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Associative memory impairment in acute stress disorder: Characteristics and time course

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ABSTRACT

Stress and episodic memory impairment have previously been associated. Acute stress disorder (ASD) is a maladaptive stress response, which develops in some individuals following traumatic life events. Recently, the authors demonstrated a specific deficit in associative memory for emotionally neutral stimuli in ASD and posttraumatic stress disorder (PTSD). This study further tested the relationship between this memory impairment and the course of ASD. We assessed new learning and memory for item and associative information in patients diagnosed with ASD ($n=14$) and matched trauma naïve controls ($n=14$). Memory performance and posttraumatic symptoms were examined for approximately 1 and 10 week periods following the traumatic experience. In the two experiments, participants studied a list of stimuli pairs (verbal or visual) and were then tested for their memory of the items (item recognition test), or for the association between items in each pair (associative recognition test). In both experiments, ASD patients showed a marked associative memory deficit compared to the control group. After 10 weeks, ASD symptoms were resolved in most patients. Interestingly, their performance on associative recognition for verbal stimuli improved, while the associative deficit for visual stimuli remained unchanged. Potential mechanisms underlying such an associative memory deficit in post-trauma patients are discussed.

1. Introduction

Individuals with posttraumatic stress disorder (PTSD) suffer from memory impairment for neutral stimuli. A recent meta-analysis investigating memory of emotionally neutral information (verbal and/or visual) in PTSD patients and healthy controls found that there was robust memory impairment among PTSD sufferers (Brewin et al., 2007). This deficit in episodic memory is more pronounced in verbal aptitudes as opposed to visual and visuo-spatial memory tasks. Yet, most of the data derived from retrospective studies include participants who have been symptomatic for many years, such as war veterans or adult survivors of child abuse, and as such presents several methodological problems. The most notable confounding factor in PTSD patients is that of co-morbidity (Danckwerts and Leathem, 2003) with depression, anxiety, panic, and other mental disorders commonly co-occurring, all of which are characterized by specific information processing and memory deficits (Williams et al., 1997). Another methodological constraint is the wide variation in the population with regard to time elapsed since the trauma. Finally, medications used for long periods by many participants to treat PTSD-related symptoms may cause changes in cognition that are not strictly related to PTSD (Savic et al., 2005). These methodological problems make it difficult to draw firm conclusions regarding the effects of traumatic stress on cognition and memory (e.g., Danckwerts and Leathem, 2003; Isaac et al., 2006, for reviews) and whether memory impairments are present at the onset of the disorder. A recent study by LaGarde et al. (2010) attempted to address this question by testing memory and executive functions in patients diagnosed with acute-PTSD (up to 3 months post-trauma) compared to trauma-exposed individuals and controls who have never been exposed to trauma.

With regard to episodic memory, they found that acute-PTSD patients perform worse than the unexposed control group for verbal and visual episodic memory in immediate and delayed recall but not on recognition memory performance. Interestingly, the trauma-exposed group (which did not develop PTSD) was not

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significantly different from the acute-PTSD group nor from the control group, and seemed to fall in between the majority of episodic memory measures. While the study described avoided some confounders of chronicity, the 3 month period cannot be taken to indicate memory impairment from the disorder onset. If this episodic memory impairment indeed exists at the onset of PTSD and is not the result of chronicity, then it is a reasonable hypothesis that this impairment exists also in acute stress disorder (ASD) patients, a group that is more homogenous and devoid of confounders compared with acute PTSD patients. Indeed, a recent paper by the current authors (Guez et al., 2011) found a specific impairment in associative memory in ASD and chronic-PTSD patients compared with control subjects.

Inappropriate associative coupling in traumatized patients may be responsible for the tendency to over-generalize the effect of traumatic cues to unrelated neutral ones as well as for the appearance of intrusive thoughts brought on by benign stimuli (Ehlers and Clark, 2000). Indeed, PTSD patients have been shown to be more susceptible to conditioning (Orr et al., 2000), which may be related to a general tendency to mistakenly associate unrelated stimuli, specifically an increase in false associations, which in turn would present as an abnormality in associative memory. This pattern of increase in false-alarms has indeed been demonstrated in studies that test false memory in PTSD patients. Although not specifically addressing associative deficit, several studies found an increased bias towards false memory for neutral verbal (Bremner et al., 2000; Zoellner et al., 2000) but not for visual stimuli (Hauschildt et al., 2012; Jelinek et al., 2009). The distinction between associative memory abnormalities for verbal as opposed to visual stimuli carries considerable theoretical and practical importance. Visual processing is a critical element in sensation-based memory abnormalities within PTSD patients, manifesting in flashbacks and vivid visual nightmares. These experiences appear to involve a breakdown in processes responsible for binding individual sensory features into stable objects or episodic memory, leading to fragmented or incomplete memories (Brewin, 2011). Thus, the study of visual processing, especially in relation to associative/binding processing, is important in the investigation of memory in post-traumatic patients. On the one hand, it might be hypothesized that an associative deficit for visual processes (e.g. encoding picture-pairs) compared to verbal material (e.g. word-pairs) would be more robust and as such be more immune to change given the fundamental visual memory impairment post-trauma. On the other hand, a large body of literature (e.g., Brewin et al., 2007; Jelinek et al., 2009; Hauschildt et al., 2012) supports a less prominent impairment of visual memory in post-traumatic patients (but see Guez et al., 2011, who found the same memory effects for verbal and visual stimuli in post-traumatic patients).

