Neuroscience Letters 486 (2010) 117–121

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

Response inhibition in adolescent earthquake survivors with and without posttraumatic stress disorder: A combined behavioral and ERP study

Jianhui Wu[a,b], Yan Ge[a,b], Zhanbiao Shi[b], Xiaoju Duan[b], Li Wang[b], Xianghong Sun[a,b], Kan Zhang[a,b,*]

[a] State Key Laboratory of Brain and Cognitive Science, Institute of Psychology, Chinese Academy of Sciences, Beijing, China
[b] Institute of Psychology, Chinese Academy of Sciences, Beijing, China

ARTICLE INFO

Article history:
Received 19 June 2010
Received in revised form 16 July 2010
Accepted 19 July 2010

Keywords:
Posttraumatic stress disorder
Response inhibition
Event-related potentials
N2

ABSTRACT

The aim of this study was to investigate whether adolescent patients with posttraumatic stress disorder (PTSD) show an impairment of executive control in a response inhibition task and to investigate its neurophysiological correlates using event-related potentials (ERPs). We analyzed data from 25 Wenchuan earthquake survivors between 15 and 19 years of age (16 diagnosed with PTSD) using a Go/NoGo task. The PTSD group made more commission errors than the non-PTSD group, indicating impairment in response inhibition. The PTSD group responded faster to Go trials and there was a significant negative correlation between their reaction time and commission/omission errors, reflecting a speed-accuracy tradeoff for the PTSD group. The PTSD group exhibited a shorter NoGo-N2 latency than the non-PTSD group, suggesting faster monitoring or detection of the response conflict. These results suggest that the impairment of response inhibition in adolescent participants with PTSD is related to their impulsive cognitive functioning.

Posttraumatic stress disorder (PTSD) "arises as a delayed or protracted response to a stressful event or situation of an exceptionally threatening or catastrophic nature.... Typical features include episodes of repeated reliving of the trauma, dreams or nightmares, occurring against the persisting background of a sense of numbness and emotional blunting, detachment from other people, unresponsiveness to surroundings, anhedonia, and avoidance of activities and situations reminiscent of the trauma" [33]. PTSD is often accompanied by cognitive deficits, e.g., in attention [5,25,30,31] and working memory [31]. PTSD might also be associated with deficits in executive function [28]. Response inhibition, a major component of executive function [21], has been reported as being impaired in adult PTSD patients for both the Haying sentence completion task [18] and the Go/NoGo task [11].

In the literature, response inhibition has been generally assessed by the Go/NoGo task [10,16,17], which consists of two stimuli: a Go stimulus which requires a response and a NoGo stimulus that requires the inhibition of the prepotent response. Falconer et al. [11] utilized this Go/NoGo task to investigate PTSD-related changes in executive inhibition using functional magnetic resonance imaging (fMRI). Their results showed that adult participants with PTSD showed more commission errors (CE) than did individuals without trauma exposure and PTSD was associated with the disruption of cortical control networks.

In contrast to fMRI, event-related potentials (ERPs) have high temporal resolution and can provide more insight into the time course of brain processes and have been repeatedly used with PTSD patients using a variety of tasks [12,15,20,22]. To our knowledge, there is only one ERP study examining response inhibition in PTSD [25], and none in adolescent patients with PTSD. In Shucard et al.'s [25] ERP study, the response inhibition in adult PTSD patients was explored with the A–X continuous performance task. Their behavioral results, however, did not show a significant difference in commission errors between PTSD and non-PTSD groups.

In previous ERP studies using the Go/NoGo task, two major components have been consistently linked with response inhibition. The first component (NoGo-N2) is an enhanced negativity at approximately 200–400 ms post-stimulus onset in response to NoGo stimuli and may represent the process of conflict monitoring or detection, i.e., the conflict between the internal representation of the Go response and the NoGo stimulus [9,17,26]. The second component (NoGo-P3) is an enhanced positivity that is elicited within a 300–500 ms time window and may represent a later stage of the response inhibitory process, i.e., response evaluation/decision or the success of inhibiting a response [4,26]. Developmental research has revealed that the N2/P3 ERPs to NoGo stimuli are also present in both children and adolescents [14,19,23].

