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

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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).

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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*

DSM-IV I and II (*SCID*, German Version: Wittchen et al., 1997) and the Clinical Administered PTSD Scale (CAPS, German version: Schnyder and Moergeli, 2002) were used.

Out of the 17 traumatized policemen, seven had to be excluded because of the above mentioned exclusion criteria. Two traumatized policemen met the exclusion criteria on psychological grounds: one participant had a prior lifetime PTSD developed during childhood and a second reported a major depression once in his life and was found to have a depressive personality trait. In addition, five policemen had to be excluded because either the fMRI data were incomplete or they were not assessable due to physical constraints ($n = 2$), movement artifacts ($n = 2$), or inattention and fatigue ($n = 1$). Finally, all the remaining 25 participants (traumatized policemen = 10; non-traumatized policemen = 15) were German and fully employed in the police service.

All traumatized policemen experienced human-induced traumata whilst on duty. However, the impact of trauma was limited as nine out of ten were short-term or sudden, and not repeated. The DSM-IV criteria A1 and A2 were fulfilled for all traumatized participants. Each was exposed to an extreme traumatic stressor involving direct personal experience of an event that involved actual or threatened death or serious injury, or other threat to one's physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate (Criterion A1). The person's response to the event must involve intense fear, helplessness, or horror (Criterion A2). Nevertheless, the nature of the trauma varied largely: two policemen had been personally involved in severe accidents involving fatalities, two were involved in life endangering rescue attempts, two were threatened physically by an aggressor, two had experienced an armed confrontation, one was deployed during war and finally, one was traumatized by a postmortem examination of an infant.

In order to control for further clinical symptoms in the group of traumatized policemen additional clinical surveys assessing current psychopathology: the *Modified PTSD Symptom Scale (MPSS*, Spitzer et al., 2001), the *Beck's Depression Inventory* (BDI, German version: Hautzinger et al., 1995), the *Beck's Anxiety Inventory* (BAI, German version: Margraf and Ehlers, 2003) and the *State Trait Anxiety Inventory* (STAI, German version: Laux, 1981) were completed. All scores were within the average range (Table 1).

Neither group differed significantly in terms of age, years of education, handedness or in verbal IQ as measured by the Vocabulary Test (Schmidt and Metzler, 1992) (Table 1). Both groups consisted mainly of right-handed men, with one ambidextrous individual in each group.

For comparing both groups on psychopathological and dissociative symptoms we used the *Revised Symptom Check List* (SCL-90-R, German version: Franke, 1984) and the *Dissociative Experience Scale* (DES, German version: Freyberger et al., 1999). None of the remaining participants met the DSM-IV criteria for PTSD or displayed any other psychiatric symptoms (see Table 1).

Before entering the study, all participants gave written informed consent. They received financial remuneration (50 Euro) for their efforts. The study was approved by the Ethics Committee of the University of Munich (see Table 2).

2.2. Assessment of stress coping styles

We used a questionnaire *(Stressverarbeitungsfragebogen, SVF-120,* Janke et al., 1985) to measure stress coping styles. It is a 120-item self-report questionnaire assessing 21 different coping strategies merged into four scales. Three subscales measuring the use of positive coping strategies were combined in the scale 'positive coping strategies'. The first subscale is a measure of devaluation and defense (minimization, disparagement, defense from guilt). The second subscale measures diversion (distraction from situations, substitute gratification, search for self-affirmation and relaxation). Finally, the third subscale measures strategies of stress control (effort to control situations and reactions and positive self-instructions). One exclusive scale records the use of negative coping strategies like escape, social withdrawal, rumination, resignation, self-pity and self-blame. Four additional subscales which do not fit into any of the higher order scales measure social support, avoidance, escape and drug use.

2.3. Neuropsychological assessment

All participants completed a neuropsychological test battery. Verbal declarative memory was examined using the following subscales of the *Wechsler Memory Scale – Revised (WMS-R*, German version: Härting et al., 2000): immediate recall *(Logical Memory I)* and delayed recall *(Logical Memory II)* of two short stories, immediate recall *(Verbal Pair-Association I)* and delayed recall *(Verbal Pair-Association II)* of word pairs. Visuospatial declarative memory was examined using the *Rey-Osterrieth Complex Figure Test* (Rey, 1941). Visuospatial memory was further assessed by two subtests

Table 1
Demographic and clinical variables: means (M) and standard deviations (SD) of the both groups, *t*- and *F*-values levels of significance (*p*-values), effect sizes (d′).

