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Working memory function is linked to trauma exposure, independently of post-traumatic stress disorder symptoms

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ABSTRACT

Introduction: The purpose of the study was to examine how working memory (WM) may be related to exposure to potentially traumatic events and symptoms of post-traumatic stress disorder (PTSD).

Method: In four studies, we measured WM function using adaptations of the running span and the reading span tasks. We compared the performance of women reporting experiences of sexual abuse to control participants (total $n = 144$ controls and 84 victims). We measured severity of the sexual abuse experiences as well as exposure to general life stress.

Results: In all studies, trauma-exposed participants showed significantly lower WM function compared to control participants. In addition to traditional null hypothesis testing, we used a mini-meta analysis to estimate the combined estimated effect size of this difference, which was in the moderate range ($d = 0.43$ with 0.15–0.70 95% confidence interval). Regression equations showed that PTSD symptoms did not mediate the relationship between trauma exposure and WM function.

Conclusions: Our results show that trauma exposure per se can be associated with important cognitive correlates even in individuals who do not develop psychopathological reactions.

KEYWORDS

Trauma; working memory; sexual abuse; executive function; PTSD

A majority of individuals will experience at least one potentially traumatic event (PTE) during their lifetime, and following this, most will not develop psychopathological reactions such as post-traumatic stress disorder (PTSD) (Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). The impact of PTEs on individuals who do not develop PTSD is not well understood. In particular, though important cognitive correlates of PTSD have been identified (Tapia, Clarys, El-Hage, & Isingrini, 2007), it is not clear if and how exposure to PTEs can in itself be related to cognitive functioning. In this paper, we report studies probing working memory (WM) in participants exposed to trauma (sexual abuse) and in non-exposed control participants.

A traumatic event is an event that is shocking and emotionally overwhelming that can involve actual or threatened death, serious injury or threat to physical integrity. A large portion of the population will be exposed to at least one PTE in their life time; estimates range from 60% to 89% (Breslau, 2009; Resnick et al., 1993). The majority of individuals exposed to PTE do not develop PTSD (lifetime prevalence of PTSD is estimated between

7% and 12%; Breslau, 2009; Resnick et al., 1993). Whereas the health correlates of PTE exposure have been examined (e.g., links between PTE and depression, Suliman et al., 2009, or general physical health), their potential cognitive correlates have not received much interest, despite increasing evidence that PTSD following PTE exposure is linked with cognitive functioning.

PTSD is associated with broad neurocognitive characteristics including alterations to verbal learning, verbal memory, attention and speed of information processing (Scott et al., 2015). In particular, a number of studies have documented lower episodic memory function in individuals suffering from PTSD compared to controls (see Isaac, Cushway, & Jones, 2006 for a review). A smaller number of studies have documented alterations of WM associated with PTSD (Schweizer & Dalgleish, 2011; Vasterling, Brailey, Constans, & Sutker, 1998). In one experiment using an auditory n-back task (Galletly, Clark, McFarlane, & Weber, 2001), participants with PTSD were found to be less accurate than control participants matched on age and education. Other studies have documented similar differences in accuracy between PTSD-suffering and control participants using various WM tasks (Bomyea, Risbrough, & Lang, 2012; Moores et al., 2008; Morey et al., 2009). Effect sizes of the difference between groups in these studies are usually large (d values between 0.96 and 1.5). In some studies, differences between groups ($d = 0.87-1.38$) were found in reaction times rather than in accuracy (Clark et al., 2003; Weber et al., 2005).

The findings of these studies seem to point to impairment in WM function related to PTSD. However, results of the broader literature are equivocal. First, a number of studies have failed to find significant differences between PTSD-suffering and control participants, sometimes using similar WM tasks (LaGarde, Doyon, & Brunet, 2010; Neylan et al., 2004; Schweizer & Dalgleish, 2011). Second, there are inconsistencies in that the same tasks sometimes produce differences in reaction times and not accuracy, and sometimes in accuracy but not reaction times (Galletly et al., 2001; Moores et al., 2008). Because generic processing speed deficits have been documented in PTSD (Nelson, Yoash-Gantz, Pickett, & Campbell, 2009), one can question whether there is a specific effect related to WM. Finally, the question of causality is one of the most difficult to settle. Lower cognitive abilities (lower IQ) is a risk factor for developing PTSD (Macklin et al., 1998). A large portion of the variance in fluid intelligence is associated with WM function (Engle, Tuholski, Laughlin, & Conway, 1999). Thus, it is not clear whether PTSD leads to WM impairments or if WM impairments predispose to or precipitate the development of PTSD.

