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BASIC RESEARCH ARTICLE

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Event-related potentials associated with cognitive control in adolescents exposed to complex childhood trauma

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ABSTRACT

Background: Complex childhood trauma (CCT), characterized by repeated and prolonged exposure to adverse experiences, disrupts cognitive, emotional, and neural development. Adolescence, a critical developmental period, is particularly vulnerable to these effects, with CCT increasing the risk of psychopathologies, including post-traumatic stress disorder (PTSD). Despite this, the neurophysiological underpinnings of trauma-related deficits in cognitive control remain insufficiently explored, particularly in the developing brains of children and adolescents. This study aimed to investigate the neurophysiological markers of cognitive control in adolescents with CCT using event-related potential (ERP) components to propose an electrophysiological phenotype associated with CCT, as a vulnerability for PTSD.

Methods: Twenty adolescents with CCT and 40 age- and gender-matched healthy controls performed a cued GO/NOGO task. ERP components – contingent negative variation (CNV), NoGo-N2, and NoGo-P3 – were analysed alongside behavioural measures such as omission and commission errors and reaction time, using a preregistered protocol. Statistical analysis included Mann–Whitney tests and cluster-based permutation tests for ERP comparisons.

Results: Adolescents with CCT showed significant impairments in both proactive (reduced CNV amplitudes) and reactive (diminished NoGo-N2 and NoGo-P3 amplitudes) control mechanisms. Behaviourally, the CCT group exhibited higher omission errors and shorter reaction times than controls. Exploratory analysis revealed reduced amplitudes in the visual negativity (VN) component, suggesting disruptions in predictive processing. Latent component analysis identified ERP markers with potential diagnostic utility, linking deficits to key neural circuits associated with cognitive control and predictive processing.

Conclusion: Study findings highlight significant impairments in cognitive control mechanisms and disrupted predictive processing in adolescents with CCT, emphasizing the importance of addressing trauma-related neural deficits during adolescence. Given that CCT is a significant risk factor for PTSD, the study provides insights into shared neurobiological pathways, supporting the development of targeted interventions. ERP markers like CNV, NoGo-N2, NoGo-P3, and VN show promise for improving diagnostic precision and monitoring therapeutic outcomes in trauma-exposed youth.

Potenciales relacionados con eventos asociados al control cognitivo en adolescentes expuestos a trauma infantil complejo

Antecedentes: El trauma infantil complejo (TIC), caracterizado por una exposición repetida y prolongada a experiencias adversas, interfiere en el desarrollo cognitivo, emocional y neuronal. La adolescencia, una etapa crítica del desarrollo, es especialmente vulnerable a estos efectos, ya que el TIC incrementa el riesgo de psicopatologías, incluyendo el trastorno de estrés postraumático (TEPT). A pesar de esto, los fundamentos neurofisiológicos de los déficits en el control cognitivo relacionados al trauma siguen estando poco explorados, especialmente en cerebros en desarrollo de niños y adolescentes. Este estudio tuvo como objetivo investigar los marcadores neurofisiológicos del control cognitivo en adolescentes con TIC utilizando componentes del potencial relacionados con evento (ERP, por sus siglas en inglés), con el fin de proponer un fenotipo electrofisiológico asociado al TIC como una vulnerabilidad para el TEPT.

Métodos: Veinte adolescentes con TIC y 40 controles sanos emparejados por edad y género realizaron una tarea con pistas previas de GO/NOGO. Se analizaron componentes ERP – variación negativa contingente (CNV), NoGo-N2 y NoGo-P3 – junto con medidas conductuales como errores de omisión y comisión y tiempo de reacción, usando un protocolo pre-registrado. El análisis estadístico incluyó pruebas de Mann–Whitney y pruebas

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Trauma infantil complejo; potenciales relacionados con eventos; control cognitivo; adolescencia; TEPT; marcadores electrofisiológicos; procesamiento predictivo

HIGHLIGHTS

- Adolescents exposed to complex childhood trauma exhibit impaired cognitive control mechanisms, revealing critical neurodevelopmental vulnerabilities that may increase the risk of PTSD.
- ERP markers (CNV, NoGo-P3, NoGo-N2, and VN), along with some of their latent components identified using a method based on joint diagonalization of covariance matrices, are proposed as promising tools for early detection of neural changes linked to complex childhood trauma.
- The study emphasizes the importance of addressing disrupted predictive processing and executive function in adolescents exposed to complex childhood trauma to improve developmental and psychological outcomes.

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de permutación basadas en clústeres para comparar los ERP.
Resultados: Los adolescentes con TIC mostraron déficits significativos tanto en los mecanismos de control proactivos (amplitudes reducidas del CNV) como reactivos (amplitudes disminuidas de NoGo-N2 y NoGo-P3). A nivel conductual, el grupo con TIC presentó más errores de omisión y tiempos de reacción más cortos que los controles. El análisis exploratorio reveló amplitudes reducidas en el componente de negatividad visual (VN), lo que sugiere alteraciones en el procesamiento predictivo. El análisis de componentes latentes identificó marcadores ERP con posible utilidad diagnóstica, vinculando los déficits a circuitos neuronales claves asociados con el control cognitivo y el procesamiento predictivo.
Conclusión: Los resultados del estudio destacan déficits significativos en los mecanismos de control cognitivo y alteraciones en el procesamiento predictivo en adolescentes con TIC, enfatizando la importancia de abordar los déficits neuronales relacionados al trauma durante la adolescencia. Dado que el TIC representa un importante factor de riesgo para el TEPT, el estudio ofrece información sobre vías neurobiológicas compartidas, lo que respalda el desarrollo de intervenciones dirigidas. Los marcadores ERP como CNV, NoGo-N2, NoGo-P3 y VN muestran potencial para mejorar la precisión diagnóstica y el seguimiento de resultados terapéuticos en jóvenes expuestos a trauma.

1. Introduction

Complex childhood trauma (CCT) refers to prolonged and repeated exposure to adverse experiences such as chronic abuse, neglect, or exposure to violence, during formative years. These experiences profoundly disrupt cognitive, emotional, and neural development, impairing the maturation of critical brain circuits, including those connecting the prefrontal cortex, amygdala, and hippocampus (Cisler & Herringa, 2021; van der Kolk, 2005). This chronic dysregulation increases vulnerability to post-traumatic stress disorder (PTSD) and other psychopathologies while diminishing the individual's ability to recover from or adapt to future stressors (Cisler & Herringa, 2021). Adolescence is particularly critical, as heightened neuroplasticity during this period amplifies the effects of trauma on executive functions, such as response inhibition and attention regulation (Milbocker et al., 2021). Over time, the cumulative burden of unresolved trauma and impaired executive functioning could create a maladaptive feedback loop (D'Andrea et al., 2012) characterized by ineffective coping mechanisms and heightened stress reactivity. Consequently, CCT has been identified as a transdiagnostic risk factor for PTSD and lifelong psychopathology (McLaughlin et al., 2020).

