

The Effect of Self-Reported REM Behaviour Disorder Symptomology on Intrusive Memories  
in Post-Traumatic Stress Disorder

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## Abstract

PTSD is characterised by severe sleep disturbances, which is increasingly recognised to in many cases consist of similar symptomology to sleep disorders such as REM Behaviour Disorder (RBD). The present study aimed to investigate whether different aspects of sleep quality influence intrusive memory development and whether PTSD status moderates this relationship. 34 PTSD, 52 trauma-exposed (TE) and 42 non-trauma exposed (NTE) participants completed an emotional memory task, where they viewed 60 images (20 positive, 20 negative and 20 neutral) and, two days later, reported how many intrusive memories they had of each valence category. Participants also completed three measures of sleep quality: the Pittsburgh Sleep Quality Index, the REM Behaviour Disorder Screening Questionnaire and total hours slept before each session. The PTSD group reported poorer sleep quality than both TE and NTE groups on all three measures, and significantly more negative intrusive memories than the NTE group. Mediation analyses revealed that self-reported RBD symptomology before the second session mediated the relationship between PTSD status and intrusive memories. Follow-up moderation analyses revealed that self-reported RBD symptomology before the second session was only a significant predictor of intrusion in the PTSD group, though with a small effect size. These findings suggest that RBD symptomology is an indicator of consolidation of intrusive memories in PTSD but not trauma-exposed or healthy participants, which supports the relevance of characterising RBD in PTSD.

**Keywords:** Posttraumatic Stress Disorder, Intrusive Memories, Rapid-Eye Movement, REM Behavior Disorder, PTSD, Sleep Disturbance

Posttraumatic Stress Disorder (PTSD) patients suffer significant sleep disturbances that affect multiple aspects of sleep architecture, including Rapid Eye Movement (REM) (Germain, 2013). Subjective reports of sleep disturbances suggest that 70%-91% of PTSD patients suffer poor sleep (Maher, Rego, & Asnis, 2006). Self-reports are corroborated by objective assessments (Germain, 2013; Kobayashi, Huntley, Lavela, & Mellman, 2012). For instance, a meta-analysis of polysomnographically measured sleep in PTSD found that PTSD was associated with less non-REM, higher REM density and overall lighter sleep than controls (Kobayashi, et al., 2007). Recognition that PTSD can be associated with the onset of distinct sleep disorders is growing, with recent reports advocating for a unique ‘Trauma-Associated Sleep Disorder’, which is characterised by symptoms of REM Behaviour Disorder (RBD) such as nightmares and restless movement behaviours during REM (Feemster, Smith, McCarter, & Louis, 2019; Mysliwiec, O’Reilly, Polchinski, Kwon, Germain, & Roth, 2014; Rachakonda, Balba, & Lim, 2018). Similarly, improvement to sleep quality in PTSD patients suffering from obstructive sleep apnoea through use of continuous positive airway pressure improves sleep quality and waking symptoms (Zhang, et al. 2017, 2019).

Disruption to sleep quality, particularly REM, has been proposed to be an important mechanism in the development of intrusive memories, which are a core feature of PTSD (APA, 2013; Germain, 2013; Spoormaker & Montgomery, 2008; Stickgold, 2002). REM sleep is believed to be critical to the affective de-coupling and healthy consolidation of memories following an emotional or traumatic experience (Goldstein & Walker, 2014; van der Helm & Walker, 2011). Neuroimaging studies have shown decreased amygdala activity to emotional cues follow REM sleep (van der Helm, et al. 2011), and restless REM sleep is associated with higher amygdala reactivity (Wassing, et al. 2019). Studies have also shown that the hippocampus, as well as amygdala and vmPFC, are abnormally activated during REM sleep in PTSD (Ebdiahad, Nofzinger, James, Buysse, Price, & Germain, 2013;

Germain, et al., 2013), suggesting that brain regions critical to the consolidation and contextualization of trauma memories are impacted during sleep in PTSD (Germain, et al., 2013; Nardo, et al., 2015). There is additional evidence that PTSD patients have reduced inter-region communication between the amygdala, vmPFC and hippocampus during REM sleep due to disturbed theta-range frequencies (Cohen, et al., 2013; Cowdin, Kobayashi, & Mellman, 2014). Non-REM sleep and some sleep spindle indices are also associated with intrusive memory development (Kleim, Wysokowsky, Schmid, Seifritz, & Rasch, 2016), with both of these aspects of sleep related to memory integration more generally (Tamminen, Lambon Ralph, & Lewis, 2013; Tamminen, Payne, Stickgold, Wamsley, & Gaskell, 2010; Ulrich, 2016).