Moreover, and central to the current investigation, it is not yet clear whether the differences documented in WM function are actually related to PTSD or to trauma exposure. In the literature on WM and PTSD, participants suffering from PTSD are most often compared to control participants who have not been exposed to trauma. Only a few studies use both a non-exposed non-PTSD and an exposed non-PTSD group for comparison (e.g., LaGarde et al., 2010). Because most studies compare groups that vary on both PTSD status and PTE exposure, it is difficult to determine if differences in WM are related to PTSD symptoms or to trauma exposure.

There are reasons to suspect that, independently of PTSD, trauma exposure itself may be linked with decreased WM function. Experimental inductions of stress (Luethi, Meier, & Sandi, 2008; Schoofs, Preuss, & Wolf, 2008) and naturally occurring acute stressors (e.g., military training; Taverniers, Van Ruyseveldt, Smeets, & von Grumbkow, 2010) can cause impairments in WM function. Laboratory-created emotional events have also been shown

to alter WM function. In a series of studies, Curci, Lanciano, Soleti, and Rimé (2013) and Curci, Soleti, Lanciano, Doria, and Rimé (2015) exposed participants to emotional videos or text (for instance, describing an episode of torture) or neutral equivalents. They tested WM function before and after this emotional event. In the neutral control condition, WM performance improved over the two tests. This was not the case when an emotional event had been experienced, especially when the WM task was verbal in nature. This negative impact of emotion exposure on WM seemed to be particularly related to the level of unintended thoughts related to the emotional event. These thoughts may take up WM capacity, which would decrease task performance. This would be consistent with theoretical and neurofunctional models of emotion-cognition interactions, for instance that of Pessoa (2005, 2009). This model suggests that the amygdala acts as a significance detector. Emotional events are flagged, because of their significance; this results in an increased representation in sensory areas (e.g., visual cortex) as well as prioritised deployment of executive resources. The amygdala relays information concerning emotional value to the anterior cingulate cortex (ACC) which integrates it with other information, prioritises it, and relays it to prefrontal structures. In particular, there are important links between the ACC and the dorsolateral prefrontal cortex, responsible for executive functions, including WM updating and inhibition. This model explains why emotional stimuli, including unintended thoughts about emotional experiences, may disrupt WM function.

Experimental studies have established that emotional events can cause decrements in WM function, and have started to examine possible mechanisms for this effect. However, these studies offer only a very partial analogy to trauma. They examine relatively mild forms of emotional events and probe very short-term effects on cognitive function. Other studies have documented an association between real-life stress exposure and long-term cognitive function. The number of important stressful events that participants report being exposed to during their lifetime appears to be negatively linked to WM performance (Klein & Boals, 2001; Stawski, Sliwinski, & Smyth, 2006). Further, there is a relationship between the amount of daily stress reported and concurrent WM accuracy (Stawski, et al., 2006) and reaction time (Sliwinski, Smyth, Hofer, & Stawski, 2006), in an n-back task. However, because most of these studies do not include a measure of PTSD symptoms, it is possible that the observed link between life stress and WM function may be explained by PTSD symptoms.

Altogether the weight of the evidence indicates that (1) there is a link between induced stress or exposure to an emotional event and short-term negative effects on WM, (2) there is a possible link between PTSD and long-term effects on WM and (3) there might be a link between life stress and long-term WM function. Our interest lies in determining whether exposure to PTEs may in itself be related to long-term WM function, independently of PTSD, or instead if PTSD symptoms necessarily mediate the effect of PTE on WM. Whereas exposure to intense stressors is a precondition for PTSD, the two also have unique variance: an important number of individuals will not develop PTSD despite high traumatic exposure. WM function might be directly associated with exposure to PTE, or to psychopathological reactions to PTE, or to both.

Overview of studies

In four studies, we examined the association between PTE exposure, PTSD symptoms and WM function. We used an approach where we compared two groups of participants, one

exposed and one non-exposed to a common potential trauma: sexual abuse. We only recruited women as the prevalence of sexual abuse is higher in this group (Gouvernement du Québec, 2014). The two groups were matched for age, gender and education. In addition, we measured self-reported life stress and PTSD symptoms. In three studies, we measured WM function using a running span task (Pollack, Johnson, & Knaff, 1959), in a fourth one we used the reading span task (Unsworth, Heitz, Schrock, & Engle, 2005). Both tasks are primarily verbal in nature. We report the methodology for the four studies together and identify distinguishing features of each. We focus our analysis on the effect size of the difference between the groups across studies, rather than null hypothesis testing, consistent with current thinking and recommendations from an important number of sources (Cumming, 2014; Miles & Field, 2007; Trafimow & Marks, 2015).

