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Latent ERP components of cognitive dysfunctions in ADHD and schizophrenia

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HIGHLIGHTS

- Latent component approach is suitable for revealing functional impairment in ADHD and schizophrenia.
- This approach provides additional information compared to the conventional ERP analysis.
- Qualitatively different patterns of cognitive deficits in ADHD and schizophrenia have been identified.

ABSTRACT

Objective: The main goal was to assess common and specific deficits of cognitive control in (Attention Deficit Hyperactivity Disorder) ADHD and schizophrenia (SZ) using event-related potentials (ERPs).
Method: Behavioral and EEG data in cued GO/NOGO task were recorded in 132 healthy controls (HC) and age, gender and education matched 63 ADHD adults, and 68 SZ patients.
Results: N2d wave in NOGO-GO contrast of ERPs did not differ between the groups while the P3d wave discriminated SZ group from two other groups. Latent components of ERPs were extracted by blind source separation method based on second-order statistics Kropotov et al. (2017) and compared between the groups. A counterpart of N2d wave of a frontally distributed latent component was smaller in SZ indicating a specific frontal dysfunction of conflict detection in SZ. Two centrally distributed P3 subcomponents were reduced in both groups indicating a non-specific dysfunction of action inhibition operations in ADHD and SZ.
Conclusion: A pattern of specific and common dysfunctions in terms of latent ERP components shows a more complex picture of functional impairment in schizophrenia and ADHD in comparison to conventional N2/P3 ERP description.
Significance: The latent component approach shows a functionally different pattern of cognitive control impairment in comparison to the conventional ERP analysis.

1. Introduction

1.1. Cognitive impairment as a core feature of ADHD and schizophrenia

A growing interest in studying clinical symptoms shared across different psychiatric conditions is reflected in the Research Domain Criteria (RDoC) project (Insel et al., 2010). The RDoC was initiated by NIMH in 2008 and aimed to find a biologically-valid framework for understanding mental disorders. In line with this general approach our study aims to find common and unique electrophysiological markers of cognitive dysfunctions in adult ADHD and schizophrenia.

A vast amount of clinical and neuroscience research shows that impaired cognitive control is a common feature of patients with ADHD and schizophrenia (Egeland et al., 2003; Egeland, 2007). Although these disorders have a different etiology, prognosis and treatment approaches patients with ADHD and schizophrenia

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behave similarly poor in neuropsychological tests on cognitive control (Egeland, 2007).

In cognitive neuroscience a theoretical construct of cognitive control defines hypothetical operations that enable human behavior to adjust flexibly to current goals and context. Proactive and reactive modes of cognitive control have been separated (Braver, 2012). The current study deals with the reactive mode of cognitive control. According to the mainstream of current research hypothetical operations of reactive cognitive control include: (1) operations of conflict detection and monitoring which occur when a current situation doesn't match a prepotent model of behavior (Botvinick, 2007; Ullsperger et al., 2014), (2) operations of action inhibition which take place when a prepared action turns inappropriate and needs suppression or overriding (for review see Bari and Robbins, 2013; Huster et al., 2013).

1.2. ERP correlates of cognitive control of healthy subjects

GO/NOGO paradigm represents one of several behavioral tasks sensitive to cognitive control (Rosvold et al., 1956; Kropotov and Ponomarev, 2009, Simson et al., 1977; Falkenstein et al., 1999; Verleger et al., 2005). In this paradigm GO stimuli elicit late parietally distributed positive fluctuations (P3 GO), whereas NOGO stimuli elicit more anteriorly distributed N2 and P3 NOGO waves associated with hypothetical operations of conflict detection and action inhibition (Bruin et al., 2001; Bekker et al., 2004; Kropotov et al., 2011, Fallgatter and Strik, 1999; Fallgatter et al., 2002). In early studies, N2 wave was considered as indicator of action inhibition, however later N2 wave was proved to be evoked by infrequent GO stimuli and consequently was associated with conflict detection operation (Randall and Smith, 2011) whereas the action inhibition operation was linked to the frontal-central P3 NOGO wave (Smith et al., 2008; Enriquez-Geppert et al., 2010; Albert et al., 2013).

However the conventional N2/P3 dichotomy has been recently questioned by a view in which ERP waves in general and ERP wave of cognitive control in particular are considered as sums of multiple spatially distributed and temporally overlapped sources (Kropotov et al., 2011; Kappenmann and Luck, 2012). Several methods for disentangling hidden sources of ERP waves (including methods of blind source separation) have been suggested (Makeig et al., 1996; Onton, and Makeig, 2006; Kropotov and Ponomarev, 2009; Ponomarev and Kropotov, 2013). In studies of our group N2d NOGO wave was decomposed into hidden components with distinct topographies and distinct functional meanings while only one of those components was associated with operation of conflict detection (Kropotov and Ponomarev, 2009; Kropotov et al., 2011, 2016, 2017).

1.3. ERP correlates of cognitive control in ADHD and schizophrenia

In most of adult ADHD studies P3 NOGO was shown to be smaller in comparison to this wave in groups of healthy subjects of the same age (Bekker et al., 2005, Wiersema et al., 2006; Fisher et al., 2011, Woltering et al., 2013, Grane et al., 2016). Similar, reduction of P3 NOGO was reported in most of schizophrenia studies (Weisbrod et al., 2000; Ford et al., 2004; Chun et al., 2013; Kiehl et al., 2000) but not in (Hoonakker et al., 2017).