There have only been four empirical studies examining the relationship between trauma, sleep and intrusive memories. Sopp and colleagues (2019) found that higher REM theta frequency was associated with less intrusive memories of a traumatic film, as well as higher intrusive memory symptoms. Kleim and colleagues (2016) found that early sleep onset was associated with fewer intrusive memories, whereas higher REM density (an irregular pattern of REM sleep typically observed in PTSD, see Kobayashi, et al., 2007) predicted more intrusive memories. Conversely, Porcheret and colleagues (2015) measured the intrusive memories of a sleep deprived group and a sleep as usual group for six days after watching a trauma film. This team found that participants who slept experienced more intrusive memories than those that did not over the first two days following the film, suggesting a protective effect of sleep deprivation on intrusive memory development (Porcheret, et al., 2015). This same group failed to replicate this finding in a more recent study, however, where sleep deprivation resulted in marginally higher intrusions two days after traumatic film viewing, but not significantly different over the total course of six days (Porcheret et al., 2019).

The present study examined the effect of sleep quality on the frequency of intrusive memories of emotional images in PTSD, trauma-exposed and non-trauma exposed participants. Based on previous research showing that between-group differences in intrusion frequency is most pronounced two days following viewing of stimuli (Porcheret, et al. 2015, 2019), we used a two-day paradigm with a memory recall session two days after watching emotionally valent images. The RBD Screening Questionnaire (RBDSQ; Stiasny-Kolster, et al., 2007) was used to measure RBD symptoms between participants and was chosen as a reliable measure of RBD due to the increasing recognition of this specific symptomology in PTSD patients (Feemster, et al. 2019; Husain, Miller, & Carwile, 2001; Mysliwiec, et al. 2014, 2018a, 2018b; Rachakonda, et al. 2018). We hypothesised that PTSD-diagnosed participants would report poorer sleep quality, would experience more negative intrusive memories than control groups, and that intrusive memories of emotional stimuli would be mediated by total sleep duration, subjective sleep quality and RBD symptomology.

## **Method**

### **Participants**

One-hundred and twenty-eight participants were recruited from the University of Tasmania and from private psychology clinics in Hobart. Four participants were excluded due to use of antipsychotics or benzodiazepines. Forty-two participants (26 female) were classified as non-trauma exposed controls (NTE), as determined by the Traumatic Experiences Questionnaire (TEQ: Vrana & Lauterbatch, 1994). Fifty-two participants (29 female) were classified as trauma exposed controls (TE) as they met criteria for exposure to a criterion A trauma according to DSM-IV-TR as measured by the TEQ, but did not meet the diagnostic criteria for PTSD or display symptoms at subsyndromal levels as determined by

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<sup>1</sup>When data collection began the DSM-IV-TR was the current manual. Use was maintained for consistency

the PTSD Checklist – Civilian Version (PCL-C: Weathers, Litz, Herman, Huska, & Keane, 1993). Thirty participants were categorized as being PTSD-positive (15 females) as recommended by a PCL-C cut-off score of 50, suggestive of a diagnosis of PTSD, or displayed symptoms at a level considered subsyndromal PTSD (PCL total >40; Weathers, et al. 2013).

Exclusion criteria included current psychiatric diagnosis or psychoactive medication use in controls, aged outside 18-65, neurological disorders or significant medical illness, history of head injury resulting in loss of consciousness for over five minutes or hospitalization, an AUDIT (The Alcohol Use Disorders Identification Test: WHO, 2001) score of over 19 or a disclosed substance dependence. These exclusions controlled for memory confounds. A small number of participants were using anti-depressant or pain relief medications (n=14). Due to the high rate of medication use in the broader PTSD community, we chose not to remove these participants to avoid biasing the sample, though reran the analyses with these participants excluded as a sensitivity analysis. The study was reviewed and approved by the local Tasmanian ethics review board.