Method

Participants

Participants were recruited from the student and staff community at the Université du Québec à Trois-Rivières (UQTR) (Québec, Canada), as well as in the local community, between 2010 and 2013. We advertised the studies as exploring the link between sexual abuse and cognitive function in women. Inclusion criteria were (1) to be aged 18 years or older, (2) to be able to speak and understand French and (3) to be a woman. Exclusion criteria were (1) to have a diagnosis of psychotic disorder, (2) to suffer from a neurological disorder or have suffered a traumatic brain injury causing a loss of consciousness of more than 10 minutes, (3) to have a substance abuse problem, (4) to have a diagnosis of Attention Deficit Disorder with or without hyperactivity, (5) to have a learning disability or (6) to present symptoms of depression more severe than symptoms of PTSD (Study 4 only). PTSD was neither an exclusion nor an inclusion criterion.

Assignment to the victim/control group was based on answers to the Early Trauma Inventory-Self Report (ETI-SR) sexual events (see later). Participants who endorsed any of the five items that meet the legal definition of sexual abuse (“agression sexuelle”, Gouvernement du Québec, 2014) were included in the exposed group.

Number, age and education level of participants in the victim and control groups in each study can be found in Table 1. Level of education could be indicated as completed primary (1), secondary (2), college (3), undergraduate university (4) or post-graduate

Table 1. Description of sample characteristics.

	Age			Education		
	Victims	Controls	<i>t</i> Value	Victims	Controls	Percentage college/university education
Study 1	30.5 (9.9) <i>n</i> = 24	24.0 (4.7) <i>n</i> = 33	3.3*	100%	100%	$\chi^2(2, N = 57) = 0.7$
Study 2	23.0 (3.8) <i>n</i> = 22	22.9 (5.8) <i>n</i> = 37	0.7	97%	95%	$\chi^2(3, N = 60) = 2.7$
Study 3	21.7 (3.7) <i>n</i> = 23	21.1 (2.9) <i>n</i> = 45	0.7	91%	82%	$\chi^2(3, N = 60) = 2.3$
Study 4	35.3 (18.4) <i>n</i> = 15	25.7 (14.5) <i>n</i> = 29	1.9	93%	100%	$\chi^2(3, N = 46) = 2.6$

* Significantly different at $p < 0.05$.

university level (5). Chi squares confirmed that educational level did not differ across groups. In terms of age, it can be seen that the groups significantly differed only in Study 1, where victims were older than controls. Where appropriate, we controlled for age in the statistical analyses.

Measures

A summary of the questionnaires used to assess the different variables in the four studies can be found in Table 2.

Severity of experiences of abuse. The sexual abuse section of the ETI-SR was used as an indicator of experiences of sexual abuse and their severity. We adapted the instructions to refer to lifetime occurrence rather than childhood occurrences only. The ETI-SR includes six questions about sexual events asking participants to indicate, yes or no, whether they have had certain types of unwanted sexual experiences, including kissing, fondling, genital and oral sexual relations. We excluded one question from our analyses, as it does not unambiguously meet the legal criterion for sexual abuse in the province of Québec, mainly because it does not mention absence of consent. A yes answer to any of the other five questions was used as a criterion for inclusion in the exposed group. Number of yes answers was summed to produce scores that represent an indication of severity of abuse.

General life stress. In all four studies, we used self-reported instruments to assess lifetime exposure to other stressful/potentially traumatic experiences. In Study 1, we used the three other sections of the ETI-SR, which measure physical abuse, emotional abuse and general trauma. In the three other studies, we used the Life Event Inventory which is a 55-item list of generally stressful life events and PTEs. Items include events such as divorce, prolonged illness or substance abuse. In both cases scores are summed and higher scores represent more exposure.

In addition, in Study 4, we also administered the Life Event Checklist (LEC), which is part of the structured interview we used in that study: the Clinician Administered PTSD Schedule (see later). The LEC assesses the occurrence of all the PTEs listed in the Diagnostic and Statistical Manual of Mental Disorders-IV.