	Traumatized group ($n = 10$)		Non-traumatized group ($n = 15$)		t or χ^2	p-Value	d'
	M	*SD*	*M*	*SD*			
Demographic variables							
Age in years	39.9	6.7	38.9	9.1	−0.306	.765	0.13
Years of education	11.0	1.3	11.5	1.5	−0.641	.605	0.36
Handedness+	9 right/1 ambidex		14 right/1 ambidex		0.091	.793	0.12
Psychiatric symptoms SCL-90-R					F	p-value	d′
GSI	0.18	0.21	0.21	0.13	0.22	0.65	−0.17
PST	13.20	13.49	16.13	9.72	0.40	0.53	−0.25
PSDI	0.90	0.70	1.16	0.21	1.87	0.18	−0–50
Dissociative symptoms DES							
DES	4.78	3.35	4.92	4.88	0.01	0.94	−0.03
Clinical control data	(Control data for traumatized group only)						
MPSS (PTSD diagnosis)	0.00	0.00					
BDI (cumulative value)	5.00	3.92			cut-off value 11		
BAI (cumulative value)	2.80	2.74			cut-off value 10		
STAI-state (raw value)	41.80	10.31			Minimum = 20 Maximum = 80		
STAI-trait (raw value)	31.90	8.28					
STAI-trait (standard T-value)	47.39	9.30			M = 50, SD = 10		

Note: ambidex = ambidextrous, + for distribution of raw values.

Table 2
Results of MANOVA on stress coping strategies of the two experimental groups (traumatized vs. nontraumatized policemen) and of an additional PTSD group.[a]

	Traumatized group (TG) $n = 10$		Non- traumatized group (NTG) $n = 15$		PTSD[a] $n = 11$		F	df	p	Significant p (post hoc)
	M	(SD)	M	(SD)	M	(SD)				
Positive strategies 1: devaluation/defense	10.49	2.67	12.79	3.05	9.52	3.00	4.284	2	.022	NTG >PTSD: .025
Positive strategies 2: diversion	10.70	2.90	12.69	3.06	11.25	3.78	1.267	2	.295	–
Positive strategies 3: stress control	14.77	3.46	17.62	1.76	15.30	3,80	3.303	2	.049	NTG > TG: .078
Positive strategies: total score	11.82	2.09	14.05	2.22	11.94	3.01	3.403	2	.045	NTG > TG: .099
Negative strategies: total score	7.16	3.95	7.77	2.99	11.62	3.33	5.711	2	.007	PTSD >TG: .014; PTSD > NTG: .021

For sample description, neurofunctional and neuropsychological data see Werner et al. (2009).
[a] The additional group with diagnosed PTSD was assessed with the similar study design as they were part of a larger project.

of the *WMS-R*: immediate recall *(Visual Pair-Association I)* and delayed recall *(Visual Pair-Association II)* of color-figure pairs. Four subtests of the computerized version of the *Testbattery for Assessment of Attention* (*TAP*, Zimmermann and Fimm, 2002) were used: *Phasic and Tonic Alertness, Divided Attention, Go/Nogo,* and *Working Memory*. Finally the subtest *Digit Span* of the *WMS-R* to assess auditory working memory was included in the test battery.

2.4. fMRI paradigm

An associative learning paradigm was used to investigate hippocampal function. This paradigm was developed based on the paradigm of Henke et al. (2003) who have already provided evidence for the sensitivity of this method to generate hippocampal activation in healthy participants. The associative learning paradigm consists of two identical encoding trials and one retrieval trial. All trials were carried out within one fMRI measurement, which lasted 12 min. An additional 9.40 min was needed for the MPRAGE sequence.

During encoding, 24 target items, which consisted of monochrome images of female full frontal portraits with a friendly facial expression, were presented (Lundqvist et al., 1998). Twenty four professions were assigned randomly to the faces and typed below the faces. About 50% of the professions belonged to the category "artists" and 50% to the category "scientists". Participants were instructed to learn these association pairs. A further 24 items which consisted of a head template without any physiognomic information were presented as controls for the experimental condition.