The goal of the current study was to investigate the response inhibition function of adolescent PTSD patients as compared with the trauma-exposed non-PTSD group under the Go/NoGo task with

* Corresponding author at: Institute of Psychology, Chinese Academy of Sciences, 4A Datun Road, Chaoyang District, Beijing 100101, China. Tel.: +86 10 6483 7096; fax: +86 10 6483 6956.
E-mail address: zhangk@psych.ac.cn (K. Zhang).

0304-3940/$ – see front matter © 2010 Published by Elsevier Ireland Ltd.
doi:10.1016/j.neulet.2010.07.040

Table 1
Subject characteristics in the PTSD and non-PTSD groups.

	PTSD ($n = 16$)		Non-PTSD ($n = 9$)		Group comparison	
	Mean	S.D.	Mean	S.D.		
Age (years)	16.0	1.0	16.3	1.1	$Z = -.71$	$p = .477$
Gender (male/female)	10/6	–	5/4	–	$\chi^2 = .12$	$p = .730$
Education (years)	9.1	.3	9.1	.3	$Z = -.10$	$p = .920$
STAI (state)	48.0	9.4	49.1	7.2	$Z = -.31$	$p = .755$
STAI (trait)	50.2	8.4	52.4	7.3	$Z = -.37$	$p = .712$
HSCL-25	58.5	11.2	57.0	12.9	$Z = -.23$	$p = .821$
PCL-C	50.0	8.9	42.1	8.5	$Z = -1.96$	$p = .051$

STAI: Spielberger State/Trait Anxiety Inventory; HSCL-25: Hopkins symptom checklist-25; PCL-C: PTSD Checklist-Civilian Version.

both behavioral and ERP measures. Consistent with the hypothesis that executive inhibition is impaired in PTSD, we predicted that the PTSD group would make more CE and that the increased CE in PTSD was not due to their lower response criteria but rather to the impaired response inhibition. Consistent with the hypothesis that the neural networks of inhibitory control are compromised in PTSD, we also predicted that the amplitudes of NoGo-N2/P3 would be reduced in the PTSD group as compared with the non-PTSD group.

Volunteers from Beichuan Vocational High School were initially screened for selection criteria. We only selected those (1) with the experience of the devastating earthquake in Wenchuan County in Sichuan Province, China on May 12, 2008, but (2) without past or current head injury, and (3) without self-reported neurological or major mental disorders, alcohol or substance use, and (4) without psychiatric treatment or medication following the earthquake. In total, 29 student survivors underwent diagnostic interviews and ERP recordings, which were performed in June, 2009, about 13 months after the earthquake, when their range was from 15 to 19 years old. Data from 25 students were used for further analysis after excluding another four students due to their self-reported and uncorrected short sight. Sixteen of them met the ICD-10 diagnostic criteria for PTSD, and nine of them did not meet the criteria. The groups were homogeneous for sex, age and years of education (see Table 1). All of them were right handed as determined by self-report. They gave informed consent and were paid for their participation.

The diagnosis of PTSD for the earthquake survivors was determined by a clinical psychologist using the ICD-10 [33], the International Classification of Diseases. In addition, all participants completed the Chinese version of the Spielberger State/Trait Anxiety Inventory (STAI) [27,36]; the Hopkins Symptom Checklist-25 (HSCL-25) [7]; and the Chinese version of the PTSD Checklist-Civilian Version (PCL-C) [32,34,35], a self-report inventory for assessing the symptoms of PTSD corresponding to the DSM-IV symptom clusters of reexperiencing, avoidance/numbing, and hyperarousal [1].