PTSD symptoms. PTSD symptoms were assessed through self-report questionnaires in Studies 1–3. In Study 1, we used the Primary Care PTSD, a four-item screening instrument used in primary care (Prins et al., 2003) and the Impact of Event Scale-Version Française (IES-VF) (Brunet, St-Hilaire, Jehel, & King, 2003). In Studies 2 and 3, participants in the exposed group completed the PTSD Checklist, Civilian Version (PCL-C) (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996).

Table 2. Summary of the measures used in the four experiments.

	WM	Severity of experiences of abuse	Other life stresses	PTSD symptoms
Experiment 1	Running span	ETI-SR – sexual abuse	ETI-SR – other	IES PC-PTSD
Experiment 2	Running span	ETI-SR – sexual abuse	LEI	PCL-C
Experiment 3	Running span	ETI-SR – sexual abuse	LEI	PCL-C
Experiment 4	Reading span	ETI-SR – sexual abuse	LEI LEC (from CAPS)	CAPS

Notes: ETI-SR, Early Trauma Inventory-Self-Report; LEI, Life Event Inventory; CAPS, Clinician Administered PTSD Schedule; PC-PTSD, Primary Care PTSD and PCL-C, Post-Traumatic Checklist, Civilian Version.

In Study 4, we used a French version of the well validated and broadly used Clinician Administered PTSD Scale (CAPS) (Saint-Onge, n.d.). The CAPS is a structured interview assessing the presence and severity of PTSD symptoms. The structure is based on criterion B, C and D of the DSM-IV: re-experiencing, avoidance and hyperarousal. Each item is scored on a scale from 0 (never) to 4 (extremely).

WM assessment. In Studies 1–3, we used a running span task adapted from Vieillard and Bougeant (2005). Participants were presented with series of consonants (from four to nine consonants), without repetition. Each consonant was presented for 850 ms visually in the centre of the screen, with a 650-ms interstimulus interval. The presentation of the last item of the series was indicated by a 950-ms beep. Following this, participants had to type in the last four items of lists of four, six or eight consonants (block 1), or the last five items of lists of five, seven or nine items (block 2). There were 12 different trials in each block. This task requires participants to update the contents of memory, because they do not know when the list will end. It therefore measures WM, rather than short-term memory. In this particular task, retaining and updating four items represents a lower load, whereas five items represents a higher load (Vieillard & Bougeant, 2005). The low load was presented in the first block and the high load in the second block. Before the start of each block, there were two practice trials. Accuracy was the dependent variable analysed. There was no time limit for participants to provide their response. A trial was coded as correct (1) if the participants recalled all items in the correct order, otherwise it was coded as incorrect (0). In Study 3, only the high load condition was used.

In Study 4, we used a locally translated and adapted version of the automated reading span (Redick et al., 2012). In this task, participants perform a storage task and a processing task. They must remember a random sequence of consonants. The presentation of these consonants is interspersed with a verbal task in which participants have to determine whether a sentence is grammatically correct or not. Participants first practiced memorising the letters (4 trials), then practiced the verbal task alone (15 trials) and then practiced 3 trials with both tasks combined. The actual experimental block comprised 15 trials, presented one at a time, with letter sets varying from 2 to 6 items to be remembered (with an equivalent number of sentences to process in between). The length of the intertrial intervals were determined by participants who pressed the space bar when they were ready to start the next trial. Again, a trial was coded as accurate if participants could recall all the letters in the correct order.

Procedure

The data reported in this paper were collected as part of a larger investigation of the cognitive correlates of trauma. Participants came for one session (Studies 1–3) or two sessions (Study 4) and completed a number of other cognitive tasks in addition to the one described here, namely, a deductive reasoning task, an attentional cueing task, an attentional network task and a task assessing the interpretation of ambiguous stimuli. Because the theoretical questions addressed with these tasks are different, these data are not reported here. The tasks, including questionnaires and cognitive tasks, were presented using EPrime.

Participants first read the information sheet and signed the consent form. They were informed that the study included questions about personal, potentially difficult and

emotional topics and that even if they agreed to participate, they could skip any question, or interrupt their participation at any time. All studies were approved by the ethics committee of UQTR.

The general procedure followed this sequence: demographic questions, cognitive tasks (always in the same order, WM task was the second to last task) and then questionnaires. Participation ended with a debriefing and referral to appropriate services if necessary. This general procedure differed only in Study 4, for which participants came to the laboratory on two different occasions. The first session consisted in administering the CAPS and filling out the self-reported questionnaires. Participants completed the cognitive tasks in a second session.

