



The relationship between childhood stress and distinct stages of dynamic behavior monitoring in adults: neural and behavioral correlates

Jianhui Wu,^{1,2} Yutong Liu,¹ Huihua Fang,¹ Shaozheng Qin,³ Nils Kohn,⁴ and Hongxia Duan^{1,2,4}

¹Center for Brain Disorder and Cognitive Science, Shenzhen University, Shenzhen 518060, China, ²Shenzhen Institute of Neuroscience, Shenzhen 518057, China, ³State Key Laboratory of Cognitive Neuroscience and Learning, McGovern Institute for Brain Research at Beijing Normal University, Beijing, China, and ⁴Donders Institute for Brain, Cognition and Behavior, Radboud University Medical Center, Nijmegen 6525 EN, The Netherlands

Correspondence should be addressed to Hongxia Duan, Center for Brain Disorder and Cognitive Science, Shenzhen University, 3888 Nanshan Ave, Nanshan, Shenzhen, Guangdong 518060, China. E-mail: duanhx@szu.edu.cn.

Abstract

Childhood adversity is a major risk factor for emotional and cognitive disorders later in adulthood. Behavior monitoring, one of the most important components of cognitive control, plays a crucial role in flexible interaction with the environment. Here, we test a novel conceptual model discriminating between two distinct dimensions of childhood adversity (i.e. deprivation and threat) and examine their relations to dynamic stages of behavior monitoring. Sixty young healthy adults participated in this study using event-related potentials and the dynamic stages of behavior monitoring including response inhibition, error detection and post-error adjustments were investigated in a classical Go/NoGo task. Multiple regression analyses revealed that participants with higher severity of childhood adversity recruited more controlled attention, as indicated by larger (more negative) conflict detection-related NoGo-N2 amplitudes and larger (more negative) error detection-related error-related negativity amplitudes. Higher severity of childhood abuse (an indicator of threat) was related to smaller (less positive) error appraisal-related error positivity amplitudes on the neural level and subsequently lower post-error accuracy on the behavioral level. These results suggested that prefrontal-supported controlled attention is influenced by universal adversity in childhood while the error-related behavioral adjustment is mainly affected by childhood abuse, indicating the dimensions of deprivation and threat are at least partially distinct.

Key words: childhood adversity; neglect; abuse; behavior monitoring; ERP

Introduction

Childhood adversity refers to a set of negative events children experience before they reach adulthood, ranging from

emotional, physical and sexual abuse to parental neglect, early life foster care and low socioeconomic status (McLaughlin *et al.*, 2014). Evidence from numerous prospective and retrospective studies demonstrates that childhood adversity has been linked

Received: 3 June 2020; Revised: 23 February 2021; Accepted: 8 April 2021

© The Author(s) 2021. Published by Oxford University Press.

This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs licence (<https://creativecommons.org/licenses/by-nc-nd/4.0/>), which permits non-commercial reproduction and distribution of the work, in any medium, provided the original work is not altered or transformed in any way, and that the work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

to poor physical and mental health outcomes (Clark et al., 2010; Shonkoff et al., 2012; Hughes et al., 2017). For example, victims of childhood maltreatment expressed a higher prevalence of substance use, affective and personality disorders as well as poorer treatment response among psychiatric patients (McLaughlin et al., 2012; Teicher and Samson, 2013). In past decades, there has been considerable interest in unraveling the underlying mechanisms linking childhood adversity to psychopathology. One factor that has been proposed to underlie these psychopathological consequences of childhood adversity is impaired executive functioning and the associated compromised neural integrity and function (Teicher, 2000; Teicher and Samson, 2013, 2016; Teicher et al., 2016). Notably, behavioral studies with tasks tapping cognitive control showed that early exposure to deprivation measured as poverty or institutional care was associated with deficits in behavior monitoring (Farah et al., 2006; Noble et al., 2007; Eigsti et al., 2011). For example, the seminal work from the Bucharest Early Intervention Project (BEIP) found that compared to controls, children who experienced prolonged institutional rearing committed more errors on response inhibition (Lamm et al., 2018) and less behavioral adjustment after an error (Buzzell et al., 2020). These deficits in behavior monitoring further mediated the relationship between early institutionalization and psychopathological symptoms (Tibu et al., 2016; Troller-Renfree et al., 2016).

Behavior monitoring or performance monitoring, the ability to monitor ongoing performance and make an adjustment by recruiting increased cognitive control when necessary, is crucial for carrying out higher-order executive functions. Behavior monitoring involves a set of highly interacting yet dissociable processes, characterized by inhibition of improper response, error detection when inhibition fails and behavioral adjustment for subsequent trials. Several key prefrontal regions including anterior cingulate cortex (ACC) and dorsal lateral prefrontal cortex (dlPFC) have been demonstrated to implement these behavior-monitoring processes (Botvinick et al., 2001; Kerns, 2004). From childhood to early adulthood, there is substantial improvement in behavior monitoring, which is supported by progressive maturation of frontal-cingulo-striatal circuitry over development (Rubia et al., 2007; Velanova et al., 2008). However, the development of this functional circuit might be altered by childhood stress even in high-functioning maltreated survivors (Marsh et al., 2008). Failures of behavior monitoring are associated with both lower survival and task success and a higher risk of psychiatric and personality disorders (Swann et al., 2002).

Event-related potentials (ERPs), with their high time resolution in milliseconds, are suitable to measure the dynamic sub-processes of behavior monitoring. In the classical Go/NoGo task, participants have to exercise inhibitory control on NoGo trials by withholding a prepotent response to Go trials. Otherwise, if response inhibition fails, an error would occur, and individuals might modify their behavior accordingly in the subsequent trial. Therefore, the Go/NoGo task is a well-established paradigm for evaluating dynamic behavior monitoring (Hester, 2004; Moser et al., 2013). In this paradigm, two ERP components have larger amplitudes in NoGo than in Go trials, which have been labeled as the NoGo-N2 and NoGo-P3 (Falkenstein et al., 1999). The NoGo-N2 is a negative shift with its maximum at frontal regions with a latency of 200–400 ms following NoGo stimuli, and a more posterior NoGo-P3 appears around 400–600 ms following NoGo stimuli (Nieuwenhuis et al., 2003; Donkers and Van Boxtel, 2004; Huster et al., 2013). The NoGo-N2 and NoGo-P3 represent the response inhibition step of behavior monitoring. The NoGo-N2 may reflect

the detection of the conflict or mismatch between the internal representation of the Go response and the NoGo stimulus before motor execution, while the NoGo-P3 reflects an evaluation of the decision or response inhibition success (Kok et al., 2004; Smith et al., 2013; Wessel and Aron, 2015).

When individuals fail to inhibit their response to a NoGo stimulus, an error occurs. Error often induces autonomic reactions and activation of defense-oriented motivation systems to protect the organism (Hajcak, 2012; Weinberg et al., 2012). The error-related negativity (ERN), a negative response-locked ERP component, appears at the frontal region approximately 0–100 ms after an erroneous response (Falkenstein et al., 1991; Gehring et al., 1993). The ERN has been proposed to reflect the automatic comparing or detection of the conflict between the intended response and the actual response (Nieuwenhuis et al., 2003; van Veen and Carter, 2006). Following the ERN, the error positivity (Pe) appears at the central-parietal region around 200–500 ms after the error commission (Falkenstein et al., 2000; Overbeek et al., 2005). It has been suggested that the Pe reflects conscious error recognition or emotional appraisal of the error and might be involved in motivated strategy adjustments after an error commission (van Veen and Carter, 2006; Hajcak and Foti, 2008; Boldt and Yeung, 2015). More negative ERNs have been found in adults with high sensitivity to punishment (Santesso et al., 2011), while diminished Pes have been associated with emotional bluntness like psychopathy (Brazil et al., 2009).

The functional significance of error processing is to subsequently modify behavior, i.e. post-error behavior adjustments, namely adapting response strategies on the trials immediately following an error to avoid more mistakes (Danielmeier and Ullsperger, 2011). Post-error behavior adjustments are fundamental to flexible behavior and crucial to survive in a complicated changing environment. Post-error behavior adjustments can be twofold: 1) a generally prolonged reaction time (RT) in trials following an error compared to RT in trials following correct Go response (post-error slowing) and accuracy change (hit rate) following an error as compared to following correct Go response (post-error accuracy change) (Danielmeier and Ullsperger, 2011). On the one hand, active processing of current errors can impose demands on limited central resources, leading to distraction or interference on the subsequent trial. The consequence of this interference can be revealed as a reduction in post-error accuracy (double errors) (Jentzsch and Dudschig, 2009; Notebaert et al., 2009; Buzzell et al., 2017). On the other hand, post-error accuracy improves when errors are consciously perceived by individuals and individuals succeed to appropriately implement top-down cognitive control (Hajcak and Simons, 2008; Danielmeier and Ullsperger, 2011). The ability to detect errors and adjust behavior after an error accordingly may be particularly important in threatening childhood environment, because error commission might be linked with more punishment.