During retrieval the previous target items were presented without the profession label. The participants were instructed to recall the professional category previously associated with each face by pressing the right button for an "artistic" profession and the left button for a "scientific" profession. During the control condition the 24 head templates were presented with slightly unequal ear size. Participants had to indicate which ear was bigger by pressing the right or the left button.

Each trial consisted of 48 stimuli (24 target items and 24 control items). The stimuli were divided into six blocks. Each block consisted of four target items and four control items and each item was presented for 5 s (Fig. 1). The stimuli were presented using Presentation® software (version 0.80, www.neurobs.com) and were projected via an LCD projector from a distance of about 3 m. Participants viewed the screen via a mirror positioned 15 cm above their eyes.

2.5. fMRI data acquisition

Imaging was carried out at 1.5 Tesla field strength (Magnetom Vision, Siemens, Erlangen, Germany) using a high-resolution T1 weighted sequence (magnetization prepared rapid gradient echo, MPRAGE) for anatomical reference and a T2$*$ weighted blood oxygen level dependent (BOLD) sensitive echo planar imaging (EPI) sequence covering the whole brain. The functional EPI-sequence consisted of 28 axial slices (slice thickness 4 mm) from the cerebellum to the cortex which was acquired parallel to the anterior commissure–posterior commissure (AC–PC). Images were acquired in interleaved order with the following MR parameters: time of repetition (TR) 5.5 s, time of echo (TE) 60 ms, flip angle (FA) 90°, matrix 64 × 64, field of view (FoV) 240 mm × 240 mm, pixel size 3.75 mm × 3.75 mm. The high-resolution MPRAGE sequence was recorded following the functional measurements using following parameters: TR 11.4 s, TE 4.4 ms, FA 8°, matrix 224 × 256, FoV 270 mm × 270 mm, 144 sagittal slices, slice thickness, 1.25 mm, pixel size 1.05 mm × 1.05 mm.

2.6. Image analyses statistical analyses of functional data

All preprocessing steps and further data analyses were carried out using Brain Voyager QX (version 1.7.4, Brain Innovation, Maastricht, The Netherlands, Goebel, 2006). After MR-scanning, the data sets were transferred to a stand-alone work station for further processing and analysis. Preprocessing of the MR images included

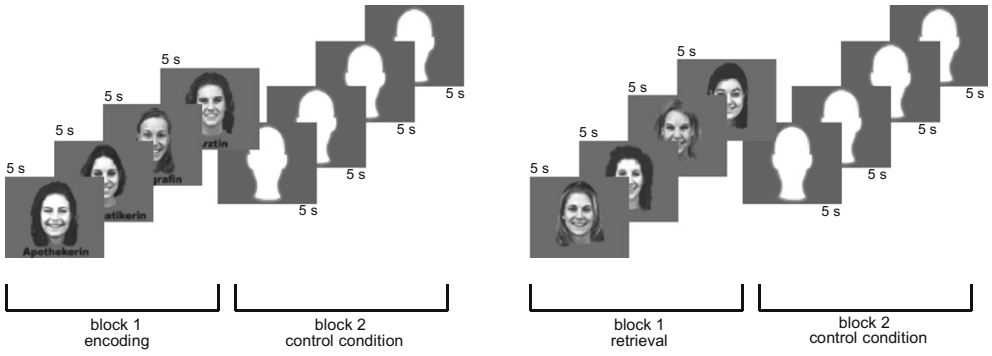

Fig. 1. Experimental design. During encoding (left) six blocks of face-profession pairs and six blocks of control stimuli were presented alternately. During retrieval (right) six blocks of faces without profession and six blocks of control stimuli with unequal ear size were presented alternately. Each stimulus lasted for 5 s.

slice-scan time correction (using sinc-interpolation) and temporal high-pass filtering (cut-off three cycles in time course) to remove low-frequency signal drifts inherent in echo planar imaging. Additionally, the functional images were 3D motion corrected using sinc-interpolation to detect and correct for small head movements. To improve the signal-to-noise ratio, spatial smoothing using a 4 mm FWHM (full width at half maximum) filter was performed. After these preprocessing steps functional data were coregistered to the anatomical images using routines implemented in BrainVoyager. The result of the alignment process was inspected visually and corrected where necessary. The anatomical data were transformed into standard Talairach space for each participant separately. Using the transformation parameters from the latter step, each functional scan could be transformed to Talairach space (Talairach and Tournoux, 1988).