Data analysis

Our main metric of interest is the effect size of the difference between WM accuracy in the exposed and control groups, which we quantify using Cohen's d . The data are combined in a mini-meta analysis used to compute the confidence interval (CI) of the estimated difference in accuracy between groups (Cumming, 2014). Although we report significance testing of the difference between the groups, we focus on effect sizes and CIs of the combined studies following recommendations of best statistical practice emanating from different sources, including the Association for Psychological Science and the American Psychological Association (Cumming, 2014; Trafimow & Marks, 2015). There are important, insoluble problems with null hypothesis testing. Focusing on estimates of effect size and combining results across studies has been recommended as a key to improving replicability in science and more meaningful data analysis.

The standardised scores on all independent and dependent variables are also combined across studies to examine the interrelations between variables. Regression models are used to determine if PTE exposure, severity of reported experiences and general life stress are related to WM function, and, importantly, if this relation is mediated by PTSD symptoms. This mediation hypothesis was tested using two methods: following the procedure described by Hayes (2013) and the Sobel test. We used the PROCESS SPSS computational tool (Hayes, 2012) to calculate the direct relation between WM performance and severity of exposure as well as the indirect relation, through PTSD symptoms. The strength of this indirect relation indicates the extent to which the link between WM function and severity of exposure is actually related to increased PTSD symptoms.

Results

Average accuracy on the WM tasks is reported for all conditions (low, high load), groups (exposed, controls) and studies in Table 3. It can be seen that participants in the exposed group consistently presented lower WM scores compared to controls, in all conditions, in all studies. This difference between groups was tested for the average span in each study separately using one-tailed independent sample t -tests, as the prediction was that participants in the exposed group would show lower WM accuracy than those in the control group. The difference was significant in all cases.

More importantly, we calculated effect sizes to quantify the extent of these differences in accuracy between groups, using Cohen's d . Results are presented in Figure 1, with black

Table 3. Average performance on WM tasks in the four studies, presented by group and difficulty level (when relevant).

	Average WM accuracy Mean (SD)	Low load condition Mean (SD)	High load condition Mean (SD)
Study 1			
Controls	0.41 (0.17)	0.55 (0.20)	0.28 (0.21)
Victims	0.34 (0.15)	0.46 (0.21)	0.22 (0.14)
	$t(55) = 1.63, p = .05$		
Study 2			
Controls	0.32 (0.14)	0.43 (0.16)	0.21 (0.14)
Victims	0.26 (0.14)	0.36 (0.18)	0.16 (0.11)
	$t(57) = 1.73, p = .04$		
Study 3			
Controls	0.28 (0.17)		0.28 (0.17)
Victims	0.20 (0.14)		0.20 (0.14)
	$t(66) = 1.85, p = .03$		
Study 4			
Controls	0.41 (0.18)		
Victims	0.27 (0.16)		
	$t(42) = 2.64, p < .01$		

squares indicating the effect size in each study (the size of the square represents the sample size) along with 95% CIs (black lines). The estimate produced from the combined data is represented by the diamond. Overall the combined studies show a moderate effect size estimated at 0.43 (weighed by sample sizes) with a CI between 0.15 and 0.70. This shows that there is a consistent difference of moderate size in the average WM accuracy of the exposed and non-exposed groups.

