the significant increase in the positive validity score in the false alarm rates in the 1:6 condition, compared to the 4:5 condition when

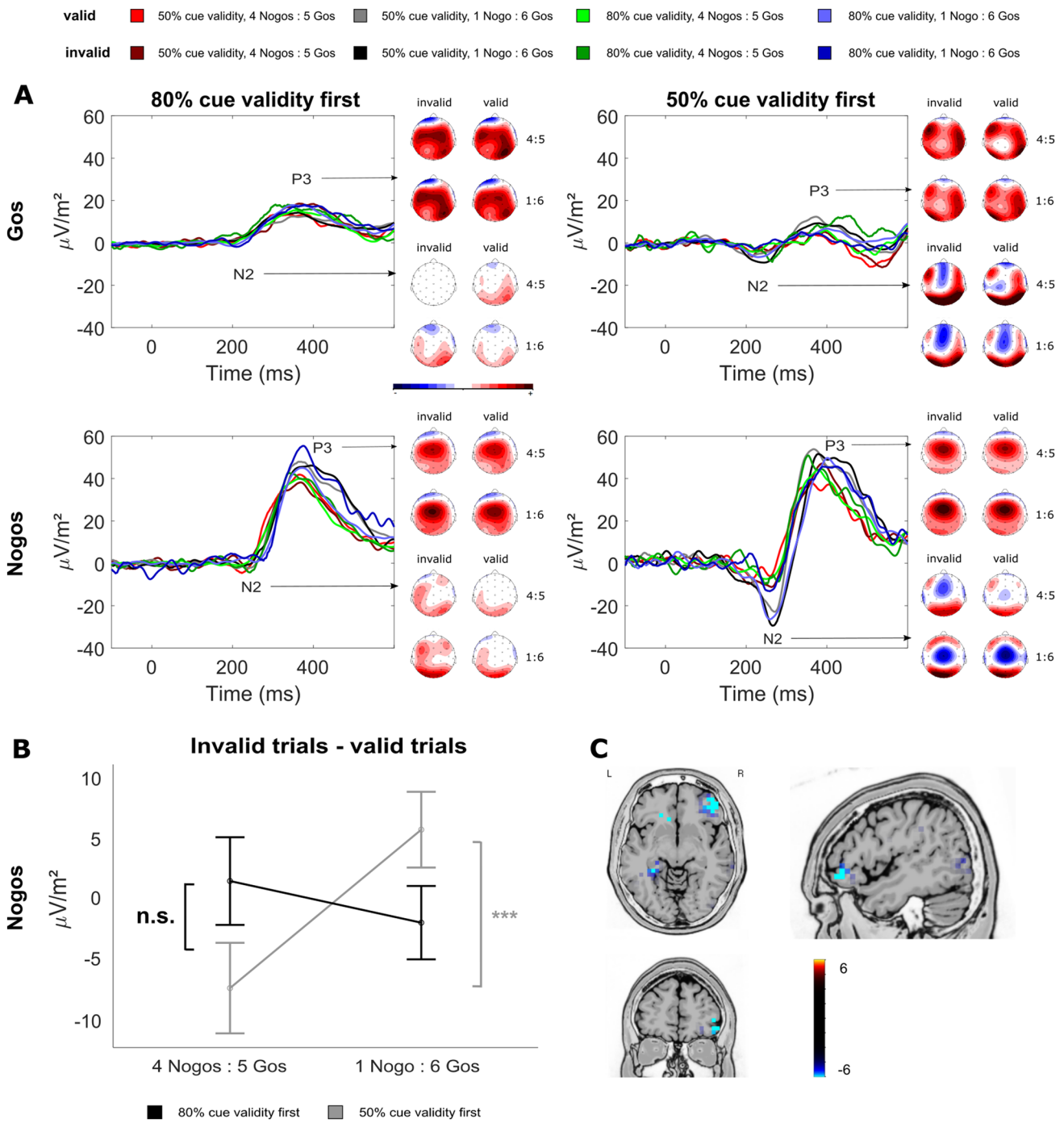

Fig. 4 **a** The C-cluster is shown for the N2 and P3 time window at electrode Cz for Go trials (top row) and NoGo trials (bottom row). The left column shows the participants starting the experiment with 80% valid cues, the right column shows the participants starting the experiment with 50% valid cues. The different colors of the curves denote the different conditions. The scalp topography plots denote the distribution of the potential at the peak of each component. Red colors denote positive values, blue colors denote negative values. **b** The interaction "ratio × order of blocks" is given for the validity effect in NoGo trials. The validity effect represents the difference invalid cue minus valid cues. For amplitudes with negative polarities, this validity parameter is negative when amplitudes are larger in invalid than valid trials; the parameter is positive when amplitudes are smaller in invalid than valid trials. The mean and standard error of the mean is shown. Red lines denote the group with 80% valid cue information in the first block. Blue lines denote the group with 50% valid cue information in the first block. The ratio of Go and NoGo is shown on the *x*-axis. (C) Results from the source localization results showing interaction "ratio × order of blocks" (corrected for multiple comparisons)

