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Individual Differences in Inhibitory Control—Relationship Between Baseline Activation in Lateral PFC and an Electrophysiological Index of Response Inhibition

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The capacity to inhibit inappropriate responses is crucial for goal-directed behavior. Inhibiting such responses seems to come more easily to some of us than others, however. From where do these individual differences originate? Here, we measured 263 participants' neural baseline activation using resting electroencephalogram. Then, we used this stable neural marker to predict a reliable electrophysiological index of response inhibition capacity in the cued Continuous Performance Test, the NoGo-Anteriorization (NGA). Using a source-localization technique, we found that resting delta, theta, and alpha1 activity in the left middle frontal gyrus and resting alpha1 activity in the right inferior frontal gyrus were negatively correlated with the NGA. As a larger NGA is thought to represent better response inhibition capacity, our findings demonstrate that lower levels of resting slow-wave oscillations in the lateral prefrontal cortex, bilaterally, are associated with a better response inhibition capacity.

Keywords: Continuous Performance Test, NoGo-Anteriorization, prefrontal cortex, response inhibition, resting EEG

Introduction

Stopping an action is required in many everyday tasks, such as stopping a vehicle at traffic lights or preventing impulsive verbal behavior. This capacity to inhibit inappropriate responses, that is, response inhibition, is typically identified as a major component of executive functions (e.g. Aron 2008; Hofmann et al. 2012). Without response inhibition, we would struggle to adapt to dynamically changing environments that call for prioritizing our actions in accordance with our internal goals and external demands. Such capacity, however, seems to come more easily to some of us than others: Individuals differ greatly in their response inhibition capacity with low capacity being a key feature of a variety of neurological and psychiatric diseases, such as attention-deficit hyperactivity disorder (ADHD) and substance abuse (e.g. Aron and Poldrack 2005; Nigg et al. 2006; for a review see Robbins et al. 2012). The goal of this study was to shed light on the largely unknown sources of individual differences in inhibitory capacity. For that purpose, we measured a reliable electrophysiological index of response inhibition, the NoGo-Anteriorization (NGA; Fallgatter and Strik 1999). We then used participants' task-independent neural baseline activation in order to explain individual variance in the NGA.

When trying to measure response inhibition capacity in the laboratory, one faces the challenge that successfully executed response inhibition leads to no observable outcome. One way to meet this challenge is to indirectly infer response inhibition capacity from behavioral performance in Go–NoGo paradigms. These paradigms require a speeded motor response to one stimulus ("Go-stimulus") and withholding a prepotent response to another stimulus ("NoGo-stimulus"). Performance indices include the reaction times in successfully executed responses after Go-stimuli and the number of errors, such as pressing a button when no response is required ("commission error") and missing a button press when a response is required ("omission error"). These behavioral performance indices, however, do not provide a direct measure of the processes executed during response inhibition and often fail to discriminate between patients characterized by a disinhibited pathology and healthy controls (e.g. Kemner et al. 1996; Karayanidis et al. 2000). Alternatively, brain activity can be quantified during the execution of response inhibition, enabling a more direct index of inhibitory capacity. A task which is ideally suited for that purpose is the cued Continuous Performance Test (CPT; Rosvold et al. 1956; Fallgatter et al. 1997). In this task, "prime" cues prompt the subject to anticipate a motor reaction, which must be executed if followed by a target stimulus ("Go-condition") and suppressed if followed by a nontarget stimulus ("NoGo-condition"). As NoGo- and Go-stimuli are equally probable, the comparison of brain responses between NoGo- and Go-stimuli is not confounded by oddball effects, that is, frequency of stimuli effects (Liddle et al. 2001; Lavric et al. 2004). Through comparing brain activation in the NoGo-condition with that in the Go-condition by means of electroencephalography (EEG), one can identify the neural mechanisms of inhibiting versus executing a motor response, capitalizing on the excellent temporal resolution of EEG on a milliseconds level.