There were inconsistencies regarding N2 NOGO wave in ADHD and schizophrenia: some studies reported reduction of this wave (Kiehl et al., 2000; Groom et al., 2008) while other studies reported intact N2 NOGO (Grane et al., 2016). Only one study directly compared NOGO ERPs in ADHD and schizophrenia patients performing a visual GO/NOGO paradigm (Groom et al., 2008). This study demonstrated reduced P3 amplitude in schizophrenia patients but not in ADHD patients and reduced N2 NOGO in both patient groups (Groom et al., 2008). The inconsistencies in different dysfunctions of N2 NOGO wave in different studies of ADHD and schizophrenia populations might be explained by inability to disentangle N2 NOGO wave into functionally distinct components.

1.4. Extracting latent components from ERP

Numerous studies demonstrate that N2/P3 dichotomy in analysis of ERPs in GO/NOGO paradigm does not correctly explain operations taking place within the brain during the tasks on cognitive control (e.g. Kropotov and Ponomarev, 2009; Kropotov et al., 2011). Different methodological approaches have been applied for extracting hidden components from ERP (for review see Kappenman and Luck, 2012). Few studies apply independent component analysis (ICA) for ERPs in adult patients with ADHD (Mueller et al., 2010, 2011) and patients with schizophrenia (Olbrich et al., 2005) in attempt to discriminate the extracted components from those of healthy controls.

Recently a new method of blind source separation based on a procedure of joint diagonalization of cross-variance matrixes was suggested (Yeredor, 2010) and adapted for ERPs (Kropotov and Ponomarev, 2015). Functional meanings of latent (hidden) components decomposed by this approach in the cued GO/NOGO paradigm were recently hypothesized (Kropotov et al., 2017). In that study (Kropotov et al., 2017) the conflict detection and action inhibition operations of cognitive control were independently manipulated in four modifications of the cued GO/NOGO paradigm. A latent component associated with the conflict detection operation was discovered. The component was characterized by frontal topography and N2-like response in trials where the prepotent model of behavior was broken (Kropotov et al., 2017). In contrast, latent components associated with action inhibition operations showed central topographies and P3-like responses in trials where a prepared action was inhibited (Kropotov et al., 2017).

1.5. Need for NOGO-GO contrast

In the present paper we used a subpopulation of healthy subjects from our earlier study (Kropotov et al., 2017) as a reference group and the same unmixing matrix for extracting latent components. Briefly, the unmixing matrix is defined from the collection of GO and NOGO ERPs computed for the large group of healthy controls so that the individual deviations of these components from the average are uncorrelated (see Methods). The components are characterized by topography and time dynamics, and, consequently, reflect activation patterns of spatially distributed neuronal networks in response to GO and NOGO stimuli.

Theoretically the activation neuronal patterns to GO and NOGO stimuli reflect three types of operations: (1) operations that are common for GO and NOGO conditions such as activation of neuronal resources needed for attention allocation, for stimulus detection and recognition, for inhibition neuronal resources that are irrelevant for both task conditions, etc. (2) operations that are unique for GO condition such reactivation of the prepotent model of behavior and motor-related processes (Verleger et al., 2005, Kropotov et al., 2017), (3) operations that are unique for NOGO condition such as conflict detection and prepared action inhibition (Bruin et al., 2001; Bekker et al., 2004; Kropotov et al., 2011, Fallgatter and Strik, 1999; Fallgatter et al., 2002; Randall and Smith, 2011; Smith et al., 2008; Enriquez-Geppert et al., 2010; Albert et al., 2013). A priory there is no reason to believe that these three types of unique and common operations show the same pattern of dependency on brain dysfunction present in ADHD and SZ. So there is a need to consider separately components for GO and NOGO conditions, as well as for NOGO-GO contrast where in NOGO-GO contrast the common operations "cancel" each other.

1.6. Aim of the study

The aim of the present study is twofold:

(1) to discover common and specific deficit of cognitive control in patients with ADHD (N = 63) and schizophrenia (N = 68) in contract to a healthy control group (N = 132) by means of extracting latent components from a collection of ERPs in the cued GO/NOGO task, and
(2) to demonstrate advantages of the latent component method in comparison to the conventional N2/P3 ERP analysis by means of comparing the results of these two approaches.

2. Methods

2.1. Participants

2.1.1. Healthy control group

The healthy control (HC) group included 132 healthy control subjects (53 males, mean age 31.8; SD 8.26; range 18–50). The healthy control subjects were from Chur, Switzerland, from students of Saint-Petersburg State University and from the staff of the N.P. Bechtereva Institute of the Human Brain of Russian Academy of Sciences. Subjects with history of head injury, with neurological or/and psychiatric conditions were excluded from the study. The control subjects were not receiving medication at the time of testing. The subjects filled out Brief Symptom Inventory (Franke, 2000), Current Symptoms Scales, and Health History questionnaire (Barkley and Murphy, 2006). The including criteria was to score lower the threshold of clinical significance on symptom checklists.