Prior to statistical analysis, the first three scans at the beginning of each trial were excluded from data analysis to minimize T1 effects. Following Boynton et al. (1996), the predictor time courses used were generated on the basis of a linear model of the relation between neural activity and hemodynamic response. To compare the BOLD responses during the experimental conditions, a general linear model (GLM) was estimated. We used the HRF as described by Boynton et al. (1996). These parameters are implemented as the default HRF in fMRI software packages (BrainVoyager). For each participant, *t*-contrasts comparing the active condition (encoding or retrieval) with the control condition were calculated. After spatial smoothing (FWHM 4 mm), contrast maps were forwarded to second level random effect analysis to compare the traumatized group with the non-traumatized group in both directions using two-sample *t*-tests (traumatized group > non-traumatized group, and vice versa). Statistical inferences of the second level analysis were based on the false discovery rate corrected voxel *p*-values (P(FDR) < 0.05). This value determines the single-voxel threshold using the FDR procedure which ensures that only 5% or less of active voxels are false-positives (cf. Genovese et al., 2002). Reporting of anatomical cluster location of significant areas was restricted to clusters containing more than 50 voxels. Anatomical assignment of local maxima within significant areas to Talairach labels was performed using LORETA (Pasqual-Marqui et al., 1994).

2.7. Statistical analysis of behavioural data

Statistical analyses apart from fMRI were performed using SPSS (version 14.0, SPSS Inc., 2005). The *t*-test was applied for the demographic comparisons of the two groups and performance in the associative learning paradigm. Neuropsychological data were clustered into the domains: verbal memory, visuospatial memory and attention. MANOVAS were applied for the basic analyses of group differences. All analyses were two-tailed and the significance level was defined as $p < .05$.

3. Results

3.1. Stress coping style

The traumatized group scored significantly lower in comparison to the non-traumatized policemen in the subscores related to the use of positive coping strategies of devaluation and defense [F (1. 23) = 3,73, $p = .066$] stress control [F (1, 23) = 7.37, $p = .012$] as well as in the sum score for positive strategies [F (1, 23) = 6.34, $p = .019$] in the Stress Processing Questionnaire (Table 4). No difference was observed in the sum score for using negative strategies [F (1, 23) = 0.19, $p > .05$].

In comparison with PTSD patients (see also Werner et al., 2009) we found significant differences in the use of positive and negative strategies: the patients with PTSD showed a reduced use of positive coping strategies in the domains 'devaluation' and 'defense' when compared to the non-traumatized policemen [F (2, 33) = 4.28, $p = .022$]. They also showed more negative coping strategies when compared to both police groups, the traumatized and non-traumatized officers [F (2, 33) = 5.71, $p = .007$]. The traumatized policemen only use less positive strategies in the domain of 'stress control' [F (2, 33) = 3.30, $p = .049$]. The general sum score for using positive strategies differed only between the non-traumatized and traumatized policemen [F (2, 33) = 3.40, $p > .045$]. No statistical differences were found for the use of positive strategies in the domain 'diversion' when comparing all three groups [F (2, 33) = 1.27, $p > .05$].

3.2. Neuropsychological performance

A significant group effect [F (3, 21) = 3.15, $p = .047$] was found in comparisons of the neuropsychological domains (verbal memory, visual memory and attention/executive functions). In regard to verbal and figural memory performance, traumatized and non-traumatized policemen only differed significantly in verbal memory [F (1, 23) = 6.22, $p = .025$]. No statistical differences between the two groups were found for either visuospatial memory: [F (1, 23) = 0.15, $p > .05$] or in the domain attention/executive function [(F (1, 23) = 2.60, $p > .05$].

3.3. fMRI activation patterns

3.3.1. Encoding

Since the encoding trials were identical and the separate analysis of the encoding trials revealed similar activation patterns they were analyzed together.

Increased fMRI activation in response to encoding of face-profession pairs was observed within the bilateral hippocampal formation, parahippocampal and fusiform gyri in non-traumatized policemen relative to the traumatized group. The activation in bilateral inferior frontal gyri was stronger for the non-traumatized policemen than the traumatized policemen.

The traumatized policemen displayed stronger BOLD responses associated with encoding in the superior frontal gyrus, insula, precentral gyrus as well as in precuneus, posterior cingulate, superior temporal and middle occipital gyrus (see Fig. 2 and Table 3).