participants started the experiment with 80% reliable cue information. When the cue information was unreliable in the first half of the experiment (i.e. when there was only 50% chance the cue indicated the upcoming target stimulus), no effect of valid and invalid cues on response inhibition performance (false alarm rate) was detectable. Thus, the behavioral data corroborate the hypothesis that the effect of valid and invalid cue information is only evident when there are high demands on inhibitory control processes, and when subjects have experienced that cue stimuli are highly likely to be valid. Especially the latter is likely to emerge from the supposed Bayesian nature of the interrelation between cue validity and target stimulus identity (Yu and Dayan 2005) and the importance of Bayesian information processing to shape learning/experience effects (Friston et al. 2017). If a cue stimulus is highly likely to be valid, a violation of this assumption leads to problems in response inhibition. If, however, subjects experienced that a cue stimulus is unlikely to be valid, it is possible that no assumptions are made for these cue stimuli whose violation could then lead to problems in response inhibition. Since cue information is most helpful when a task is highly demanding/difficult (Snowden et al. 2001), it seems reasonable that above-mentioned effects are only evident when there was a low probability that an upcoming target stimulus is a NoGo stimulus. The neurophysiological (RIDE) and source localization data detail what information coding levels and functional neuroanatomical structures are affected.

The RIDE data revealed a very specific pattern of results because no interactive effects in line with the behavioral data were observed in the S-cluster. The lack of effects in the S-cluster suggests that bottom-up perceptual gating or attentional selection processes, reflected in the P1 and N1 time window (Herrmann and Knight 2001), are not important to consider. Even for the C-cluster, interactive effects and modulations depending on (1) cue information/validity, (2) demands on inhibitory control processes and (3) the subject's experience about the reliability of cue stimuli were confined to the N2 time window. It is important to keep in mind that a constant number of NoGo trials ($N = 60$) across the experimental conditions ensured that systematic differences in the signal-to-noise ratios between conditions are highly unlikely to explain the observed neurophysiological effects. The sLORETA analysis showed that these interactive effects were associated with activity modulations in the right inferior frontal gyrus (rIFG) (BA47). Regarding the debate whether stimulus-dependent attentional processes or motor response-inhibition processes play an important role during inhibitory processes (Simmonds et al. 2008; Sharp et al. 2010; Hampshire et al. 2010; Dodds et al. 2011; Hampshire 2015; Hampshire and Sharp 2015; Aron et al. 2015), the dissociation between the S-cluster and the C-cluster suggest that motor processes may be more important than stimulus-related processes—at least for the combination of factors examined in this study.

As outlined in "Results" and shown in Fig. 4, the validity effect (i.e. invalid minus valid) is negative for C-cluster N2 amplitudes, when the C-cluster amplitude on the target stimulus is smaller (i.e. less negative) after valid than invalid cues. The results show that there was an amplitude modulation of the cue validity effect in the C-cluster between the 1:6 and the 4:5 condition, when there was no such modulation at the behavioral level (i.e. when participants started the experiment with 50% reliable cue information). Importantly, however, when behavioral performance was modulated and false alarm rates were higher after invalid cues in the 1:6 condition when participants started the experiment with 80% reliable cue information, there was no modulation in C-cluster amplitudes in the N2 time window. Bayesian analyses support this specific pattern of a lack of effects (see Table 1).

How may this neurophysiological effect specific to the N2 time window be explained, especially given that previous research identified the P3 component as a marker for response inhibition (Smith et al. 2008; Albert et al. 2013; Wessel 2018)? Several lines of evidence suggest that neurophysiological processes in the N2 time window in NoGo trials may reflect conflict monitoring (Nieuwenhuis et al. 2004) or pre-motor inhibitory control processes (Falkenstein et al. 1999; Bokura et al. 2001). The latter refers to mechanisms related to the inhibition or revision of motor program before the actual motor response (Beste et al. 2010; Huster et al. 2013). According to that, high amplitudes in the N2 time window have been shown to be associated with better response inhibition performance (Beste et al. 2010; Huster et al. 2013). However, in the N2 time window, stimulus and response-related aspects have been shown to overlap (Folstein and Van Petten 2008). This is also the case during response inhibition, where 'stimulus codes' and 'response selection codes' can co-exist over extended time periods during the inhibition of responses (Mückschel et al. 2017b). It has been suggested that modulations of the C-cluster during response inhibition reflect a 'braking function' (Ouyang et al. 2015b; Bluschke et al. 2016; Friedrich et al. 2018; Bodmer et al. 2018)—a behavioral brake that is switched on when it is necessary to pause an action (Aron et al. 2014; Bianco et al. 2017). In view of the behavioral results, the C-cluster data in the N2 time window suggest that braking processes cannot be sufficiently used if relatively reliable cue information (reliable in 80% of cases) turns out to be invalid and the rarity of situations in which response inhibition is necessary (1:6 NoGo:Go trials) places high demands on these processes. These effects do not occur if only one of these aspects does not apply, i.e. if either the demands on inhibitory control are lower or the cue information is not considered reliable. If cue information is generally considered unreliable, it makes sense to leave response

inhibition processes "pre-activated", which then facilitates the application of these processes when they are required. If the requirements for the response inhibition processes are lower because they have to be used more frequently, it is also easier to apply them.