Disruptions in neural function underlying cognitive control have been widely reported in institutionalized children from the BEIP (e.g. McDermott et al., 2012; Loman et al., 2013). A few studies also revealed the persistent effects of childhood maltreatment on cognitive control in adolescence and adulthood, although the results are mixed in the literature. Compared to healthy controls, for instance, adolescents with different types of early life stress showed increased activation in the inferior frontal cortex and ACC during response inhibition (Mueller et al., 2010). Another study found that adolescents, but only girls growing up in poverty, had hyper-activation in the ACC and decreased ACC-dlPFC coupling during response inhibition (Spielberg et al., 2015). In contrast, Lim et al. (2015) found that compared

to a healthy group, the physically abused group showed no difference in activation patterns during response inhibition, but hyper-activation in the dorsomedial frontal cortex and supplementary motor area during error processing (Lim et al., 2015). By using electrophysiological measures, Lackner et al. (2018) found that adolescents with greater than fewer adverse childhood experiences showed larger ERN differentiation (Error-Correct difference), while better self-regulation measured by questionnaire reduced the association between adverse childhood experiences and ERN amplitudes. Compared to adolescents who experienced a time-limited form of institutional deprivation but were subsequently adopted into nurturing families, those who experienced prolonged institutional rearing exhibited decreased error-related mediofrontal theta power and were less likely to adjust their behavior following errors (post-error accuracy) (Buzzell et al., 2020).

The mixed findings might be partially explained by the different types of psychopathology in the maltreated group, as well as the variability in the approaches to measuring childhood stress. Prevailing research approaches rely on specific types of adversity (such as physical or sexual abuse, or poverty), while ignoring the fact that different types of adversity usually coexist (Kessler et al., 2010; McLaughlin et al., 2012). Consequently, it is difficult to differentiate whether a particular outcome (e.g. cognitive impairment) is due to the focal adversity or other co-occurring adversities. By distilling complex adverse experiences into core dimensions, the recently proposed dimensional approach suggested two dimensions across different types of childhood adversity: (i) adversity in the form of deprivation, due to the absence of cognitive stimulation and multi-sensory and social experiences, which is central to children exposed to neglect and poverty and (ii) adversity in the form of direct (physical or emotional) threat such as abuse (McLaughlin et al., 2014; Sheridan and McLaughlin, 2014; Humphreys and Zeanah, 2015; Teicher and Samson, 2016; Teicher et al., 2016). Furthermore, exposure to early deprivation was thought to be associated with volume reductions in the association cortex (including PFC, and superior and inferior parietal cortex), which in turn might produce difficulties in multiple domains of executive functioning (McLaughlin et al., 2014, 2017; Sheridan and McLaughlin, 2014). In the case of threat dimension, threatening experience during childhood was supposed to be associated with alterations in the circuits involved in processing salient information like the amygdala, hippocampus and vmPFC, leading to an abnormal emotional response, emotional learning and behavior (McLaughlin et al., 2014; Sheridan and McLaughlin, 2014).

Recently, a few studies with the dimensional method have provided preliminary but direct evidence for the distinction between deprivation (a core feature of neglect) and threat (a core feature of abuse). In a behavioral study, Lambert et al. (2017) revealed that deprivation (poverty) was linked with poor cognitive inhibitory control measured by a Flanker task but not automatic emotion regulation measured by emotional Stroop, while threat (child abuse and community violence) was linked with deficits in automatic emotion regulation but not cognitive control (Lambert et al., 2017). Sheridan et al. (2017) found that deprivation (low parental education and child neglect) rather than threat (community violence and abuse) was associated with poor working memory performance at the behavioral level as well as widespread and inefficient neural recruitment in the parietal cortex and PFC at the neural level (Sheridan et al., 2017). Furthermore, the distinction was also demonstrated at the physiological level, such that threat rather than deprivation was

associated with blunted sympathetic and cortisol reactivity to acute stress (Busso et al., 2017).

The aim of the present study was to investigate the relationship between childhood adversity and the dynamic process of behavior monitoring (inhibition, error processing and post-error adjustments) in the Go/NoGo task using ERPs and behavioral measurements. We further explored whether the two dimensions of childhood adversity (i.e. threat and deprivation) have a distinct association with behavior monitoring. Considering that both healthy participants and patients with psychopathologic disorders were recruited in the maltreated group in many of the previous studies (e.g. Mueller et al., 2010; Lim et al., 2015), this calls into question whether the findings indicate the neurodevelopmental consequences of childhood adversity or rather reflect the characteristics of resilience to maltreatment. Therefore, we recruited healthy adults and measured their childhood adverse experiences by the Childhood Trauma Questionnaire. According to the dimensional model of childhood adversity, we hypothesized that (i) exposure to childhood deprivation (indicated by neglect) would be associated with deficits in response inhibition and (ii) exposure to threat (indicated by abuse) would be associated with disrupted emotion processing, in this case, error as salient information especially in abusive early environment, and thus altered error monitoring and appraisal, and consequently worse post-error adjustments.

Methods

Participants

As part of a larger project addressing the relationship between cognition and stress/cortisol, this study sought to specifically investigate the impact of childhood stress and behavior monitoring. The results between cortisol and cognition will be reported elsewhere. Participants were recruited from Shenzhen University through online and public postings. All postings specified the inclusion criteria of the study: (i) male [to control sex effect on cortisol response (Liu et al., 2019) and sex-based differences in the development of the hippocampus and amygdala (Fish et al., 2020)]; (ii) normal hearing ability and physically healthy; (iii) no habit of staying up late and (iv) no psychiatric disorders including depression, etc. All eligible participants were further screened by the following exclusion criteria: (i) history of psychiatric or neurological disease, epilepsy or migraine; (ii) history of endocrine disorders (such as Cushing's syndrome); (iii) history of other major chronic physiological diseases, such as diabetes, heart disease, meningitis, severe kidney disease and malignant tumors; (iv) history of brain injury (such as brain surgery, cerebral hemorrhage and severe head trauma); (v) long-term use of antipsychotic, neurological or adrenocortical hormone drugs; (vi) long-term use of other medications; (vii) major operation in the past 6 months; and (viii) smoking (more than five cigarettes a day) and drinking habit (more than two alcoholic drinks daily). Participants were first screened based on the inclusion criteria at the time of recruitment and further screened by the exclusion criteria by self-reported questionnaire. A total of 60 healthy young male students met the criteria for inclusion in the study and analyses. The mean age was 21.10 years (s.d.: 0.838) and the mean year of education was 13.55 years (s.d.: 0.699). All participants gave written informed consent and were paid for their participation. This experiment was approved by the Ethics Committee of Human experimentation at the Medical Department of Shenzhen University.

General procedure

After arriving at the laboratory, participants were informed of the experimental procedure and completed demographic information collection and questionnaires including the Perceived Stress Scale (10-item version, PSS10). After application of the electrodes, participants were seated in a dimly lit, sound-attenuated, electrically shielded room and completed the acoustic Go/NoGo task while behavioral and electroencephalogram (EEG) data were recorded. To avoid the effect of recall-induced negative emotion on performance, participants filled in Negative Affect Scale (NAS) and the Childhood Trauma Questionnaire (CTQ) as described in the Questionnaires section after they completed the experiment.

Go/NoGo task

For the auditory Go/NoGo task, the targets (Go stimuli) were 1000 Hz pure tones and the non-targets (NoGo stimuli) were 1032 Hz pure tones (10 ms rise and fall times). The sound volume was adjusted to a comfortable level. All the tones were presented binaurally over headphones. After an initial practice block of 10 trials, two experimental blocks each including 310 trials (20% NoGo and 80% Go probability) were completed with 2-min breaks between blocks. The duration of the tones was 200 ms and the stimulus-onset asynchrony randomized from 900 to 1100 ms. During each trial, one of the two tones was presented. Participants were instructed to respond as fast and accurately as possible by pressing a button on the keyboard once they heard the Go stimuli. The sequence of Go and NoGo stimuli was pseudorandom, thus the consecutive presentation of two NoGo stimuli was avoided.

Questionnaires

Childhood stress was assessed by 28-item version CTQ on a 5-point Likert scale (1, never true; 5, very often true) (Bernstein and Fink, 1998). The CTQ is a self-reported scale, which was used as a quantitative measure of the severity of childhood adversity within different populations with or without psychopathology (Viola et al., 2016). The exemplary item is 'when I was growing up, I got hit so hard by someone in my family that I had to see a doctor or go to the hospital'. Besides the CTQ total (CTQ-t) score, originally there are five subscales including emotional, physical and sexual abuse, and emotional and physical neglect. However, different types of adverse early experiences usually coexist, and recent model and empirical evidence suggested distinctions between abuse dimension and neglect dimension (McLaughlin et al., 2014; Sheridan and McLaughlin, 2014; Humphreys and Zeanah, 2015; Teicher and Samson, 2016; Teicher et al., 2016). Therefore, abuse total score (as a proxy of threat) was calculated by summing up scores from emotional, physical and sexual abuse subscales, and neglect total score (as a proxy of deprivation) was calculated by summing up scores from emotional and physical neglect subscales.

The 10-item NAS from the Positive and Negative Affect Schedule was used to assess the negative mood state at the time of filling in the CTQ. The exemplary items are 'Indicate to what extent you feel this way right now: Irritable, Ashamed, Nervous'. Each item is rated on a 5-point scale of 1 (not at all) to 5 (very much) (Watson et al., 1988). NAS was used as a covariate in our data analysis to correct for possible recollection bias, which is common among individuals who suffered from traumatic events (Amir et al., 1996; Tapia et al., 2012). In turn, the recollection bias might result in an overestimation of childhood stress.

