

Metacognitive and Metamemory Beliefs in the Development and Maintenance of Posttraumatic Stress Disorder

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Abstract

Can metacognition increase trauma sufferers' risk for developing and maintaining posttraumatic stress disorder (PTSD)? We assessed the role of a range of cognitive and metacognitive belief domains—including metamemory—on PTSD symptoms. Adult participants reported their existing meta/cognitions and lifetime exposure to trauma, then 12 weeks later, they reported meta/cognitions and PTSD symptoms in relation to new trauma exposure since the initial assessment. Participants with more PTSD symptoms held more problematic metacognitions than participants with fewer distress symptoms. Moreover, people who endorsed maladaptive metacognitions before trauma exposure were more likely to experience symptoms of PTSD after exposure. Metacognition predicted the maintenance of elevated PTSD symptoms over the 12-week delay. Our findings support the metacognitive model of PTSD and highlight the importance of metamemory, an understudied factor in PTSD research.

Keywords

well-being, trauma, posttraumatic stress disorder, cognitive processes, cognition

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Exposure to a sudden or sustained stressful experience can lead to psychological problems such as posttraumatic stress disorder (PTSD). Hallmark symptoms of PTSD include repeated and unwanted reexperiencing of the event, negative alterations in arousal, reactivity, cognition and mood, and avoidance of trauma reminders (American Psychiatric Association [APA], 2013). Yet, attesting to human resilience, not all trauma-exposed people develop PTSD (Lee, 2006). Determining why some trauma-exposed people develop serious psychopathology when others do not is of critical clinical significance. Recently, metacognition—beliefs about thinking that guide our thinking and coping—has received attention for its role in PTSD (Wells, 2000). PTSD sufferers who endorse maladaptive metacognitive beliefs posttrauma tend to exhibit more PTSD symptoms (e.g., Roussis & Wells, 2006). However, research to date has not examined the role metacognition might play in trauma reactions over time. Here, we examined whether dysfunctional metacognition

pretrauma predicted PTSD symptomatology posttrauma, and whether metacognitive beliefs predicted the maintenance of elevated PTSD symptom levels over time.

Wells's (2000; Wells & Sembi, 2004) metacognitive model focuses on how people's metacognitive beliefs can lead to PTSD. It stipulates that intrusions, startle responses, and increased arousal are normal responses to trauma, forming part of a self-righting, reflexive adaptation process (RAP) that initiates automatically and determines adjustment and recovery. The RAP's goal is to simulate plans for future threats. Thus, symptoms should subside once a satisfactory plan is established. However, metacognitive beliefs that encourage dysfunctional thinking styles or maintain focus on danger or the person's

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unsatisfactory reactions to trauma—worry/rumination, thought suppression, threat monitoring—can obstruct the RAP and thus interfere with spontaneous recovery from trauma.

Researchers have investigated a range of maladaptive cognitive and metacognitive beliefs trauma-exposed people hold. We know people who negatively appraise their traumatic experience are at increased risk of pathology (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999) and people who interpret their intrusive memories negatively are less able to overcome their posttraumatic symptoms (e.g., Ehlers, Mayou, & Bryant, 1998). Recent evidence suggests that training people to adopt a positive appraisal style regarding their ability to appropriately respond to trauma leads to fewer analogue symptoms (Woud, Holmes, Postma, Dalgleish, & Mackintosh, 2012). Moreover, Kleim et al. (2013) found that changes in dysfunctional trauma-related appraisals led to decreased PTSD symptoms among PTSD patients who received trauma-focused cognitive behavior therapy.

Indeed, people who hold maladaptive beliefs *pre*-trauma may be predisposed to develop PTSD. Bryant and Guthrie (2005) found that trainee firefighters with a pre-existing tendency for negative self-appraisal—but not cognition concerning self-blame or the world as unsafe—were more symptomatic 6 months later. These results suggest that studying people's cognition—and perhaps their metacognition—pretrauma may help identify people most at risk for PTSD symptomatology.

Bennett and Wells (2010) found student nurses who endorsed negative metamemory beliefs (“having gaps in memory of the event means I am not normal”) were more likely to exhibit PTSD symptoms after a distressing event during their training. Moreover, such beliefs predicted PTSD better than objective indicators of memory problems in participants' recall narratives. These data suggest that metacognitive beliefs about memorial problems may be an important area for further research.