## Measures

**Pittsburgh Sleep Quality Index (PSQI: Buysse, et al., 1989).** The PSQI subjectively evaluates quality of sleep over the past month. PSQI scores are reported on a 19-item inventory, with items pertaining to sleep quality on four-point scales from “Not during the past month” to “Three or more times per week”. The PSQI has adequate internal consistency, with a Cronbach’s alpha of .83 (Buysse, et al., 1989) and was used to measure retrospective sleep quality in the present study.

**Sleep Diary.** Sleep quality was further indexed using an acute sleep diary, where total hours of sleep before each testing session was subjectively reported. Participants were

asked to estimate the total hours of last night's sleep using this questionnaire, with number of hours as an estimated numerical value.

**REM Behaviour Disorder Screening Questionnaire.** An adapted measure of the REM Behaviour Disorder Screening Questionnaire (RBDSQ: Stiasny-Kolster, et al., 2007) was used to evaluate RBD symptomology on the nights preceding each testing session. The RBGSQ is a validated self-reported measure that diagnoses aspects of RBD, which include nightmares and nocturnal movements characteristic of REM disturbance. The RBGSQ has a high internal consistency of Cronbach's alpha of .885 (Stiasny-Kolster, et al., 2007). We adapted it to the current study by including a Likert rating scale (scores ranging from 1-5) for each question, which was different from the original design that consisted only of dichotomous options for each of the thirteen questions. Recent meta-analysis of 10 RBGSQ evaluative articles and almost 2,000 participants found that this measure has exemplary diagnostic sensitivity and specificity in the general population for predicting RBD, which here we generalise to the broader symptomology in trauma-associated sleep disturbances (Husain, et al. 2001; Li, Li, Su, & Chen, 2017; Mysliwiec, et al. 2014). A Cronbach's alpha of .806 was obtained in the current dataset between PSQI, sleep duration and RBGSQ scores of participants.

**PTSD CheckList – Civilian Version (PCL-C: Weathers, et al. 1993).** The PCL-C provides diagnostic information and an ordinal measure of PTSD symptom severity, based on the DSM-IV-TR (American Psychiatric Association, 2000). 17 self-report items, which are rated on five-point Likert scales, range from 1 ("Not at all") to 5 ("Extremely") and each index intrusive memory, hyperarousal and avoidance behaviour symptomatology over the past month. The PCL-C has high internal consistency, with a reported Cronbach's alpha of .90 (Foa, et al., 1997) and an alpha of .88 in the current dataset.

**Traumatic Experiences Questionnaire (TEQ: Vrana & Lauterbatch, 1994).** The TEQ indexes the number of events classified as criterion A trauma by the DSM-IV-TR (APA, 2000). 11 dichotomous answer (yes/no) items also assess the type of trauma experienced. The TEQ has high internal consistency, with a reported Cronbach's alpha of .91 (Vrana & Lauterbatch, 1994).

**Medical Questionnaire.** Basic medical background was self-reported using a medical questionnaire. This questionnaire included questions relating to medication, psychiatric condition, head injury, loss of consciousness, substance and tobacco use, neurological conditions and other questions of a medical nature.

**Depression, Anxiety and Stress Scale (DASS-21: Lovibond & Lovibond, 1995).** The DASS-21 is a brief questionnaire that assesses depression, anxiety and stress levels over the past week. The DASS-21 has good internal consistency for its depression (Cronbach's  $\alpha = .91$ ), anxiety ( $\alpha = .84$ ) and stress ( $\alpha = .90$ ) scales (Lovibond & Lovibond, 1995). In the current dataset these scales had a Cronbach alpha level of .855.

**Alcohol Use Disorders Inventory Test (AUDIT: WHO, 2001).** The AUDIT is scored by self-report and is a 10-item index of alcohol use and dependency. The AUDIT has adequate internal consistency, with a reported Cronbach's alpha of .83 (Hays, Merz, & Nicholas, 1995) and an alpha of .80 in the current dataset. The AUDIT was used for screening purposes in the current study.