Recent perceived stress was assessed by Cohen's PSS (10-item version) (Cohen, 1988). The scale measures perceived stress over the last month on a 5-point Likert scale ranging from 0 (never) to 4 (always). The exemplary item is 'In the last month, how often have you felt that you were unable to control the important things in your life?'. PSS10 was further controlled to exclude the effect of chronic stress during the last month on behavioral monitoring (Wu et al., 2019).

EEG recording and preprocessing

The EEG was recorded with Ag/AgCl electrodes from 64 scalp sites mounted in an elastic cap (Neuroscan Inc., Charlotte, NC, USA) according to the international 10–20 system, with online reference to the left mastoid. Data were re-referenced offline to the average of both mastoids. One pair of electrodes placed above and below the left eye was used to record vertical eye movement and another pair placed 10 mm from the outer canthi of each eye to monitor horizontal eye movement. The impedance from all electrodes was below 5 K Ω . Signals were amplified with a bandpass filter at 0.05–100 Hz and digitized at 1000 Hz.

The EEG data were processed by Scan 4.3 software (Neuroscan, USA). The data were further filtered with a 30-Hz lowpass filter. Ocular artifacts were removed from the EEG signal by a regression procedure implemented in Neuroscan software (Semlitsch et al., 1986). Trials with artifacts were automatically rejected with a criterion of $\pm 100 \mu\text{V}$.

For the response inhibition, two stimulus-locked (N2 and P3) components were analyzed. EEG data were epoched into periods of 1000 ms (including 200 ms before stimulus onset as baseline) time-locked to the onset of the stimulus. Considering that mean amplitude is more reliable than peak amplitude (Clayson et al., 2013), all the ERP components were measured as mean amplitude in the current study. The mean amplitude of the frontal N2 was measured at the Fz site in the time window between 200 and 400 ms after stimulus onset. The mean amplitude of the P3 was measured at the Pz site in the time window between 400 and 600 ms after stimulus onset. These sites and time windows were chosen according to the maximum amplitude in our data and in line with previous research (Falkenstein et al., 1999; Sehlmeier et al., 2010).

For the error processing, two response-locked (ERN and Pe) components were analyzed. Error responses to the NoGo trials (false alarm) were averaged for the ERN and Pe. EEG data were epoched into periods of 1000 ms, including 400–200 ms before response as baseline according to previous studies (Olvet and Hajcak, 2009), and time-locked to the onset of the button press. The ERN focused on the electrode placed at the FCz site and the Pe on the electrode of the CPz site. Mean ERN amplitude was measured 50 ms before and 150 ms after a false alarm response, and mean Pe amplitude was measured between 300 and 500 ms after a false alarm response. These sites and time windows were chosen according to our own data where they have the maximum amplitude and in line with previous research (Falkenstein et al., 2000; O'Connell et al., 2007).

Data preparation and preprocessing

For the behavioral performance of response inhibition, the false alarm rate of the NoGo trials was calculated. Trials with RT below 50 ms and above 1000 ms were excluded. The hit rate of the Go trials and the RT of the correct Go trials were also calculated.

For post-error adjustments, the behavioral performance after committing an error was calculated. Specifically, 'post-error'

refers to Go trials following an error response to NoGo trials, and 'post-correct' as a control condition refers to Go trials following a correct response to Go trials. Post-error costs were indexed by post-error slowing and post-error accuracy change. Post-error slowing was calculated as the difference between mean RTs on 'post-error' vs those on 'post-correct'. Post-error accuracy change was calculated as the difference between hit rates on 'post-error' vs those on 'post-correct'.

All statistical analyses were conducted by SPSS software (version 22.0). Considering that the bootstrapping method provides better control over type I error as well as a better representation of the probability distribution which makes it a robust statistical test (Wilcox, 2011), we performed all the analyses with bootstrap ($n = 1000$ times of resampling with replacement) to improve the robustness of our inference. For the response inhibition-related ERP components (N2 and P3 amplitudes), a paired-samples t -test was carried out on stimulus type (Go vs NoGo). For the error processing-related ERP components (ERN and Pe amplitudes), a paired-samples t -test was carried out on response outcomes (correct hit to Go vs false alarm to NoGo). Greenhouse-Geisser correction for degrees of freedom was applied when the sphericity assumption was violated.

Data analysis

First of all, we investigated the relationship between the severity of childhood adversity and dynamic stages of behavior monitoring by using multiple linear regression. The primary predictor was CTQ-t score. The demographic variables (age and education years) and current mood state (NAS), as well as chronic perceived stress (PSS10) were treated as covariate variables. The dependent variables were the dynamic stages of behavior monitoring, i.e. response inhibition (false alarm rate, and NoGo-N2 and NoGo-P3 amplitudes), error processing (ERN and Pe amplitudes) and post-error cost (post-error slowing and post-error accuracy change).

To further explore the differential influence of the two dimensions of childhood adversity on the dynamic stage of behavior monitoring in adulthood, we repeated the multiple regression analysis with the primary predictors of abuse and neglect scores from CTQ. Results from the regression analysis were described as regression coefficients with 95% CI and standard errors from the bootstrap analysis.

In order to test the hypothesis that abuse and neglect distinctly influence behavior monitoring, we used the conservative method by examining the overlap of CI from abuse and neglect. In the event that the CIs overlapped by less than 50% of one CI arm, the beta weights would be considered statistically significantly different from each other ($P < 0.05$; Cumming, 2009, 2014).

Additionally, to explore the relationship between inhibition-related ERP components (NoGo-N2 and NoGo-P3 amplitudes) and behavior performance of response inhibition, the Pearson correlation was conducted between the inhibition-related ERP components and the false alarm rate. Considering one of the functional roles of error processing is to prevent future mistakes, a correlational analysis was conducted between the error-related ERP components (ERN and Pe amplitudes) and post-error costs (post-error slowing and post-error accuracy change).

Results

Subjective measurements

The average CTQ-t score was 35.07 (s.d.: 6.88, range: 25–57). When the CTQ was subdivided into abuse and neglect, the

Table 1. The behavioral performance for the Go/NoGo task

	Mean (s.d.)	Post-trial RT (s.d.)	Post-trial accuracy (s.d.)
False alarm	25.17% (16.23)	445.18 ms (102.08)	76.75% (23.46)
Hit	91.14% (8.95)	403.69 ms (65.88)	92.92% (7.70)

average abuse score was 18.33 (s.d.: 3.35, range: 15–28) and the average neglect score was 16.73 (s.d.: 4.85, range: 10–29). The mean score of NAS was 16.07 (s.d.: 5.96, range: 10–36), and the mean score of PSS10 was 16.08 (s.d.: 5.35, range: 7–28).

Behavioral performance

Table 1 shows the behavioral performance for the Go/NoGo task. For the response inhibition performance, the mean false alarm rate for NoGo trials was 25.17%, with an average of 30.2 (s.d.: 19.48) false alarms to NoGo trials per participant. The mean RT for correct Go trials was 403.58 ms (s.d.: 66.39). For post-error performance, the paired t -test showed that the post-error RT was slower than post-correct RT ($t = 4.01$, $P < 0.01$), and the post-error accuracy was lower than post-correct accuracy ($t = 5.97$, $P < 0.01$). The mean post-error slowing was 41.50 ms (s.d.: 78.92 ms) and mean decreases in post-error accuracy (double error) was 16.17% (s.d.: 20.98%).

ERPs

Figure 1 shows the response inhibition-related ERP waveforms time-locked to stimulus onset. N2 amplitudes ($t = 11.72$, $P < 0.01$) and P3 amplitudes ($t = 9.68$, $P < 0.01$) were larger for NoGo than for Go stimuli, indicating the classical response inhibition effects on ERPs. Mean NoGo-N2 amplitudes were $-7.23 \mu\text{V}$ (s.d.: $2.97 \mu\text{V}$). Mean NoGo-P3 amplitudes were $4.06 \mu\text{V}$ (s.d.: $3.36 \mu\text{V}$).

Figure 2 shows the error processing-related ERP waveforms time-locked to correct hit response to Go trials and false alarm response to NoGo trials. ERN amplitudes ($t = 11.27$, $P < 0.01$) and Pe amplitudes ($t = 3.77$, $P < 0.01$) were larger for the erroneous response to NoGo trials than for the correct hit to Go trials, indicating the classical error processing on ERPs. Mean ERN amplitudes were $-6.74 \mu\text{V}$ (s.d.: $4.87 \mu\text{V}$). Mean Pe amplitudes were $4.36 \mu\text{V}$ (s.d.: $5.11 \mu\text{V}$).

