Available online at www.sciencedirect.com
ScienceDirect
Psychiatry Research 157 (2008) 299–302
PSYCHIATRY RESEARCH
www.elsevier.com/locate/psychres

Brief report

No evidence for verbal learning impairments in women with a history of childhood abuse

Marko Jelicic*, Elke Geraerts, Harald Merckelbach

Department of Clinical Psychological Science, Maastricht University, The Netherlands

Received 15 August 2006; received in revised form 27 June 2007; accepted 30 June 2007

Abstract

We examined verbal learning in 54 women with a history of childhood abuse and 40 women without trauma history. Although women with a history of abuse reported higher levels of psychological distress than controls, the two groups did not differ in their verbal learning performance.

Keywords: Traumatic stress; Childhood abuse; Verbal learning; Memory

1. Introduction

Some authors (e.g. Bremner, 1999) have argued that exposure to traumatic stress may lead to memory impairments. According to these authors, excessive levels of cortisol, released during severe stress, would result in damage to the hippocampus, thereby undermining the vital role this brain structure plays in storing new information in memory. Meanwhile, the evidence for memory impairments following traumatic stress is somewhat mixed. Although several studies found that traumatized patients who developed posttraumatic stress disorder (PTSD) show worse performance on standard memory tests than control participants, other investigations were unable to document memory impairments in PTSD patients (for reviews, see: Horner and Hamner, 2002; Isaac et al., 2006).

It is tempting to take memory impairments in PTSD as evidence for the idea that traumatic stress, via structural brain damage, leads to problems in learning new information. However, there are several problems with this line of reasoning (Horner and Hamner, 2002; Jelicic and Merckelbach, 2004; Isaac et al., 2006). Some studies failed to exclude patients with neurological injury or did not control for alcohol and/or drug abuse (see Horner and Hamner, 2002, for a list of studies with methodological problems). Therefore, one could argue that memory impairments in traumatized people who developed PTSD may, at least in some cases, be the result of neurological injury and/or substance abuse rather than exposure to traumatic events per se. In order to circumvent the problem of neurological injury and/or substance abuse, Twamley et al. (2004) studied neuropsychological function in undergraduate students with or without a history of trauma. Using a non-clinical sample, the authors failed to find evidence for neuropsychological impairments in undergraduates who had been exposed to traumatic events. Thus, their study seems to suggest that exposure to traumatic events does not invariantly lead to memory problems.

* Corresponding author. Department of Clinical Psychological Science, Maastricht University, P.O. Box 616, 6200 MD Maastricht, The Netherlands.
E-mail address: m.jelicic@psychology.unimaas.nl (M. Jelicic).

0165-1781/$ - see front matter © 2007 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.psychres.2007.06.027

Inspired by the study by Twamley et al. (2004), we decided to examine learning of new information in participants from the general population with and without a history of childhood abuse. In contrast to Twamley and colleagues, we opted for participants from the general population because the use of undergraduate students is sometimes associated with problems such as expectancy effects and demand characteristics (Schulz, 1969). Verbal learning was chosen because most studies on memory in PTSD used verbal learning tests (cf. Isaac et al., 2006). Although we focused on one aspect of memory, i.e., learning of new semantic information, it should be noted here that verbal learning tests play a prominent role in the evaluation of memory disorders (Lezak et al., 2004). For instance, such tests can be used to distinguish between normal cognitive aging and neurodegenerative disorders. Our expectation was that there would be no differences in verbal learning between people with trauma exposure and those without a background of abuse.

2. Methods

2.1. Participants

We recruited participants through advertisements in local newspapers. Women with and without a history of childhood sexual abuse were invited to volunteer in a study on memory and personality. The study was approved by the local ethical committee. All participants were studied after informed consent. The trauma group consisted of 54 women (mean age = 43.0 years, S.D. = 14.5) with clear and continuous memories of childhood sexual abuse (CSA). That is, these women had never forgotten that they were victims of CSA, and in many cases they could provide us with sources of corroborative evidence for the abuse (e.g., siblings who could confirm the abuse). CSA was defined as physical sexual contact ranging from fondling to penetrative acts. The control group comprised 40 women (39.1 years, S.D. = 11.1) who said that they had, neither during childhood nor during adulthood, ever been sexually abused. Participants with traumatic experiences other than CSA were excluded from the study.