In summary, extant research demonstrates that dysfunctional meta/cognition may render people more vulnerable to increased PTSD symptomatology. Does metacognition also play a role in *maintaining* posttraumatic stress? Some studies show that posttrauma cognition independently predicts PTSD when measured between several months and 1 year posttrauma (e.g., Ehlers et al., 1998; Halligan, Michael, Clark, & Ehlers, 2003) and, in children, mediates the relationship between initial and longer term PTSD symptoms (Meiser-Stedman, Dalgleish, Glucksman, Yule, & Smith, 2009). However, to our knowledge, no study has investigated whether metacognition contributes to the *maintenance* of persistent PTSD symptoms in adults over time.

The Present Study

We investigated the role of meta/cognitive factors in predicting and maintaining PTSD among a nonclinical adult population. Even short research time frames can capture a high prevalence of trauma in nonclinical populations (e.g., 8 weeks; Frazier et al., 2009). However, to increase the likelihood that we would capture trauma, we assessed participants over 12 weeks. We measured participants' trauma-related cognition, metacognition, and PTSD symptoms pre- and post-any recent trauma exposure. Our aims were threefold. First, we examined the cross-sectional relationship between a range of cognitive and metacognitive belief domains—including positive and negative metamemory beliefs—and PTSD reactions to traumatic events. Second, we examined whether pre-existing metacognitive beliefs (Time 1 [T1]) increased PTSD symptomatology after trauma (Time 2 [T2]). Third, we investigated whether metacognition predicted the maintenance of elevated PTSD symptom levels over time.

Method

Participants

Participants ($N = 664$) were residents of Australia ($n = 74$), Canada ($n = 17$), New Zealand ($n = 4$), the United Kingdom ($n = 51$), and the United States ($n = 518$), at least 18 years old, and fluent in English. We recruited participants from Amazon Mechanical Turk ($n = 372$; they received \$0.75 at T1 and \$0.75 at T2, in keeping with rates of compensation by time for other psychology studies), the Flinders University Psychology Research Participation Pool ($n = 33$, course credit), and standard research recruitment and social networking sites ($n = 259$, voluntary participation). Of the 683 who completed the T1 survey, 315 also participated at T2. We embedded manipulation checks to ensure participants paid attention and excluded participants who failed more than one (T1: $n = 13$, T2: $n = 10$). From T1, we also excluded 5 participants who did not meet the eligibility criteria and 1 who completed the survey twice. From T2, we excluded 1 participant who had failed attention checks at T1, 1 who could not be matched to his or her T1 data, and 3 who completed T2 18 weeks or more after T1. The final sample comprised 664 T1 participants and 300 T2 participants. Table 1 shows their demographics and descriptive information for the main measures. Notably, at T1, participants who did not complete T2 were older ($p < .01$), reported more PTSD symptoms ($p < .01$), and had higher problematic cognition and metacognition on all but positive metamemory beliefs ($ps < .05$), compared with completers.

Table 1. Demographic, Trauma-Related Characteristics, and Main Measures for the Total Sample Including Cross-Sectional Pearson's Correlation Coefficients and Confidence Intervals With PTSD Symptoms