The relationships between severity of childhood adversity and dynamic stages of behavior monitoring

'For the response inhibition', a multiple linear regression (Table 2) was conducted with primary predictor (CTQ-t) and control variables (age, education year, NAS and PSS10). The model explained 13.7% of the variance in NoGo-N2 amplitudes [$R^2 = 0.14$, $F(5,59) = 1.71$, $P > 0.10$], and only CTQ-t score significantly predicted NoGo-N2 amplitudes (Table 2). Therefore, more negative N2 amplitudes were associated with higher CTQ-t scores. However, the CTQ-t was not associated with any behavior index (i.e. false alarm rate) or the late stage (NoGo-P3 amplitudes) of response inhibition (P values > 0.10). To explore the neural effect of the NoGo-N2 on behavior, we further investigated the association between CTQ-t and false alarm rate after controlling for mean NoGo-N2 amplitudes. We first ran a bootstrapping partial correlation (sampling $n = 1000$) between CTQ-t and false alarm rate with mean NoGo-N2 amplitudes

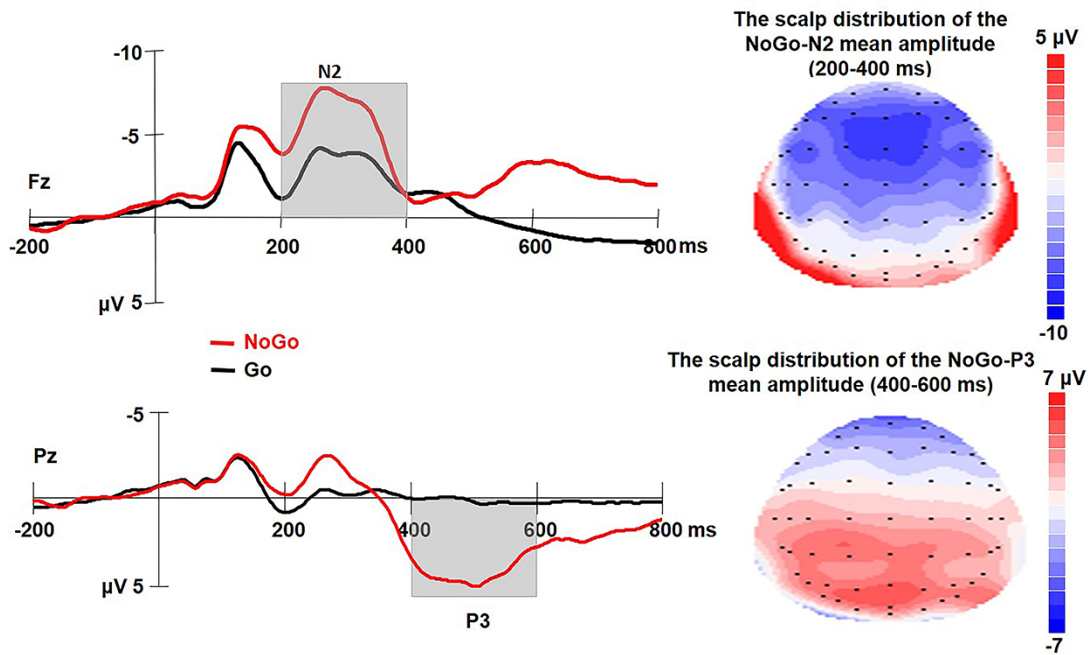


Fig. 1. Left: The response inhibition-related ERP waveforms time-locked to Go and NoGo stimuli. Right: The scalp distribution of the NoGo-N2 and NoGo-P3.

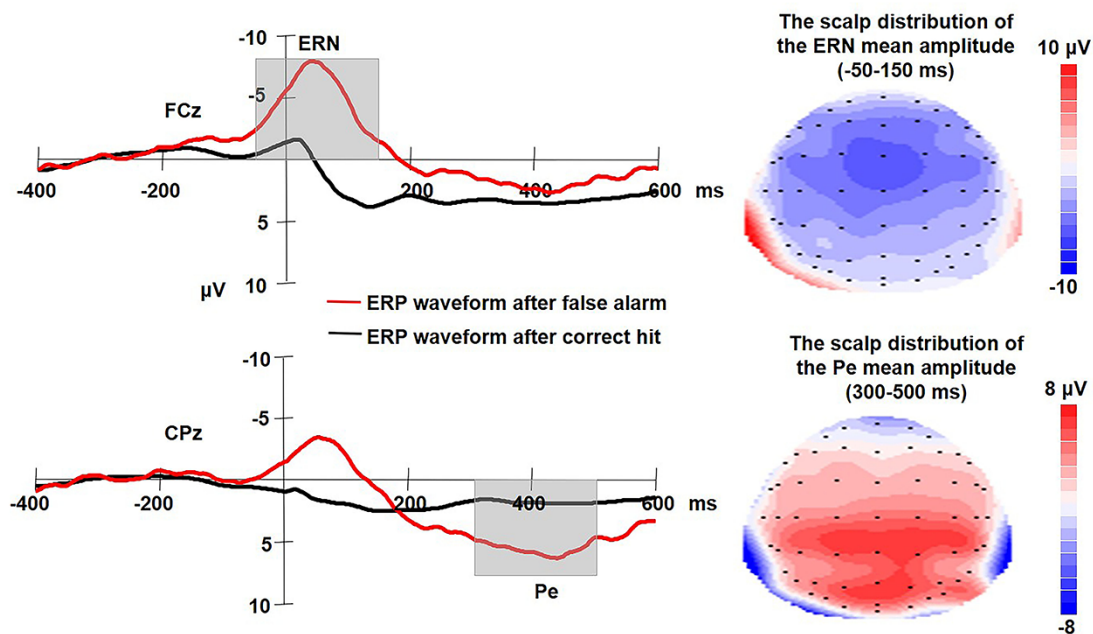


Fig. 2. Left: The error processing-related ERP waveforms time-locked to correct hit response to Go trials and false alarm response to NoGo trials. Right: The scalp distribution of the ERN and Pe.

controlled, and the partial correlation coefficient was not significant (partial $r=0.05$, $P>0.10$, 95% confidence interval (CI) $[-0.15, 0.27]$). Next, we conducted a stricter partial correlation of CTQ-t and false alarm rate, controlling for five variables: the demographic variables (age and education), the psychological variables (negative affect and PSS) and mean NoGo-N2 amplitudes. However, the partial correlation coefficient was still not significant (partial $r=0.06$, $P>0.10$, 95% CI $[-0.01, 0.11]$).

‘For the error processing’, the result from multiple linear regression [Table 3, $R^2=0.09$, $F(5,57)=0.97$, $P>0.10$] showed that the CTQ-t score significantly predicted ERN amplitudes. More negative ERN amplitudes were associated with higher CTQ-t scores. The regression model on Pe amplitudes [Table 4, $R^2=0.21$, $F(5,57)=2.69$, $P<0.05$] also showed that higher CTQ-t score significantly predicted less positive Pe amplitudes.

‘For post-error adjustments’, the CTQ-t was not related with either post-error slowing or post-error accuracy (P values >0.10).

Table 2. Bootstrapping regression of CTQ-t score on NoGo-N2 amplitudes

	Standardized regression coefficient B	95% CI	Standard error (bootstrap)	P
Age	-0.08	[-0.44, 0.23]	0.17	0.64
Education years	0.03	[-0.27, 0.30]	0.14	0.79
NAS	-0.09	[-0.45, 0.17]	0.15	0.52
PSS10	-0.14	[-0.51, 0.21]	0.19	0.45
CTQ-t	-0.30	[-0.55, -0.04]	0.13	0.03

Table 3. Bootstrapping regression of CTQ-t score on ERN amplitudes

	Standardized regression coefficient B	95% CI	Standard error (bootstrap)	P
Age	-0.10	[-0.53, 0.13]	0.17	0.50
Education years	0.05	[-0.25, 0.62]	0.22	0.82
NAS	-0.00	[-0.38, 0.29]	0.16	0.97
PSS10	0.01	[-0.27, 0.35]	0.16	0.93
CTQ-t	-0.29	[-0.62, -0.07]	0.14	0.05

Table 4. Bootstrapping regression of CTQ-t score on the Pe amplitude

	Standardized regression coefficient B	95% CI	Standard error (bootstrap)	P
Age	-0.04	[-0.35, 0.33]	0.18	0.83
Education years	-0.05	[-0.50, 0.22]	0.17	0.74
NAS	0.19	[-0.24, 0.63]	0.23	0.46
PSS10	0.26	[-0.08, 0.57]	0.17	0.13
CTQ-t	-0.29	[-0.57, -0.03]	0.14	0.04

Distinct relationships between two dimensions of childhood adversity and dynamic stages of behavior monitoring

We found that the severity of childhood adversity is associated with more allocation of controlled attention (indicated by the more negative NoGo-N2 and ERN amplitudes) as well as less conscious error assessment (indicated by the less positive Pe amplitudes). In the next step, we explored whether different dimensions of childhood adversity (i.e. abuse and neglect) have a distinct relationship with behavior monitoring. The same multiple linear regression was repeated on dynamic stages of behavior monitoring with primary predictors (abuse and neglect) and control variables (age, education year, NAS and PSS10).

'For the response inhibition', the results from the multiple regression with bootstrap revealed that neither abuse nor neglect was significantly associated with an index of response inhibition (i.e. false alarm rate and NoGo-N2/P3 amplitudes, P values > 0.10).