2.1.2. ADHD group

The ADHD group consists of 63 adult patients (30 males, mean age 33.1; SD 7.84; range 18–50) diagnosed with ADHD by an independent psychiatrist. We recruited ADHD subjects from other psychiatrists and ADHD associations as well by advertising in the local media. The subjects were included in the ADHD group on the basis of DSM-IV criteria for ADHD (Barkley et al., 2008) if they recalled retrospectively the presence of at least 4 inattention and/or hyperactivity/impulsivity symptoms in childhood, and if at least 4 inattention and/or hyperactivity/impulsivity symptoms had been often present during the last 6 months. ADHD subjects filled out a series of questionnaires, including the Brief Symptom Inventory (Franke, 2000), the Current and Childhood Symptoms Scales (Barkley and Murphy, 2006), and the health history questionnaire (Barkley and Murphy, 2006). The rating scales were also obtained from the subject's parents (Childhood Symptoms Scale) and partners (Currant Symptoms Scale) when available. 18 subjects were diagnosed as inattentive ADHD type, 3 subjects as hyperactive/impulsive type, and 42 subjects as combined ADHD type. No discrimination between ADHD types was made in this study because the goal was to find brain dysfunctions common for the adult ADHD group as a whole. The patients were either unmedicated or refrained from taking medication at least 1 day before the testing. Also, subjects with head injury, with neurological or other psychiatric conditions were excluded from the study.

2.1.3. SZ group

68 SZ patients (47 males, mean age 30.6; SD 7.17; range 18–50) participated in our study. The patients were diagnosed by a consensus of senior staff psychiatrists of Institute of the Human Brain of Russian Academy of Sciences (an author of the paper Yury I. Poliakov was one of them) and met ICD-10 criteria for diagnosis of schizophrenia (World Health Organization, 1992). Exclusion criteria were recent drug abuse, a history of neurological disorder and of benzodiazepines treatment. All SZ patients had normal or corrected-to-normal visual acuity. The patients had an illness of at least 6 months of duration and demonstrated moderate to severe degree of social and cognitive impairment at the time of the testing. 18 patients received atypical antipsychotic medication (Risperidone, Aripiprazole, Clozapine, Quetiapine, Amisulpride), 19 patients received a typical antipsychotic medication (Haloperidol), 31 patients received no neuroleptic medication. The positive and negative syndrome scale for schizophrenia scores (PANSS; Kay et al., 1987) for the patients selected for our study were as follow: the negative symptoms scale: 20.1 ± 4.4 (mean ± SD), the positive symptoms scale: 26.1 ± 4.6 (mean ± SD). The patients had a score of 47.9 ± 5.9 on general psychopathology scale. These symptoms' values were similar to those presented in (Kay et al., 1987). No distinction between the subgroups of schizophrenia was taken into account because the goal of the study was to find dysfunctions common for the schizophrenia group as a whole.

The groups of participants did not differ significantly in age, handedness and years of education. The study was approved by the local ethical committee. A written informed consent was obtained from all participants after an explanation of the procedure.

2.2. Task and stimuli

The cued GO/NOGO task was used for studying brain correlates of cognitive control (Kropotov and Quantitative, 2008; Kropotov and Ponomarev, 2009). During a single task 400 trials with 3 s inter-trial intervals were presented to a participant. Each trial consisted of sequential presentation of two visual stimuli with 1 s inter-stimulus interval and 0.1 s stimulus duration. Different images of animals (*a*), plants (*p*) and humans (*h*) were selected as stimuli. The overall luminance and image size of stimuli were approximately equal. The stimuli were randomly presented in the following pairs *a-a, a-p, p-p and p-h* with equal probabilities. The subjects were instructed to press a button to *a-a* trials as fast as possible and as precise as possible. In order to keep alertness novel sounds were presented simultaneously with images of humans in ignore trials. These 'novelty' trials produced an orientation reaction reflected in P3 novelty ERP wave. In this paper we analyzed only *a-a* (GO) trials and *a-p* (NOGO) trials. We also dealt with reactive cognitive control operations taking place after the second stimulus presentation in *a-a* and *a-p* pairs.

The trials were presented in four equal blocks. In each block a unique set of five different *a* images, five different *p* images, and five different *h* images were presented. To avoid habituation effect subjects practiced the task before EEG recording. After 200 trials participants rested for a few minutes. Subjects sat upright in a comfortable armchair. Stimuli were presented on a 17-inch CRT computer screen located 1.5 meters from the subjects' eyes and occupied 3.8° of the visual field. All trials were presented using a software *Psytask* designed by one of the authors (VAP).

2.3. Recording EEG and subject's responses

19-channel EEG was digitally filtered in 0.53–50 Hz frequency band and sampled at 250 Hz. A digital notch filter (45–55 Hz) was used to remove 50 Hz artifact. The electrodes were applied according to the International 10–20 system. Impedances were kept below 5 kOhm. The EEG was referenced to linked ears. The same equipment in the same standard conditions was used in all recordings. The equipment included (1) a 19-channel electroencephalograph Mitsar-201 (CE 0537) manufactured by Mitsar, Ltd. (http://www.mitsar-medical.com), (2) a software for acquisition and analysis of EEG and ERPs - WinEEG, written by one of the authors (Ponomarev Valery A). (3) a software for stimulus presen-

tation Psytassk, (4) tin electrodes assembled in caps manufactured by Electro-cap International, Inc. (http://www.electro-cap.com/caps.htm), (5) ECI ELECTRO-Gel for contacting electrodes with the scalp.