3.3.2. Retrieval

The two groups did not differ in temporomesial activation during retrieval. However, whereas the non-traumatized group showed a stronger BOLD response within the frontal brain areas (middle frontal gyrus, insula, precentral gyrus and superior frontal gyrus), the traumatized policemen's parietal areas (postcentral gyrus, cingulate gyrus, lingual gyrus, fusiform gyrus, Fig. 3) were more activated (Table 4).

3.4. Error rates and reaction times during fMRI paradigm

Error rates during the fMRI paradigm were not significantly different between the groups (errors: traumatized policemen: $M = 9.6$, $SD = 2.3$; non-traumatized policemen: $M = 10.4$, $SD = 3.3$, t (21) = −0.76, $p > .05$). (t = -.583, p > .05). However, one exception illustrates that traumatized participants showed longer reaction times (RT) when assigning profession to face stimuli during retrieval (RT measured in seconds: traumatized policemen: $M = 3.9$ s, $SD = 2.2$ s; non-traumatized policemen: $M = 2.47$ s, $SD = 0.65$ s; t (21) = −2.26, $p = .034$).

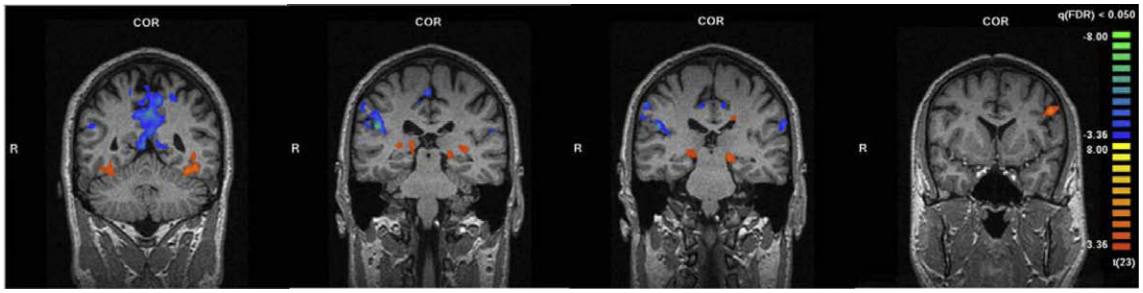

Fig. 2. Results of the group comparison for encoding ($p < .05$, FDR corrected; Talairach coordinates from the left to the right: $x = -42$, $y = -48$, $z = -20$; $x = 11$, $y = -28$, $z = -7$; $x = -16$, $y = -24$, $z = -9$; $x = -42$, $y = 10$, $z = 31$). Red/yellow colors correspond to stronger fMRI BOLD activations of the non-traumatized group, blue/green colors correspond to stronger activations of the traumatized group.

Table 3
Significant results of the contrast of the non-traumatized > traumatized group (and vice versa) for encoding (FDR < .05).

Region	R/L	BA	Talairach coordinates			*T*	Number of voxels
			x	*y*	*z*		
Non-traumatized >traumatized group							
Frontal							
Inferior frontal gyrus	L	9	−48	11	34	6.42	1806
Inferior frontal gyrus	L	45	−39	23	3	5.51	351
Precentral	L	6	−43	−4	58	5.41	258
Temporal							
Fusiform gyrus	R	19	33	−64	−20	7.10	6572
Fusiform gyrus	L	37	−36	−49	−20	5.75	3018
Hippocampus	R		30	−34	7	5.14	125
Hippocampus	L		−33	−28	−2	4.13	83
Parahippocampal gyrus	R	28	18	−25	−2	4.06	67
Parahippocampal gyrus	R	27	15	−28	4	4.71	96
Parahippocampal gyrus	L	28	−15	−22	−8	4.98	233
Parietal							
Precuneus	R	19	30	−67	34	6.86	1154
Angular gyrus	L	39	−33	−64	37	9.42	1339
Traumatized >non-traumatized group							
Frontal							
Insula	R	13	45	−13	10	5.11	237
Superior frontal gyrus	R	8	21	26	43	4.68	72
Precentral gyrus	R	4	39	−13	46	4.51	75
Temporal							
Superior temporal gyrus	R	29	48	−25	18	7.81	803
Superior temporal gyrus	R	39	48	−58	28	5.34	183
Superior temporal gyrus	L	41	−48	−30	13	4.05	55
Parietal/sub-Parietal							
Precuneus	L/R	7/31	−1/1	−49	31	6.76	9339
Posterior cingulate	L	29	0	−50	9	6.76	
Occipital							
Middle occipital gyrus	R	18	12	−88	16	5.30	192
Middle occipital gyrus	L	18	−12	−88	13	6.20	358