Despite its profound impact, CCT remains unrecognized as a formal diagnosis, complicating efforts to understand how children and adolescents uniquely express trauma-related symptoms. Subtle deficits in attention regulation and response inhibition often precede PTSD symptoms, serving as potential risk factors linked to symptom severity (Aupperle et al., 2012). However, research exploring these cognitive processes in younger populations, especially during adolescence, is limited. Studies on event-related potentials (ERPs) associated with operations of cognitive control offer a promising avenue to identify neurophysiological markers of these deficits, that might be reflected in specific electrophysiological patterns of ERPs (Askovic et al., 2020; Häger et al., 2024; Ogrim & Kropotov, 2020). ERP components such as contingent negative variation (CNV, first described by Walter, 1967), N2, and P3 are associated with preparatory, conflict-monitoring, and inhibitory processes and have been extensively studied in trauma and PTSD populations (for a review see Polich, 2007; Di Russo et al., 2019).

CNV reflects anticipatory attention and preparatory motor control, and was associated with energization (Brunner et al., 2015). Alterations in CNV amplitudes have been observed in trauma-exposed individuals, though findings are mixed. For instance, Duan et al. (2016) reported increased CNV amplitudes in subjects with PTSD, with a significant positive correlation between CNV amplitude and symptom severity (Duan et al., 2016). Conversely, other studies have documented reduced CNV amplitudes in trauma-exposed populations, suggesting impaired proactive control and compromised task preparation (Kimble et al., 2004). These discrepancies may reflect variability in trauma type, symptom severity, and neural adaptation mechanisms.

The N2 wave, particularly the NoGo-N2 component, is thought to reflect conflict monitoring and early inhibitory processing. Some studies interpret the N2 primarily as a marker of conflict monitoring rather than inhibition per se (Donkers & van Boxtel, 2004; Nieuwenhuis et al., 2003). Decreased NoGo-N2 amplitudes may indicate impaired cognitive flexibility and difficulties in detecting response conflict (S. Kim et al., 2018; Kropotov et al., 2011). However, trauma-related findings are mixed (Miller et al., 2021): while some report reduced N2 amplitudes in trauma-exposed individuals (Duan et al., 2018) others found no significant group with PTSD (Shucard et al., 2008) or increased N2 amplitudes in maltreated adolescents (Bruce & Kim, 2022). This variability may suggest developmental or context-specific variability.

NoGo-P3 is typically associated with the evaluation (allocation of attentional resources) and inhibition of responses (Brunner et al., 2015; Randall & Smith,

2011). Reduced amplitudes in trauma-exposed individuals suggest impaired inhibitory control and broader attentional deficits (Butt et al., 2019; S. Kim et al., 2018; Miller et al., 2021; Min et al., 2020). These neural alterations often correspond to behavioural outcomes such as heightened impulsivity and longer reaction times (Min et al., 2020; Veltmeyer et al., 2005).

Collectively, alterations in CNV, NoGo-N2, and NoGo-P3 provide a promising neurophysiological profile for trauma-related executive function impairments, potentially reflecting a shift from proactive to reactive control mechanisms (Kropotov et al., 2011; Vuillier et al., 2016). Additionally, behavioural results like increased commission errors suggest impulsivity and impaired response inhibition in trauma-exposed individuals (Shucard et al., 2008; Wu et al., 2010). ERP abnormalities in PTSD are considered acquired rather than preexisting (Metzger et al., 2009), with frontoparietal components proposed as biomarkers, reflecting attention dysregulation and hyperactivation (Butt et al., 2019).

While adult studies provide foundational insights into these neural changes, the impact of CCT on ERPs during adolescence remains unclear. Developmental differences in neural plasticity, prefrontal-amygdala connectivity, and inhibitory control suggest distinct trajectories for trauma-related ERP changes in youth (Cisler & Herringa, 2021). Limited findings indicate that early adversity, such as institutional care, is associated with reduced CNV and P3 amplitudes, reflecting deficiencies in preparatory and attentional processing (McDermott et al., 2012). However, the lack of adolescent-specific studies, often due to methodological inconsistencies and small sample sizes, leaves critical gaps in understanding the neurophysiological correlates of trauma during this developmental stage (Javanbakht et al., 2011).

The cued GO/NOGO paradigm has proven particularly useful in investigating executive function deficits in trauma-exposed populations. Enhanced motor readiness, reflected in increased CNV amplitudes (Kropotov et al., 2011; Vuillier et al., 2016), and diminished NoGo-N2 and NoGo-P3 responses (Kropotov et al., 2011; Ponomarev & Kropotov, 2024) indicate a reliance on reactive rather than proactive control mechanisms. Similarly, trauma-exposed adolescents show poorer inhibitory control and heightened impulsivity compared to non-traumatized peers, with these behavioural deficits mirrored in ERP measures such as diminished P3 amplitudes and delayed reaction times (Bruce & Kim, 2022).

This study aims to address existing gaps by investigating the neurophysiological markers of cognitive control in adolescents with CCT through the cued GO/NOGO paradigm. It will specifically examine CNV, NoGo-N2, and NoGo-P3 amplitudes, along with behavioural measures such as omission and commission errors and reaction time, to propose an electrophysiological phenotype associated with CCT. By examining these markers, the study aims to advance the understanding of trauma-related neurodevelopmental disruptions, enabling timely interventions to prevent PTSD and reduce the progression of maladaptive responses in this vulnerable population.

2. Methods

2.1. Participants

Twenty adolescents (ages 11-16) with CCT (12 females) performed a cued GO/NOGO task while a 19-channel electroencephalography (EEG) was recorded. For comparing the recorded ERPs and behavioural parameters in the task with the normative data, the corresponding data from 40 healthy subjects of similar age and gender were selected from the HBI normative database recorded by a similar hardware and software equipment (Mitsar EEG amplifier and WinEEG ver 3.13.26, https://www.mitsar-eeg.ru/). The study was conducted in accordance with the Declaration of Helsinki, and all participants provided informed consent. The whole procedure of the participant's selection, ethical approval, and neuropsychological assessment was preregistered in an open registry (https://osf.io/m3fdv/?view_only=cc6d743714504749a85cc093c85039cc).