Participants were seated in a normally illuminated room. Electrophysiological data were obtained under three experimental paradigms: Go/NoGo, S1-S2, and auditory oddball paradigm. Only data for the first paradigm are presented in this paper. These paradigms were counterbalanced for order of presentation. For the Go/NoGo paradigm, we used the same stimuli and procedure as a previously published paper [10].

Working with EEG recording and pre-processing generally is same as described in the previously published paper [10] with two modifications. (1) The EEG data were epoched into periods of 700 ms (including a 100 ms prestimulus baseline) time-locked to the onset of the presented number. (2) Trials with various artifacts were rejected, with a criterion of $\pm 70\,\mu V$.

Differences between the groups for subject characteristics and behavioral data, such as the reaction time (RT) of the correct trials, the rate of omission errors (OE) in Go trials, and the rate of CE in NoGo trials, were compared using non-parametric Mann–Whitney tests. We also calculated response criterion (β) as the ratio of the ordinate of the hit rate to the ordinate of the CE rate according to signal detection theory, $\beta = [y(\text{hit rate})]/[y(\text{commission errors rate})]$, and the d-prime (d') as a difference between the z-score values for the hit rate and CE rate [29]. For ERP data, the peak amplitude and latency of the N2 and P3 were measured in each condition at the following five sites: Fz, FCz, Cz, CPz and Pz. Measurements of a peak at different electrodes in a single subject and experimental condition were taken at the same latency. If the peak was maximal at one electrode location, its latency at this location was used [24]. With the relatively small sample size, non-parametric distribution-free tests were employed for ERP data analysis including the Mann–Whitney tests for between-group comparison (PTSD and non-PTSD) and the Wilcoxon signed-ranks tests for within-group comparisons (Go and NoGo). Behaviorally incorrect trials were not included in the ERP averages. All p values $\leq .05$ were considered statistically significant.

As shown in Table 1, the scores of PCL-C were marginally significantly higher in the PTSD group compared to the non-PTSD group. The STAI and HSCL-25, however, did not show any significant differences between the two groups.

As shown in Table 2, the PTSD group committed significantly faster and made marginally significant more OE in Go trials, and made significantly more CE in NoGo trials than did the non-PTSD group. There was no significant difference between the two groups in the response criterion (β), but the non-PTSD group showed a higher d' than did the PTSD group.

The behavioral results revealed that PTSD subjects made more CE to NoGo trials but responded faster to Go trials, thus we developed a post hoc hypothesis that these behavioral differences between the two groups could be explained by the difference in speed-accuracy tradeoff (SAT) characteristics. SAT analysis would help to elucidate the mechanism underlying the predicted impaired response inhibition in PTSD [13]. Here, we used the two-tailed Spearman correlation coefficient to investigate SAT characteristics in both groups. For example, if the speed-up is achieved at the expense of accuracy, a negative correlation should be found between rate of OE/CE and RT. The results are illustrated in Fig. 1: a negative correlation was found between RT and OE or CE for the PTSD group, but not for the non-PTSD group. We also statistically compared correlation coefficients between the two groups, but did not find significant differences (correlation of OE and RT: $Z = .66$, $p > .05$; correlation of CE and RT: $Z = 1.04$, $p > .05$).

As illustrated in both Fig. 2 and Table 3, the amplitude of N2 was larger for NoGo than for Go stimuli for both groups, and the stimulus difference effect were maximal at the CPz and Pz sites and minimal or absent at the Fz and FCz sites. There was no significant difference between two groups for both Go and NoGo stimuli. For the latency, the PTSD group elicited a shorter latency of NoGo-N2 than that of the non-PTSD group.

The amplitude of P3 was larger for NoGo than for Go stimuli at Fz but was smaller at CPz and Pz for the PTSD group. For the non-PTSD group, the amplitude of P3 was smaller for NoGo than for Go stimuli at Pz, but the frontal NoGo effect did not achieve statistical

Table 2
Behavioral performance compared between the PTSD and non-PTSD groups.