Characteristic	Time 1 (<i>n</i> = 664)		Time 2 (<i>n</i> = 300)		Time 1 (<i>n</i> = 664)		Time 2 (<i>n</i> = 300)	
	<i>M</i> (<i>SD</i>), <i>R</i>	<i>M</i> (<i>SD</i>), <i>R</i>	<i>M</i> (<i>SD</i>), <i>R</i>	<i>M</i> (<i>SD</i>), <i>R</i>	<i>r</i> [CI] With PCL	<i>r</i> [CI] With PCL	<i>r</i> [CI] With PCL	<i>r</i> [CI] With PCL
Demographics								
Age in years	32.38 (12.34), 18–75	35.32 (12.96), 18–75						
Female sex, % (<i>n</i>)	68.5 (455)	67.7 (203)						
Ethnicity								
Caucasian, % (<i>n</i>)	83.4 (554)	85.0 (255)						
Minority, % (<i>n</i>) ^a	16.6 (110)	14.7 (42)						
Level of education achieved								
School, % (<i>n</i>)	31 (206)	28 (84)						
College/university, % (<i>n</i>)	65.5 (435)	68.7 (206)						
TAFE/trade, % (<i>n</i>)	3.5 (23)	3.3 (10)						
Employment								
Employed, % (<i>n</i>)	63.2 (420)	70.7 (212)						
Unemployed, % (<i>n</i>)	16.7 (111)	15.6 (47)						
Student, % (<i>n</i>)	20 (133)	13.7 (41)						
Trauma characteristics and symptoms								
Age at trauma								
Age at trauma	20.80 (11.56), 0–70	35.32 (12.96), 18–75			-.19 [-.27, -.11]	***	-.18 [-.29, .07]	**
Time since trauma ^b	12.12 (11.44), 0–57	26.04 (22.02), 0–105			-.08 [-.16, -.01]	***	.05 [-.15, .26]	***
Number of traumas	4.06 (2.72), 0–13	4.36 (2.99), 0–15			.38 [.31, .44]	***	.41 [.32, .51]	***
Distress at trauma	4.59 (.79), 1–5	3.93 (1.38), 1–5			.21 [.14, .28]	***	.43 [.26, .58]	***
HADS total	16.67 (7.06), 0–42	12.80 (8.29), 0–42			.68 [.64, .72]	***	.74 [.70, .77]	***
HADS anxiety	10.40 (4.25), 0–21	7.61 (4.62), 0–21			.64 [.59, .70]	***	.70 [.66, .74]	***
HADS depression	6.27 (3.63), 0–21	5.18 (4.52), 0–21			.56 [.49, .62]	***	.64 [.59, .68]	***
PCL total	37.09 (17.38), 17–85	32.52 (14.97), 17–84			—		—	
Metacognition measures								
BAMQ positive	14.38 (6.27), 8–32	15.34 (6.75), 9–36			.44 [.38, .50]	***	.56 [.48, .63]	***
BAMQ negative	10.01 (3.98), 7–28	10.72 (3.98), 8–32			.58 [.52, .62]	***	.60 [.52, .67]	***
MCQ-30 positive worry	10.84 (4.35), 6–24	10.60 (4.10), 6–24			.24 [.17, .31]	***	.30 [.19, .40]	***
MCQ-30 uncontrollability	13.2 (5.17), 6–24	12.29 (4.87), 6–24			.54 [.48, .59]	***	.53 [.44, .61]	***
MCQ-30 thought control	11.84 (4.03), 6–24	11.04 (4.14), 6–24			.43 [.37, .49]	***	.39 [.29, .48]	***
PTCI self	2.65 (1.42), 1–7	2.46 (1.37), 1–7			.71 [.67, .74]	***	.76 [.71, .80]	***
PTCI world	4.23 (1.65), 1–7	3.92 (1.69), 1–7			.57 [.51, .61]	***	.56 [.48, .63]	***
PTCI self-blame	2.53 (1.58), 1–7	2.32 (1.56), 1–7			.46 [.40, .52]	***	.43 [.33, .52]	***
RIQ intrusions	14.81 (9.36), 6–42	13.86 (8.79), 6–42			.70 [.64, .74]	***	.76 [.69, .81]	***

Note: In the first two columns, values are *M* (*SD*), *R*, unless otherwise noted. BAMQ = Beliefs About Memory Questionnaire; CI = confidence interval; HADS = Hospital Anxiety and Depression Scale; MCQ-30 = Metacognitions Questionnaire-30; PCL = PTSD Checklist-Specific Version; PTCI = Posttraumatic Cognitions Inventory; PTSD = posttraumatic stress disorder; RIQ = Response to Intrusions Questionnaire; TAFE = Technical and Further Education.

^aMinority, e.g., African American, Hispanic, Middle Eastern, Asian American, multiracial. There were no significant differences in PTSD symptoms between White and non-White participants at either time point, and we therefore excluded ethnicity from further analysis. ^bTime since trauma = years at T1, days at T2. ***p* < .01. ****p* < .001.