2.2. Tasks

A cued GO/NOGO task, described in several papers (Brunner et al., 2015; Kropotov et al., 2011; Kropotov & Ponomarev, 2009) was employed in the present study. In the task, participants were presented with various images of animals (*a*), plants (*p*), and humans (h) randomly paired together. The pairs were presented with equal probabilities, resulting in four types of pairs: *a-a, a-p, p–p, and p-h*. The image of humans was accompanied by a novel sound to generate an orienting response. The duration of stimuli was 100 ms, the inter-stimulus interval in pairs was 1s, and the trial-trial interval was 3s. Participants were instructed to press a button with their right index finger in response *a-a* trials. Both speed and precision were equally emphasized in the instructions. The images were selected from school textbooks to ensure that the overall luminance and the image sizes of the stimuli were approximately equal. In total, 400 trials were presented, with 100 trials in each trial category. Participants practiced the task with around 20-30 trials before the recording started, and they rested for a few minutes after completing the first 200 trials. Participants sat upright in a comfortable chair, facing a computer screen. Stimuli were presented on 17-inch

CRT computer screen positioned 1.5 m in front of the participants, occupying ~3.8° of the visual field. Psytask was used to present visual stimuli and record responses (Kropotov & Ponomarev, 2009).

2.3. Data recording

EEG was recorded from 19 scalp sites (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, O2) according to the 10-20-system (Jasper, 1958). The signals were bandpass-filtered between direct current (DC) and 50 Hz and digitized at a rate of 250 samples per second, per channel. The EEG was online referenced to linked ears using a 19-channel PC-controlled electroencephalographic system, the 'Mitsar-202' (CE 0537), manufactured by Mitsar Co., Ltd (https://mitsar-eeg.com/ 24.01.2023). Electrodes were applied using the Medcap EEG Cap (Spes Medica), featuring embedded silver/silver chloride electrodes in the international 10–20 system, similar to the Electro-Cap used in the HBI Database recordings (www.hbimed.com/en/hbi-database). Further, the EEG was off-line refiltered to a frequency band of 0.5–50 Hz (1st order 0.5 Hz high-pass and 2nd order 50 Hz low-pass Butterworth IIR filters). All ERP analysis procedures were performed by WinEEG software.

Subject responses were recorded on separate channels. A response to GO stimulus was considered correct if it was made to appropriate stimulus and initiated within a 100–1000 ms time interval. Responses to NOGO and other irrelevant trials were considered correct if no button presses were detected during these trials. Average response latency and variance were calculated individually for each subject's correct trials. Omission errors (failures to respond to GO trials) and commission errors (failures to suppress a response to NOGO trials) were computed for each subject separately.

2.4. ERP computing

First, eyeblink artefacts were corrected using independent component analysis (ICA) by zeroing the activation curves of one individual independent component associated with eye blinks (Jung et al., 2000; Tereshchenko et al., 2009). Subsequently, epochs with excessive EEG amplitude and/or excessive frequency activity were excluded from the analysis (Kropotov & Ponomarev, 2009). Empirically selected exclusion thresholds were set as follows: (1) 100 μV for non-filtered EEG, (2) 50 μV – for slow waves in the 0–1 Hz band, (3) 35 μV – for fast waves in the 20–35 Hz band (Kropotov & Ponomarev, 2009). Channels were not deleted, all analysed data contained 19-channel EEG signals. Individual ERPs were obtained by averaging all correct trials separately the corresponding task conditions. The time interval for computing ERPs included 300 ms before the first stimulus in pair (S1) and 1100 ms after the second stimulus in pair (S2). The baseline was not corrected. To maintain a high signal-to-noise ratio, participants with fewer than 30 trials for any trial category were excluded from analysis (Table 1).

Table 1. The percentage of the average number of artefact-free trials across the groups and trial categories. Recall that number of trials in each category was 100.

Category	CCT	HC
GO	56 ± 23	67 ± 24
NOGO	80 ± 18	82 ± 82

2.5 Group decomposition of ERPs into hidden components

In this study, we employed a blind source separation method for decomposing group ERPs into hidden components (Kropotov & Ponomarev, 2015). Specifically, a joint diagonalization method for covariance matrixes was applied to the large collection of ERPs of age group 11–16 year presented in the HBI database in the tasks (Ponomarev & Kropotov, 2024). In more detail, the procedure is described in 'Group decomposition of ERPs into hidden components' of the Supplementary Materials.

2.6. Statistical analysis

Analysis of behavioural data. Omission and commission errors, as well as reaction times, were evaluated using the nonparametric Mann–Whitney test.

Analysis of raw ERPs. Prior to assessing hidden components statistically, a preliminary analysis of ERPs was conducted to demonstrate the statistical significance of ERPs across task conditions. ERPs and ERP difference waves were compared using a cluster-based permutation test (Marcu, Szekely-Copîndean, et al., 2024) implemented in WinEEG (v. 3.13.26). This approach addressed the issue of multiple comparisons by clustering the data based on temporal and spatial proximity.

While similar to the procedure implemented in the FieldTrip MATLAB toolbox for M/EEG analysis (freely available at http://fieldtrip.fcdonders.nl/, 24/01/2023; Oostenveld et al., 2011), this analysis differed in several aspects: (1) Wilcoxon signed-rank nonparametric tests were used for comparing ERP waveforms under different conditions instead of dependent sample t-tests, (2) a normal approximation for the Wilcoxon signed rank test and the sum z-score within a cluster were utilized for the cluster-level statistics instead of the sum of the t-values. Nonparametric statistics were preferred due to their robustness against outliers.

In particular, two comparisons were conducted using the whole head, cluster-based, Wilcoxon signed rank test for GO and NOGO separately. Electrodes located at the border of the electrode grid were considered to have 3 neighbours, while others had 4 neighbours. Clusters were defined as samples with statistics corresponding to a p-value smaller than a critical value ($p < .01$) and were based on temporal and spatial adjacency. Cluster-level statistics were calculated by summing the z-score within every cluster. Subsequently, 10,000 data permutations were performed by shuffling the condition labels, and for each permutation, clustering and cluster-level statistics were recalculated.

The cluster-corrected threshold was determined as the permutation distribution of the maximum cluster-level statistics (Maris & Oostenveld, 2007). The assessment of ERP components latency was not conducted in this investigation because the tools employed were insufficient for precise measurement. As a result, no comparative analyses pertaining to this parameter were performed.

Analysis of hidden components. The cluster-based analysis described above was applied to each component separately.