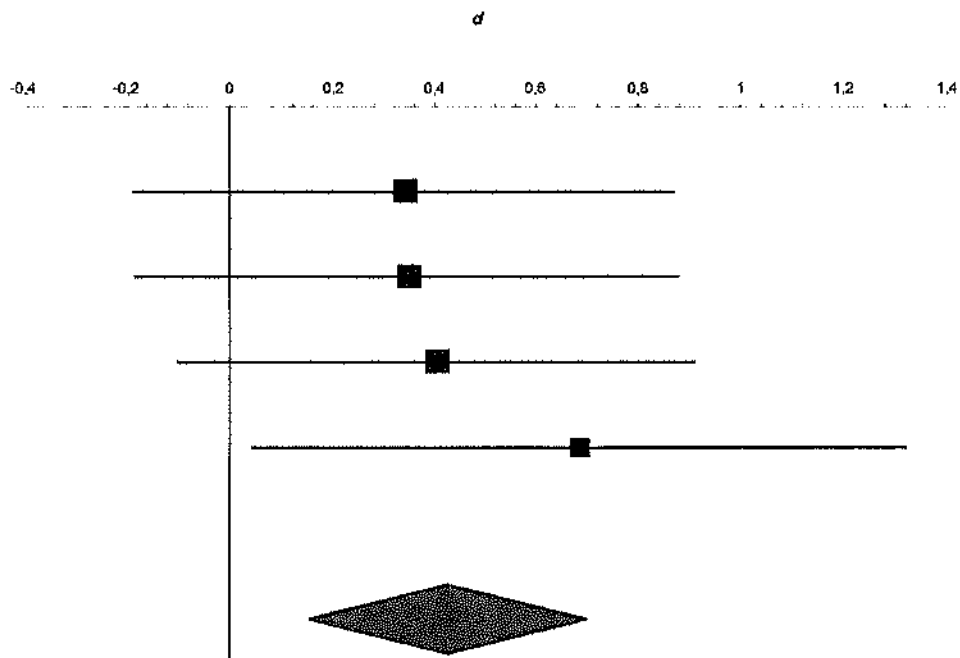


Figure 1. Effect size of the difference in WM function between exposed and control groups in the four studies (squares) and combined (diamond), weighed by sample size and with 95% CIs.

Table 4 reports the correlations between the different variables in the combined data, using standardised scores from each study. It can be seen that WM accuracy correlated significantly with the reported severity of experiences of sexual abuse as well as with general life stress. WM accuracy did not, however, correlate with level of PTSD symptoms.¹ Intercorrelations between the questionnaire measures show that general life stress was positively related to experiences of sexual abuse and that level of PTSD symptoms was positively related to general life stress and to the severity of experiences of abuse, as would be expected.

We classified participants as PTSD or non-PTSD, using the recommended clinical cut-off scores for the respective instruments used in the different studies. The number of victims reaching this criterion was 10 (41.7%), 5 (23.8%), 6 (26.1%) and 4 (26.7%) for Studies 1 through 4, respectively. Overall, the proportion of participants reaching the PTSD cut-off was greater in the victim group (30.1%), compared to the control group (4.3%), $\chi^2(1, N = 177) = 21.5, p < .01$. We compared WM performance (using the standardised scores of accuracy) of the PTSD group ($M = 0.10, SD = 1.07, n = 28$) and non-PTSD group ($M = -0.06, SD = 1.01, n = 147$). The means did not differ significantly between groups, $t(173) = 0.73, p = .47, d = -0.14$.

The final step in our analyses was to investigate whether trauma and stress exposure (sexual abuse experience and general life stress) was directly related to WM accuracy or whether the relationship was mediated by PTSD symptoms. We considered exposure using three measures: group (dummy coded as 0 for controls and 1 for victims of sexual abuse), severity of reported experiences of sexual abuse and general life stress. We first examined whether these three variables together predicted WM function, above and beyond age which was entered in a first step. The model was significant, $F(4,222) = 6.85, p < .01, R^2 = .11$. More importantly, the second step (including the exposure variables after age) provided incremental significance, $F(3,222) = 7.16, p < .01$, incremental $R^2 = .09$. Group ($\beta = -.25, p = .02$) and general life stress ($\beta = -.19, p < .01$) were the more important predictors, compared to severity ($\beta = .11, p = .31$). Following this, we examined whether this relationship between stress exposure and WM was mediated by PTSD symptoms. We used the procedure described by Hayes (2013) to test for mediation. This procedure determines mediation by examining the strength of the indirect relation between trauma exposure and WM, through PTSD symptoms. We used the predicted values from the first model, therefore a composite score representing severity of exposure, as our independent variable, PTSD symptoms as the mediator, and WM accuracy as the dependent variable. The direct effect, between exposure and WM was estimated at .34

Table 4. Correlations between WM performance and other variables.

	WM accuracy	Severity of abuse	General life stress
Severity of abuse	-.16* <i>n</i> = 228		
General life stress	-.27** <i>n</i> = 227	.35** <i>n</i> = 230	
PTSD	-.08 <i>n</i> = 144	.52** <i>n</i> = 145	.40** <i>n</i> = 145

Notes: *R* values of correlations between the different variables, with valid *n* for each. Values remained similar when controlling for age (WM and severity, $r = -.13, p = .06$; general life stress $r = -.02, p < .01$ and PTSD $r = -.09, p = .27$).

*Significant at $p < .05$.