This research was approved by the Social and Behavioural Research Ethics Committee at Flinders University and conducted in accordance with the provisions of the World Medical Association Declaration of Helsinki.

Measures

We administered all measures at T1 and T2, 12 weeks apart.

Personal information. Participants completed demographic questions (age, ethnicity, country of residence, highest education level achieved) and provided their email address.

Depression and anxiety. The 14-item Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) assessed anxiety and depression symptoms. Participants rated each item from 0 to 3 according to how they felt during the previous week (anchors vary by item). The Depression subscale focuses mainly on the reduced pleasure response aspect of depression (“I feel cheerful”; 0 = *most of the time*, 3 = *not at all*), whereas the Anxiety subscale focuses on generalized anxiety and panic (“worrying thoughts go through my mind”; 0 = *only occasionally*, 3 = *a great deal of the time*). For our sample, internal consistency was as follows: Anxiety T1 = .81, T2 = .88; Depression T1 = .73, T2 = .87.

Traumatic events. The Trauma History Screen (Carlson et al., 2011) assessed lifetime exposure to potentially traumatic events at T1; at T2, participants indicated whether they had experienced any of the events since T1. Participants also specified their age (or days since the event for T2) and how emotionally distressed they were at the time of the event (*not at all*, *a little*, *somewhat*, *much*, *very much*). Next, participants nominated their worst event and described that event briefly. The psychometric properties of this scale are comparable or better than longer measures of trauma exposure (Carlson et al., 2011).

Posttraumatic stress symptoms. The PTSD Checklist-Specific Version (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993) assessed posttraumatic stress. Participants responded to 17 items regarding their self-nominated worst event at T1 and T2 and their symptoms within the past 2 weeks, using a 5-point scale (1 = *not at all*, 5 = *extremely*). Internal consistency in the current sample was .95 at T1 and T2. To investigate symptom maintenance over the T1 to T2 delay, we classified participants’ PTSD symptoms as “persistent” if they displayed elevated PCL scores (>44) at T1 and T2 and “recovered” if they displayed elevated scores at T1 but not at T2 (i.e., we

followed previous research: Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Bonanno, 2005; Robinaugh et al., 2011).

Trauma-related cognitions. The Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999) measured negative cognitions about the self (“I can’t rely on myself”) and the world (“you can never know what or who may harm you”) and self-blame (“the event happened because of the way I acted”). Participants rated their agreement with each statement (1 = *totally disagree*, 7 = *totally agree*). Here, the PTCI demonstrated adequate reliability for each subscale (Self: .96 and .97; World: .92 and .91; Self-Blame: .88 and .94).

Metacognitive beliefs. We selected three subscales of the Metacognitions Questionnaire-30 (MCQ-30; Wells & Cartwright-Hatton, 2004) due to their established association with PTSD (Roussis & Wells, 2006): positive beliefs about worry (“worrying helps me cope”), beliefs about the uncontrollability and danger of thoughts (“my worrying is dangerous for me”), and beliefs about the need to control thoughts (“it is bad to think certain thoughts”). Participants rated their agreement with each statement on a 4-point scale (1 = *do not agree*, 4 = *agree very much*). The MCQ-30 is well validated in adults (Wells & Cartwright-Hatton, 2004). Cronbach’s alpha ranged from .77 to .92. The 10-item Response to Intrusions Questionnaire (RIQ; Clohessy & Ehlers, 1999) measured negative inferences about the meaning of intrusive memories.¹ Participants who had experienced intrusions rated what they thought their intrusions meant (“my life is ruined”) on a 7-point scale (1 = *totally disagree*, 7 = *totally agree*). The scale has previously shown good reliability and predictive validity (e.g., Clohessy & Ehlers, 1999; Halligan et al., 2003). Here, internal consistency was .92 at T1 and .91 at T2.

Metamemory. The Beliefs About Memory Questionnaire (BAMQ; Bennett & Wells, 2010) assessed metacognition about traumatic memory. The 15 items measured positive beliefs about needing a complete trauma memory (“I must try to remember all of the details of the event so that I can understand why it happened”) and negative beliefs about the consequences of not having a complete memory (“gaps in my memory for the event are preventing me from getting over it”). Participants rated their agreement with each belief on a 4-point scale (1 = *do not agree*, 4 = *agree very much*). Bennett and Wells (2010) found preliminary support for the convergent validity of the BAMQ subscales with subscales of the MCQ-30. Here, the internal consistency was as follows: Positive Beliefs: T1 = .91, T2 = .92; Negative Beliefs: T1 and T2 = .85.