3. Results

3.1. Behavioural data

The behavioural results of participants in the cued GO/NOGO task revealed significant differences between adolescents with CCT and healthy controls (HC). The CCT group exhibited a higher rate of omission errors in the Go condition (18.2% ± 20.0%) compared to the HC group (4.3% ± 9.1%, $p < .002$). Additionally, reaction times in the Go condition were significantly shorter in the CCT group (313.8 ms ± 79.4) relative to the HC group (442.0 ms ± 87.1, $p < .00001$). The calculated Cohen's d for reaction time is −1.51, indicating a very large effect size for the difference between the CCT and HC groups. Additionally, Cohen's d for omission errors is 1.02, suggesting a large effect size. No significant differences were observed in commission errors between groups during NOGO trials. Although it is well documented that CCT affects impulsivity (Kim & Choi, 2020) the present study doesn't reveal any statistically significant group difference in commission errors. This fact might reflect a low sensitivity of this behavioural parameter for reflecting impulsivity as a behavioural trait. Indeed, in our previous study no differences between commission errors in patients with ADHD, schizophrenia, and healthy controls performed the same cued GO/NOGO tasks were found (Kropotov et al., 2019).

Table 2 provides descriptive statistics of the participants' behaviour for each trial type and each group.

3.2. Electrophysiological data – ERP comparisons

Grand averaged ERPs for the HC group are presented in Figure 1. Three conditions are depicted: GO (a-a) trials (green lines), NOGO trials (a-p) (red lines) in comparison to Ignore (p–p) trials (black lines). Canonical ERP waves such as Visual Negativity (VN) (first described in Di Russo et al., 2019), CNV (first described by Walter, 1967), P3-GO, N2 NOGO, and P3-NOGO (Kropotov & Ponomarev, 2009) are marked by black arrows. The topographies of the waves at the marked latencies are presented at the bottom. The peak latencies and time windows of the ERP waves are presented in Table 3.

Grand-averaged ERPs revealed distinct deviations in neural processing between the CCT and HC groups across task conditions (Figure 2) in both cognitive control components:

Proactive Control (Cue-Related Components): The CCT group exhibited significantly reduced amplitudes of the contingent negative variation (CNV) component during the preparatory interval compared to the HC group. This reduction suggests deficits in proactive control, reflecting impaired anticipatory attention and motor preparation (Kimble et al., 2004).

Reactive Control (NOGO Components): The amplitude of the NoGo-N2 component was significantly lower in the CCT group compared to the HC group, indicating reduced conflict monitoring

Table 2. Behavioural parameters in GO and NOGO conditions and their comparison by means of non-parametric Mann–Whitney U test between the groups: participants with CCT (CCT group) and healthy controls (HC group). The values of p and U are presented only for the statistically significant differences between the groups.

Trial name	Number of participants	Omission errors in %	Commission errors in %	Reaction time in ms	Standard error of RT in ms
CCT Group					
GO	20	18.2 ± 20.0	–	313.8 ± 79.4	13.2 ± 8.1
NOGO	20	–	3.3 ± 5.2	–	–
HC Group					
GO	40	4.3 ± 9.1	–	442 ± 87.1	11.3 ± 4.0
NOGO	40	–	1.9 ± 4.3	–	–
Difference between CCT and HC (p-value/U)					
GO	-	<0.002/593	-	<0.00001/678	NS
NOGO	-	-	NS	-	-

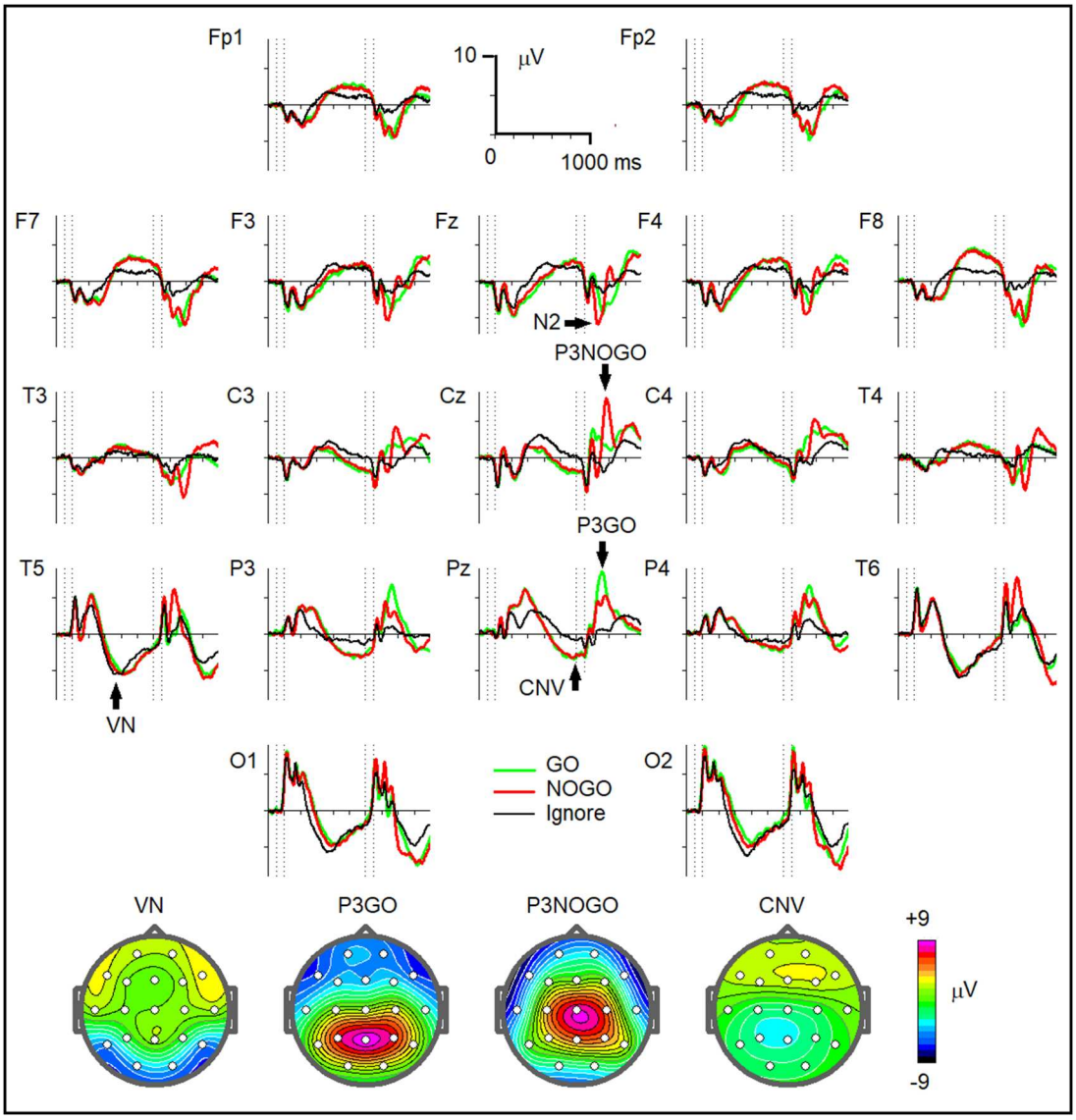

Figure 1. Grand averaged 19-channel ERPs for the HC group for GO (green lines) and NOGO trials (red) in comparison to Ignore trials (black lines). Canonical ERP waves are marked by black arrows: Visual Negativity (VN), Contingent Negative Variation (CNV), P3 GO and P3 NOGO. The topographies of the waves at the marked latencies are presented at the bottom.

capabilities (Kropotov et al., 2011). NoGo-P3 Amplitudes were significantly diminished in the CCT group, highlighting impairments in late-stage inhibitory control and the ability to implement corrective responses.