In particular, the comparison of EEG scalp maps between the NoGo- and Go-condition around 300 ms after stimulus onset consistently shows an anteriorization of the positive centroid (i.e., the center of gravity of the positive brain electrical field) in the NoGo-condition when compared with the Go-condition. This is referred to as NGA. The NGA has been proposed to be a reproducible and temporally stable (Fallgatter et al. 2001; Fallgatter, Aranda et al. 2002) electrophysiological index of inhibitory brain function, with a larger NGA representing a better response inhibition capacity (Fallgatter and Strik 1999). Evidence for the validity of this index has been supplied by studies that report a smaller NGA in patients with ADHD (e.g. Fallgatter et al. 2005) and schizophrenia (e.g. Fallgatter and Mueller 2001). Further, a smaller NGA has also been observed in individuals possessing risk alleles of genes putatively associated with these same disorders, which are well known for involving a disturbed response inhibition capacity (e.g. Ehlis et al. 2007; Baehne et al. 2009).

Subjects strongly differ in their response inhibition capacity as measured by the NGA (e.g. Fallgatter et al. 2001). But where do these individual differences come from? Only weak

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associations have been found between demographic or personality variables and the NGA (Fallgatter et al. 1999; Fallgatter and Herrmann 2001). A number of candidate gene studies have identified certain genetic sources of variability in the NGA (for a recent example, see Heinzel et al. 2012). However, no study has yet examined whether dispositional neural markers predict individual differences in the NGA. Such neural markers can be captured by measuring an individual's neural baseline activation with EEG, while he or she is at rest and not engaged in any specific task (resting EEG). A number of studies have demonstrated that individual resting EEG is stable over a period of years (e.g. Smit et al. 2005; Napflin et al. 2007). Therefore, resting EEG allows the measurement of stable individual differences in neural functioning at rest and can be used to explain individual differences seen either in behavioral responses (e.g. Pizzagalli et al. 2006; Gianotti et al. 2012) or in event-related potentials (ERPs; e.g. Polich 1997; Lee et al. 2011; Nash et al. 2012).

In this study, we investigated whether neural baseline activation might explain individual differences in inhibitory brain function as measured by the NGA. For that purpose, we first measured participants' EEG at rest. Secondly, we quantified participants' inhibitory capacity in the cued CPT by means of the NGA. As it has been consistently shown that frontocingulate regions encompassing the lateral prefrontal cortex (PFC) and anterior cingulate cortex (ACC) are more active during the NoGo- compared with the Go-condition (e.g. Fallgatter, Bartsch et al. 2002; Garavan et al. 2002; Swick et al. 2011), we hypothesized that baseline activation in these areas would be related to the NGA.

Materials and Methods

Participants

Participants were recruited at the University of Basel. Inclusion criteria were: age between 18 and 40 years, right-handedness, normal, or corrected-to-normal vision. Participants were excluded if they reported current or past neurological or psychiatric illness. Two hundred and ninety-seven participants were enrolled. Twenty-seven participants were excluded from analyses because of excessive artifacts in the EEG recording. Seven participants were excluded from analyses because of missing behavioral data due to technical problems, extreme reaction times, and/or error rates in the CPT. Mean age of the remaining 263 participants (172 females) was 23.4 years (SD = 3.8). The study was approved by the local ethics committee. Participants were remunerated with 30 Swiss francs (1 Swiss franc = $1 US) for participating.

Procedure

Upon arriving at the laboratory, participants signed an informed consent form. Participants were seated comfortably in a dimly lit, quiet room, with intercom connection to the experimenters. In a first step, EEG was recorded during rest with open or closed eyes. The protocol consisted of 20-s eyes open followed by 40-s eyes closed, repeated 5 times. We analyzed data only from the 200-s eyes-closed condition. In a second step, we recorded EEG while participants performed the cued version of the CPT (Rosvold et al. 1956; Fallgatter et al. 1997).

Continuous Performance Test

This task requires the preparation and execution of responses to predefined target stimuli and the inhibition of the anticipated response to nontarget stimuli. Participants were instructed to press a response button whenever the letter O (primer) is directly followed by the letter X (target; "Go-condition"). If the letter O is followed by any other letter than X (nontarget), participants were instructed not to respond ("NoGo-condition"). Participants were told to give their answers as quickly and accurately as possible. The stimulus set consisted of 400 letters (12 different letters: A, B, C, D, E, F, G, H, J, L, O, and X). Of those, 80 were primer stimuli, followed by 40 target stimuli and 40 nontarget stimuli. The remaining stimuli were 240 distractor letters (other letters, or X without a preceding O). Letters were presented on a computer screen in a pseudorandomized order one at a time for 200 ms with an interstimulus interval of 1650 ms. The task lasted for about 13 min.