Because responses on the PTCI, BAMQ, and RIQ are anchored to a specific traumatic event, we asked participants to respond in reference to their worst event or, if they had not described one, a negative event they had experienced.

Procedure

At T1, participants completed the questionnaire battery online. Approximately 12 weeks later, we emailed participants who consented to be recontacted with the link to the T2 survey (completion time = 77–91 days after T1). Participants received full debriefing information at the study conclusion.

Results

All analyses were two-tailed and alpha was set at .05. Several subscale scores at T1 and T2 were positively skewed. Although square root and log transformations reduced the skew, the overall pattern of results was the same; thus, we retained the original untransformed data for analysis. We replaced missing items on questionnaires using mean substitution (by subscale).

Consistent with prior studies, most ($n = 633$, 95.3%) participants reported experiencing at least one lifetime trauma (Breslau et al., 1998; Frazier et al., 2009).² The most frequent self-nominated worst events were the unexpected death of a loved one ($n = 173$), “other” trauma ($n = 105$, e.g., childhood emotional abuse, stillbirths, kidnappings), child sexual assault ($n = 67$), sudden abandonment ($n = 48$), and transport accident ($n = 46$). At T2, 35% ($n = 107$) reported experiencing at least one “new” trauma; all but one also reported a lifetime trauma at T1. Of these recent events, “other” events were most commonly nominated as the worst ($n = 48$, e.g., elementary school shootings, Boston bombings, Hurricane Sandy). Of participants displaying elevated (>44) PTSD symptoms at T1 ($n = 214$, of whom 78 also completed T2), 56.4% ($n = 44$) showed persistently elevated PTSD symptoms at T2, and 43.6% ($n = 34$) were classified as recovered.

Table 1 shows the cross-sectional relationships among the demographic, cognitive, metacognitive, and symptom variables at T1 and T2. Females reported significantly more PTSD symptoms than males at T1, $t(662) = -2.84$, $p < .01$, $d = .24$. Age at trauma was negatively related to PTSD. Number of prior traumas, emotional distress at the time of the trauma, anxiety, depression, and all types of cognition and metacognition were positively associated with PTSD symptoms at both time points. Participants’ negative cognitions about the self were highly correlated with other variables; in particular with PTSD ($r = .70$) and RIQ ($r = .83$). Thus, we excluded

this variable from our regression analyses due to multicollinearity.³

We next conducted a (forced entry) hierarchical regression to examine whether certain demographic, cognitive, and metacognitive variables predicted the degree of concurrent PTSD symptomatology at T1.⁴ In Step 1, we entered five control variables—age at trauma, distress at the time of recent worst trauma exposure, gender, number of prior traumas, and depression; these variables accounted for a significant proportion of the variance in symptoms (43%). All were independent predictors except gender and distress; depression, which is often comorbid with PTSD (APA, 2013), was the strongest predictor. In Step 2, preexisting cognitive beliefs accounted for a significant additional proportion of symptom variance (10%); depression, age at trauma, and number of prior traumas remained significant predictors, and world beliefs and self-blame were also independent predictors. In Step 3, metacognitive beliefs also accounted for a significant proportion of symptom variance over and above the control and cognitive variables (11%). Depression, prior traumas, and world beliefs from Step 2 remained significant predictors, along with beliefs concerning the uncontrollability and danger of thoughts, positive and negative metamemory beliefs, and negative inferences about intrusive memories. The overall model explained 64% total variance in PTSD, $F(13, 348) = 47.96$, $p < .001$.