** Significant at $p < .01$.

(95% CI [.18, .49]). The estimated magnitude of the indirect relation, between exposure and WM through PTSD, was small ($-.02$, 95% CI [$-.09$, $.04$]) and the CI included 0, suggesting that the relationship between exposure and WM function was not mediated by PTSD symptoms. Moreover, though not significant, the direct relationship between PTSD and WM was actually positive, in contrast to the negative relationship between stress exposure and WM. An alternative method for testing mediation, the Sobel test, also confirms that the relationship between exposure and WM function is not mediated by PTSD symptoms, $z' = 0.15$, $p = .88$.

Discussion

We examined the possible cognitive correlates of exposure to one PTE: sexual abuse. We found that women reporting experiences of sexual abuse generally showed lower WM performance compared to non-exposed women. Moreover, WM function was more generally systematically negatively related to reports of stressful life experiences. Our findings, therefore, confirm the existence of a link, of moderate size, between trauma/stress exposure and one important cognitive function: WM. Importantly, our results indicate that this link is not necessarily mediated by PTSD symptoms. This suggests that trauma exposure per se can be associated with cognitive correlates, independently of psychopathological symptoms following PTE.

Our studies are correlational, necessarily, since it is impossible to randomly assign participants to either a trauma-exposed or control group. We cannot, therefore, conclude that trauma exposure causes WM deficits. Nevertheless, there are potential cognitive and physiological mechanisms to support an impact of trauma or stress on WM. Cognitively, the experience of emotional events is associated with intrusive thoughts and rumination; trauma can be associated with the activation of irrelevant semantic contents generally (Falsetti, Monnier, Davis, & Resnick, 2002). According to this account, trauma would be associated with increased task-irrelevant thoughts that would take up WM capacity, leading to decreased accuracy or longer reaction times on WM tasks. This is consistent with experimental studies exposing participants to emotional event in the laboratory. Curci et al. (2013, 2015) observed that such exposure altered performance on a WM task, and that these emotion-related alterations were largely related to unintended thoughts linked to the emotional event.

Rumination or unintended thoughts, therefore, represents one possible mechanism to account for the link between trauma exposure and WM function. Studies have shown that most individuals experience unintended thoughts about negative life experiences, often many years after their occurrence (Lehman, Wortman, & Williams, 1987; Tait & Silver, 1989). These emotional thoughts may be prioritised, given their significance, and mobilise the allocation of executive resources, consistent with Pessoa's model of emotion-cognition interactions. This suggests that the tendency to ruminate may be an important variable moderating the link between exposure to trauma and WM function. Studies have shown that individuals who ruminate may have particular difficulties inhibiting irrelevant negative contents from WM, leading to more task interference (Joormann & Gotlib, 2008). This may be even more characteristic of brooding, a type of rumination that involves a passive comparison of one's current situation to unachieved goals (Bernblum & Mor, 2010). In that study, WM updating was examined in particular. Whereas all participants

showed a tendency to update negative stimuli more readily, consistent with the attentional prioritisation of emotional contents, brooders showed interference from the mere presence of negative stimuli, even when these were not task relevant. In the context of our study, this suggests that participants with a tendency to ruminate or brood may be especially likely to have task-irrelevant trauma-related thoughts which would interfere with WM performance. Such individual differences could be measured in future studies.

Concerning the link between inhibition of task-irrelevant thoughts and WM function, it is worth noting that the relation should be considered bidirectionally. One experimental study has shown that induced emotional events had a greater impact on task performance of low WM individuals, compared to high WM capacity individuals (Curci et al., 2013). Individuals lower in executive function may be less able to inhibit task-irrelevant thoughts, especially if these are emotional. Indeed, that study showed that low WM individuals reported more rumination following, and up to 24 hours after experiencing the emotional events. The involvement of executive processes can reduce the impact of task-irrelevant emotional distractors (Cohen, Henik, & Mor, 2011). In a modified version of the attentional network task, emotional cues impacted performance only in congruent trials (when distractors and targets evoked the same response). These trials do not require the involvement of executive processes as much as incongruent trials where distractors lead to a response that must be inhibited. In these incongruent, more difficult trials, the impact of emotional task-irrelevant cues was reduced, showing that involving executive processes attenuates the impact of task-irrelevant emotions. In another study (Cohen et al., 2011), it was shown that this effect is particularly pronounced for individuals who tend to use reappraisal as an emotion regulating strategy. This is consistent with the idea of an important link between controlled executive processes and the impact of task-irrelevant emotional stimuli on cognitive performance. Concerning the link between trauma exposure and WM function, both individual differences in executive function and emotion regulation may be important moderators to consider. Specifically, high executive abilities or high WM capacity may act as a protective factor attenuating the link between task-irrelevant emotional thoughts and cognitive performance.