Electrophysiological Equipment

A continuous EEG was recorded at a sampling rate of 512 Hz (24 bit precision; bandwidth: 0.1–100 Hz) from 64 Ag–AgCl active electrodes positioned according to the 10/10 system montage (Nuwer et al. 1998). During the recordings, the signals were referenced to a common-mode sense, while driven right leg served as ground. Horizontal and vertical electro-oculographic signals were recorded with electrodes at the left and right outer canthi and left infraorbital. Eye-movement artifacts were corrected by independent component analysis. EEG signals from channels with corrupted signals were interpolated.

Resting EEG Data Processing

A computerized artifact rejection was applied to the EEG collected at rest (maximal allowed voltage step: 15 μV/ms; minimal allowed activity in intervals of 100-ms length: 0.5 μV; maximal allowed amplitude: ±100 μV). Data were additionally examined visually to eliminate residual artifacts (e.g. large movement-related artifacts). All available artifact-free 2048-ms EEG epochs were extracted and recomputed against the average reference. On average, there were 84.5 epochs (SD = 16.1) available per subject. A fast Fourier Transformation (using a square window) was applied to each epoch and channel to compute the power spectra with 0.5-Hz resolution. The spectra for each channel were averaged over all epochs for each participant. Absolute power spectra were integrated for the following 7 independent frequency bands (Kubicki et al. 1979): Delta (1.5–6 Hz), theta (6.5–8 Hz), alpha1 (8.5–10 Hz), alpha2 (10.5–12 Hz), beta1 (12.5–18 Hz), beta2 (18.5–21 Hz), and beta3 (21.5–30 Hz).

ERP Data Processing

EEG data collected during the CPT were filtered offline with a bandpass from 0.1 to 30 Hz. After a computerized artifact rejection (only amplitudes <70 μV in all EEG channels within 200 ms before and 1000 ms after stimulus presentation were allowed), data were additionally examined visually to eliminate residual artifacts. All available artifact-free EEG epochs after correct responses were re-referenced to an average reference and averaged to Go and NoGo ERPs. All participants had at least 20 artifact-free and correct-response Go and NoGo epochs. On average, 34.3 Go epochs (SD = 4.4) and 33.7 NoGo epochs (SD = 5.1) were available for averaging. Two-dimensional positive area centroids (Koenig and Gianotti 2009) of P300 field maps were calculated for the Go- and NoGo-conditions using individual P300 peaks. P300 peaks were defined as the most positive deflection within the P300 microstate (240–484 ms for the Go-condition and 304–444 ms for the NoGo-condition) at electrodes Pz (Go) and Cz (NoGo), respectively. Briefly, the term "microstates" refers to short time periods of relatively stable electrical field configurations that are assumed to correspond to different steps of information processing (for further explanation of the methodology see, e.g., Michel et al. 2009).

The location of each individual centroid was quantified on an anterior–posterior axis of a coordinate system, resulting from the planar projection of the electrode array onto a rectangular grid. Centroids could obtain values between 1 (position of the electrode Fpz) and 9 (position of Oz) as illustrated in Supplementary Fig. 1. Smaller values of centroid locations indicate a more anterior localization. Finally, the NGA was calculated individually as the difference

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between Go and NoGo centroids on this anterior–posterior axis such that more positive numbers indicate a larger NGA.

Source Localization

Standardized low-resolution brain electromagnetic tomography (sLORETA; Pascual-Marqui 2002) was used to estimate the intracerebral electrical sources that generated the scalp-recorded activity. sLORETA computes electrical neuronal activity as current density (A/m^2) without assuming a predefined number of active sources. The sLORETA solution space consists of 6239 voxels (voxel size: 5 × 5 × 5 mm) and is restricted to cortical gray matter and hippocampi, as defined by the digitized Montreal Neurological Institute probability atlas. Using the option automatic regularization method in the sLORETA software, we chose the transformation matrix with the signal-to-noise ratio set to 10. To reduce confounds that have no regional specificity, for each subject, sLORETA images were normalized to a total power of one and then log-transformed before statistical analyses.