'For the error processing', the multiple regression model for ERN amplitudes showed that neither abuse nor neglect was associated with ERN amplitudes (P values > 0.10). The multiple regression model explained 23.4% of the variance in Pe

Table 5. Bootstrapping regression of CTQ two dimensions on the Pe amplitude

	Standardized regression coefficient B	95% CI	Standard error (bootstrap)	P
Age	-0.04	[-0.34, 0.33]	0.17	0.82
Education years	-0.07	[-0.53, 0.17]	0.17	0.62
NAS	0.18	[-0.25, 0.56]	0.21	0.43
PSS10	0.24	[-0.07, 0.57]	0.16	0.15
Abuse	-0.33	[-0.64, -0.02]	0.16	0.04
Neglect	-0.05	[-0.40, 0.31]	0.18	0.78

Table 6. Bootstrapping regression of CTQ's two dimensions on decreases in post-error accuracy

	Standardized regression coefficient B	95% CI	Standard error (bootstrap)	P
Age	-0.10	[-0.47, 0.13]	0.15	0.44
Education years	0.12	[-0.11, 0.55]	0.18	0.44
NAS	-0.08	[-0.33, 0.22]	0.14	0.53
PSS10	-0.10	[-0.45, 0.27]	0.18	0.59
Abuse	0.37	[-0.01, 0.77]	0.20	0.06
Neglect	-0.20	[-0.52, 0.01]	0.17	0.22

amplitudes [Table 5, $R^2 = 0.23$, $F(6,57) = 2.59$, $P = 0.03$], and CTQ-abuse score significantly predicted Pe amplitudes (Figure 3 Left). To evaluate the hypothesis more precisely, standardized beta coefficients and CIs were compared between CTQ-abuse and CTQ-neglect. As shown in Figure 4 Left, CIs of Pe mean amplitudes for abuse and neglect overlapped substantially (over 50%), demonstrating that the regression coefficient for CTQ-abuse was not considered significantly different from the CTQ-neglect regression coefficient (Cumming, 2009).

'For post-error adjustments', the multiple regression model explained 16% of the variance in decreases in post-error accuracy [Table 6, $R^2 = 0.16$, $F(6,59) = 1.68$, $P > 0.10$], and only CTQ-abuse score marginally positively predicted decreases in post-error accuracy (Figure 3 Right). Furthermore, as shown in Figure 4 Right, CIs of post-error accuracy for abuse were clearly distinguished from neglect (less than 50%), indicating that the relationships for decreases in post-error accuracy were significantly different between CTQ-abuse and CTQ-neglect (Cumming, 2009). However, the multiple regression model for post-error slowing showed that neither abuse nor neglect was associated with post-error slowing (P values > 0.10).

The relationships between ERP components and behavioral performance

Correlations between NoGo-N2 amplitudes or latencies and response performance (hit rate, false alarm rate and RT to correct hit trials) were not significant (r values = 0.03 to 0.14, P values > 0.10). NoGo-P3 amplitudes were positively correlated with hit rate ($r = 0.38$, $P = 0.00$) and negatively correlated with RT to correct hit trials ($r = -0.31$, $P = 0.019$). There was also a marginally negative relationship between NoGo-P3 amplitudes and false alarm rate ($r = -0.23$, $P = 0.076$). That is, more positive NoGo-P3 amplitudes were associated with the faster and more

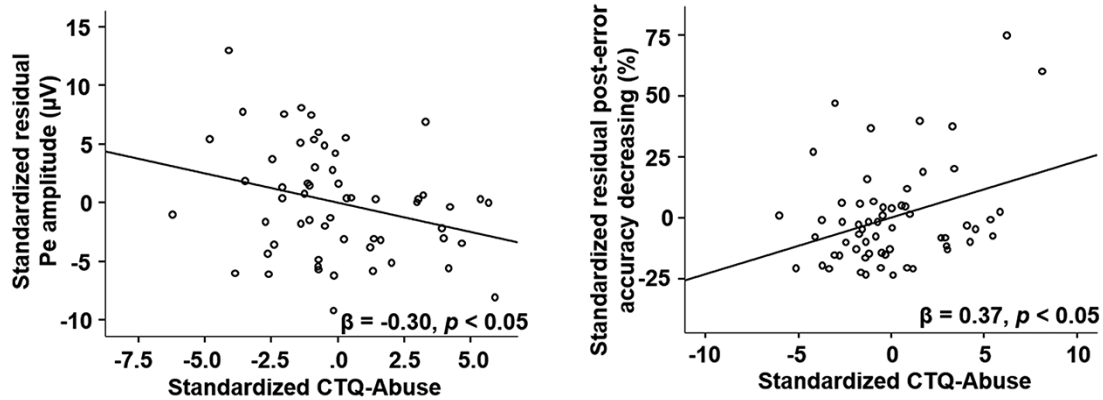


Fig. 3. Left: The relationship between standardized CTQ-abuse score and the standardized residual Pe mean amplitude. Right: The relationship between standardized CTQ-abuse score and the standardized residual of decreases in post-error accuracy.

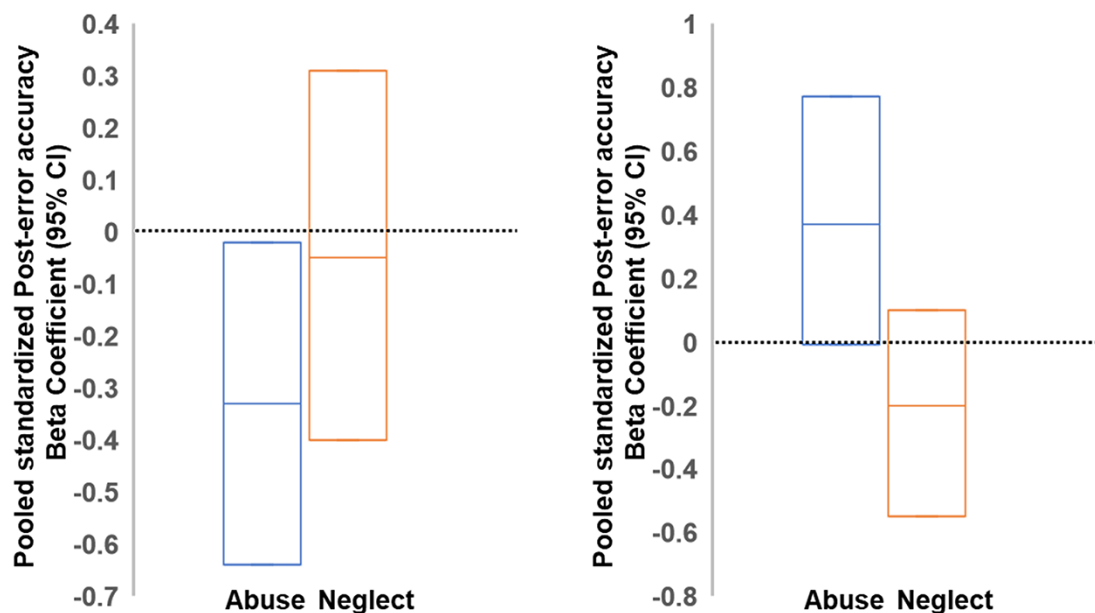


Fig. 4. Left: Pooled standardized beta coefficients (with 95% CIs), representing the magnitude of Pe mean amplitudes, by CTQ-abuse and CTQ-neglect. Right: Pooled standardized beta coefficients (with 95% CIs), representing post-error accuracy, by CTQ-abuse and CTQ-neglect.

accurate response to the Go stimulus as well as the marginally lower false alarm response to the NoGo stimulus.

Correlations between ERN amplitudes or latencies and post-error performance (post-error slowing and decreases in post-error accuracy) were not significant (r values = 0.017–0.22, P values > 0.10). The Pe amplitudes were negatively correlated with post-error slowing ($r = -0.52$, $P = 0.00$) as well as decreases in post-error accuracy ($r = -0.44$, $P = 0.00$). In short, more positive Pe amplitudes were associated with less post-error slowing and lower decreases in post-error accuracy (i.e. better behavioral adjustment after making an error).

Discussion

The present study investigated the relationships between childhood adversity (and its two dimensions) and dynamic behavioral monitoring in healthy young adults. There were three major findings in this study: (i) Adults with higher severity of childhood adversity deployed more controlled attention (indicated by more

negative NoGo-N2 and ERN amplitudes) in the Go/NoGo task; (ii) Higher severity of childhood abuse (as a proxy of threat) was related with less awareness/appraisal of errors (indicated by less positive error-related Pe amplitudes); and (iii) Higher severity of childhood abuse was significantly and distinctively related with poorer post-error adjustment (i.e. lower post-error accuracy). These associations were emerged in rather healthy individuals during adulthood, even by controlling potential factors such as age, education, current negative affect and perceived chronic stress.

Adults with higher severity of childhood adversity displayed larger (more negative) response inhibition-related NoGo-N2 amplitudes and larger (more negative) ERN amplitudes. In the response inhibition domain, NoGo-N2 component has been interpreted in terms of conflict detection, i.e. detection of conflict between the internal dominant representation of response and external nonresponse cue (Nieuwenhuis *et al.*, 2003; Donkers and Van Boxtel, 2004; Huster *et al.*, 2013). The ERN reflects an automatic comparing or detection of the

conflict between the intended response and the actual response (Nieuwenhuis et al., 2003; van Veen and Carter, 2006). By using a novel Go/NoGo paradigm, van Noordt et al. (2015) found that simply presenting individuals with cues signaling the potential need for a relative increase in response control without the involvement of errors, response conflict resolution or inhibition is sufficient to elicit a medial frontal N2-like component, further overlapping with the ERN and NoGo-N2. Their finding was in line with other studies suggesting that ERN and N2 components from different paradigms have significantly overlapping topographical scalp maps and that the modulation of controlled attention is the underlying principle of medial frontal activation in performance paradigms with different task demands (Gruendler et al., 2011; Wessel et al., 2012; van Noordt et al., 2015). Our results, to some extent, supported this literature by showing that childhood adversity is associated with more negative NoGo-N2 as well as ERN amplitudes, indicating that childhood adversity might have a universal influence on controlled attention. Our results were consistent with those of Lackner et al. (2018), who also found that adolescents with greater adverse childhood experiences showed larger ERN differentiation (error-correct difference). The positive relationship between higher childhood adversity and more allocation of controlled attention echoed with findings from a neuroimaging stop-signal task in which adopted adolescents with a history of neglect and maltreatment had higher activation in brain regions involved in conflict monitoring (dorsal ACC) and inhibitory and response control (inferior PFC and striatum) but equal behavioral performance compared to the control group (Mueller et al., 2010). Similarly, we found that behavioral performance was not influenced by childhood adversity (even after controlling the N2 level), suggesting that more controlled attention does not lead to better behavioral performance in individuals exposed to childhood adversity. In other words, compared to individuals with a low level of childhood adversity, individuals who suffered from more severe childhood adversity are less neurally efficient when conducting executive tasks.