A separate channel recorded subject's responses. It was used for computing behavior parameters during the task: reaction time (RT), RT variance, omission errors (failure to respond in GO trials), and commission errors (failure to inhibit a response in NOGO trials). EEG epochs with erroneous responses were also automatically excluded from analysis. The subject's response was considered correct if it was made in the time interval 200–1000 ms after the relevant stimulus.

2.4. Artifact correction

Eye blink artifacts were corrected by an Independent Components Analysis (ICA) method applied to raw EEG fragments, i.e. by zeroing activation curves of independent components with topographies related to individual eye blinks. The method was described in (Vigario, 1997; Jung et al., 2000; Li et al., 2006) and was shown to fit an EOG regression technique in (Tereshchenko et al., 2009). Further, epochs with excessive EEG amplitude (>100 μV) and/or excessive fast waves (>35 μV for EEG filtered in 20–35 Hz band) and/or slow frequency waves (>50 μV for EEG in 0–1 Hz band) were automatically excluded from analysis. Further analysis was carried out only with EEG records, for which the number of artifact-free trials was not less than 30 for each of the conditions.

2.5. Latent components separation

The model of blind source separation methodology from a collection of ERPs is presented in the following equation:

$$x_i^j(t) = \sum_{k=1}^{K} a_{i,k} s_k^j(t) + \varepsilon_i^j(t)$$

where $x_i^j(t)$ is ERP at i-electrode (i = 1, …, M) of j-subject (j = 1, …, P) at time point t (t = 1, …, T), ε is the noise which is supposed to be stationary, additive, isotropic and independent of the signals $s_k^j(t)$. a_{ik} – weights that constitute $M \times K$ mixing matrix.

A method of blind source separation based on second-order statistics was used in the present study. In a general form it was described in (Yeredor, 2010) and adapted for ERPs in (Ponomarev and Kropotov, 2013; Kropotov and Ponomarev, 2015). The main assumption of the method is that the deviations of individual components from the group average components are low correlated signals. One of drawbacks of this method is that for obtaining reliable components a large number of individual ERPs is required. In order to fit this requirement the elements a_{ik} of the mixing matrix A in the present study were taken from our previous study of 454 healthy adult subjects performed the same GO/NOGO task (Kropotov et al., 2016). The method allowed us to obtain low-correlated components with a high level of the split-half reliability of the components (Kropotov et al., 2016). Using this matrix, the ERP data were separately converted for each subject of the present study into latent components as $\hat{S} = A^{+}X$, where A^{+} is the Moore-Penrose pseudoinverse of A.

To verify how well the mixing matrix explains the present ERP data, a normalized mean square error (NMSE) for each subject separately as $NMSE = \| X - A\hat{S} \|_2^2 / \| X \|_2^2$ was calculated in time interval from −100 ms to 1600 relative to the second stimulus in the trials. Then the averaged value of NMSE and its standard deviation were estimated for each group of subjects separately. In all cases the value of *NMSE* was relatively small: *NMSE* = 0.0040, *SD* = 0.0026 for HS, *NMSE* = 0.0051 and *SD* = 0.0045 for ADHD, and *NMSE* = 0.0098 and *SD* = 0.0124 for SZ and did not differ significantly between the groups.

A first example of application of the method was presented in (Kropotov and Ponomarev, 2015). The goal of this study was to decompose ERPs into latent components associated with hypothetical processes of category discrimination, comparison to working memory and action-related operations. With this goal we designed five variants of the delayed match-to-sample s1-s2 task by independently manipulating instructions and associated operations. The BSS method was applied to the collection of ERPs. The category discrimination operation was attributed to latent components generated in different parts of the prestriate cortex with peak latencies of 130–170 ms. The comparison to working memory operation was attributed to a latent component generated in the temporal cortex with a peak latency of 250 ms.

The latest our attempt of using the group BSS method based on joint diagonalization of covariance matrices of ERPs was described (Kropotov et al., 2017). We designed four variants of a frequently used go/no-go paradigm in which operations of reactive cognitive control were independently manipulated. The results showed that a latent component, generated in the anterior cingulate cortex, induced N2/P3 fluctuation only in conditions in which the prepotent model was violated, and thus ccould be associated with conflict detection operations. In contrast, the two latent components generated in the vicinity of the central sulcus induced P3-like fluctuations in conditions in which the prepared action was suppressed, and thus could be associated with action inhibition operations.

2.6. Statistical analysis

2.6.1. Analysis of behavioral data

Errors of omission and commission, as well reaction times (RT) were assessed by one-way ANOVA, factor with two levels – pair of groups.

2.6.2. Analysis of raw ERPs and latent components

ERPs have been analyzed within time intervals (windows) corresponding to N2 and P3 waves. The boundaries of these windows approximately corresponded to half amplitude of the peak value of the NOGO-GO difference for all groups of participants. Three time interval were selected: 220–270 ms, 260–430 ms and 300–450 ms. The first time interval approximately corresponded to N2 wave in our experimental conditions, the second time interval corresponded to parietal P3 and the third – to frontal P3. The mean values of the potentials were calculated for each of selected time intervals in each individual ERP and each of the conditions and used for further statistical analysis. ANOVA with (1) the between-subjects factor – "group" (three levels: healthy subjects, ADHD and SZ), and (2) the within subjects factor "experimental condition" (GO and NOGO), and (3) the within subjects factor "electrode position" (19 channels) was performed separately for each of selected time intervals. Degrees of freedom were corrected with the Greenhouse-Geisser epsilon coefficient.

