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Short report

Verbal memory and treatment response in post-traumatic stress disorder

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Summary
Post-traumatic stress disorder (PTSD) is often associated with verbal memory deficits, which could influence treatment outcome. We assessed neuropsychological functioning in individuals with PTSD and their response to cognitive–behavioural therapy (CBT). Treatment non-responders had significantly poorer performance on measures of verbal memory compared with responders and demonstrated narrative encoding deficits. Differences were not explained by IQ, performance on tasks of attention, initial PTSD severity, depression, time since trauma, or alcohol/substance misuse. Verbal memory deficits seem to diminish the effectiveness of CBT and should be considered in its implementation.

Declaration of interest
None.

Individuals with post-traumatic stress disorder (PTSD) often demonstrate verbal memory deficits,[1–3] which may increase risk of developing the disorder.[4] Alcohol misuse, depression, lower IQ and impairments in attention contribute to verbal memory problems. However, the link between memory problems and PTSD remains after controlling for comorbidity, attentional difficulties and intellectual functioning.[5] It is unclear whether verbal memory influences outcome of cognitive–behavioural therapy (CBT) for PTSD, an effective intervention[6] recommended by the National Institute for Health and Clinical Excellence (NICE) as a first-line treatment. Between 8 and 12 sessions are typically offered, but not all people with PTSD recover with this regimen.[6] Our purpose was to examine whether verbal memory relates to outcome of CBT for individuals with PTSD.

Method

Twenty-three people (10 women; mean age=34.8 years, s.d.=8.3) were recruited from consecutive referrals to a PTSD clinic in London. All participants had PTSD as a result of interpersonal trauma or a road traffic accident. Two participants had physical injury: one had suffered the amputation of two toes, and one had leg scarring. Diagnoses were ascertained with the Structured Clinical Interview for DSM–IV (SCID)[7] and the Clinician Administered PTSD Scale (CAPS).[8] There was full agreement in PTSD diagnoses at assessment and follow-up. Two participants met criteria for SCID-assessed alcohol misuse. Severity of depression and anxiety were measured using the Beck Depression Inventory (BDI)[9] and the Beck Anxiety Inventory.[10] Participants underwent neuropsychological assessment before commencing a standard course of eight sessions of CBT for PTSD and were re-assessed for PTSD after session eight. One of two clinical psychologists with extensive training in CBT delivered the treatment. All participants were native English-speaking, free from illicit drug use, psychotropic medications, major medical illness, neurological disorder, and history of head trauma with loss of consciousness.

The Vocabulary and Block Design tests of the Wechsler Adult Intelligence Scale–Revised[11] were administered to estimate verbal and non-verbal IQ.[12] Memory was assessed with the Adult Memory and Information Processing Battery.[13] Two variables that reflect the ability to register meaningful narrative and retain it, hypothesised as relevant to CBT, were analysed: story recall immediate (immediate verbal memory) and percentage retained (retention of verbal information). The digit span subtest of the WAIS–R was administered to measure encoding, and three subtests of the Test of Everyday Attention (TEA)[14] were selected: map search (focused attention), visual elevator (attentional switching), and lottery (sustained attention). These latter four subtests measure components of attention.[15]

T-tests were performed to assess differences between responders and non-responders on pre-treatment severity, age, years of education, alcohol intake, time since trauma, memory, attention and intellectual functioning. There was a trend for non-responders to have higher scores on the BDI (P=0.077) and the CAPS (P=0.098) at the start of treatment compared with responders. These variables were used as covariates in subsequent analyses.

Partial correlations were performed to assess the relationship between improvement in symptoms and the memory and attention variables controlling for pre-treatment severity. To test whether verbal memory functioning predicted improvement after controlling for pre-treatment severity and differences in attention, sequential regression was employed.