If an error is detected immediately after a false alarm response to NoGo trials, post-error behavioral adjustments may occur. In general, the accuracy rate for the post-error trials in our study was lower compared to the post-correct trials (decreases in post-error accuracy). One possible explanation for such double error is bottom-up error-induced blindness, i.e. error processing interferes with task performance when subsequent decisions must be made rapidly (Jentsch and Dudschig, 2009; Notebaert et al., 2009; Buzzell et al., 2017), as the response-stimulus interval in our study design was relatively short (approximately 400–600 ms). Another possibility is the impaired top-down control after error detection. In support of this, we found that it is Pe amplitude rather than ERN amplitude that is associated with post-error behavior, i.e. the more positive the Pe amplitude, the higher the accuracy of the post-error trials. According to previous literature, the Pe component reflects error awareness or emotional appraisal of the error and might be involved in motivated strategy adjustments after an error is committed (van Veen and Carter, 2006; Hajcak and Foti, 2008; Boldt and Yeung, 2015). Therefore, both the bottom-up attentional bottleneck and the impaired top-down control might contribute to the reduction in post-error accuracy.

Our result showed that the severity of childhood abuse was associated with late stage of error processing: the more severe the childhood abuse, the smaller (less positive) the Pe amplitude. On the one hand, the association between childhood abuse and less positive Pe amplitude suggested that

higher severity of childhood abuse is associated with less pronounced conscious recognition and/or emotional/motivated appraisal of an error. On the other hand, the less salient appraisal of the error leads to less behavior adjustments after an error commission. This speculation further receives evidence from our own data: the positive association between the severity of childhood abuse and decreases in post-error accuracy. A similarly decreased Pe amplitude and poor post-error adjustments have been found in individuals with externalizing disorders, characterized as impulsive or antisocial behaviors (Wilkowski and Robinson, 2008; Luijten et al., 2014), which are common comorbidities of childhood abuse (Duffy et al., 2018). It is possible that impaired error assessment mediates the relationship between childhood abuse and externalizing disorders. More specifically, childhood abuse might increase the risk for externalizing disorder by means of a decreased saliency of error assessment and subsequent incapability of behavioral adjustments in response to the error. However, due to the lack of assessment of externalizing symptoms in our study, more empirical researches are needed to test this hypothesis.

As the theoretical models have proposed, the dimensions of threat and deprivation should be distinguished among childhood adversities and may have differential impacts on the brain and cognition (McLaughlin et al., 2014; Sheridan and McLaughlin, 2014; Humphreys and Zeanah, 2015; Teicher and Samson, 2016; Teicher et al., 2016). This view is also aligned with the proposal by the Research domain criteria, which emphasizes fundamental dimensions and validates particular dimensions by exploring their associated behavioral and neural underpinnings (Insel et al., 2010; Cuthbert and Insel, 2013). Partially consistent with this proposal, we found that abuse (as a proxy of threat) was numerically associated with error assessment. Furthermore, only abuse rather than neglect (as a proxy of deprivation) was distinctively associated with post-error adjustment (post-error accuracy change). However, contrary to the dimensional model that threat (experiences of violence at an early age) would give rise to alterations in cortical and sub-cortical circuits involved in emotional processing, leading to heightened reactivity to threat information in the environment (McLaughlin et al., 2014), our result showed that abuse was associated with attenuated rather than an exaggerated assessment of errors, a specific type of emotional event from an early abusive environment. This effect, to some extent, was consistent with previous findings that abused children tended to avoid negative information both on the behavioral level (bias attention away from threat information) (Pine et al., 2005) and on the neural level (decreased activation of amygdala and insula to social rejection) (Puetz et al., 2016). Results of both the blunted error assessment in the current study and avoidance to threat in the previous two studies (Pine et al., 2005; Puetz et al., 2016) could be explained by the cortical and cognitive adaptation mechanism, i.e. downregulation of emotional response to negative events following iterative exposure to threat (Heim et al., 2013). Another interpretation is disengagement coping, i.e. responses oriented away from the stressor or one's emotions, which is a typical coping strategy among individuals with childhood abuse (Compas et al., 2001; Leitenberg et al., 2004). While initially protective, such decreased motivated error processing might represent a direct biological substrate for the development of behavioral problems in later life, such as failure to adjust behavior in the face of repeated errors, as our study showed. From the perspective of behavioral cost of error, our results suggest that childhood abuse is associated with poor behavioral adjustment after

making an error, which is partially consistent with the hypothesis by McLaughlin et al. (2014).

Behavior monitoring is a key component of cognitive control, which is crucial to mature adaptive behavior. Although childhood adversity is widely accepted as an important risk factor for the development of psychopathology (Kessler et al., 2010; McLaughlin et al., 2012), it is worthwhile to mention that all the participants in our study are mentally healthy despite exposure to varying degrees of childhood adversity. Individuals who demonstrate stable and healthy levels of functioning on one or more domains after experiences of adversity are commonly referred to as 'resilient' (Kalisch et al., 2019). By comprehensively reviewing the literature, Moreno-López et al. (2020) proposed that resilient functioning after childhood adversity is characterized by strong executive control, dampened threat processing and habituation of stress responses potentially through stronger connectivity between the central executive network and limbic regions. Therefore, rather than vulnerability, the more controlled attention and less motivated assessment of errors in our study might represent resilient factors to childhood adversity. However, the lack of a comparison group (adults reporting similar childhood adversity with current psychopathology) limits us from making a strong inference.

Except for the lack of comparison group, there are some other limitations to the current study that should be addressed. First, all the participants in the current study were young healthy males. This specific sample, on the one hand, mitigates potential confounding factors such as hormonal contraceptive use or menstrual cycle, but on the other hand, limits the generalizability. Thus, we do not know whether childhood adversity influence males and females differently. This will be addressed in future studies. Second, we only used the self-reported past experiences of childhood maltreatment instead of a detailed interview or documentary recording. However, we controlled both the concurrent negative affect and chronic stress level, which, to some extent, may control the recall bias effects on traumatic events. Third, although the CTQ is a reliable, valid and commonly used instrument for retrospective assessment of childhood adversity, it limits our understanding of the specific developmental stage(s) (i.e. infancy, childhood or adolescence) of trauma exposure, which is a very important factor on the effect of early adversity on brain development (McLaughlin et al., 2014; Teicher et al., 2016; Dunn et al., 2019).

Conclusion

In summary, our study demonstrates that the severity of childhood adversity is associated with more allocation of controlled attention, as shown by both higher level of response conflict during response inhibition (as indicated by more negative NoGo-N2 amplitudes) and automatic error detection during error processing (as indicated by more negative ERN amplitudes). Furthermore, abuse is associated with the less emotional assessment of errors and poorer behavioral adjustment after making an error (as indicated by less positive Pe amplitudes and more double errors). Our findings provide preliminary evidence for the distinct consequences of childhood adversity on the neurocognitive processes of behavior monitoring later in adult life.

Acknowledgement

We thank Dr. Xiaohu Peng for ERP technical support.

Funding

This work was supported by the National Natural Science Foundation of China (31771246, 31920103009, 31522028 and 81571056), Shenzhen-Hong Kong Institute of Brain Science-Shenzhen Fundamental Research Institutions(2019SHIBS0003), the Guangdong Grant 'Key Technologies for Treatment of Brain Disorders' (2018B030332001), and the Guangdong Pearl River Talents Plan Innovative and Entrepreneurial Team Grant (2016ZT06S220).

Conflict of interest

The authors declare that they have no conflict of interest.