Notably, modulations in the C-cluster during inhibitory control have previously been reported to be associated with activity modulations in the right inferior frontal gyrus (rIFG) using a SART paradigm (Mückschel et al. 2017b). Exactly this cortical region is revealed by the current source localization analysis using sLORETA. Interestingly, it has been shown that the rIFG likely mediates the above-mentioned 'braking function' (Gillies and Willshaw 1998; Aron et al. 2014, 2015) supposed to be reflected by the C-cluster. Based on that, and the lack of modulations seen in the S-cluster, the results suggest that especially response braking functions associated with the rIFG are modulated by the interplay of (1) cue information, (2) demands on inhibitory control processes and (3) the subject's experience about the reliability of cue stimuli during inhibitory control. This interplay of processes is a new functional facet of the rIFG during response inhibition. However, this new functional aspect of the rIFG seems reasonable in view of existing findings. First, the rIFG is involved in the response inhibition network (Chambers et al. 2007; Bari and Robbins 2013; Aron et al. 2014; Di Russo et al. 2016; Allen et al. 2018) especially when inhibitory control is highly demanding (Mückschel et al. 2017b). Second, there is plenty of evidence that the rIFG is activated when behaviorally relevant cues are detected (Vossel et al. 2006; Chi et al. 2014) that may be used to inform inhibitory control (Hampshire et al. 2010; Lenartowicz et al. 2011). Finally, evidence suggests that the rIFG encodes beliefs about the nature and predictive value of external stimuli that are shaped by learning experiences (Ostwald et al. 2012; d'Acremont et al. 2013; Meyniel and Dehaene 2017). Since (1) cue information processing, (2) motor inhibitory control and (3) the subject's experience about the reliability of cue stimuli during inhibitory control seems to be associated with the function of the rIFG for themselves, it is reasonable that the combination of these processes is also modulating processes of the rIFG.

As outlined in "Materials and methods", we varied different NoGo ratio blocks within the cue validity blocks but not the other way around. Though there are good reasons to do so, it is, currently, not clear whether this limits the conclusions that can be drawn from the results. This needs to be investigated in the future. Another opportunity for future research might be to test different cue-target intervals. We chose a relatively short interval in order to make conscious efforts to ignore the cue information more difficult. Consequently, a temporal analysis of neurophysiological processes occurring during this interval was not feasible. However, other studies, by independently modulating this parameter, might determine whether such a short interval duration (making the cue stimulus a kind of conscious prime for task sets) is indeed necessary for the observed effects. Future research might also focus on the neurobiological implementation of the processes described in this study. As pointed out in the introduction, Yu and Dayan (2005) proposed that two distinct neurobiological systems interact to bring about this specific behavioral pattern as a function of cue information and NoGo likelihood. These are namely the acetylcholine system and the norepinephrine system. While acetylcholine is hypothesized to signal expected uncertainty, in this case, the cue validity with a fixed value below 100%, norepinephrine system activity is thought to be elevated when there is unexpected uncertainty, as in the actual identity of the stimulus when both Gos and NoGos are about equally likely. The exact contribution of these systems to the observed behavior thus is a relevant topic for future research.

In summary, the study shows that response inhibition performance is modulated by interactive effects between (1) cue information/validity, (2) demands on inhibitory control processes and (3) the subject's experience that cue information is valid/invalid during response inhibition processes. Only if demands on inhibitory control processes are high and when participants acquainted the experience that cue information is very likely to be valid, invalid cue information compromised response inhibition performance. The neurophysiological data shows that processes likely reflecting braking processes, but not stimulus-related processes during response inhibition are modulated. It seems that braking processes cannot be sufficiently deployed if cue information that has been experienced to be highly valid turns out to be invalid and the rarity of situations in which response inhibition is necessary places high demands on these processes. Source localization data shows that these joined effects of factors specifically modulate processes in the right inferior frontal gyrus (BA47). This suggests that the rIFG is a hub region integrating different factors modulating response inhibition.

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Compliance with ethical standards

Conflict of interest There are no conflicts of interest.

Ethical statement The study was approved by the IRB of the TU Dresden.

Informed consent Written informed consent was obtained from all subject before any of the study's procedures were commenced.

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