Exploratory Finding: An additional exploratory analysis revealed that the VN component, an early-stage processing wave associated with the predictions and prediction errors in the visual domain (Häger et al., 2024) exhibited reduced amplitudes in the CCT group across both task conditions (Cue and NoGo). This suggests that trauma exposure may affect sensory integration and initial attentional allocation based on prior expectations. A recent review supports this suggestion by demonstrating that trauma exposure and resultant PTSD symptoms are indeed associated with altered structure and function of the ventral visual stream (Harnett et al., 2025). New models of PTSD propose that dysregulation of sensory (including visual) systems may influence fear learning in threat-related neurocircuitry and consequently symptom expression in PTSD (Harnett et al., 2025; Kearney & Lanius, 2022).

Table 3. Peak latencies and time windows measured at 50% of peak amplitude of the canonical ERP waves in the cued GO/NOGO task in a group of healthy subjects. Localization of the components is indicated by arrows in Figure 1.

ERP wave	Peak latency in ms	Peak amplitude in µV	Time window in ms
VN	700	5.1	540–950
CNV	-	1.8	0–200
P3 GO	310	8.6	240–390
N2 NOGO	250	5.9	220–320
P3 NOGO	360	8.2	320–430

The results of the most significant deviations from the reference of CCT group are presented in Figure 2. Left of Figure 2: represents ERPs to Cue stimuli (marked by arrows) in the interval prior the second stimulus for CCT group (green lines), for HC group

(grey lines) and the difference waves for CCT-HC (blue lines) with indicators of the levels of statistical significance of the difference below the curves. Right of Figure 2 represents ERPs to NOGO stimuli (marked by arrows) in the interval after the second stimulus for CCT group (red lines), for HC group (grey lines) and the difference waves for CCT-HC (blue lines) with indicators of the levels of statistical significance of the difference below the curves. The maps below are taken at the latencies (indicated below the maps) of the largest deviations from the references.

Five **latent components** with large were extracted from the group ERPs of the HBI database for age 11–16 (see 'Split-half reliability of the latent components' of the Supplementary material). Figure 3 illustrates the significant differences (grey areas) identified between the CCT and HC groups in the components associated with early VN, CNV and P3 NoGo waves, reflecting hypothetical operations of visual prediction, motor inhibition, and conflict detection/monitoring.

The C1 (associated with VN wave) component, which is characterized by a temporal-occipital topography, exhibited significant attenuation in the CCT group following both S1 (the cue) and S2 (NOGO stimuli) compared to the HC group, suggesting impaired visual prediction processing (Häger et al., 2024) in the CCT group.

The C5 P3 (associated with early NoGo part of NOGO wave) component, with a central positive topography and generated in the supplementary motor cortex, was also significantly reduced in the CCT group. The observed attenuation in the CCT group highlights deficits in inhibitory control mechanisms, including the ability to withhold inappropriate motor responses (Kropotov, 2016). In addition, the parietal component (C2), associated with the CNV wave, exhibited significant attenuation in the CCT group during the cue (S1) condition. This component, with a posterior topography centred at the Pz electrode, may indicate compromised action preparation operations (Brunner et al., 2015).

Figure 4 demonstrates discriminability between the HC and CCT groups on the basis of amplitude of the two selected components from Figure 3. sLORETA images (Pascual-Marqui, 2002) are presented together with the maps. According to sLORETA, the C1 (VN) component is generated in Brodmann area 20 (Fusiform Gyrus), the C5 (NoGo-P3, early) is generated in Brodman area 6 (Superior Frontal Gyrus). On the plot: *X*-axis – an amplitude of an individual C1 component averaged over time interval indicated by grey area, *Y*-axis – an amplitude of an individual C5 component averaged over time interval indicated by grey area.

4. Discussions

The current study investigated electrophysiological markers of cognitive control in adolescents with complex childhood trauma (CCT) using a cued GO/NOGO task. The findings reveal significant deficits in both proactive and reactive control mechanisms and suggest broader disruptions in sensory prediction processes. These results contribute to the growing body of evidence on trauma-related neurodevelopmental impairments and their potential underlying neural mechanisms.

4.1. Proactive and reactive control impairments in CCT

The reduced CNV amplitudes in the CCT group highlight deficits in proactive control, particularly in anticipatory attention and motor preparation (Liebrand et al., 2017). These findings align with prior research suggesting that trauma disrupts frontoparietal circuits critical for planning and goal-directed behaviour (Cisler & Herringa, 2021; McDermott et al., 2012). Proactive control deficits are of particular concern during adolescence, a period characterized by heightened neuroplasticity and ongoing maturation of the prefrontal cortex (Milbocker et al., 2021), and literature continues to face challenges in detecting developmental differences in this process (Vuillier et al., 2016). Impairments in these mechanisms may hinder the ability to predict and prepare for environmental demands, increasing susceptibility to maladaptive coping strategies. Additionally, given that the adolescent brain is still developing capacities for risk assessment and hazard identification, deficits in proactive control may further exacerbate vulnerability to physical harm, including accidental injury or death – concerns that extend beyond psychological outcomes and carry important real-world implications for adolescents with CCT.

Reactive control, reflected in the NoGo components, also showed significant impairments in the CCT group. The reduced amplitudes of the NoGo-N2 and NoGo-P3 components indicate challenges in conflict monitoring and response inhibition, consistent with findings in PTSD and other trauma-related conditions (S. Kim et al., 2018; Min et al., 2020). These reactive control deficits may result in increased impulsivity and difficulties suppressing inappropriate responses, as evidenced by the higher commission error rates observed behaviourally. These impairments are likely mediated by disruptions in the brain circuitry playing a critical role in managing reactive responses to threats and other environmental stimuli (Carrion et al., 2008).