2.2. Measures

2.2.1. Self-report scales

All participants were asked to fill out four questionnaires. The Childhood Trauma Questionnaire (CTQ; Bernstein and Fink, 1998) is a widely used self-report scale of traumatic childhood events. The short form consists of 25 items that address five areas of childhood maltreatment, namely emotional, physical, and sexual abuse, and emotional and physical neglect. Higher scores on the CTQ reflect higher levels of childhood trauma. The Impact of Event Scale (IES; Horowitz et al., 1979) is a 15-item self-report scale measuring the present level of trauma-related distress. It comprises two subscales tapping symptoms of intrusion and avoidance. Higher scores on the IES indicate higher levels of traumatic distress. The Beck Depression Inventory (BDI; Beck and Steer, 1987) consists of 21 items focusing on the behavioral manifestations of depression. Higher scores on the BDI reflect more depressive symptoms. The Dissociative Experiences Scale (DES; Bernstein and Putnam, 1986) is a 28 self-item self report scale pertaining to dissociative experiences. Participants have to indicate on visual analogue scales the degree to which they experience these feelings. Higher scores on this instrument indicate stronger dissociative experiences.

2.2.2. Memory measures

Participants were given a modified version of the 30-word Verbal Learning Task (30WVLT; Smeets et al., 2006). Instead of 30 neutral words, 10 positive emotional, 10 negative emotional, and 10 neutral words were used. The different types of words were completely intermixed. Participants were read the words at a rate of one per 2 s and asked to recall as many words from the list as possible. This procedure was repeated twice (with the same words). Thirty min after the third trial, participants again were asked to recall the study words. We used the total number of recalled words at trial 3 as an index of immediate recall (IR) and the number of words recalled after 30 min as an index of delayed recall (DR).

3. Results

The scores of the two groups on the self-report scales and the memory measures can be found in Table 1. As

Table 1
Mean (S.D.) scores on the self-report scales and the memory measures in women with and without a history of childhood abuse

Measure	Women with abuse	Women without abuse	t (92)	P
CTQ	37.3 (11.4)	24. 2 (4.7)	6.87	<0.001
IES	23.0 (8.4)	7.9 (5.3)	10.00	<0.001
BDI	13.5 (9.1)	7.0 (4.4)	4.19	<0.001
DES	22.3 (12.7)	13.9 (7.1)	3.76	<0.001
IR	16.4 (5.3)	16.1 (4.6)	0.38	0.71
DR	11.8 (6.4)	11.3 (5.4)	0.38	0.69

Note: CTQ = Childhood Trauma Questionnaire; IES = Impact of Event Scale; BDI = Beck Depression Inventory; DES = Dissociative Experiences Scale; IR = immediate recall; DR = delayed recall.

expected, participants with a history of CSA had significantly higher scores on the CTQ, IES, BDI, and DES than those without a history of abuse. All these differences yielded large effect sizes (all Cohen's d's > 0.84). There were no differences between the two groups with respect to memory performance. For the whole sample, age correlated negatively with the two memory measures (both r's > −0.24; both P's < 0.05), while dissociation was not significantly related to the memory parameters (both r's < −0.12). In the group with a history of CSA, 35 (65%) had an IES score that exceeded the clinical cut-off for trauma-related distress (>19; see Joseph, 2000). None of the controls had an IES score above the cut-off. Note that IES scores were not significantly related to the memory parameters (both r's < −0.10).

4. Discussion

Our findings as well as those reported by Twamley et al. (2004) suggest that trauma exposure does not invariantly lead to problems in learning new information. Thus, it may well be the case that memory problems in patients with PTSD are a side effect of neurological injury and/or substance abuse. According to the glucocorticoid toxicity hypothesis, traumatic stress may lead to hippocampal damage through elevated levels of cortisol. This hypothesis has been criticized on a number of grounds (McNally, 2003; Jelicic and Merckelbach, 2004). For example, although many magnetic resonance imaging studies show smaller hippocampi in PTSD patients, most of these investigations suffer from methodological weaknesses such as the failure to include an adequate control group. Also, a small hippocampus may be a risk factor for PTSD rather than a consequence of exposure to trauma. Moreover, cortisol levels are often lowered, rather than elevated, in PTSD patients (Yehuda, 2002). The present findings along with those of Twamley et al. (2004) pose additional problems for the glucocorticoid toxicity hypothesis. Note that there is little reason to doubt that the CSA survivors in our study had been confronted with serious abusive events in childhood. Their relatively high CTQ and IES scores underline this point. The finding that age was inversely related to memory performance shows that our memory parameters had reasonable sensitivity. Given the relatively large sample size, our failure to detect suboptimal memory performance in those with a history of CSA cannot be easily dismissed as a power problem. Rather, it casts doubt on the idea that traumatic exposure in itself undermines storing of new information in memory. Much the same is true for our failure to find a correlation between dissociation and verbal learning. Apparently, the idea that dissociation is accompanied by memory deficits does not possess the empirical credibility some authors assume it has (e.g., Prohl et al., 2001).

