Journal of Psychiatric Research 43 (2009) 1146–1155

Contents lists available at ScienceDirect

Journal of Psychiatric Research

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

After facing traumatic stress: Brain activation, cognition and stress coping in policemen

Kristina Hennig-Fast [a,*], Natalie S. Werner [b], Ramona Lermer [a], Knut Latscha [c], Franziska Meister [a], Maximilian Reiser [d], Rolf R. Engel [a], Thomas Meindl [d]

[a] Department of Psychiatry, Section of Neurocognition, Ludwig-Maximilians-University of Munich, Nussbaumstr. 7, D-80336 München, Germany
[b] Department of Psychology, Ludwig-Maximilians-University of Munich, Germany
[c] Police University of Baden-Würtemberg, Germany
[d] Department of Radiology, Ludwig-Maximilians-University of Munich, Germany

ARTICLE INFO

Article history:
Received 5 December 2007
Received in revised form 16 January 2009
Accepted 1 March 2009

Keywords:
Trauma
Stress coping
Resilience
fMRI
Associative memory and cognition

ABSTRACT

Introduction: Resilience can be defined as the capacity to recover following stress or trauma exposure by adopting healthy strategies for dealing with trauma and stress. Although the importance of stress resilience has been recognized, the underlying neurocognitive mediators have not yet been identified. Thus, the primary goal of this study was to investigate memory-related brain activity in traumatized policemen who attended a pre-traumatic general stress coping program.

Method: Ten traumatized male police officers were compared to demographically matched non-traumatized officers ($n = 15$) on associative memory by using a block design paradigm. Participants with either another psychiatric comorbidity or neurological disorder were excluded.

During functional brain imaging (1.5-Tesla), face-profession pairs had to be encoded twice. For subsequent retrieval the faces were presented as cue stimuli for associating the category of the prior learned profession. Additionally, clinical pattern, stress coping style, and cognitive parameters were assessed.

Results: Less BOLD activation was found in the hippocampus, parahippocampal gyrus and fusiform gyrus in the trauma group when compared with the non-trauma group during encoding. This was accompanied by slower reaction times in the trauma group during retrieval. Further impairments were found in context memory and in the use of positive cognitive coping strategies.

Discussion: Support was provided for the presence of memory-related disturbances in brain activity associated with trauma even in a resilient population. The contribution of the changes in stress coping ability needs to be further examined in longitudinal studies.

1. Introduction

Psychological trauma is an inevitable part of human experience and affects many dimensions of a person producing a correspondingly wide range of psychological symptoms (Scaer, 2005). There are individual differences in vulnerability as well as in resilience factors for traumatic stress. Measuring and understanding resilience therefore involves understanding the relationships between vulnerability, trauma exposure and the development of psychopathology.

From a psychological perspective resilience is defined as the capacity to recover following exposure to stress or trauma through the flexible adoption of healthy strategies in the face of trauma, adversity and stress (Block and Kremen, 1996; Lazarus, 1993; Masten, 2001). A common approach to studying the effects of trauma and stress involves identification of risk factors that make individuals especially vulnerable to stress-related disorders. This approach is useful in many respects; however, still little is known about participants who either do not demonstrate similar trauma and stress responses to people with PTSD (i.e., stress resistant people) or those whose traumatic response is of shorter duration to that of people with PTSD and does not lead to long-term responses (i.e., stress or trauma resilient people) in spite of being exposed to comparable traumatic situations. Therefore, identifying mechanisms that make individuals less vulnerable to traumatic stimuli is important. Active and instrumental coping strategies have been associated with a good adaptation to traumatic stress (positive stress coping), while more passive or avoidant strategies are often considered as maladaptive negative coping strategies (e.g. Resnick et al., 1992; for review: Linley and Joseph, 2004). In general, strategies involving disengagement from coping with the trauma increase the likelihood of experiencing ongoing distress and of developing a post-traumatic stress disorder (PTSD).

* Corresponding author. Tel.: +49 89 5160 5565; fax: +49 89 5160 5562.
E-mail address: kristina.fast@med.uni-muenchen.de (K. Hennig-Fast).

0022-3956/$ - see front matter © 2009 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jpsychires.2009.03.001

Several trauma theorists suggest that cognitive factors have an important impact on the trauma response (Foa et al., 1989; Ehlers and Steil, 1995). A fundamental assumption of many of these cognitive models of PTSD is the notion that perception of a stressful event as a threat may be at least as important as trauma severity and variations in pre-trauma experience in the development and maintenance of PTSD (Janoff-Bulman, 1985; Horowitz, 1986; Foa et al., 1989; Ehlers and Clark, 2000). In addition to appraisal of the traumatic event, a link between appraisals of acute symptoms and PTSD has been postulated (Foa and Riggs, 1993; Ehlers and Steil, 1995), principally because this leads to a sense of serious, current threat (Ehlers and Clark, 2000).