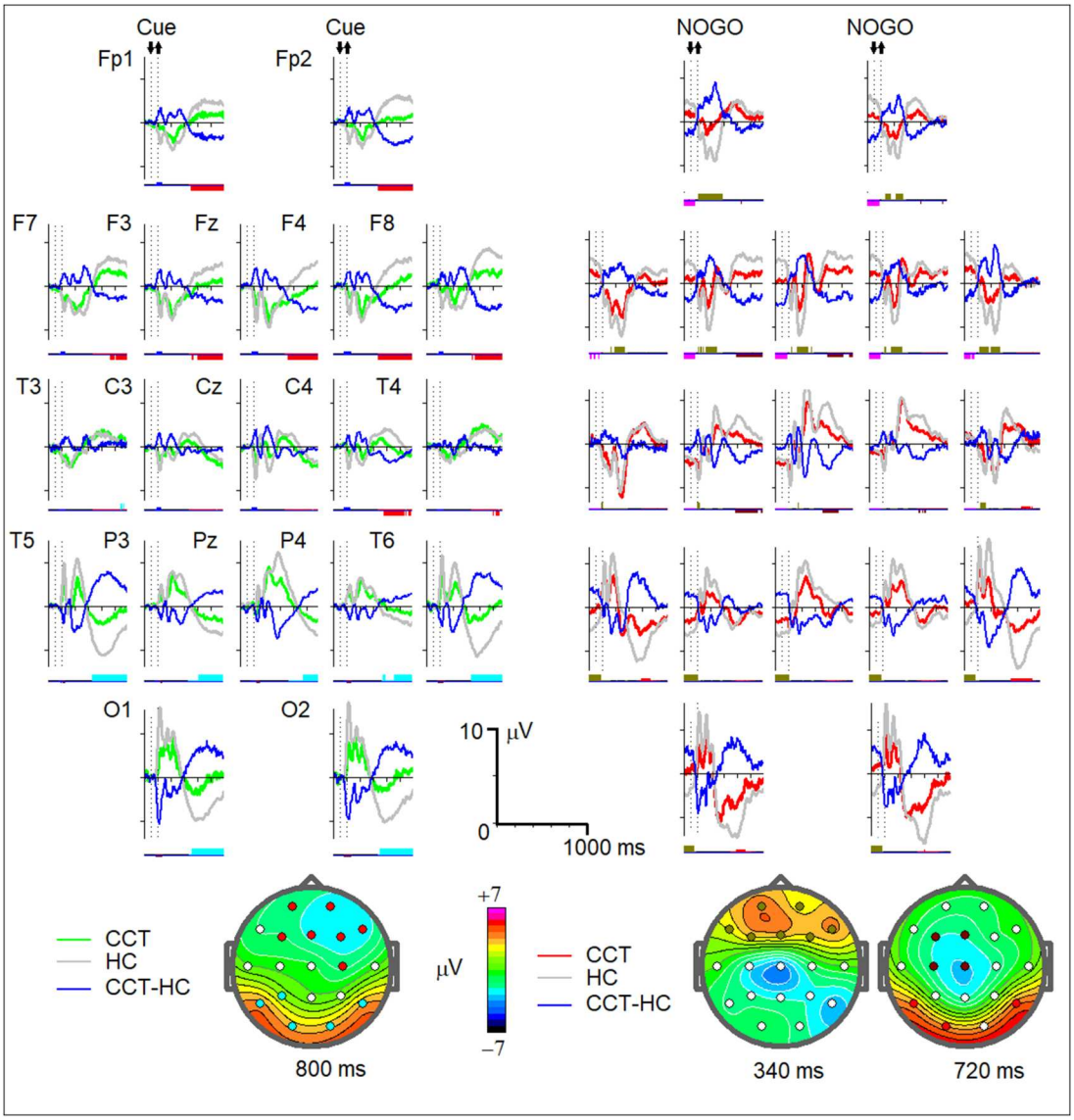

Figure 2. Grand averaged ERPs for CCT, HC groups, and CCT-HC difference waves. Left: ERPs to Cue stimuli (marked by arrows) in the interval prior the second stimulus for CCT group (green lines), for HC group (grey lines) and the difference waves for CCT-HC (blue lines) with indicators of the levels of statistical significance of the difference below the curves. Right: ERPs to NOGO stimuli (marked by arrows) in the interval after the second stimulus for CCT group (red lines), for HC group (grey lines) and the difference waves for CCT-HC (blue lines) with indicators of the levels of statistical significance of the difference below the curve. The maps below are taken at the latencies (indicated below the maps) of the largest deviations from the references.

4.2. Impairments in predictive processing

An exploratory analysis revealed significantly reduced amplitudes of the VN component in the CCT group across both Cue and NoGo conditions. VN is associated with early sensory processing and prediction error detection, a critical aspect of the brain's predictive coding framework (Friston, 2010; Kropotov, 2016b). Within this framework, the brain continuously generates and refines predictions about incoming sensory information, updating these predictions in response to discrepancies or errors. The VN reflects the brain's ability to detect discrepancies between expected and actual visual sensory inputs, serving as a neural marker of prediction error. Reduced VN amplitudes in the CCT group suggest a diminished capacity to detect and adapt to prediction errors, potentially impairing attentional allocation and increasing vulnerability to attentional biases. This aligns with evidence from PTSD populations, where disrupted prediction error signalling has been linked to reduced capacity to filter irrelevant information or adapt to changing environments (Leone et al., 2022; Veltmeyer et al., 2005; Zukerman et al., 2018).

Furthermore, the reduced value of the VN in the CCT group may suggest a neural basis for broader disruptions in predictive coding, indicating a potential transdiagnostic mechanism that might underlie various psychopathologies. This finding aligns with the observation of overlapping symptoms among different psychiatric disorders, such as borderline personality disorder, depression, and anxiety, in individuals exposed to