Results

At follow-up, 16 patients had recovered (70%) and 7 (30%) continued to meet DSM–IV[16] criteria for PTSD. The online Table DS1 summarises sample characteristics, pre-treatment symptom scores and neuropsychological scores, and post-treatment improvement on the CAPS. The PTSD-persistent group had poorer performance on immediate and delayed verbal recall compared with the recovered group. Groups were similar on percentage of information retained. There was a trend (P=0.081) for the PTSD-persistent group to have more difficulty with sustained attention, although their scores fell in the average range. Groups did not differ in age, years of education, gender, time since trauma, trauma type, alcohol intake and misuse, pre-treatment PTSD symptomatology, intellectual functioning, and rates of physical injury and prior trauma.

Controlling for pre-treatment severity, improvement on the CAPS was associated with story recall immediate (r=0.688, d.f.=19, P=0.001) but not with percentage retained (r=0.000, d.f.=19, P=0.998). As the two groups did not differ on this variable, it was not included in subsequent analyses. Improvement on the CAPS was unrelated to attention: encode (r=0.177, d.f.=19, P=0.442), focus (r=0.213, d.f.=19, P=0.355), shift (r=0.072, d.f.=19, P=0.755) and sustain (r=0.072, d.f.=19, P=0.755).

Sequential regression revealed that even after controlling for differences in pre-treatment severity and attention, story recall immediate predicted improvement on the CAPS. The BDI, CAPS, and TEA sustained attention, entered at step 1, failed to predict improvement in symptoms (r^2=0.246; adjusted r^2=0.126; F=2.06; d.f.=3,22; P=0.139) but adding story recall immediate at step 2 significantly increased r^2 (r^2=0.604; adjusted r^2=0.516; F=6.87; d.f.=4,22; P=0.002), accounting for 36% of the variability in improvement on the CAPS. Non-responders performed in the low average to abnormal range on this task.

Discussion

Memory difficulties did not characterise all patients with PTSD, but did distinguish patients who failed to improve in treatment from those who recovered. In particular, registration and recall of meaningful narrative predicted outcome. This relationship was sustained after controlling for depression, PTSD severity and differences in attention, highlighting the role of verbal memory in treatment-related recovery from PTSD. These findings extend reports that verbal memory difficulties predict the development of PTSD in trauma-exposed adults.[4] Once PTSD is established, verbal memory may constrain recovery.

The recovered and PTSD-persistent groups did not differ on the long delay/short delay savings ratio, consistent with current research.[1] What patients did initially register, they retained, suggesting that the problem lies in the ability to initially learn meaningful information. Differences in memory function could be related to encoding difficulties. The PTSD-persistent group had lower scores on the digit span test of the WAIS–R. However, poorer performance on digit span was unrelated to improvement in symptoms. The ability to encode and manipulate numerical information (digit span) may have little relevance to CBT, whereas the ability to encode and remember meaningful narrative (story recall immediate) is relevant. There was a trend for the PTSD-persistent group to have more difficultly sustaining attention, although patients still performed in the average range and attention was unrelated to improvement in symptoms. This may relate to the likelihood that clinicians will notice and appropriately address patients who are unable to sustain attention. The groups were similar in their ability to focus and shift attention, consistent with previous research[1] and findings that suggest memory functioning is a specific predictor of PTSD status separate from attention.[5]

The main limitation of our study is its small sample size. However, the differences in memory between our groups remain significant after correcting for multiple comparisons and controlling for possible contributing variables. Given the small sample size, further research is encouraged. Additional limitations include limited information on participants' premorbid psychiatric history and their memory functioning. The memory deficits may have existed prior to trauma exposure, or could have developed in its aftermath in vulnerable individuals. Regardless of the origin of memory dysfunction, our study indicates that patients with poor verbal memory make less progress with CBT than patients with similar levels of PTSD without memory deficits. This finding has implications for improved treatment for PTSD as it may guide adjustments to CBT to compensate for verbal memory deficits, such as summarising key points frequently. Alternatively, patients with poor verbal memory and PTSD may benefit from other NICE recommended treatments that are possibly less reliant on verbal memory capacity, such as eye movement and desensitisation reprocessing therapy, or drug treatment in cases with severe depression.

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EXTRA CONTENT ONLINE

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