Note. The coordinates (*x*, *y*, *z*) localize the maximum of the BOLD response within one structure, *T*-values correspond to the FDR correction reflecting the peak of activation. The anatomical areas and Brodmann Areas (BA) were identified using LORETA (Pasqual-Marqui et al., 1994). R: right, L: left.

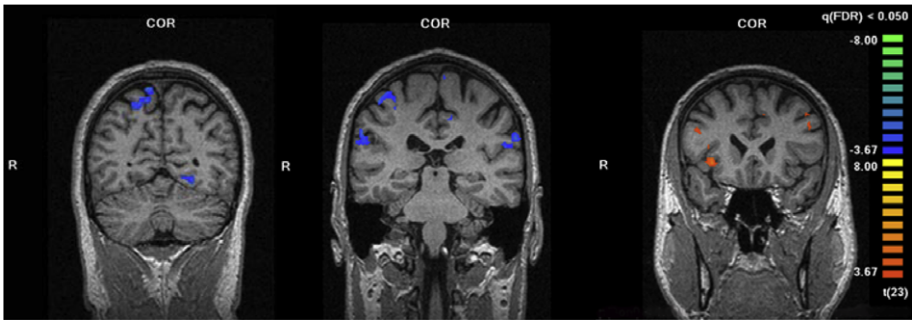

Fig. 3. Results of the group comparison for retrieval ($p < .05$, FDR corrected; Talairach coordinates from the left to the right: $x = -22$, $y = -61$, $z = -12$; $x = 34$, $y = -28$, $z = -13$; $x = 33$, $y = 20$, $z = 1$). Red/yellow colors correspond to stronger fMRI BOLD activations of the nontraumatized group, blue/green colors correspond to stronger activations of the traumatized group.

Table 4
Significant results of the contrast of the non-traumatized >traumatized group (and vice versa) for retrieval (FDR <.05).

Region	R/L	BA	Talairach coordinates			*T*	Number of voxels
			x	*y*	*z*		
Non-traumatized > traumatized group							
Frontal							
Middle frontal gyrus	R	9	48	17	31	4.76	469
Insula	R	13	33	20	1	5.24	271
Precentral gyrus	L	9	−42	26	34	6.50	3342
Superior frontal gyrus	L	6	−6	12	52	4.85	712
Temporal							
Fusiform gyrus	R	37	33	−61	−23	4.25	248
Parietal							
Precuneus	L	19	−33	−71	40	4.60	660
Occipital							
Fusiform gyrus	R	18	21	−88	−12	5.04	199
Inferior occipital gyrus	L	17	−27	−91	−8	6.18	204
Traumatized > non-traumatized group							
Frontal							
Paracentral gyrus	R/L	5	0	−31	55	5.28	348
Temporal							
Fusiform gyrus	R	37	45	−58	−8	4.65	207
Superior temporal gyrus	L	22	−66	−40	22	7.80	2385
Parietal/sub-Parietal							
Precuneus	R	7	18	−67	31	6.33	4742
Postcentral gyrus	R	40	63	−25	19	5.98	585
Postcentral gyrus	R	2	45	−29	52	5.08	773
Postcentral gyrus	R	3	12	−34	70	4.69	273
Cingulate gyrus	L	31	−6	−37	37	5.47	1680
Inferior parietal lobule	L	40	−42	−37	52	4.95	117
Occipital							
Lingual gyrus	R	18	6	−79	4	4.76	55
Lingual gyrus	R	19	12	−58	−2	4.47	258
Fusiform gyrus	L	19	−24	−61	−11	4.94	90

Note. The coordinates (*x, y, z*) localize the maximum of the BOLD response within one structure, *T*-values correspond to the FDR correction reflecting the peak of activation. The anatomical areas and Brodmann Areas (BA) were identified using LORETA (Pasqual-Marqui et al., 1994). R: right, L: left.