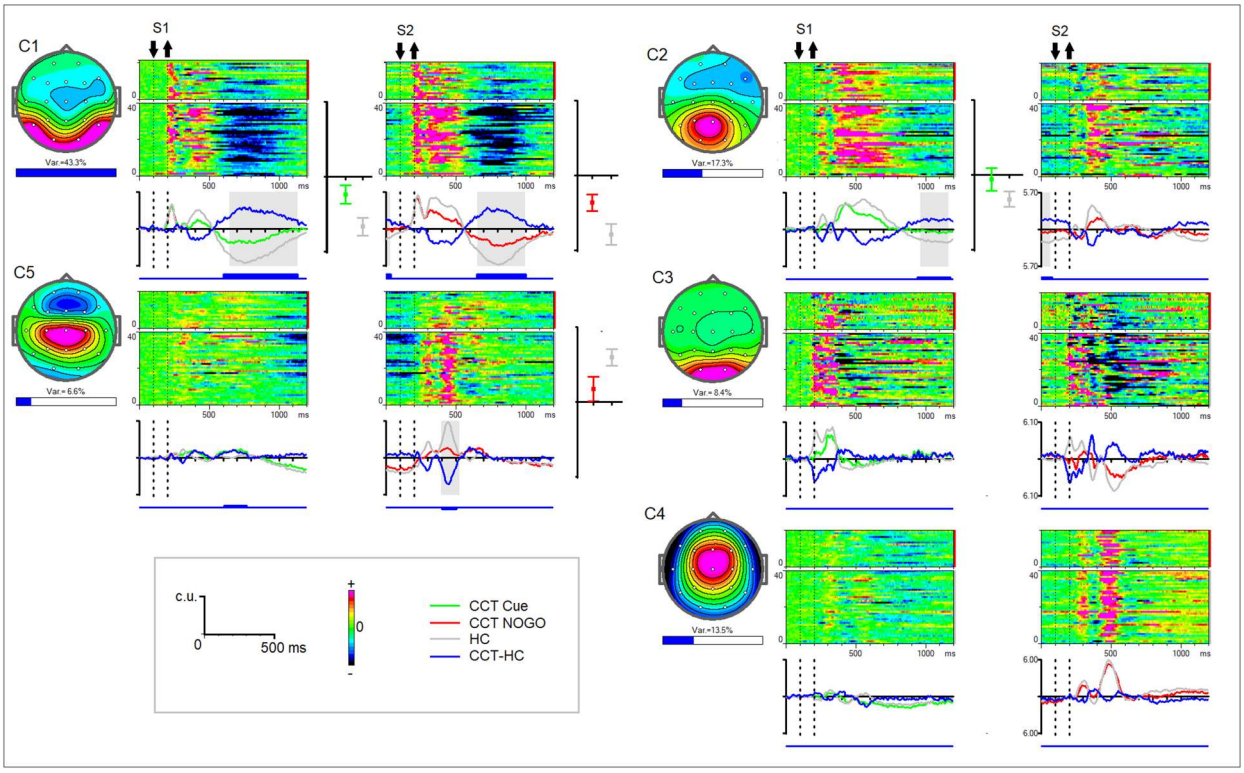

Figure 3. Latent ERP components for CCT and HC groups. For each component: left – topography with the relative power of the component below; middle – response of the component to the cue (S1) where the colour raster for CCT group is presented at the top and marked by red colour to the right, the colour raster for HC group is presented at the bottom and marked by grey colour at the right, and where every line on the raster represents a colour coded component for a single participant, below the raster – grand averaged time dynamics of the component in conventional units, to the right of the raster – mean values and 95% confidence intervals averaged over the time windows corresponding to the statistically significant differences between the groups and depicted on the graphs with gray colour, right column – response of the component NOGO stimuli (S2). In grey box – time, mapping scales, and colours for the groups and group differences.

CCT. Predictive processing in trauma highlights how the brain's generative model, which continuously predicts sensory inputs and minimizes errors, becomes dysregulated following traumatic experiences. For example, in PTSD, the brain develops hyperprecise threat priors, meaning that predictions of danger are excessively rigid and persist even in the absence of actual threats (Linson & Friston, 2019). In CCT, the maladaptive predictive processing seems to take the opposite direction, as indicated by reduced amplitude in sensory-related components. Both responses fail to properly weight prediction errors, preventing the brain from updating its internal model (Greco et al., 2024) in response to new, non-threatening sensory information (Nave et al., 2020). These impairments may underlie broader deficits in integrating sensory information with prior experiences, contributing to the heightened sensitivity and maladaptive responses observed in trauma-affected individuals. These findings align with research suggesting that trauma may occupy cognitive resources, limiting the brain's capacity to process new information. Anders et al. (2015) reported reduced mPFC modulation in trauma-exposed individuals, indicating persistent engagement in fear-related processing, limiting the capacity to integrate and respond to new stimuli. Similarly, Glazebrook et al. (2023) found that, in contrast to those developing PTSD, individuals with higher post-traumatic growth showed greater neural flexibility, enabling more effective filtering of irrelevant information. The increased omission errors in our study may reflect similar impaired attention allocation in trauma-exposed adolescents.

4.3. Implications of latent component analysis

The analysis of latent components showed that three out of five components, corresponding to three of the four ERP components that were weaker in the CCT group, exhibited significant differences between the CCT and HC groups. The smaller number of significantly altered latent components in the CCT group, compared to the number of impaired ERP components, indicates that despite the neurophysiological impact of CCT, some cognitive control mechanisms remain relatively intact during adolescence. This selective impairment, as highlighted in a previously published case study on adolescent siblings with CCT (Marcu, Szekely-Copîndean, et al., 2024), may reflect the heightened neuroplasticity and ongoing maturation of the brain during this developmental period, particularly

