



# The effects of child abuse and neglect on cognitive functioning in adulthood

Felicia Gould<sup>a</sup>, Jennifer Clarke<sup>a,b</sup>, Christine Heim<sup>c</sup>, Philip D. Harvey<sup>a</sup>, Matthias Majer<sup>c</sup>, Charles B. Nemeroff<sup>a,\*</sup>

<sup>a</sup> Department of Psychiatry and Behavioral Sciences, University of Miami Leonard M. Miller School of Medicine, Miami, FL, USA

<sup>b</sup> Department of Epidemiology and Public Health, University of Miami Leonard M. Miller School of Medicine, Miami, FL, USA

<sup>c</sup> Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, Atlanta, GA, USA

## ARTICLE INFO

### Article history:

Received 7 October 2011

Received in revised form

23 December 2011

Accepted 5 January 2012

### Keywords:

ELS

Cognition

Child abuse

## ABSTRACT

**Aims:** Recent research has revealed that early life trauma (ELS), including abuse (sexual and/or physical) and neglect, produce lasting changes in the CNS. We posited that cognitive deficits, often observed in psychiatric patients, result, in part, due to the neurobiological consequences of ELS. Additionally, we hypothesized that the nature and magnitude of cognitive deficits would differ according to the subtype of ELS experienced.

**Method:** The Cambridge Neuropsychological Test Automated Battery (CANTAB) was used to assess neurocognitive functioning in 93 subjects (60 with ELS and 33 without). In the patients with a history of ELS, 35% and 16.7%, respectively, met criteria for current major depression and PTSD.

**Results:** Significant associations between ELS status and CANTAB measures of memory and executive and emotional functioning were found.

**Conclusions:** These data suggest that exposure to ELS results in a cascade of neurobiological changes associated with cognitive deficits in adulthood that vary according to the type of trauma experienced.

© 2012 Elsevier Ltd. All rights reserved.

## 1. Introduction

There is evidence that trauma exposure early in life (referred to as early life stress or ELS) markedly increases the risk for major depression, bipolar disorder, schizophrenia, and post traumatic stress disorder (PTSD), all syndromes characterized by cognitive dysfunction. Numerous studies have observed deficits in executive functioning, attention and concentration, memory and processing speed in patients with depression (McClintock et al., 2010; Nebes et al., 2000), yet it is clear that not all depressed persons suffer such impairment (Porter et al., 2007), and trauma exposure may offer one explanation for variability in these findings. In addition to neuropsychological dysfunction, depressed patients are well known to display deficits in emotional processing (Elliott et al., 2002; Fales et al., 2008). Mounting evidence from functional neuroimaging studies suggests that neural circuit abnormalities

underlie cognitive and emotional processing deficits in patients with major depressive disorder (MDD). Specifically, hypoactivity in the dorsal lateral prefrontal cortex, and the anterior cingulate cortex have been reported (Fales et al., 2008; Davidson et al., 2002). A growing body of literature suggests cognitive abnormalities including executive and attentional deficits, deficits in short-term and working memory for both visual and verbal material and psychomotor speed dysfunction characterize MDD even in young adulthood (Castaneda et al., 2008). Amongst patients with MDD, executive and psychomotor impairments are more pronounced in the elderly, severely melancholic, psychotic and bipolar populations (Porter et al., 2007). Deficits in patients with MDD appear to persist even in the remitted state (Hasselbalch et al., 2010). Among patients with anxiety disorders in general, deficits in episodic memory and executive functioning have been found (Castaneda et al., 2008), but significant heterogeneity exists amongst these patients depending on their specific anxiety disorder subtype. For example, a relatively broader range of impairments in intellectual functioning, attention/working memory, processing speed, learning, and executive functioning have been found in patients with PTSD (Yehuda et al., 1995; Vasterling et al., 2002; Liberzon and Sripada, 2008). Functional

\* Corresponding author. Department of Psychiatry and Behavioral Sciences, University of Miami Leonard M. Miller School of Medicine, 1120 NW 14th Street, #1455, Miami, FL 33136, USA. Tel.: +1 404 727 8382.

E-mail address: [cnemeroff@med.miami.edu](mailto:cnemeroff@med.miami.edu) (C.B. Nemeroff).

and structural imaging correlates of cognitive deficits in PTSD have also been identified (Liberzon and Sripada, 2008; Woodward et al., 2009). There may be a bidirectional and temporal relationship between PTSD, the hippocampus and neuropsychological functioning in that structural and functional brain abnormalities may either precede the development of PTSD, or emerge after the onset of PTSD (Woodward et al., 2009; Vasterling and Verfaellie, 2009; Yehuda et al., 2007). However, no consistent link between hippocampal volume and cognition has emerged (Woodward et al., 2009) and alterations in hippocampal volumes may be a risk factor for the development of PTSD (Yehuda et al., 2007). Instead, a more fronto-striatal pattern of neuropsychological impairment has emerged (Golier and Yehuda, 2002; Twamley et al., 2009) and implicates the importance of anterior brain regions such as the prefrontal cortex.