Recent findings indicate an association between maladaptive coping styles and autonomic reactivity (Bonanno et al., 2003; Mason et al., 2001). Preliminary data suggested an association between adaptive coping styles (self-enhancement) and salivary cortisol levels supporting a neuroendocrine response that is related to resilience. In PTSD a low secretion of cortisol and a high secretion of catecholamine in urine, with a norepinephrine/cortisol ratio was found to be higher than in comparable non-diagnosed individuals (Mason et al., 1988). According to Marshall et al. (2002) the functional hypothalamic-pituitary-adrenal (HPA) axis and noradrenergic profiles of PTSD appear unambiguously different from those of panic disorder. PTSD has been characterized by lower baseline cortisol levels, baseline 3-metoxy-4-hydrophenylglycol (MHPG) and reduced MHPG volatility and marginally reduced cortisol volatility compared to patients with panic disorder (Marshall et al., 2002). The HPA axis abnormalities are likely predicated on strong negative feedback inhibition of cortisol (Yehuda, 2001). This is in contrast to the normative fight/flight response, in which both cortisol- and catecholamine levels are elevated after exposure to a stressor (Bonanno, 2004, for review: Olff et al., 2005).

Together these findings give a pathophysiological explanation for PTSD by a maladaptive learning pathway to fear response through a hypersensitive, hyperreactive and hyperresponsive HPA axis. (e.g. Delahanty et al., 2005; Morgan et al., 2004; Schelling et al., 2004; Yehuda, 2002).

In addition to biochemical changes, PTSD also involves changes in brain morphology. Hippocampal volume has been previously linked with both PTSD and PTSD risk: hippocampal volume was frequently reported to be reduced in PTSD (e.g. Bremner, 2006; Hull, 2002; Karl et al., 2006; Smith, 2005). Although several studies investigating PTSD following stress exposure failed to find smaller hippocampal size (e.g. Bonne et al., 2001; DeBellis et al., 2002), evidence from other studies add support to the notion that it is a significant factor in PTSD. In a study by Gurvits et al. (1996), Vietnam veterans with PTSD showed a 20%-reduction in the volume of their hippocampus compared with those who did not suffer PTSD-like symptoms.

Two further studies illustrate the link between reduced hippocampal size and PTSD, albeit with contrasting theoretical interpretations. Gilbertson et al. (2002) suggest that a preexisting reduced hippocampal size could cause cognitive-emotional dysregulation and thereby increase vulnerability to PTSD (Gilbertson et al., 2002). A study Winter and Irle (2004) strengthens the case that hippocampal volume reduction in trauma-exposed individuals is the result of traumatic stress. By their attempt to differentiate the biological correlates of risk, PTSD and resilience following trauma exposure Yehuda and Flory (2007) found that people classified as more resilient had a better capacity to cope than people classified as vulnerable. The putative measure of PTSD risk, a small hippocampal volume, was inversely associated with the measure of resilience, good coping capacity.

It is generally accepted that the hippocampus is involved in the generation and recollection of episodic memories, in the formation of spatial and temporal associations and the consolidation of associative material into long-term storage (e.g. Aggleton and Brown, 1999; Mayes et al., 2002; Henke et al., 2003). Several studies provide support for the idea that memory deficits in PTSD exist and that they are associated with hippocampal damage (e.g. Bremner et al., 1995a,b, 2003; Geuze et al., 2007; Gurvits et al., 1996). Based on those findings it has been postulated that exposure to a traumatic event may itself result in severe alterations of trauma-associated memory functioning, including memory fragmentation, memory disorganization and dissociation of trauma-related memories from other memories (e.g. Brewin, 2001; Zoellner et al., 2000). However, little is currently known about brain activity for neutral, non-trauma-related associative memory in PTSD. One fMRI study seeking to address this issue provided evidence that in PTSD patients a deactivation of the frontal cortex, together with increased activation of the temporal cortex, were neural correlates for the encoding of neutral (non-trauma-related) associative words (Geuze et al., 2007). Two imaging studies investigating hippocampal size and memory in traumatized policemen with and without PTSD (Lindauer et al., 2006, 2004) demonstrated reduced hippocampus size in traumatized policemen with PTSD, but no association between hippocampal volume and memory performance. Thus, they concluded that memory impairment in PTSD does not seem to be a direct consequence of hippocampal size.

Taking these findings in PTSD and trauma together, it is still not possible to say whether trauma exposure is associated with altered brain activity, memory and stress coping style in resilient populations. Hence, we performed a study on participants drawn from the police force to investigate the effects of trauma exposition in a resilient population on the functional role of the hippocampus, parahippocampus and other brain structures during an episodic associative memory task. As well as this, cognitive functioning and the use of coping strategies for stress were assessed.

We hypothesized that (a) resilient traumatized policemen would show different coping styles when compared to non-traumatized policemen, (b) that the resilient traumatized policemen would not show the typical coping pattern of patients developing PTSD, and (c) the fMRI response to a memory paradigm would differ between the traumatized and non-traumatized participants despite normal level of cognitive functioning.

2. Methods and materials

2.1. Participants

A sample of 35 policemen was recruited from the Central Psychological Service of the Bavarian police force: 17 officers who had experienced a traumatic event and 18 healthy policemen who never had experienced a trauma. For all participants, exclusion criteria included substance dependence, current or prior psychiatric or neurological diagnoses, a history of major head trauma, excessive weight and any magnetic metals in their body. Inclusion criteria encompassed an age range between 18 and 55, an at least average level intelligence (above 85 in a German version of a vocabulary test [Wortschatztest, Schmidt and Metzler, 1992]) and German as their first language. All included participants attend a stress management training course (PAKET, Polizeiliches Antistress Kommunikatives Einsatztraining) that is offered by the Bavarian police to enhance stress coping ability before being exposed to traumatic stress. This training is based on behavioural therapy designed to enhance social skills, stress and conflict management (Murck and Schmalzl, 1992).

To decide whether a policeman could be assigned to the trauma group, the German version of the *Structured Clinical Interview for*