The present study has some limitations. Because all our participants were female, our findings are silent about memory performance in men with a trauma history. However, there is no a priori reason to assume that trauma affects memory functioning in women and men differently (cf. Twamley et al., 2004). Furthermore, our participants were recruited via advertisements in local newspapers. As a result, only resilient people with a trauma history might have volunteered in our study. It is possible that people with memory impairments brought about by traumatic events did not respond to our advertisement. While our study may suffer from a selection bias, it should be noted here that the participants with a trauma history had elevated scores on the IES and BDI, suggesting that, at present, they still suffer from the traumatic events that took place in their childhood. Finally, our study focused on learning of new semantic information. There are many indicators showing that when it comes to retrieval of autobiographical memory, trauma survivors exhibit abnormalities (e.g., Wessel et al., 2002).

Acknowledgements

This study was supported by a grant from the Dutch Organization for Scientific Research N.W.O. (grant # 452-02-006-A).

References

Beck, A.T., Steer, R.A., 1987. Beck Depression Inventory Manual. Psychological Corporation, San Antonio TX.

Bernstein, E.M., Putnam, F.W., 1986. Development, reliability and validity of a dissociation scale. Journal of Nervous and Mental Disease 174, 727–735.

Bernstein, D.P., Fink, L.A., 1998. CTQ: Childhood Trauma Questionnaire: A Retrospective Self-Report. Psychological Corporation, San Antonio TX.

Bremner, J.D., 1999. Does stress damage the brain? Biological Psychiatry 45, 797–805.

Horner, M.D., Hamner, M.B., 2002. Neurocognitive functioning in posttraumatic stress disorder. Neuropsychology Review 12, 15–30.

Horowitz, M., Wilner, N., Alvarez, W., 1979. Impact of Event Scale: a measure of subjective stress. Psychosomatic Medicine 41, 209–218.

Isaac, C.L., Cushway, D., Jones, G.V., 2006. Is posttraumatic stress disorder associated with specific deficits in episodic memory? Clinical Psychology Review 26, 939–955.

Jelicic, M., Merckelbach, H., 2004. Traumatic stress, brain changes, and memory deficits: a critical note. Journal of Nervous and Mental Disease 192, 548–553.

Joseph, S., 2000. Psychometric evaluation of Horowitz' Impact of Event Scale: a review. Journal of Traumatic Stress 13, 101–113.

Lezak, M.D., Howieson, D.B., Loring, D.W., 2004. Neuropsychological Assessment, 4rd edition. Oxford University Press, Oxford.

McNally, R.J., 2003. Remembering Trauma. Belknap Press/Harvard University Press, Cambridge, MA.

Prohl, J., Resch, F., Parzer, P., Brunner, R., 2001. Relationship between dissociative symptomatology and declarative and procedural memory in adolescent psychiatric patients. Journal of Nervous and Mental Disease 189, 602–607.

Schulz, D.P., 1969. The human subject in psychological research. Psychological Bulletin 72, 214–228.

Smeets, T., Jelicic, M., Merckelbach, H., 2006. The effect of acute stress on memory depends on word valence. International Journal of Psychophysiology 62, 30–37.

Twamley, E.W., Hami, S., Stein, M.B., 2004. Neuropsychological function in college students with and without posttraumatic stress disorder. Psychiatry Research 126, 265–274.

Wessel, I., Merckelbach, H., Deckers, T., 2002. Autobiographical memory specificity, intrusive memory, and general memory skills in Dutch–Indonesian survivors of the World War II era. Journal of Traumatic Stress 15, 227–234.

Yehuda, R., 2002. Current status of cortisol findings in posttraumatic stress disorder. Psychiatric Clinics of North America 25, 341–368.