	PTSD (n = 16)		Non-PTSD (n = 9)		Group comparison	
	Mean	Q.D.	Mean	Q.D.	Z	p
RT (ms)	336	21	378	34	−2.49	.013
CE (%)	10.0	5.3	4.0	2.4	−2.10	.036
OE (%)	8.7	3.3	2.3	1.7	−1.80	.072
β	.66	.38	.59	.43	−.34	.690
d'	4.08	.85	6.41	3.25	−2.04	.041

RT: reaction time of Go trial; CE: rate of commission error; OE: rate of omission error; β: response criterion; d': d-prime; Q.D.: quartile deviation.

significance. For the latency, the Go stimulus elicited a significantly shorter latency of P3 than that of the NoGo stimulus for both groups.

This study represents the first demonstration of both ERP and behavioral performance associated with the impairment of response inhibition in adolescent PTSD patients. The main findings can be summarized as follows. Behaviorally, the PTSD group had more CE in NoGo trials but responded faster in Go trials than those trauma-exposed non-PTSD subjects. The RT in Go trials negatively correlated with both OE and CE for the PTSD subjects. This correlation, however, did not occur for non-PTSD subjects. For the indexes according the signal detection theory, the two groups had the same level of response criteria (β), and non-PTSD showed a higher sensitivity (d') than PTSD. For ERP, the PTSD group elicited a shorter latency of NoGo-N2 than that of the non-PTSD group.

The PTSD group made more CE on the Go/NoGo task. This result replicated the previous research in adults subjects [11] and confirmed the impairment of response inhibition for adolescent PTSD subjects. In Falconer et al.'s study [11], PTSD subjects made more commission errors as compared with individuals without trauma exposure. In the present study, we found that this difference also occurred between PTSD subjects and trauma-exposed non-PTSD subjects. Further analysis revealed that the increased CE in PTSD was not due to their lower response criteria. Analysis using signal detection theory also revealed that the PTSD group had a lower sensitivity than non-PTSD, indicating their impaired ability to discriminate Go stimulus (signal) from NoGo stimulus (noise).

Surprisingly, our results also showed that the PTSD group responded faster to Go trials than did non-PTSD subjects. In the present study, both PTSD and non-PTSD subjects were instructed to respond as quickly and accurately as possible, but the behavioral results varied between the two groups in different directions for speed and accuracy, i.e., greater speed but lower accuracy for PTSD subjects. Further analysis revealed a negative correlation between RT and OE/CE for PTSD subjects but not for non-PTSD subjects. These results suggest the speed-accuracy tradeoff characteristics for the PTSD subjects, i.e., the greater speed is at the cost of lower accuracy and sensitivity, which might reflect impulsive responsiveness for PTSD individuals during the task [8]. The impulsivity might be associated with "a state of autonomic hyperarousal with hypervigilance, and enhanced startle reaction" in PTSD [33].

Interestingly, the latency of the N2 NoGo effect followed the same trend as the RT to Go trials, i.e., the PTSD group elicited a shorter latency of NoGo-N2 than that of the non-PTSD group. According to the literature, the N2 NoGo effect may represent the earlier step of response inhibition, i.e., the monitoring or detection