The analysis of ERP differences across the groups was performed using one-way ANOVA with two levels (pairs of the groups). This analysis was carried out for each pair of the groups, each condition (GO and NOGO), as well as for difference wave (NOGO-GO) and each electrode (Fz, Cz, Pz) separately. Only midline electrodes were selected because the largest differences between the groups were observed around those electrodes. The time intervals for analysis were selected visually and corresponded to time windows in which the ERP differences across the groups revealed larger values. In total, six time intervals were selected. Input data for statistical analysis were mean values of potentials in the

selected time intervals for each condition, each electrode and each subject separately. In total, 54 statistical comparisons (3 electrodes × 3 pairs of the groups × 6 time intervals) were performed. To eliminate false positives Bonferroni correction was applied. Consequently, the results were reported with $p < 0.01/54 = 0.0002$.

The analysis of latent components was performed in similar way. In this case the time intervals were also selected visually according the criteria described above. Since the waveforms of ERPs and latent components are not identical a distinct set of time intervals for analysis was selected for a given component. In total, 10 time intervals were selected and 30 statistical comparisons were performed. Corresponding Bonferroni correction was applied. Consequently the results were reported with $p < 0.01/30 = 0.0004$.

3. Results

3.1. Performance data

Table S1 shows behavioral data for GO and NOGO conditions and each experimental group and the results of their comparison across the groups.

3.2. ERP measures: GO, NOGO waves and NOGO-GO difference waves

Three-way ANOVA analysis with the between-subjects factor group (three levels: healthy subjects, ADHD and SZ) and with the within subjects factors experimental condition (GO and NOGO) and electrode position (19 channels) performed for N2 and P3 latency windows revealed significant differences for 220–270 ms ($F[(36, 4680] = 2.18$, $p < .05$), 260–430 ms ($F[(36, 4680] = 6.98$, $p < .001$) and 300–450 ms ($F[(36, 4680] = 8.48$, $p < .001$) intervals.

Fig. 1 shows grand average ERPs for correct GO trials, NOGO trails and NOGO-GO differences for the midline electrodes (Fz, Cz, and Pz), and for the groups of patients with ADHD (red line), schizophrenia (blue line) in contrast to ERPs of healthy controls (black line).

As one can see GO stimuli for all groups elicit a positive fluctuation of potential at peak latency of approximately 320 ms and of parietal distribution (Fig. 1b), while NOGO stimuli elicit a positivity at about 370 ms with more anterior distribution (Fig. 1c). On the NOGO-GO contrats a frontally distributed negativity N2d is seen for all groups at latency of about 240 ms which is followed by a frontal positivity P3d at around 400 ms (Fig. 1a).

The results of the statistical comparison of ERPs waveforms in three groups are presented in Table S2. No significant differences bewteen the groups were found for N2 window. In contrast P3 amplitude showed statistically significant differences between the SZ and two other groups with smallest values in SZ group and largest values in HC group.

3.3. Decomposition of ERPs into latent components

Fig. 2 represents the results of decomposition of the central-frontal NOGO wave for the three groups of participants (black line – HC, red line – ADHD, blue line – SZ) into latent components. The decomposition was made on the basis of our previous study by applying method of joint diagonalization of covariance matrixes (Kropotov et al., 2017). In this previous study four modifications of the GO/NOGO paradigm were designed so that the hypothetical operations of conflict detection and action inhibition were independently manipulated. According to this study the NOGO-GO contrast of the component on the top of Fig. 2 appeared only in trials in which a current condition did not match the prepotent model of behavior, and was associated with conflict detection operation. The NOGO-GO contrasts for the two other components (middle and bottom rows on Fig. 2) appeared only in trials in which the subjects had to suppress a prepared action and were associated with action inhibition.

3.3.1. Component of conflict detection

Fig. 2 top row demonstrates grand averages of the component of conflict detection operation. One can see that N2d/P3d of this frontally distributed component does not differ between ADHD and HC groups but differentiates SZ group from HC group. Statistical significance of the differences is presented in Table S3.

3.3.2. Components of action inhibition

Fig. 2 (middle and bottom rows) demonstrates grand averages of two components of action inhibition for the three groups separately for GO, NOGO conditions, as well as for the NOGO-GO contrast. The pattern of deviation from the healthy controls for ADHD and SZ groups is different. In particular, for NOGO condition and late time window one component (Action inhibition 1) is smaller in comparison to HC both in ADHD and SZ groups, the other component (Action inhibition 2) is significantly smaller only for SZ group.

4. Discussion

4.1. Behavior: Attention deficit in patient groups

The present study shows that both patient groups in comparison to healthy controls have clear behavioral pattern of attention deficit: an excessive number of omission errors (due to missing targets) and an excessive variance of reaction time (due to attention lapses). This attention deficit fits behavioral abnormalities observed in numerous studies on ADHD and schizophrenic patients in continuous performance tasks (Egeland, 2007; Pallanti and Salerno, 2015). In the present study the direct comparison of the behavioral parameters between the patient groups indicates, however, that this deficit is quantitatively larger in SZ group in comparison to ADHD (see also Frith and Done, 1989).