We next examined whether preexisting metacognitive beliefs predicted degree of PTSD symptomatology following recent trauma exposure (i.e., exposure between T1 and T2); we conducted a (forced entry) hierarchical regression. Here, we included only participants who had experienced a novel trauma ($n = 107$) between T1 and T2, with T2 PCL as the outcome variable. Of these, 25 participants had no score for the RIQ, because it was administered only to participants who reported intrusions. To maximize the available sample size, we used the Expectation-Maximization technique in SPSS to replace missing values for participants who did not report age at the time of the trauma ($n = 3$), distress relating to the trauma ($n = 7$), or either of these variables ($n = 2$). We entered six control variables—age, distress at the time of recent trauma exposure, depression, gender, number of prior traumas, PTSD symptoms at T1—simultaneously in Step 1. We added the cognitive variables (T1) in Step 2 and the metacognitive variables (T1) in Step 3.

Table 2 displays the regression statistics for each step of the model. As shown in Step 1, the control variables accounted for a significant proportion of variance in T2 PTSD symptoms (58%). In Step 2, preexisting cognitive beliefs did not explain significant additional variance (1%). However, as predicted, in Step 3 preexisting metacognitive beliefs did account for a significant proportion

Table 2. Summary of Hierarchical Regression of Control Variables, Cognitive Beliefs, and Metacognitive Beliefs at Time 1 in Predicting Posttraumatic Stress Symptoms at Time 2

Predictor	<i>B</i>	<i>SE</i>	β		Squared semipartial correlation	VIF
Step 1						
Constant	-1.99	7.82				
Age at time of trauma	-0.04	0.10	-.03		.001	1.04
Number of traumas	0.38	0.46	.06		.004	1.19
Distress at trauma	3.69	1.59	.19	*	.030	1.25
Gender	-0.82	3.13	-.02		<.001	1.29
Time 1 PTSD	0.43	0.10	.45	***	.111	1.82
Time 1 depression	1.44	0.47	.30	**	.052	1.75
$R^2 = .58, F(6, 75) = 17.53, p < .001$						
Step 2						
Constant	3.52	8.70				
Age at time of trauma	-0.04	0.10	-.03		.001	1.05
Number of traumas	0.54	0.47	.10		.007	1.25
Distress at trauma	3.07	1.65	.16		.019	1.35
Gender	-0.23	3.14	-.01		<.001	1.31
Time 1 PTSD	0.49	0.11	.52	***	.112	2.39
Time 1 depression	1.68	0.49	.35	**	.064	1.94
PTCI world	-1.89	1.23	-.16		.013	1.97
PTCI self-blame	-0.09	0.97	-.01		<.001	1.68
$R^2_{\text{Change}} = .01, F_{\text{Change}}(2, 73) = 1.26, p = .289$						
Step 3						
Constant	3.51	8.91				
Age at time of trauma	-0.00	0.10	-.00		<.001	1.27
Number of traumas	1.03	0.44	.18	*	.025	1.34
Distress at trauma	2.02	1.62	.11		.007	1.56
Gender	0.40	3.15	.01		<.001	1.59
Time 1 PTSD	0.25	0.12	.26	*	.020	3.37
Time 1 depression	1.46	0.49	.31	**	.041	2.30
PTCI world	-2.89	1.17	-.25	*	.028	2.14
PTCI self-blame	-0.40	0.94	-.04		.001	1.89
RIQ intrusions	0.44	0.17	.28	*	.032	2.36
BAMQ positive metamemory	0.20	0.22	.08		.004	1.68
BAMQ negative metamemory	0.14	0.38	.04		.001	2.39
MCQ-30 positive worry	0.15	0.30	.04		.001	1.36
MCQ-30 uncontrollability/danger	1.04	0.36	.31	**	.038	2.52
MCQ-30 control thoughts	-0.84	0.41	-.22	*	.020	2.48
$R^2_{\text{Change}} = .10, F_{\text{Change}}(6, 67) = 3.43, p = .005$						

Note: BAMQ = Beliefs About Memory Questionnaire; MCQ-30 = Metacognitions Questionnaire-30; PTCI = Posttraumatic Cognitions Inventory; PTSD = posttraumatic stress disorder; RIQ = Response to Intrusions Questionnaire; VIF = variance inflation factor.