Statistical Analysis

The main goal of this study was to assess links between neural baseline activation and the NGA. Accordingly, sLORETA was applied to estimate the intracerebral electrical sources generating the scalp-recorded activity, and a voxel-wise correlation approach was used to identify brain regions whose baseline activations correlate with the NGA, separately for each EEG frequency band. We restricted our voxel-by-voxel correlation analyses to all voxels encompassing prefrontal regions [Brodmann areas (BAs) 8, 9, 10, 11, 44, 45, 46, and 47; 1331 voxels] and anterior cingulate regions (BAs 24, 32, and 33; 313 voxels). Correction for multiple testing (for all voxels of the frontocingulate regions) was implemented by means of a nonparametric randomization approach (Nichols and Holmes 2002). The nonparametric randomization approach was used to estimate empirical probability distributions and the corresponding corrected (for multiple comparisons) critical probability thresholds.

sLORETA was also used in order to identify brain activation during the CPT that significantly contributed to the NGA. Current density images were computed at individual P300 peaks for Go- and NoGo-conditions, respectively. Descriptive *t*-statistic whole-brain images of the differences between the Go- and NoGo-conditions at individual P300 peaks were computed.

Results

Behavioral Data

On average, participants made 0.4 (SD = 0.8) errors of omission (misses) in the 40 Go-trials and 0.2 (SD = 0.5) errors of commission (false alarms) in the 40 NoGo-trials. Mean reaction times for correct responses were 387.4 ms (SD = 77.4).

Electrophysiological Data

ERP Data

The latencies of the individual P300 peaks within the P300 microstate in the NoGo-condition (365.4 ± 30.9 ms) were significantly prolonged, compared with the P300 peaks in the Go-condition (328.9 ± 40.6 ms; $t_{262} = 13.0$, $P < 0.0001$). The topographical analysis at the individual P300 peaks revealed a more anterior location of the positive centroid in the NoGo-condition (5.0 ± 0.7), compared with the Go-condition (6.7 ± 0.5; $t_{262} = 37.3$, $P < 0.0001$). This well-established anteriorization of the positive centroid in the NoGo- compared with the Go-condition (NGA) was consistently found in 99% of our participants (260 of 263). The NGA correlated negatively with the reaction time for correct responses ($r_{261} = -0.14$, $P = 0.02$), suggesting that a better response inhibition capacity is correlated with faster reactions in Go trials. Whole-brain analyses with sLORETA were applied to identify brain regions contributing to the NGA. The source localization for the contrast NoGo- versus Go-condition indicated stronger activity in the lateral PFC, bilaterally, and in the ACC [peak voxel: MNI (*x*, *y*, *z*) 0, 15, 35 in the cingulate gyrus, see Supplementary Fig. 2).

Relationship Between EEG Baseline Activation and NGA

Using sLORETA to estimate intracerebral sources underlying scalp-recorded resting EEG, we found that, in the delta (1.5–6 Hz), theta (6.5–8 Hz), and alpha1 (8.5–10 Hz) frequency bands, there were voxels showing negative significant correlations between current density and NGA ($P < 0.05$, corrected for multiple testing). In the delta band, all 53 significant voxels fell into one cluster in the BAs 8, 9, 44, 45, and 46 in the left hemisphere [peak voxel: MNI (*x*, *y*, *z*) −50, 15, 40 in the middle frontal gyrus, BA 9, Fig. 1*A*]. In the theta band, all the 13 significant voxels fell into one cluster in the BA 9 in the left hemisphere [peak voxel: MNI (*x*, *y*, *z*) −55, 15, 35 in the middle frontal gyrus, BA 9, Fig. 1*B*]. In the alpha1 band, the 14 significant voxels fell into 2 clusters: one in BAs 9 and 44 in the left hemisphere [peak voxel: MNI (*x*, *y*, *z*) −50, 25, 35 in the middle frontal gyrus, BA 9, Fig. 1*C*] and another in BA 46 in the right hemisphere [peak voxel: MNI (*x*, *y*, *z*) 50, 40, 20 in the inferior frontal gyrus, BA 46, Fig. 1*D*]. The significant negative correlations between current density within the clusters (i.e., averaged current density across voxels within each cluster) in the left lateral PFC and NGA were: delta: $r_{261} = -0.19$, $P = 0.003$; theta: $r_{261} = -0.18$, $P = 0.003$; and alpha1: $r_{261} = -0.20$, $P = 0.002$. In the right lateral PFC, the significant negative correlation between current density within the cluster in alpha1 and NGA was $r_{261} = -0.19$, $P = 0.003$. Our results thus demonstrate that lower levels of resting slow-wave oscillations in the lateral PFC were associated with a larger NGA, that is, a better response inhibition capacity.