4.2. N2d and P3d waves

In the present study operations of cognitive control are discussed within a theoretical concept of the pre-potent model of behavior (Isoda and Hikosaka, 2011; Ridderinkhof et al., 2011). Following this concept, we presume that NOGO stimulus in GO/NOGO paradigm doesn't fit the prepotent model resulting in conflict detection operation (Larson et al., 2014) followed by action inhibition (Bari and Robbins, 2013; Huster et al., 2013). Previous studies show that schizophrenia and ADHD are associated with deficits in establishing prepotent responses in complex paradigms and failures to inhibit the prepotent responses (Senderecka et al., 2012; Becerril and Barch, 2013).

Difficulty in inhibition is reflected in commission errors while brain representations of cognitive control are reflected in the NOGO-GO ERP contrast (Gemba and Sasaki, 1989; Randall and Smith, 2011). In this contrast N2d wave is currently associated with conflict detection and P3d wave is associated with action inhibition (Randall and Smith, 2011; Smith et al., 2008; Enriquez-Geppert et al., 2010; Albert et al., 2013). As we see in our data (Fig. 1) P3d wave is smaller in SZ than in ADHD and HC. However N2d wave is indistinguishable between the groups.

This result fits some studies and contradicts the other studies (Bekker et al., 2005, Wiersema et al., 2006; Fisher et al., 2011, Woltering et al., 2013, Grane et al., 2016; Weisbrod et al., 2000; Ford et al., 2004; Chun et al., 2013; Kiehl et al., 2000; Groom et al., 2008). The main reason of this discrepancy might be that

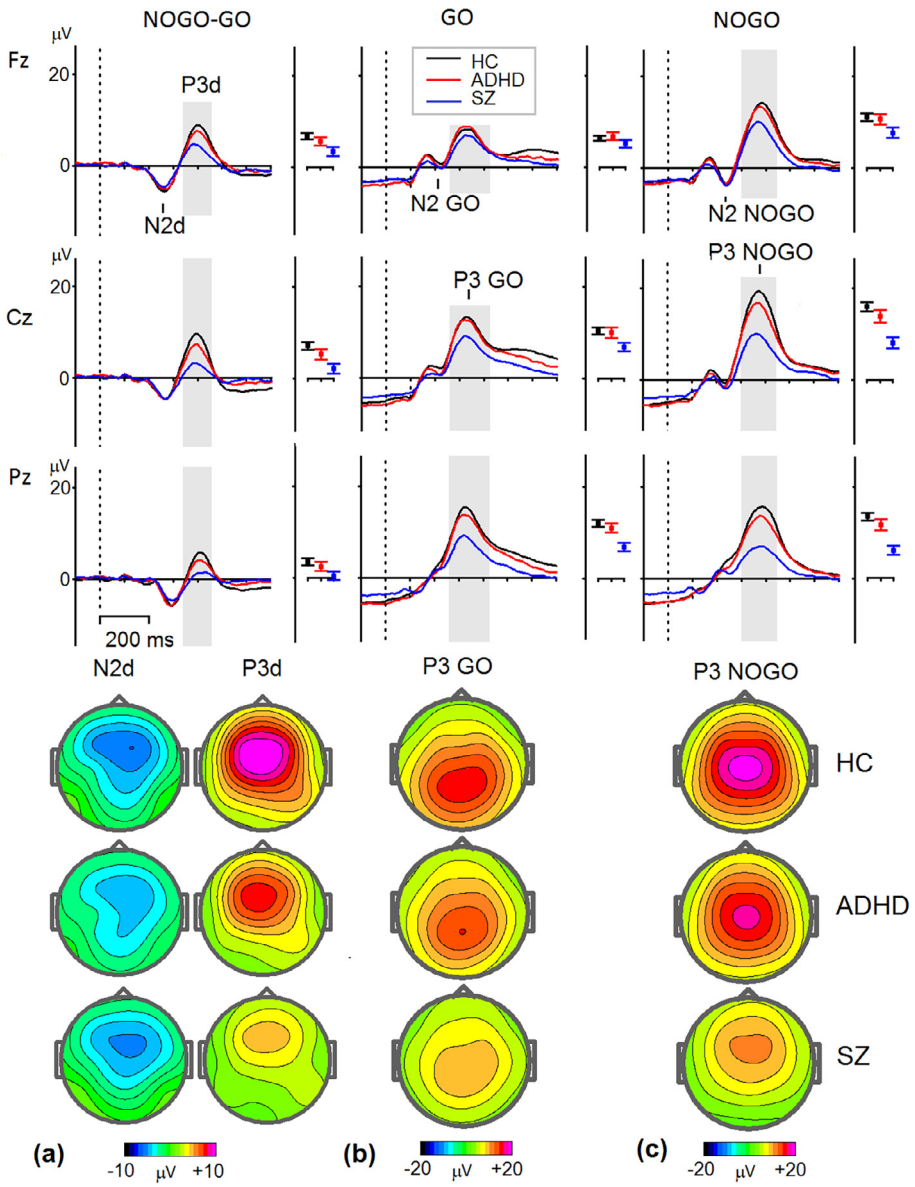

Fig. 1. ERPs for midline electrodes (Fz, Cz, and Pz) for NOGO-GO differences (a), GO (b), NOGO trials (c) and and their maps for for healthy controls (HC), patients with ADHD, and patients with schizophrenia (SZ). Top: (a) grand averaged ERP for NOGO-GO, (b) GO and (c) NOGO conditions for the group of healthy controls (N = 132, black lines), patients with ADHD (N = 63, red lines), patients with schizophrenia (N = 68, blue lines). Time windows corresponding to P3 waves (GO, NOGO and NOGO-GO) are marked by grey color. The mean values and 95% confidence levels of the potential in the corresponding time windows are presented to the right of the curves. N2 and P3 GO, N2 and P3 NOGO, as well as N2d (NOGO-GO) and P3d (NOGO-GO) waves are indicated. Bottom: topograpies of P3 waves taken at their maximums. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