* $p < .05$. ** $p < .01$. *** $p < .001$.

of additional variance (10%). In particular, preexisting beliefs concerning the uncontrollability/danger of thoughts and negative inferences about intrusive memories independently predicted PTSD symptoms after recent trauma. These variables are important contributors to the final model, uniquely accounting for 3.84% and 3.24% of the variance, respectively. Other critical

variables are depression (4.08%) and prior traumas (2.46%). However, contrary to our hypothesis, people who, at T1, were less likely to believe the world is dangerous and that they needed to control thoughts had more symptoms of PTSD at T2 (2.82%, 1.99%). The overall model explained 69% total variance in PTSD, $F(14, 67) = 10.77, p < .001$.

We also predicted that maladaptive meta/cognitions would drive the *persistence* of PTSD and hence differentiate people who experienced elevated PTSD symptoms between T1 and T2 and people whose symptoms abated. Using only those participants whose symptoms were classified as “persistent” or “recovered” at T2 ($n = 78$, less 12 participants with no RIQ score), we entered the cognitive and metacognitive variables into a (forced entry) hierarchical multivariate logistic regression analysis.⁵ This model accurately predicted 83.3% of cases and contained only negative metamemory beliefs (OR = 1.35 [1.01, 1.81], $p < .05$) and negative inferences about the meaning of intrusions (OR = 1.17 [1.03, 1.33], $p = .01$) as significant predictors of persistent PTSD symptoms ($\chi^2 = 38.61$, $p < .01$).

Discussion

We examined the relationship between specific types of metacognition and the development and maintenance of posttraumatic stress symptoms. Considering the correlational data, at T1 and T2, the more our participants held unhelpful beliefs, the more PTSD symptomatology they showed. It is interesting that cognition about the self was the most important correlate of PTSD symptomatology, corroborating previous research (vs. world/self-blame; Bryant & Guthrie, 2005; Foa & Rauch, 2004; Moser, Hajcak, Simons, & Foa, 2007). This result is not surprising, given substantial conceptual overlap between negative self-related cognition and symptoms, as well as with our other key variables. Of the metacognitive beliefs, at T1 negative metamemory beliefs were the most important, whereas at T2 it was beliefs about intrusions. However, these particular results do not speak to whether pretrauma metacognition predicts later PTSD symptomatology.

Thus, we next examined whether cognitive and metacognitive factors predicted PTSD symptomatology after trauma exposure. We found that preexisting cognitive and metacognitive beliefs (T1) predicted PTSD symptom levels after exposure to a novel trauma (T2). Specifically, people reported more PTSD symptoms when exposed to trauma if they had showed concern that their thoughts were dangerous and made negative inferences about the meaning of their intrusive symptoms. In addition, people who, pretrauma, believed the world to be more safe and predictable, and did not believe that they should control their thoughts, were more likely to report more symptoms posttrauma. Perhaps for people with overly optimistic views of the safety and fairness of the world, trauma shatters their basic beliefs and leads them to shift those beliefs to the other extreme (Foa & Riggs, 1993; Janoff-Bulman, 1992). Our data, like those of Bryant and Guthrie (2005), suggest beliefs that the world is dangerous and that it is bad to think certain thoughts

are activated following—rather than prior to—trauma exposure (cf. Foa & Riggs, 1993). Finally, we found the extent to which people held metamemory beliefs that fragmentary trauma memory was pathological and made negative inferences about the meaning of intrusions played a significant role in *maintaining* elevated PTSD symptoms over 12 weeks. These results support the hypothesis that metacognition plays an important role in PTSD maintenance among adults.

Taken together, our results support the metacognitive model of PTSD (Wells, 2000): Problematic metacognitions likely blocked participants’ adaptation process, increasing their focus on threat such that anxiety and a sense of ongoing danger persisted, which in turn maintained symptoms (e.g., Roussis & Wells, 2006). Our data also contribute to a growing literature showing the importance of metamemory beliefs (about intrusive memories and memory deficits) to PTSD symptoms. In particular, negative beliefs that a fragmented memory means something bad, or is abnormal, predicted PTSD maintenance.