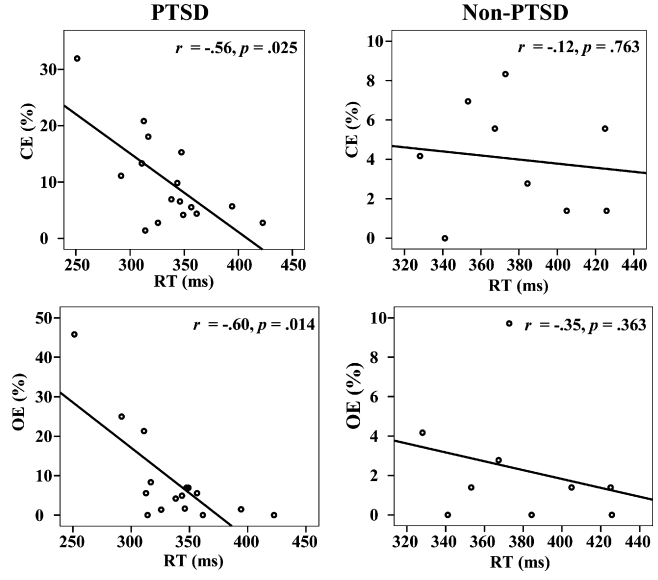

Fig. 1. Scatter plots and Spearman correlation coefficients showing the negative correlations between RT and CE/OE for PTSD but not for non-PTSD subjects. RT: reaction time of Go trial; CE: rate of commission error; OE: rate of omission error.

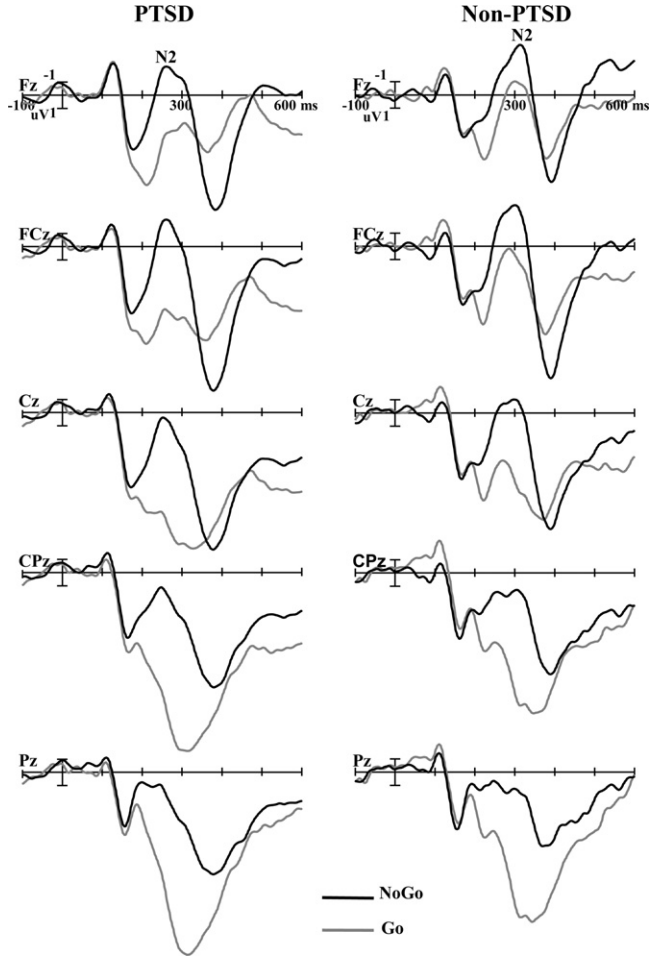

Fig. 2. Grand averaged ERPs illustrating Go and NoGo trials for both PTSD (left panel) and non-PTSD subjects (right panel).

Table 3
Means (standard deviations of the mean) for the peak amplitude and latency of N2/P3 components to Go and NoGo stimuli for the PTSD and non-PTSD groups, and comparisons between two stimuli for both groups and between two groups for both stimuli.