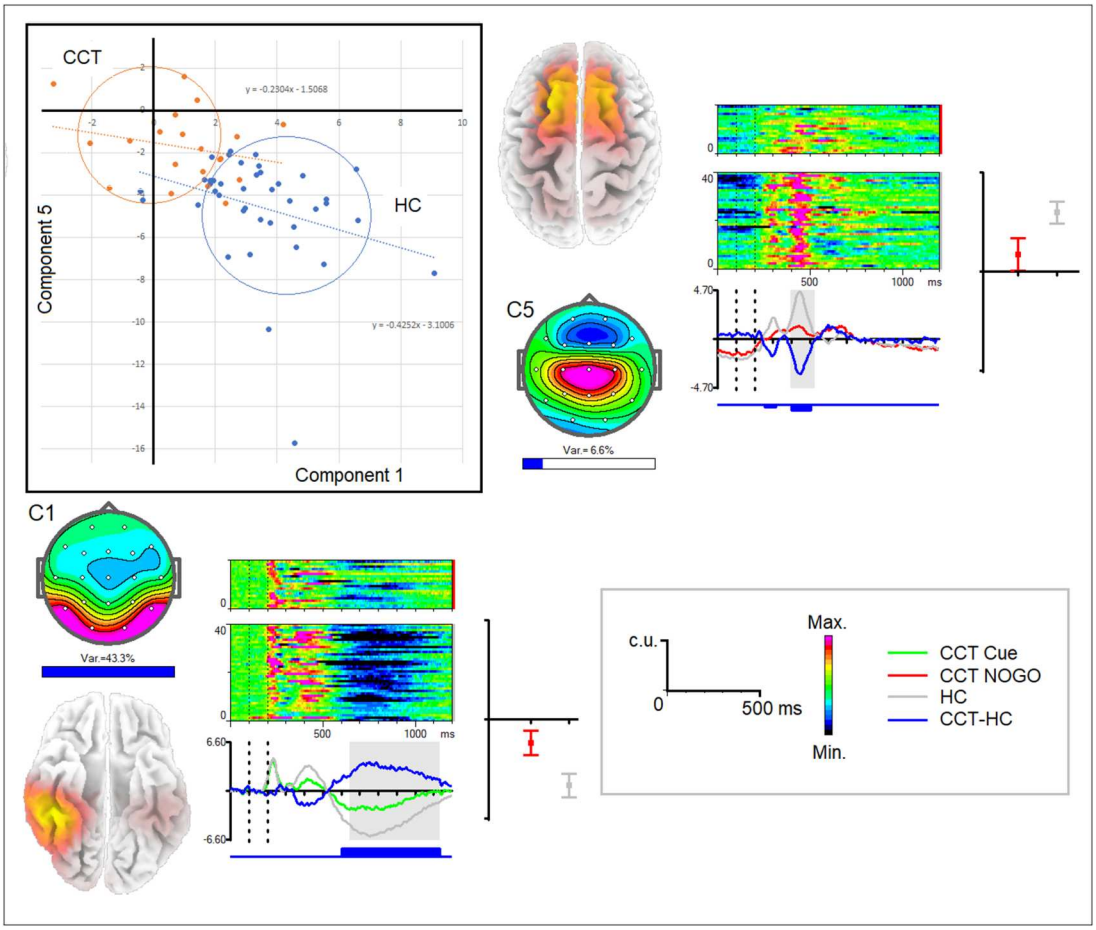

Figure 4. Discrimination of CCT group from HC group in two dimensional space of the amplitudes of the two latent components. The two latent components are as in Figure 3. sLORETA images are presented in addition to the topography of the components. On the plot: *X*-axis – averaged amplitude of the component corresponding to P3 NOGO wave (marked by grey colour on the curve) and generated in Brodmann area 6, Superior Frontal Gyrus, Frontal Lobe ($X = -15$, $Y = 20$, $Z = 60$), MNI coords; *Y*-axis – averaged amplitude of the component corresponding to VN wave (marked by grey colour on the curve) and generated Brodman area 20, Fusiform gyrus, temporal lobe ($X = 45$, $Y = -35$, $Z = -25$) (MNI coords); blue – dots HC participants, gold – CCT patients.

in regions such as the prefrontal cortex and related networks involved in cognitive control. These findings reinforce the idea that adolescence is a critical period where targeted interventions could potentially reverse or mitigate some of the trauma-related neural disruptions.

Furthermore, the latent component analysis revealed distinct patterns of neural activity that effectively discriminated the CCT group from healthy controls in two-dimensional space. Specifically, differences in the latent components associated with VN and NoGo-P3 show potential as biomarkers for trauma-related cognitive control impairments. VN reflects anticipatory brain activity that integrates sensory inputs to prepare for future stimuli, which makes it a key component for predictive coding (Spratling, 2008). The significant reduction in amplitude of the latent component associated with VN, observed in the CCT group, suggests a hypothetically impaired predictive processing, that might be further reflected in a diminished capacity to anticipate and adaptively prepare for task demands. The action suppression (NoGo-P3 associated) component reflects the ability to inhibit prepotent responses and correlates with the neuropsychological domain of energization (Kropotov, 2016), together with parameters such as reaction time (Brunner et al., 2015). The observed attenuation of this component in the CCT group highlights deficits in inhibitory control mechanisms, including the ability to withhold inappropriate motor responses. These findings align with behavioural markers of increased impulsivity and reduced response inhibition in trauma-exposed individuals (Johnson et al., 2013; S. Kim et al., 2018). The sLORETA analysis localized these two components to Brodmann areas associated with sensory integration (BA20, fusiform gyrus) and inhibitory control (BA6, superior frontal gyrus), further suggesting vulnerable developmental areas that might be addressed in intervention formulations.

4.4. Temporal-Posterior regions in trauma

The VN component and its associated independent component (IC) topography can be corroborated with prior research (Marcu, Băcilă, et al., 2024),

which identified attenuated alpha power in temporal-posterior regions in adolescents with complex childhood trauma (CCT), suggesting a specific neural vulnerability in the **temporal-posterior** regions, particularly around the T5/T6 electrode sites. Alpha rhythms, which are crucial for temporal coordination (Schoffelen et al., 2024) and sensory gating (Kizuk & Mathewson, 2017) in the brain, are often linked to the readiness of neural circuits to process incoming information (Sadaghiani & Kleinschmidt, 2016). The diminished alpha power previously observed in trauma-exposed adolescents (Marcu, Băcilă, et al., 2024) aligns with the current study's findings of reduced VN amplitude, further supporting the hypothesis that trauma disrupts the neural substrates responsible for integrating and predicting sensory information. These findings collectively highlight the temporal-posterior regions as a critical site of vulnerability in adolescents with CCT, suggesting that interventions aimed at regulating neural activity or connectivity in these areas, potentially through neurofeedback or other neuroregulation modalities, could address the underlying vulnerabilities and improve cognitive outcomes in trauma-exposed youth.

4.5. Limitations and future directions

This study represents a preliminary investigation, and while the findings are promising, they should be interpreted with caution until validated in larger, more diverse samples. The study sample size (20 CCT participants and 40 healthy controls), although consistent with other exploratory studies in this area, is rather small, which limits its statistical power and the ability to detect small but potentially meaningful effects. Additionally, this limitation might impact the generalizability of the findings to broader populations and can further lead to discrepancies in results with similar research (Larson, 2020; Larson & Carbine, 2017). Future studies with larger cohorts are needed to validate current results and explore potential subgroup differences within the CCT population. Future studies could add insights into the longitudinal trajectories of the founded deficits to better understand their developmental course and potential for intervention. Investigating the relationship between the ERP markers and specific clinical outcomes, such as PTSD severity, and further functional impairments, would also be valuable. The study lacks validation against established neural correlates (e.g. fMRI) or behavioural measures. The lack of component latency analysis in this study should be acknowledged as a limitation. Without replication or cross-method consistency, interpreting utility of latent components as biomarkers remains premature. In future research, using a larger sample of CCT patients, we plan to conduct a latency analysis similar to that performed in our previous study (Brunner et al., 2013).

5. Conclusions

This study highlights significant impairments in proactive and reactive control mechanisms and exploratory evidence of disrupted predictive processing in adolescents with CCT. These findings emphasize the importance of addressing trauma-related neural deficits during adolescence, a critical period for intervention, to enhance recovery and foster resilience. Among observed ERP markers, NoGo-P3 and VN, showed promising utility in identifying trauma-related disruptions and monitoring intervention outcomes. Given that CCT is a significant risk factor for the development of PTSD, these findings provide important insights into the shared neurobiological pathways that link early trauma to long-term psychopathology. By using ERP markers like those of cognitive control, future research can deepen our understanding of how CCT contributes to PTSD vulnerability. Additionally, the study's findings indicate that latent ERP components may offer a deeper understanding of the neural substrates impacted by CCT. This could aid in developing more accurate diagnostic tools for developmental samples that are at risk of PTSD due to CCT.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Data availability statement

The data supporting the findings of this study can be accessed upon request made to the corresponding author.

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