4. Discussion

Our main results were: (a) resilient traumatized policemen showed decreased responsiveness in the temporomesial regions during encoding, relative to the non-traumatized policemen; (b) traumatized and non-traumatized policemen showed no significant differences in either episodic associative memory or on most measures of cognitive performance; (c) both traumatized and non-traumatized officers used a comparable number of negative coping strategies, however the traumatized policemen used fewer positive coping strategies.

In this study we compared resilient traumatized and non-traumatized policemen on the following measures: fMRI responses to a memory paradigm, cognitive abilities and coping styles. In comparison to non-traumatized policemen, traumatized policemen showed reduced fMRI BOLD activation within the temporomesial brain regions during encoding. This partly contradicts recent data concerning verbal associative memory in fully developed PTSD (Geuze et al., 2007). In this study behavioural measures showed a trend towards a deficit in memory performance in PTSD patients that was not present in our study. In addition, Geuze's PTSD group exhibited reduced activity, relative to controls, in regions of the frontal lobe, together with larger activation in the temporal lobe during the encoding phase.

Additionally, during encoding, our traumatized group display increased activation only within the superior temporal gyrus. This partly corresponds with Geuze's findings. Unlike Geuze's PTSD-group however, they also show reduced BOLD activation in temporomesial regions (fusiform gyrus, hippocampus, parahippocampus) and increased BOLD activation within differentiated prefrontal and parieto-occipital areas when compared to non-traumatized policemen.

The mentioned prefrontal regions that showed less activation in the traumatized group during encoding include left-hemispherical prefrontal regions (BA 6, 9, 45). This prefrontal network is known to be critically involved in working memory (BA 9, 45) and in updating verbal information (BA 6, e.g. Wagner, 2002). On the other hand, the trauma group displayed an elevated right-hemisphere activation of the insula (BA 13) and BA 8/4 during encoding. Paulus and Stein (2006) reported that activation in the anterior insula is modulated by an individual's degree of anxiety. They emphasise that functional neuroimaging studies have linked insular cortex activation to the anticipation of aversive stimuli, cognitive and affective processes during learning and error related brain activation. This and other studies show clearly that the insular cortex is important for linking emotions to cognitive processes and behavioural responses (see also Nitschke et al., 2006). Hence, the insular activation could reflect either a pre-conscious level of anxiety that is not consciously reported (see BAI and STAI-values, Table 1) or a subjective response uncertainty.

Concurrently, we found an increased activation of the angular gyrus in the non-traumatized sample, whereas the traumatized patients displayed an elevated activation of the posterior cingulate gyrus. These two different areas perform different cognitive functions. It is thought that the angular gyrus is involved in the transcription of the written word into an internal monologue (Geschwind, 1965). In contrast, the posterior cingulate cortex is involved in spatial orientation and memory. It is likely that connections between posterior cingulate and parahippocampal cortices contribute to these processes. The increased BOLD activation in the occipital bilateral BA 18 in the traumatized group indicates the active processing of visual information. These differences in activation could reflect different encoding strategies: while the

non-traumatized would use more internal language to encode the stimuli, the traumatized may use more visual strategies. An alternative interpretation is that activation differences between these groups could reflect different encoding steps resulting from disparities in certainty during encoding. The fact that the traumatized group shows slight verbal memory deficits strengthens the case for the first explanation, whereas the finding of elevated reaction times during later retrieval supports the second explanation. Thus the first interpretation suggests that traumatized participants show weakened verbal information processing, the second interpretation suggests that there are emotional disturbances in traumatized participants.

In contrast to Geuze et al. (2007) we did not find any group differences in fMRI BOLD activation within the hippocampus and parahippocampal gyrus during retrieval. At retrieval, Geuze' PTSD-group showed decreased activity in areas of the frontal lobe, hippocampus, parahippocampal gyrus, middle and superior temporal gyrus.

Instead, our traumatized participants displayed only reduced activation in frontal brain regions accompanied by increased activation in the paracentral gyrus and within the parietal and occipital lobes. This finding could have two different explanations that parallel our two possible interpretations of the encoding data: On one hand the increased activation within the posterior brain regions relative to frontal areas may reflect a reduced involvement of brain areas related to executive control functions, and an increased demand on regions associated with visual-spatial processing. On the other hand it could just reflect the longer time needed for information processing in the traumatized group (which is also reflected by elevated response times).