Whether people with PTSD have fragmentary and disorganized memories for their traumatic experience has long been debated (e.g., Ehlers, Ehling, & Kleim, 2012). Our data suggest that negative metacognition about traumatic memory might contribute to trauma-exposed people’s struggle to resolve their symptoms. It is interesting that people’s metacognition becomes more apparent the more they report recurrently thinking about an event. The more we think about an event, the more we might think details are missing from memory. Recovering previously inaccessible details creates an impression of partial amnesia (e.g., Berntsen & Rubin, 2014). Yet, nonclinical research suggests this is not unique to traumatic memory (Read & Lindsay, 2000). Thus, it is unclear whether disorganization in traumatic memories contributes significantly to PTSD symptoms, or if metamemory beliefs and strategies concerning trauma memory (e.g., rumination, “gap filling”) play a greater role in maintaining symptoms. We do know, however, that rumination mediates the relationship between metamemory beliefs and intrusive PTSD symptoms (Bennett & Wells, 2010).

There are several limitations to our study. First, although we gathered data prospectively, the study lasted only 12 weeks. In addition, because not all participants met the one-month duration criterion for PTSD with symptoms at T2, it is possible that, among this group, symptomatology represented normal stress reactions to trauma. It is important that we did not include a clinical diagnostic interview to assess PTSD or any other psychopathology at T1 that could potentially account for our results. Also, we used a convenience sample of students and a broad section of Internet users, who were predominantly Caucasians from the United States, which

may limit the generalizability of our findings. Moreover, because the participants reporting the highest levels of problematic metacognition and PTSD symptoms at T1 were less likely to complete the entire study, our final T2 sample was not representative of those at the more extreme end. Also, although we initially recruited a large sample, due to incomplete data on some measures, the final sample size we used for our main regression analyses was limited. Finally, in interpreting the separate contribution of *metacognitions* to the development and maintenance of posttraumatic stress symptoms, it is important to acknowledge that the RIQ could potentially also be indexing cognitions, rather than just pure metacognitions.

Considering our key findings, and the limitations of our study, we would suggest two future research areas. To advance our understanding of how metacognitive beliefs may lead to PTSD after new trauma exposure, we suggest conducting a similar longitudinal study with people at high risk of trauma exposure, such as emergency service personnel (e.g., Bryant & Guthrie, 2005). Research could also examine whether cognitive bias training (Woud et al., 2012) directed specifically at changing problematic metacognitive beliefs halts the development of symptoms after new trauma or reduces symptoms relating to previous trauma. This method would help determine whether metacognitive beliefs are *causal* risk factors for PTSD (Kraemer et al., 1997). In both lines of research, we suspect it will be important to employ a longer delay when assessing the role of metacognition in maintaining PTSD symptoms over time and to attempt to capture metacognition and symptoms immediately after trauma exposure.

In summary, our results indicate that metacognition—particularly regarding traumatic memory, a new area of investigation—plays an active role in adult PTSD. Specifically, this evidence adds to growing empirical support that metacognitive beliefs are important to predicting PTSD symptomatology.

Author Contributions

M. K. T. Takarangi and D. Strange developed the study concept. All authors contributed to the study design. R. A. Smith performed data collection; R. A. Smith performed initial analyses and interpretation; M. K. T. Takarangi and H. D. Flowe performed additional analysis. R. A. Smith and M. K. T. Takarangi drafted the manuscript, and D. Strange and H. D. Flowe provided critical revisions. All authors approved the final version of the manuscript for submission.

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Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information may be found at <http://cpx.sagepub.com/content/by/supplemental-data>.

Notes

1. An initial coding error on the survey meant we were unable to use T1 RIQ data for 92 participants.
2. Table S1 in the Supplemental Material available online shows the lifetime prevalence of each potentially traumatic event and participants' exposure to these events between T1 and T2.
3. We tested our hypotheses using ordinary least squares (OLS) regression and logistic regression analyses. To address the concern that the predictors in our models could be highly correlated, we verified each analysis using ridge regression (Hoerl & Kennard, 1970; Millsap & Maydeu-Olivares, 2009). Ridge regression improves a model's predictive accuracy in cases where there are more predictors than observations and/or when the predictors are highly correlated. The ridge regression results did not differ from those obtained with OLS and logistic regression; hence, we report only the latter ones for ease of interpretation.
4. Table S2 in the Supplemental Material available online displays the full regression table.
5. With the available sample size reduced, here we included only the theoretical predictors of interest (i.e., meta/cognitive factors). Table S3 in the Supplemental Material available online displays the full regression table.

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