the conflict detection operation and its ERP representation are contaminated by other operations that occur in the same time window during the GO/NOGO paradigm (Kropotov et al., 2011). To resolve this discrepancy in the present study the latent components for the three groups of participants (HC, ADHD, and SZ) have been extracted and compared with each other.

4.3. Latent components

By simply comparing Figs. 1 and 2 one can see that the pattern of the cognitive control deficit in ADHD and SZ groups in terms of latent components (Fig. 2) qualitatively differs from that in terms of N2/P3 ERP waves (Fig. 1). More specifically:

4.3.1. Component of conflict detection

As has been demonstrated in our previous paper the frontal latent component (Fig. 2 top row) fits the definition of conflict detection representation, i.e. shows clear N2/P3 pattern in conditions when the prepotent model is violated (Kropotov et al., 2017). According to sLORETA this component is localized in the anterior congulate cortex (Kropotov et al., 2017) which fits well to other studies regarding localization of conflict detection operation within the brain (Botvinick, 2007; Ullsperger et al., 2014). The early part, N2 fluctuation, seems to represent the conflict detection operation. In the present study N2d part of this latent component is found to be smaller in SZ group but does not differ between ADHD and HC groups. The fact that N2 effect in SZ is seen only after decomposing the N2 wave into latent components demonstrates an advantage of using the component decomposition approach in comparison to the conventional N2/P3 description.

The observed effect might explain why SZ patients lose the feeling of reality because they simply do not differentiate between expectation (prepotent model) and reality (Frith and Done, 1989). Our data also fit numerous studies demonstrated that indi-

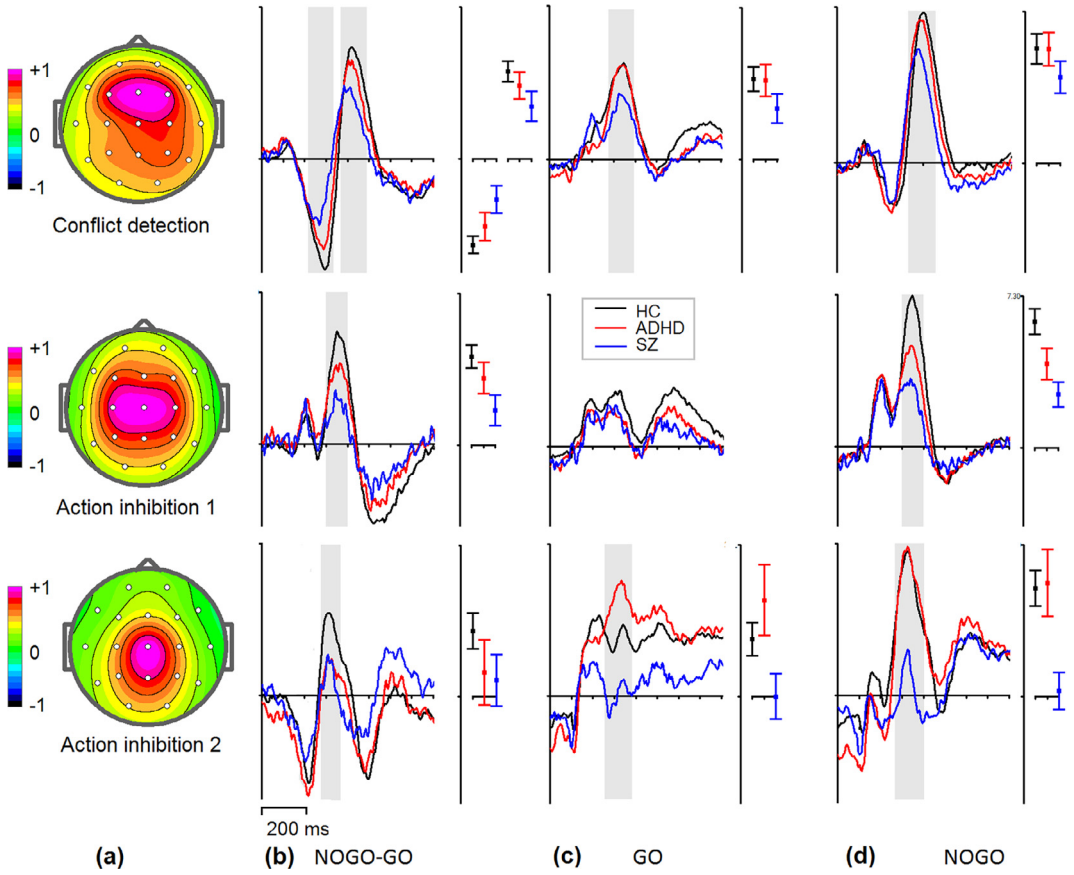

Fig. 2. Latent components associated with conflict detection operation and action inhibition operations in three groups of participants (black line – HC, red line – ADHD, blue line – SZ). (a) topographies of components with numbers below indicating the relative variance (power) of the component; (b) component differences for NOGO-GO contrast; (c) components for GO condition, (d) components for NOGO condition. Note that because of the ambiguity of component's amplitude the Y axis is in conventional units. X-axis – time, zero point – onset of the second stimulus. To the right of the curves – mean values and 95% confidence intervals of the corresponding components (or component contrasts) computed for the time windows depicted as grey columns. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