In the current study, we further tested the distinction between an associative deficit for visual and verbal stimuli in post-traumatic patients and their relation to the course of ASD symptoms. These are experienced during or immediately after the traumatic event, last for at least 2 days, and are resolved within the following 4 weeks. If these persist, they merit a PTSD diagnosis (Diagnostic and Statistical Manual of Mental Disorders, DSM-IV; APA, 1994).

Though we have previously shown that the memory pattern of ASD patients is similar to that of PTSD patients, some critical questions remain. The question of causality still remains because a pre-post design is virtually unattainable. Does an associative memory deficit reflect a pre-existing cognitive predisposition to maladaptive stress response, or is it the result of post-traumatic processes? Furthermore, are memory deficits in traumatized patients reversible as the post-traumatic symptoms resolved? One study by Vermetten et al. (2003) found that patients with PTSD (but not ASD) showed a significant improvement in PTSD symptoms under paroxetine treatment (a selective serotonin reuptake inhibitor) that was also associated with a significant improvement in verbal declarative memory. This finding favors stress response rather than predisposition as an explanation for post-traumatic memory impairment. By testing ASD patients, most of whom recover within 1 month, we were able to conduct a current-post design of memory performance as it relates to the course of ASD symptoms.

1.1. Current study design and hypotheses

The current study had two main objectives: first, to test whether the associative memory deficit we previously reported in ASD patients might resolve when the post-traumatic symptoms improve. Second, to test for differential effects of stimulus type (verbal vs. visual) on episodic memory over time in ASD patients compared with control subjects.

Based on our previous results, we hypothesized that ASD patients would present a specific associative memory impairment. We further postulated that memory impairment in ASD is a result of trauma exposure and not a pre-existing predisposition; thus we hypothesized that memory impairment would get resolved with symptom resolution. Regarding stimuli type (verbal vs. visual), there is support for two opposing hypotheses: that associative memory impairment for visual stimuli is more robust relating to visual memory disturbances post-trauma or that associative memory for visual stimuli is less affected, in line with previous studies of visual memory in similar populations.

We tested patients diagnosed with ASD presenting the full PTSD spectrum but differing from PTSD with respect to time course (and presence of dissociative symptoms). These patients present the acute effects of trauma but do not manifest long-term effects resulting from chronic stress. In the current study, ASD patients were screened and excluded in the presence of additional psychopathology. Specifically, in the ASD population, co-morbidities have not yet developed, the time from trauma is relatively homogeneous and short, and medical treatment has not been initiated during the initial period of assessment.

Two experiments were conducted, comparing a group of ASD patients to matched healthy controls in a paradigm that directly assessed memory for item and associative information for verbal and visual stimuli. Patients were tested twice, once within 2 weeks from their traumatic experience and again at 10–12 weeks following the event.

2. General method

2.1. Participants

The two experiment comprised of 28 adults: 14 with ASD[1] and 14 healthy controls unexposed to trauma matched for age and education. By telephone interview, 250 trauma exposed individuals were screened upon admission to the emergency department. About one-third of these were classified as potentially having acute stress symptoms and approximately half of these were admitted to the trauma clinic. Initially, 30 patients from this group were tested, however, only 14 completed the follow-up visit 10–12 weeks later. The ASD participants had experienced a life-threatening traumatic event within the 2 weeks prior to the first testing (11 motor vehicle accidents, and three war-related events). These participants had been admitted to a medical emergency department after the traumatic event, and met DSM-IV diagnostic criteria for ASD. The study was approved by the institutional review board of the Soroka University Medical Center. All participants gave their written informed consent for study participation.

[1] Some of these participants were included in our previous article (Guez et al., 2011). Here, we report their follow-up testing.