With regard to cognitive functions, we had hypothesized a priori a realm of cognitive functions, reflected by a dissociation of cognitive and fMRI BOLD patterns in the traumatized group, with almost normal cognitive profiles but impaired underlying brain activation. Our results largely concurred with this hypothesis. We found that the traumatized policemen did not differ from non-traumatized policemen on the majority of cognitive performance measures. Only verbal memory and rapid mobilization of resources to process expected stimuli (as measured by reaction times during the fMRI experiment) were shown to be weaker for the traumatized policemen. However, although reaction times for traumatized policemen were slower, the accuracy of performance on the associative learning task was unaffected. Since our experimental task comprised of both verbal and visual stimuli for investigating associative memory, the dissociation of the assessed verbal memory performance and the associative learning accuracy could be explained by the different stimulus material. Those findings are in agreement with previous studies that only revealed mild memory impairments in PTSD patients (e.g. Gilbertson et al., 2006) and specific verbal memory impairments in traumatized combat veterans with and without PTSD (Tischler et al., 2006). Tischler and colleagues posited that poorer memory performance in their sample of combat veterans reflects some preexisting factor that is related to a person's psychological response to trauma rather than being the result of interference effects of current symptoms on cognitive performance. Our findings on memory and reaction time have implications for these theoretical questions: In line with Yehuda (2004) it could be argued that both the impaired verbal memory performance and the deceleration of reaction reflect the psychological response to trauma. This response can be modified by pre-traumatic differences in cognitive-behavioural pattern (see also Gilbertson et al., 2006).

Beyond our assumptions on general cognitive functioning we hypothesized that the resilient traumatized policemen would show different stress coping styles from those of the non-traumatized policemen (and from those shown by PTSD sufferers). Consistent with our hypotheses and prior findings we found a specific pattern that differentiated the three groups' ability to cope with stress. The traumatized policemen reported using fewer positive, adaptive coping strategies in comparison to the non-traumatized police officers. In contrast, both traumatized and non-traumatized officers used a similar number of negative coping strategies. However PTSD-patients used more negative, maladaptive strategies than both traumatized and non-traumatized officers (see also Werner et al., 2009). Our findings indicate that traumatized policemen without PTSD use fewer positive coping strategies than non-traumatized policemen. They used fewer control strategies, as well as less positive self-instruction. They also more readily used denial strategies like minimization and disparagement, to defend themselves from stressful events. However, they were successful in making use of a number of coping strategies that could be helpful in overcoming the negative influence of traumatic stress. Traumatized policemen's use of maladaptive negative coping strategies is much more limited than PTSD patients. For example PTSD patients use more rumination and more self-blaming behaviour (Southwick et al., 2005; see Table 2). This difference in the use of stress coping strategies may reflect an intermediate use of adaptive coping strategies in traumatized policemen.

One limitation of our study is its cross-sectional design. A longitudinal design would be the ideal method for examining pre- and post-traumatic differences in the traumatized group. It might also be argued that the study is limited because it only investigates effects of trauma exposure in a small group of selected participants, who may not be representative of the general population. In answer to this potential objection we believe that it is useful to investigate these resilient individuals because our data provides a possible framework for developing a model of interacting factors of traumatic effects. Nevertheless, a comparison between traumatized resilient and traumatized non-resilient participants is missing. Such a study would yield further information about the relationship between resilience and exposure to stress. In accordance with many prior studies, disentangling verbal memory and deficits in information processing speed is problematic, irrespective of functional changes in the limbic system. Although we found no general attentional deficits in the traumatized sample, a slowed reaction time during the experimental task was shown, which probably indicates an impaired alertness or ability to initiate actions. Thus, we have to acknowledge that a change in processing speed and memory could be confounded. This could be reflected by altered brain activation.

In conclusion, our results converge with previous findings, showing that trauma-related disturbances in brain activity can be seen even in resilient populations, without significant concomitant deficits in cognitive-behavioural performance. Additionally, individual differences in pre-traumatic cognitive-behavioural resources play a role in regulating brain activity after trauma and have an effect on the likelihood of developing PTSD. Our findings reinforce the importance of providing preventative programs for employees in stressful occupations such as policing, in order to strengthen their cognitive-behavioural coping strategies and thereby minimize future incidents of PTSD.

5. Conflict of interest statement

None declared.

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