Whereas the reviewed studies provide important suggestions concerning possible cognitive mechanisms underlying the relation between trauma exposure and WM function, it is also possible to envisage physiological mechanisms. A study by Evans and Schamberg (2009) suggests that the link between chronic stress and WM function is mediated by allostatic load (in this case including resting blood pressure, overnight cortisol and catecholamines, and body mass index). In that study they observed a negative correlation between the proportion of childhood spent in poverty (0–13 years) and performance on a WM task, which disappeared when allostatic load was entered as a mediator in the regression equation. Moreover, stress induces the release of hormones such as cortisol which have an impact on neurocognitive function (de Quervain, Aerni, Schelling, & Roozendaal, 2009), in particular activity of the dorsolateral prefrontal cortex, a structure that is importantly implicated in WM (Curtis & D'Esposito, 2003). Whereas more research is needed to identify the causal chain, there is a possible neurophysiological pathway between stress/trauma exposure and WM, importantly involving the dorsolateral prefrontal cortex.

Whereas there are possible mechanisms to support the view that trauma exposure affects WM, the reverse relation is also possible. As highlighted previously, our study is

correlational in nature and hence we cannot draw conclusions concerning the direction of the link. In the PTSD literature, lower cognitive abilities (IQ) have been shown to predate trauma exposure (Breslau, Lucia, & Alvarado, 2006). Though it is uncomfortable to conceive, and maybe implausible, our data do not allow us to rule out the theoretical possibility that pre-existing differences in WM abilities could predispose individuals to become victims of sexual abuse. Some prospective studies have shown that IQ can serve as a protective factor against both exposure to PTEs (Breslau et al., 2006) as well as against the development of PTSD symptoms following traumatic exposure (Gilbertson et al., 2006). Generally, studies also provide evidence that lower IQ is a risk factor for PTSD (Gale et al., 2008). WM is highly correlated with general cognitive functioning including IQ (Qureshi et al., 2011). It is possible that our results highlight one facet of this general link between cognitive functioning and trauma. However, we do not know of any data suggesting a link between IQ and the likelihood of being the victim of sexual abuse.

Another, maybe more plausible account of the inverse causal path (from WM function to increased trauma exposure) is that pre-existing cognitive differences may lead to differences in self-reports of negative experiences. Individuals with lower WM capacity may be more likely to report negative experiences, including experiences of sexual abuse, than individuals with higher WM capacity. Though there is no direct evidence to support this, future studies will be needed to rule out this possibility.

Our investigation presents a number of limitations. Because we focused on trauma exposure, few of our participants exhibited clinically significant levels of PTSD symptoms. Moreover, half of the participants reaching the criterion for PTSD came from one of the four studies, which used a very brief self-report measure of PTSD symptoms. Thus results concerning the comparisons between PTSD and non-PTSD groups must be interpreted with caution. Moreover, PTSD was assessed mostly with self-report questionnaires; only Study 4 included an exhaustive clinical assessment of PTSD (using the CAPS). Another important limitation is that we did not measure when the abuse last occurred, hence we do not know the time that has elapsed since the events. This may be a crucial variable moderating the link between exposure and WM function. It could be expected that the correlation may be stronger when events are more recent. This will need to be assessed directly and considered in future studies. Finally, as mentioned, we cannot conclude that exposure to trauma is causally linked to lower WM function, as our study is correlational and we did not have a pre-trauma measure of WM function. Prospective studies will be necessary to further investigate this link.

Overall, these data have provided important new insights concerning the relation between trauma and cognitive function. Results establish a clear link between WM function and exposure to a PTE. This is important to consider when working with different populations that have been exposed to potentially traumatic experiences or stressful life circumstances. Using a novel analytic strategy, recommended to solve the many problems associated with null hypothesis testing, our results show that this effect is of moderate size. Obviously these results will need to be replicated and confirmed using multiple methodologies and data analytic strategies. Importantly, longitudinal studies should be conducted to determine the direction of this relationship. Our results also clearly show that this relationship between trauma and WM is not mediated by PTSD symptoms. This does not mean that there are no WM deficits specifically associated with PTSD (and indeed

this has been shown to be the case, see Qureshi et al., 2011), but it does mean that trauma exposure can be associated with altered WM function even in the absence of PTSD symptoms.

Note

1. In Study 1, we used IES score as our measure of PTSD symptoms.

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