References

- Amir, N., McNally, R.J., Wiegartz, P.S. (1996). Implicit memory bias for threat in posttraumatic stress disorder. *Cognitive Therapy and Research*, *20*(6), 625–35.
- Bernstein, D.P., Fink, L. (1998). *Childhood Trauma Questionnaire: A Retrospective Self-report: Manual*. San Antonio, TX: Psychological Corporation.
- Boldt, A., Yeung, N. (2015). Shared neural markers of decision confidence and error detection. *Journal of Neuroscience*, *35*(8), 3478–84.
- Botvinick, M.M., Braver, T.S., Barch, D.M., Carter, C.S., Cohen, J.D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*(3), 624–52.
- Brazil, I.A., de Bruijn, E.R., Bulten, B.H., et al. (2009). Early and late components of error monitoring in violent offenders with psychopathy. *Biological Psychiatry*, *65*(2), 137–43.
- Busso, D.S., McLaughlin, K.A., Sheridan, M.A. (2017). Dimensions of adversity, physiological reactivity, and externalizing psychopathology in adolescence: deprivation and threat. *Psychosomatic Medicine*, *79*(2), 162–71.
- Buzzell, G.A., Beatty, P.J., Paquette, N.A., Roberts, D.M., McDonald, C.G. (2017). Error-induced blindness: error detection leads to impaired sensory processing and lower accuracy at short response-stimulus intervals. *Journal of Neuroscience*, *37*(11), 2895–903.
- Buzzell, G.A., Troller-Renfree, S.V., Wade, M., et al. (2020). Adolescent cognitive control and mediofrontal theta oscillations are disrupted by neglect: associations with transdiagnostic risk for psychopathology in a randomized controlled trial. *Developmental Cognitive Neuroscience*, *43*, 100777.
- Clark, C., Caldwell, T., Power, C., Stansfeld, S.A. (2010). Does the influence of childhood adversity on psychopathology persist across the lifecourse? A 45-year prospective epidemiologic study. *Annals of Epidemiology*, *20*(5), 385–94.
- Clayson, P.E., Baldwin, S.A., Larson, M.J. (2013). How does noise affect amplitude and latency measurement of event-related potentials (ERPs)? A methodological critique and simulation study. *Psychophysiology*, *50*, 174–86.
- Cohen, S. (1988). Perceived stress in a probability sample of the United States. In: Spacapan, S., Oskamp, S., editors. *The Social Psychology of Health: Claremont Symposium on Applied Social Psychology*, pp. 31–67. Newbury Park, CA: Sage Publications, Inc.
- Compas, B.E., Connor-Smith, J.K., Saltzman, H., Thomsen, A.H., Wadsworth, M.E. (2001). Coping with stress during childhood and adolescence: problems, progress, and potential in theory and research. *Psychological Bulletin*, *127*(1), 87–127.

- Cumming, G. (2009). Inference by eye: reading the overlap of independent confidence intervals. *Statistics in Medicine*, *28*(2), 205–20.
- Cumming, G. (2014). The new statistics: why and how. *Psychological Science*, *25*(1), 7–29.
- Cuthbert, B.N., Insel, T.R. (2013). Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Medicine*, *11*(1), 126.
- Danielmeier, C., Ullsperger, M. (2011). Post-error adjustments. *Frontiers in Psychology*, *2*, 233.
- Donkers, F.C., Van Boxtel, G.J. (2004). The N2 in go/no-go tasks reflects conflict monitoring not response inhibition. *Brain and Cognition*, *56*(2), 165–76.
- Duffy, K.A., McLaughlin, K.A., Green, P.A. (2018). Early life adversity and health-risk behaviors: proposed psychological and neural mechanisms. *Annals of the New York Academy of Sciences*, *1428*(1), 151–69.
- Dunn, E.C., Soare, T.W., Zhu, Y., et al. (2019). Sensitive periods for the effect of childhood adversity on DNA methylation: results from a prospective, longitudinal study. *Biological Psychiatry*, *85*(10), 838–49.
- Eigsti, I.-M., Weitzman, C., Schuh, J., de Marchena, A., Casey, B.J. (2011). Language and cognitive outcomes in internationally adopted children. *Development and Psychopathology*, *23*(2), 629–46.
- Falkenstein, M., Hohnsbein, J., Hoormann, J., Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography and Clinical Neurophysiology*, *78*(6), 447–55.
- Falkenstein, M., Hoormann, J., Hohnsbein, J. (1999). ERP components in Go/Nogo tasks and their relation to inhibition. *Acta Psychologica*, *101*(2–3), 267–91.
- Falkenstein, M., Hoormann, J., Christ, S., Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: a tutorial. *Biological Psychology*, *51*(2–3), 87–107.
- Farah, M.J., Shera, D.M., Savage, J.H., et al. (2006). Childhood poverty: specific associations with neurocognitive development. *Brain Research*, *1110*(1), 166–74.
- Fish, A. M., Nadig, A., Seidlitz, J., et al. (2020). Sex-biased trajectories of amygdalo-hippocampal morphology change over human development. *NeuroImage*, *204*, 116122.
- Gehring, W.J., Goss, B., Coles, M., Meyer, D.E., Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science*, *4*(6), 385–90.
- Gründler, T., Ullsperger, M., Huster, R.J., Verdejo García, A. (2011). Event-related potential correlates of performance-monitoring in a lateralized time-estimation task. *PLoS One*, *6*(10), e25591.
- Hajcak, G. (2012). What we've learned from mistakes insights from error-related brain activity. *Current Directions in Psychological Science*, *21*(2), 101–6.
- Hajcak, G., Foti, D. (2008). Errors are aversive: defensive motivation and the error-related negativity. *Psychological Science*, *19*(2), 103–8.
- Hajcak, G., Simons, R.F. (2008). Oops!.. I did it again: an ERP and behavioral study of double-errors. *Brain and Cognition*, *68*(1), 15–21.
- Heim, C.M., Mayberg, H.S., Mletzko, T., Nemeroff, C.B., Pruessner, J.C. (2013). Decreased cortical representation of genital somatosensory field after childhood sexual abuse. *American Journal of Psychiatry*, *170*(6), 616–23.
- Hester, R. (2004). Individual differences in error processing: a review and reanalysis of three event-related fMRI studies using the GO/NOGO task. *Cerebral Cortex*, *14*(9), 986–94.
- Hughes, K., Bellis, M.A., Hardcastle, K.A., et al. (2017). The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. *The Lancet Public Health*, *2*(8), e356–66.
- Humphreys, K.L., Zeanah, C.H. (2015). Deviations from the expectable environment in early childhood and emerging psychopathology. *Neuropsychopharmacology*, *40*(1), 154.
- Huster, R.J., Enriquez-Geppert, S., Lavallee, C.F., Falkenstein, M., Herrmann, C.S. (2013). Electroencephalography of response inhibition tasks: functional networks and cognitive contributions. *International Journal of Psychophysiology*, *87*(3), 217–33.
- Insel, T., Cuthbert, B., Garvey, M., et al. (2010). Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, *167*(7), 748–51.
- Jentsch, I., Dudschig, C. (2009). Short article: why do we slow down after an error? Mechanisms underlying the effects of posterror slowing. *Quarterly Journal of Experimental Psychology*, *62*(2), 209–18.
- Kalisch, R., Cramer, A., Binder, H., et al. (2019). Deconstructing and reconstructing resilience: a dynamic network approach. *Perspectives on Psychological Science*, *14*(5), 765–77.
- Kerns, J.G. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*(5660), 1023–6.
- Kessler, R.C., McLaughlin, K.A., Green, J.G., et al. (2010). Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. *The British Journal of Psychiatry*, *197*(5), 378–85.
- Kok, A., Ramautar, J.R., de Ruiter, M.B., Band, G., Ridderinkhof, K.R. (2004). ERP components associated with successful and unsuccessful stopping in a stop-signal task. *Psychophysiology*, *41*(1), 9–20.
- Lackner, C.L., Santesso, D.L., Dywan, J., O'Leary, D.D., Wade, T.J., Segalowitz, S.J. (2018). Adverse childhood experiences are associated with self-regulation and the magnitude of the error-related negativity difference. *Biological Psychology*, *132*, 244–51.
- Lambert, H.K., King, K.M., Monahan, K.C., McLaughlin, K.A. (2017). Differential associations of threat and deprivation with emotion regulation and cognitive control in adolescence. *Development and Psychopathology*, *29*(3), 929–40.
- Lamm, C., Troller-Renfree, S.V., Zeanah, C.H., Nelson, C.A., Fox, N.A. (2018). Impact of early institutionalization on attention mechanisms underlying the inhibition of a planned action. *Neuropsychologia*, *117*, 339–46.
- Leitenberg, H., Gibson, L.E., Novy, P.L. (2004). Individual differences among undergraduate women in methods of coping with stressful events: the impact of cumulative childhood stressors and abuse. *Child Abuse & Neglect*, *28*(2), 181–92.
- Lim, L., Hart, H., Mehta, M.A., Simmons, A., Mirza, K., Rubia, K. (2015). Neural correlates of error processing in young people with a history of severe childhood abuse: an fMRI study. *American Journal of Psychiatry*, *172*(9), 892–900.
- Liu, Y., Fang, H., Duan, H. (2019). The relationship between chronic perceived stress and error processing: evidence from event-related potentials. *Scientific Reports*, *9*(1), 1–8.
- Loman, M.M., Johnson, A.E., Westerlund, A., Pollak, S.D., Nelson, C.A., Gunnar, M.R. (2013). The effect of early deprivation on executive attention in middle childhood. *Journal of Child Psychology and Psychiatry*, *54*(1), 37–45.
- Luijten, M., Machielsen, M.W., Veltman, D.J., Hester, R., de Haan, L., Franken, I.H. (2014). Systematic review of ERP and