viduals with schizophrenia often showed impairments in ability to monitor their ongoing behavior (Frith and Done, 1989; Malenka et al., 1986), and to adjust their responses on the basis of advance information or feedback (Elliott et al., 1995). In line with the observed effect SZ patients show reduction of an ERP index of error detection (error-related negativity) (Minzenberg et al., 2014) and significant fMRI hyperactivation to non-targets in brain areas activated by targets (Liddle et al., 2013).

It should be stressed here that our data demonstrate intact N2d part of the frontal component in ADHD group which in turn indicates undamaged conflict detection operation in this disorder (see also Herrmann et al., 2010).

4.4. Components of action inhibition

Our previous study (Kropotov et al., 2017) has shown that there are several latent ERP components associated with action (response) inhibition operations (Fig. 2 middle and bottom rows), i.e. these components demonstrate P3-like fluctuations when the subject inhibits the prepared response. According to sLORETA, these components are generated in the vicinity of mesial parts of the central sulcus. The involvement of neuronal populations in the supplementary/pre-supplementary motor areas during response inhibition has been demonstrated in many fMRI studies with stop-signal and GO/NOGO tasks (de Wit et al., 2012; Warren et al., 2013). Neuronal representation of response inhibition have been decomposed into sub-components with different functional meanings, for example, global inhibition vs. selective inhibition, early vs. late (Ridderinkhof et al., 2014).

In the present study the two components of action inhibition show central (Fig. 2 middle raw) and central-parietal (Fig. 2 middle raw) distributions with peak latency of about 20 ms shorter for the central-parietal component. Local lesion studies provide strong evidence that separate subregions of the prefrontal cortex make critical contributions to different aspects of response inhibition (Picton et al., 2007). Neuroscience studies have demonstrated the existence of several neuronal networks involved in response inhibition operation such as (1) the indirect pathway of the basal ganglia-thalamo-cortical motor circuit that in case of NOGO condition inhibits neurons in the ventro-lateral nucleus of the thalamus projecting to the pre-supplementary motor and pre-motor cortical areas (Alexander, 1994), (2) the right prefrontal cortex (Levy and Wagner, 2011; Zhang et al., 2017); (3) the inferior frontal cortex and pre-supplementary motor area (Duann et al., 2009).

The available data don't allow us to associate these circuits with the decomposed latent ERP components but the comparison of the components between SZ and ADHD groups shows that amplitude of two action inhibition components is reduced in SZ both for NOGO-GO difference and for NOGO activation patterns while ADHD group shows a different pattern: only one component is selectively impaired. This observation allows us to speculate that response inhibition brain dysfunction in ADHD is more localized than that in SZ and is confined by a local network.

4.5. Limitations of the study

1. The study does not take into account the heterogeneity of patients in both groups but rather analyzes the overall patterns of ERP components that characterize each disease as a whole. Further studies are needed to demonstrate how different subtypes of ADHD and SZ groups can be differentiated by the latent component approach.
2. The validity of application of the unmixing matrix from a collection of ERP in a large group of healthy subjects (N = 454) obtained in our previous study (Kropotov et al., 2016) must be considered as a hypothesis. A priory we don't know if the component structure of healthy subjects is the same in the both patient groups. To prove this hypothesis more patients with ADHD and schizophrenia must be recorded.
3. The blind source separation method used in this paper does not allow localization of components in left and right hemispheres separately. This must be considered as a limitation of the approach because some studies indicate left/right asymmetry in ERPs of SZ patients (Renoult et al., 2007). To overcome this limitation a higher order statistics must be used for decomposing ERPs into latent components (De Lathauwer, 2010).

5. Conclusions

Our study demonstrates that application of a blind source separation method to ERPs (described in Kropotov and Ponomarev, 2015) provides a qualitatively different view to neuronal mechanism of cognitive deficit in patients with ADHD and schizophrenia (SZ) in comparison to the description given by the conventional N2/P3 dichotomy ERP analysis.

Indeed, the N2/P3 approach indicates no deficit in conflict detection operation and deficit in response inhibition which is larger in SZ group than in ADHD. A qualitatively different picture is given by the latent component approach indicating a selective conflict detection deficit in SZ. In particular, a counterpart of N2d wave of the frontally distributed latent component is significantly smaller in SZ (but not ADHD) group in comparison to HC indicating a specific frontal dysfunction of the conflict detection mechanism in SZ. The P3d parts of centrally distributed components were reduced in both groups indicating a non-specific dysfunction of action inhibition operations in ADHD and SZ.

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Conflict of interest

The authors claim no conflict of interest.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.clinph.2019.01.015.

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