	PTSD ($n = 16$)				Non-PTSD ($n = 9$)				Group comparison			
	Go	NoGo	Comparison		Go	NoGo	Comparison		Go		NoGo	
			Z	p			Z	p	Z	p	Z	p
N2 amp (μV)												
Fz	.0 (7.7)	−4.6 (6.6)	−2.69	.007	−2.9 (3.7)	−4.6 (4.6)	−.89	.374	−.74	.462	−.11	.910
FCz	2.2 (7.8)	−4.8 (7.3)	−3.52	.000	−1.0 (3.4)	−4.5 (6.7)	−1.60	.110	−1.70	.089	−.11	.910
Cz	6.0 (7.9)	−2.5 (6.6)	−3.52	.000	3.5 (3.4)	−2.2 (6.5)	−2.07	.038	−1.08	.282	−.00	1.000
CPz	9.1 (8.5)	−1.5 (6.2)	−3.52	.000	7.6 (4.2)	.6 (4.8)	−2.31	.021	−.34	.734	−.45	.651
Pz	8.8 (8.8)	−1.1 (6.4)	−3.52	.000	8.6 (3.9)	1.0 (2.7)	−2.55	.011	−.23	.821	−.85	.396
N2 lat (ms)	275 (44)	263 (41)	−1.51	.132	302 (33)	298 (25)	−.71	.477	−1.50	.133	−2.35	.019
P3 amp (μV)												
Fz	5.5 (5.8)	10.1 (4.0)	−2.33	.020	3.1 (6.6)	7.1 (6.9)	−1.60	.110	−1.02	.308	−1.25	.213
FCz	9.1 (5.4)	12.2 (4.5)	−1.81	.070	5.7 (6.8)	10.3 (6.9)	−1.48	.139	−1.25	.213	−.79	.428
Cz	13.1 (5.1)	11.7 (3.8)	−.83	.408	9.5 (5.6)	9.2 (6.2)	−.42	.678	−1.81	.070	−1.13	.258
CPz	16.2 (5.6)	9.8 (4.6)	−3.26	.001	12.8 (5.1)	8.2 (5.1)	−1.72	.086	−1.25	.213	−.51	.610
Pz	16.0 (6.4)	8.4 (5.5)	−3.36	.001	13.2 (3.8)	5.8 (4.9)	−2.43	.015	−1.19	.234	−1.02	.308
P3 lat (ms)	332 (43)	387 (33)	−3.18	.001	335 (33)	391 (18)	−2.67	.008	−.37	.712	−.45	.650

amp: amplitude; lat: latency.

of response conflict [9,17,26]. Thus the shorter latency of N2 for the PTSD group suggests that PTSD subjects are faster in the timing of conflict monitoring, and at first glance this inference contrasts with the hypothesis of cognitive deficits for PTSD subjects.

Results from brain imaging and neuropsychology indicate the enhanced motor cortical activation in PTSD in response to both fear/trauma-related stimuli [2,3] and NoGo neutral stimuli [11] and increased excitability of the motor cortex in response to transcranial magnetic stimulation [6], suggesting there may be "an enhanced motor readiness or an increased prepotency to respond", and then an "increased demand on inhibitory control systems" and "more urgent inhibition" in PTSD [11]. The ERP results of the present study, i.e., the shorter latency of the N2 NoGo effect in the PTSD subjects, provide the temporal information about brain activity in PTSD subjects during response inhibition and suggest the impulsive cognitive and brain function which lead to rapid RT in Go trials and more CE in NoGo trials.

There are some limitations to this study that need to be addressed. Firstly, the sample sizes were relatively small, which might lead to the failure of other indexes to reach significant differences. Secondly, these results found here may be explained by co-morbid disorders such as major depression, generalized anxiety disorder and panic disorder [25,31]. Due to time limitations and poor local clinical practice, participants in this study were not formally examined for co-morbid disorders, apart from that none of our subjects reported they have suffered from neurological or major mental disorders, alcohol or substance use. The insignificant differences between groups on the scores of STAI (state and trait) and HSCL-25 suggest the possibility that group differences found here are not confounded by co-morbid disorders.

In conclusion, adolescent PTSD subjects showed an impairment of executive control in a response inhibition task as compared with those trauma-exposed non-PTSD subjects. Both the shorter latency of the N2 NoGo effect and the speed-accuracy tradeoff characteristics suggest impulsive cognitive functioning in the adolescent PTSD subjects which might underlie their impairment of response inhibition.

Acknowledgements

This work was supported by the NSF China (90924017, 30900442). We express great appreciation to the teachers and students of Beichuan Vocational High School for their courage after experiencing the earthquake and their support in this study. We also acknowledge Richard Carciofo for his editorial assistance.

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