- fMRI studies investigating inhibitory control and error processing in people with substance dependence and behavioural addictions. *Journal of Psychiatry & Neuroscience*, **39**(3), 149–69.
- Marsh, R., Gerber, A.J., Peterson, B.S. (2008). Neuroimaging studies of normal brain development and their relevance for understanding childhood neuropsychiatric disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, **47**(11), 1233–51.
- McDermott, J.M., Westerlund, A., Zeanah, C.H., Nelson, C.A., Fox, N.A. (2012). Early adversity and neural correlates of executive function: implications for academic adjustment. *Developmental Cognitive Neuroscience*, **2**, S59–66.
- McLaughlin, K.A., Green, J.G., Gruber, M.J., Sampson, N.A., Zaslavsky, A.M., Kessler, R.C. (2012). Childhood adversities and first onset of psychiatric disorders in a national sample of US adolescents. *Archives of General Psychiatry*, **69**(11), 1151–60.
- McLaughlin, K.A., Sheridan, M.A., Lambert, H.K. (2014). Childhood adversity and neural development: deprivation and threat as distinct dimensions of early experience. *Neuroscience and Biobehavioral Reviews*, **47**, 578–91.
- McLaughlin, K.A., Sheridan, M.A., Nelson, C.A. (2017). Neglect as a violation of species-expectant experience: neurodevelopmental consequences. *Biological Psychiatry*, **82**(7), 462–71.
- Moreno-López, L., Ioannidis, K., Askelund, A.D., Smith, A.J., Schueler, K., Van Harmelen, A.-L. (2020). The resilient emotional brain: a scoping review of the medial prefrontal cortex and limbic structure and function in resilient adults with a history of childhood maltreatment. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, **5**(4), 392–402.
- Moser, J., Moran, T., Schroder, H., Donnellan, B., Yeung, N. (2013). On the relationship between anxiety and error monitoring: a meta-analysis and conceptual framework. *Frontiers in Human Neuroscience*, **7**, 466.
- Mueller, S.C., Maheu, F.S., Dozier, M., et al. (2010). Early-life stress is associated with impairment in cognitive control in adolescence: an fMRI study. *Neuropsychologia*, **48**(10), 3037–44.
- Nieuwenhuis, S., Yeung, N., Van Den Wildenberg, W., Ridderinkhof, K.R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: effects of response conflict and trial type frequency. *Cognitive, Affective & Behavioral Neuroscience*, **3**(1), 17–26.
- Noble, K.G., McCandliss, B.D., Farah, M.J. (2007). Socioeconomic gradients predict individual differences in neurocognitive abilities. *Developmental Science*, **10**(4), 464–80.
- Notebaert, W., Houtman, F., Van Opstal, F., Gevers, W., Fias, W., Verguts, T. (2009). Post-error slowing: an orienting account. *Cognition*, **111**(2), 275–9.
- O’Connell, R.G., Dockree, P.M., Bellgrove, M.A., et al. (2007). The role of cingulate cortex in the detection of errors with and without awareness: a high-density electrical mapping study. *European Journal of Neuroscience*, **25**(8), 2571–9.
- Olvet, D.M., Hajcak, G. (2009). The stability of error-related brain activity with increasing trials. *Psychophysiology*, **46**(5), 957–61.
- Overbeek, T.J., Nieuwenhuis, S., Ridderinkhof, K.R. (2005). Dissociable components of error processing: on the functional significance of the Pe vis-à-vis the ERN/Ne. *Journal of Psychophysiology*, **19**(4), 319–29.
- Pine, D.S., Mogg, K., Bradley, B.P., et al. (2005). Attention bias to threat in maltreated children: implications for vulnerability to stress-related psychopathology. *American Journal of Psychiatry*, **162**(2), 291–6.
- Puetz, V.B., Viding, E., Palmer, A., et al. (2016). Altered neural response to rejection-related words in children exposed to maltreatment. *Journal of Child Psychology and Psychiatry*, **57**(10), 1165–73.
- Rubia, K., Smith, A.B., Taylor, E., Brammer, M. (2007). Linear age-correlated functional development of right inferior fronto-striato-cerebellar networks during response inhibition and anterior cingulate during error-related processes. *Human Brain Mapping*, **28**(11), 1163–77.
- Santesso, D.L., Dzyundzyak, A., Segalowitz, S.J. (2011). Age, sex and individual differences in punishment sensitivity: factors influencing the feedback-related negativity. *Psychophysiology*, **48**(11), 1481–9.
- Sehlmeyer, C., Konrad, C., Zwieterlood, P., Arolt, V., Falkenstein, M., Beste, C. (2010). ERP indices for response inhibition are related to anxiety-related personality traits. *Neuropsychologia*, **48**(9), 2488–95.
- Semlitsch, H.V., Anderer, P., Schuster, P., Presslich, O. (1986). A solution for reliable and valid reduction of ocular artifacts, applied to the P300 ERP. *Psychophysiology*, **23**(6), 695–703.
- Sheridan, M.A., Peverill, M., Finn, A.S., McLaughlin, K.A. (2017). Dimensions of childhood adversity have distinct associations with neural systems underlying executive functioning. *Development and Psychopathology*, **29**(5), 1777–94.
- Sheridan, M.A., McLaughlin, K.A. (2014). Dimensions of early experience and neural development: deprivation and threat. *Trends in Cognitive Sciences*, **18**(11), 580–5.
- Shonkoff, J.P., Garner, A.S., Siegel, B.S., et al. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, **129**(1), e232–46.
- Smith, J.L., Jamadar, S., Provost, A.L., Michie, P.T. (2013). Motor and non-motor inhibition in the Go/NoGo task: an ERP and fMRI study. *International Journal of Psychophysiology*, **87**(3), 244–53.
- Spielberg, J.M., Galarce, E.M., Ladouceur, C.D., et al. (2015). Adolescent development of inhibition as a function of SES and gender: converging evidence from behavior and fMRI. *Human Brain Mapping*, **36**(8), 3194–203.
- Swann, A.C., Bjork, J.M., Moeller, F.G., Dougherty, D.M. (2002). Two models of impulsivity: relationship to personality traits and psychopathology. *Biological Psychiatry*, **51**(12), 988–94.
- Tapia, G., Clarys, D., Bugajska, A., El-Hage, W. (2012). Recollection of negative information in posttraumatic stress disorder. *Journal of Traumatic Stress*, **25**(1), 120–3.
- Teicher, M.H. (2000). Wounds that time won’t heal: the neurobiology of child abuse. *Cerebrum*, **2**(4), 50–67.
- Teicher, M.H., Samson, J.A., Anderson, C.M., Ohashi, K. (2016). The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience*, **17**(10), 652–66.
- Teicher, M.H., Samson, J.A. (2013). Childhood maltreatment and psychopathology: a case for ecophenotypic variants as clinically and neurobiologically distinct subtypes. *American Journal of Psychiatry*, **170**(10), 1114–33.
- Teicher, M.H., Samson, J.A. (2016). Annual research review: enduring neurobiological effects of childhood abuse and neglect. *Journal of Child Psychology and Psychiatry*, **57**(3), 241–66.
- Tibu, F., Sheridan, M.A., McLaughlin, K.A., Nelson, C.A., Fox, N.A., Zeanah, C.H. (2016). Disruptions of working memory and inhibition mediate the association between exposure to institutionalization and symptoms of attention deficit hyperactivity disorder. *Psychological Medicine*, **46**(3), 529.
- Troller-Renfree, S., Nelson, C.A., Zeanah, C.H., Fox, N.A. (2016). Deficits in error monitoring are associated with externalizing but not internalizing behaviors among children with a history

- of institutionalization. *Journal of Child Psychology and Psychiatry*, *57*(10), 1145–53.
- van Noordt, S.J., Desjardins, J.A., Segalowitz, S.J. (2015). Watch out! Medial frontal cortex is activated by cues signaling potential changes in response demands. *NeuroImage*, *114*, 356–70.
- van Veen, V., Carter, C.S. (2006). Error detection, correction, and prevention in the brain: a brief review of data and theories. *Clinical EEG and Neuroscience*, *37*(4), 330–5.
- Velanova, K., Wheeler, M.E., Luna, B. (2008). Maturation changes in anterior cingulate and frontoparietal recruitment support the development of error processing and inhibitory control. *Cerebral Cortex*, *18*(11), 2505–22.
- Viola, T.W., Salum, G.A., Kluwe-Schiavon, B., Sanvicente-Vieira, B., Levandowski, M.L., Grassi-Oliveira, R. (2016). The influence of geographical and economic factors in estimates of childhood abuse and neglect using the childhood trauma questionnaire: a worldwide meta-regression analysis. *Child Abuse & Neglect*, *51*, 1–11.
- Watson, D., Clark, L.A., Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063–70.
- Weinberg, A., Riesel, A., Hajcak, G. (2012). Integrating multiple perspectives on error-related brain activity: the ERN as a neural indicator of trait defensive reactivity. *Motivation and Emotion*, *36*(1), 84–100.
- Wessel, J.R., Danielmeier, C., Morton, J.B., Ullsperger, M. (2012). Surprise and error: common neuronal architecture for the processing of errors and novelty. *Journal of Neuroscience*, *32*(22), 7528–37.
- Wessel, J.R., Aron, A.R. (2015). It's not too late: the onset of the frontocentral P3 indexes successful response inhibition in the stop-signal paradigm. *Psychophysiology*, *52*(4), 472–80.
- Wilcox, R.R. (2011). *Introduction to Robust Estimation and Hypothesis Testing*. Boston: Academic Press.
- Wilkowski, B.M., Robinson, M.D. (2008). Putting the brakes on antisocial behavior: secondary psychopathy and post-error adjustments in reaction time. *Personality and Individual Differences*, *44*(8), 1807–18.
- Wu, J., Feng, M., Liu, Y., et al. (2019). The relationship between chronic perceived stress and error processing: evidence from event-related potentials. *Scientific Reports*, *9*(1), 11605.