Discussion

Using resting EEG in 263 participants to explain individual differences in the NGA, we found that slow-wave oscillations originating from lateral prefrontal regions were related to this electrophysiological index of response inhibition. More specifically, lower delta, theta, and alpha1 current density in the left middle frontal gyrus and lower alpha1 current density in the right inferior frontal gyrus were associated with a more pronounced NGA.

Previous studies have demonstrated that overall power in slower frequency bands of resting EEG is a valid source of individual variability in amplitude and latency of various ERP components (e.g. Polich 1997; Ramos-Loyo et al. 2004; Lee et al. 2011). Our study went a step further, as we estimated the intracerebral sources of baseline activation in the distinct frequency bands by means of sLORETA (Pascual-Marqui 2002). Thus, we were able to identify brain regions with activation levels at rest that were significantly correlated with the NGA. Because resting slow-wave oscillations are primarily considered to be inversely related to cortical activation (e.g. Shagass 1972; Oakes et al. 2004), our results indicate that higher baseline activation in the lateral PFC, bilaterally, was

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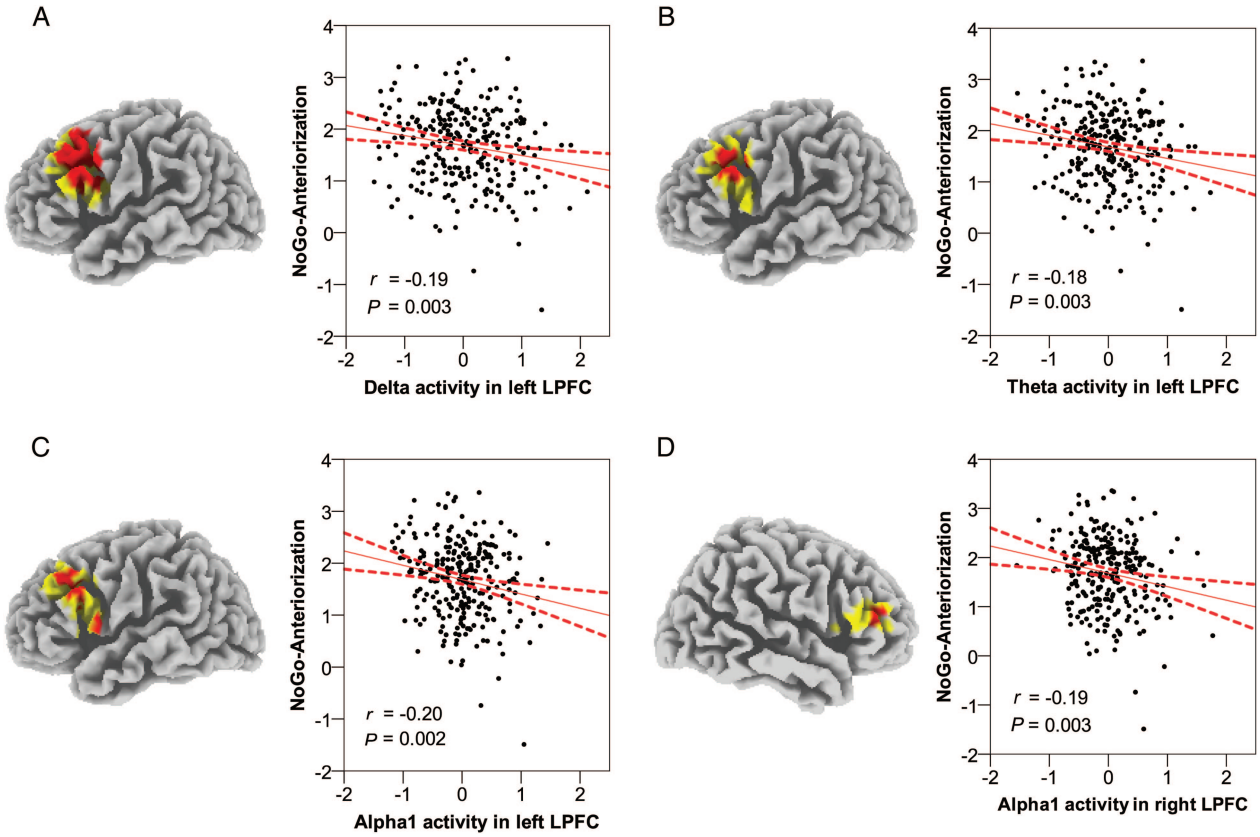