Trauma exposure can occur throughout the lifespan and the time-frame for exposure may have critical implications for life-long functioning. Previous research has indicated that prenatal stress and stress throughout childhood both alter stress reactivity. Such abnormal development and subsequent increases in reactivity have been found to result in vulnerability to mood and anxiety disorders (Nemeroff, 2004). Nonhuman primate studies have indicated that sensitization to fear cues, corticotropin-releasing factor (CRF), neuronal hyperactivity and hypercortisolism may all result from early life stress (Coplan et al., 1996). Heim et al. (2000) found that adult women with a history of ELS display increased sensitivity of the HPA axis and autonomic nervous system responses to stress. Twamley et al. (2009) investigated cognitive functioning in women with PTSD related to intimate partner violence and found that they demonstrated slower processing speed, poor reasoning performance, and dissociative symptoms proportionate to PTSD severity. Recent data suggests that the full impact of ELS on the HPA axis may not be observed until adolescence, but gender and timing of stress exposure are likely to modify neurobiological vulnerability to mood and anxiety disorders, yet more data is needed to determine precise critical periods for the maximum impact of trauma exposure (Andersen and Teicher, 2008; Heim and Nemeroff, 2009). Children with a history of ELS have been found to perform more poorly on intellectual, cognitive and achievement tasks and ELS exposure is broadly associated with both a high probability of neurocognitive dysfunction and real world functional impairment (Twamley et al., 2009; De Bellis et al., 2009). A range of disparate cognitive deficits have been associated with functional impairment in psychiatric patients across different neuropsychiatric conditions (Twamley et al., 2009; Wingo et al., 2009). Memory, executive functioning and psychomotor speed deficits have been uncovered by the majority of studies investigating neuropsychological dysfunction in patients with MDD (Porter et al., 2007), which is consistent with the fronto-striatal profile of cognitive impairments seen in other conditions where trauma exposure may be an etiological factor, including PTSD and, perhaps, schizophrenia. Inconsistent findings may have arisen from the variation in potential etiological factors, including ELS, the influence of which has not been well characterized in investigations of neuropsychological functioning in MDD.

Only two previous studies (Majer et al., 2010; Pluck et al., 2011) have explicitly tested whether the cognitive deficits observed in abused or neglected children persist into adulthood. The Majer et al. (2010) study excluded patients with both ELS and current depression in adulthood. The Pluck et al. (2011) study included a sample comprised exclusively of homeless individuals with subaverage IQs. Our current study examined the impact of ELS on relatively healthy, community dwelling adults via a battery of

cognitive tests selected to measure the pattern of deficits observed in previous studies of PTSD and MDD. The present study included a predominantly female civilian population with a history of ELS as defined by a history of either sexual, physical, emotional abuse, or neglect. We hypothesized that deficits in memory, executive functioning, processing speed, and emotional processing would distinguish ELS patients from controls.

## 2. Method

### 2.1. Participants

The sample was comprised of 93 subjects (28 men and 65 women) 18–45 years old ( $M = 29.83$ ,  $SD = 7.54$  years) who had valid assessment data. Subjects were recruited as part of the Conte Center for the Psychobiology of Early-Life Trauma (MH58922) and included subjects with or without exposure to ELS before the age of 13 years and with or without a diagnosis of MDD. MDD was defined by a diagnosis of current MDD according to the Diagnostic and Statistical Manual for Mental Disorder 4th Edition (DSM-IV). For the present analysis, subjects were classified into those with and without a history of ELS on Childhood Trauma Questionnaire (CTQ) scores (First et al., 1997) as described below. Exclusion criteria were current unstable medical illness, lifetime history of psychosis or bipolar disorder, meeting DSM-IV criteria for alcohol or substance abuse within 6 months, heavy smoking ( $>20$  cigarettes/day) or eating disorders within the past year. None of the participants was currently receiving psychotherapy or medications. All subjects were recruited from responses to advertising in local newspapers and in the public transportation system and screened for eligibility. Eligible subjects were invited and paid for their time if they participated. After description of the study to participants, written informed consent was obtained. The study was approved by the Institutional Review Board of Emory University School of Medicine.

### 2.2. General procedure

Subjects were admitted as inpatients for 2.5 days to the Atlanta Clinical and Translational Science Institute, Emory University Hospital Clinical Interaction Site, which provides a standardized setting for clinical studies. For the diagnosis of lifetime and current MDD and other psychiatric disorders the Structured Clinical Interview for DSM-IV (SCID) (First et al., 1997) was administered to patients and controls. Depression symptom severity was measured using the 21-item Hamilton Depression Rating Scale (HAM-D-21) (Williams, 1988). Exposure to ELS was assessed using the CTQ.

The CTQ is a self-report questionnaire that measures five categories of childhood trauma experience, including emotional, physical, and sexual abuse, and emotional and physical neglect. Each subscale is measured in 5 items rated on a 5-point Likert scale from 1 = “Never true” through 5 = “Very often true”. In addition, scores for none–low, low–moderate, moderate–severe, and severe–extreme exposure have been developed for each subscale. Good specificity and sensitivity of cutoff scores to classify maltreated subjects has been reported (Bernstein and Fink, 1998).

We used the moderate–severe cutoff scores for each subscale to classify subjects as positive for a history of childhood trauma in that category (Bernstein and Fink, 1998). Cases classified as positive on any one subscale were considered to have exposure to ELS, whereas cases classified as negative on all subscales were designated as not exposed to trauma. A total of 60 subjects were classified as positive and 33 were classified as negative for a history of ELS. On the

Download English Version:

<https://daneshyari.com/en/article/327409>

Download Persian Version:

<https://daneshyari.com/article/327409>

[Daneshyari.com](https://daneshyari.com)