Figure 1. Relationship between the NGA and EEG baseline activation (A/m^2). In each figure, on the left side, locations of the voxels that showed significant correlations are indicated in red ($P < 0.05$) or in yellow ($0.05 < P < 0.10$) and, on the right side, scatter plots are shown demonstrating the relationship between the NGA and EEG baseline activation in the respective frequency band and region, including regression lines and confidence intervals (95%). We found significant negative correlations between the NGA and current density in the left middle frontal gyrus in the delta (*A*; BAs 8/9/44/45/46), theta (*B*; BA 9), and alpha1 (*C*; BAs 9/44) frequency bands and in the right inferior frontal gyrus in the alpha1 (*D*; BA 46) frequency band. Note that resting delta, theta, and alpha activity are inverse indicators of cortical activation, meaning that higher baseline activation in the lateral PFC predicts a larger NGA, that is, a better response inhibition capacity.

associated with a larger NGA, that is, a better response inhibition capacity (a word of caution is appropriate here as recent literature suggests a complex interpretation of the functional role of EEG slow-waves at rest, see e.g. O'Gorman et al. 2012). This finding is consistent with converging evidence that lateral prefrontal regions are important in inhibiting responses. These regions are activated when subjects execute response inhibition (e.g. Garavan et al. 2002; Aron et al. 2004; Swick et al. 2011). Both patients with lesions in the lateral PFC (e.g. Aron et al. 2003), and healthy subjects in which these regions were temporarily disrupted by means of repetitive transcranial magnetic stimulation (e.g. Chambers et al. 2006), show deficits in response inhibition. Moreover, an individual's degree of inhibitory control across a wide range of regulatory processes is related to baseline activation level in the lateral PFC measured by resting EEG (Gianotti et al. 2009; Knoch et al. 2010; Gianotti et al. 2012).

We additionally wondered whether baseline activation in the ACC would also explain individual variance in response inhibition capacity, as measured by the NGA. Although the ACC is more strongly activated during the NoGo- compared with the Go-condition in the range of the P300 (i.e. the period in which the NGA is calculated; see Supplementary Fig. 2), we did not find a significant relationship between this region's baseline activation and the NGA. We want to stress the fact that the direct comparison between the 2 conditions in the range of the P300 might have not only revealed brain activity related to the process of response inhibition, as several other processes are likely taking place in parallel, including performance monitoring and conflict detection. Indeed, the ACC has been more consistently related to these latter processes than to response inhibition (e.g. Braver et al. 2001; Botvinick et al. 2004; Berkman et al. 2012).

The moderate relationship between baseline activation in the PFC and the NGA, while a relevant observation, suggests that PFC baseline activation may not unerringly index variance in response inhibition capacity. We encourage direct manipulations of PFC activation in subsequent research to bolster the findings presented here. Moreover, future studies could try to identify variables (e.g. personality traits, genes, or patterns of baseline activation in those subcortical regions, which are not detectable with EEG) that potentially moderate the link between PFC baseline activation and the NGA.

Future research could also combine the measurement of both task-independent neural baseline activation and task-dependent NGA in patient populations. It has been repeatedly shown that patients with a disturbed response inhibition capacity (e.g. ADHD and schizophrenia) have a smaller NGA (e.g. Fallgatter and Mueller 2001; Fallgatter et al. 2005; Dresler et al. 2010). Other studies showed abnormal resting-state activity in both ADHD and schizophrenic patients, namely increased power in the slow frequency bands (e.g.

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Koehler et al. 2009; Hong et al. 2012). Based on the present results, it would be worth examining whether a patient's inhibitory deficits (reflected in a smaller NGA) are linked to lower baseline activation in the lateral PFC (reflected in increased power in the slow frequency bands). If this appears to be true, our findings could even serve to promote neurofeedback training to increase baseline activation in lateral frontal regions to enhance the NGA, that is, response inhibition capacity. Indeed, neurofeedback treatments are already applied with some success to treat ADHD (e.g. Kropotov et al. 2005; Arns et al. 2009; Lofthouse et al. 2012). The results of our study could help to inform these efforts and to increase treatment precision by using tomographic neurofeedback (e.g. Congedo et al. 2004; Liechti et al. 2012) in order to specifically target certain slow-wave oscillations originating from particular lateral prefrontal regions.

Supplementary Material

Supplementary material can be found at: http://www.cercor.oxfordjournals.org/.

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