## **Procedure**

Participants attended two afternoon testing sessions, which were two days apart. Sessions were conducted in the afternoon to control for the circadian effects of cortisol, which are known to impact intrusive memory consolidation (Nicholson, Bryant, & Felmingham, 2014). In the first session, participants provided informed consent before



completing the DASS-21, the PCL-C and the TEQ, at which point they were categorised as NTE, TE or PTSD-diagnosed participants. They were informed that the purpose of the study was to investigate the influence of arousal on viewing emotional images and were not informed that the study examined memory until the second session to prevent priming of intrusions or rehearsal of images. Participants were shown three blocks of International Affective Pictures System (IAPS) images on a laptop computer. According to standardized procedure, 20 emotionally neutral images (mean valence: 4.99, mean arousal: 2.75), 20 positive images (mean valence: 7.49, mean arousal: 4.42), and 20 negative images (mean valence: 2.30, mean arousal: 6.18) were presented in a counterbalanced procedure. These images were chosen on the basis of normative data and stimuli from IAPS (Lang, Bradley, & Cuthbert, 2008). Each image appeared for six seconds on the screen. To complete the first session, participants filled in the medical questionnaire, the AUDIT, the PSQI and sleep diary (including RBDSQ) for the prior night.

Participants returned for a brief follow-up session two days later. In a surprise memory task, they were asked to write down as many of the images they could recall from the first session. They next completed an intrusive memory diary adapted from Holmes, Brewin, & Hennessy 2004, where they reported the frequency and distress of intrusive memories of emotional images during the time after the first session. Reports of intrusive memories were verified by the researcher to be both adhering to the definition of an intrusion, as well as to be specifically of the images rather than other content. Similarly, images that were deliberately recalled were verified to be recollections of the original images by two independent raters after the study. Any ambiguity in written reports given by participants was verbally clarified by the researcher. Participants then completed a second sleep diary (including the RBDSQ) for the night prior to this follow-up session. Participants then rated all previously viewed IAPS images in terms of arousal and valence, according to standard

procedure (means reported above, Lang, et al., 2008). Participants were thanked for their time, debriefed on the complete goals of the study, and reimbursed in either a \$30 payment or university course credits.

## **Analyses**

All analyses were conducted on SPSS 24 for Windows. Gender dispersion between groups was assessed using a chi square test of independence. Demographics and clinical scores, such as age, TEQ and PCL were analysed using univariate analyses of variance (ANOVA). Significant results were followed up with Sidak-adjusted pairwise comparisons. We also used univariate ANOVAs and sidak post-hoc analyses to compare sleep quality scores between groups on all sleep measures. Both deliberate recall and intrusive memory frequency between groups was analysed using a 3 (Group: PTSD, TE, HC) x 3 (Valence: negative, positive, neutral) mixed model repeated measures ANOVA, and followed-up with sidak post-hoc analyses. Statistical significance was set at  $p < .05$ , and effect sizes as well as 95% confidence intervals (CIs) were reported. Greenhouse-Geisser (GG) corrections were applied when sphericity in data was detected.

Using Andrew Hayes's PROCESS macro v3 for SPSS 24 (Hayes, 2018a), separate simple mediation analyses were used to identify mediating influences of sleep quality scores on the relationship between PTSD status (PTSD, trauma-exposed, non-trauma exposed) and number of negative intrusive memories of IAPS images. Mediating measures of sleep quality were hours of sleep before the second session (HRS2), self-reported RBD symptomology before the second session (REM2) and PSQI (total) scores. Relative direct effects and partially standardized indirect effects (5000 bootstrapped confidence intervals) were examined. Using PROCESS, we entered group status as a multi-categorical predictor variable, and the TE group was entered as the comparative group. Exploratory mediation and

moderation analyses were used to explore the effects (see results).  $R^2$  and  $\Delta R^2$  were used as measures of effect size in our moderation analyses. We used mean-centered variables to improve the interpretability of our conditional effects (Hayes, 2013) and heteroscedasticity-consistent inference to protect from lack of homoscedasticity (Hayes & Cai, 2007).

## Results

### Demographics and Clinical Data

Mean scores, standard deviations and test statistics are summarized in Table 1 for demographic and clinical data across NTE, TE and PTSD groups. Gender distribution between groups was equal; however, age, AUDIT, DASS, PCL and TEQ scores were all significantly different between groups. Sidak post-hoc analyses indicated that PTSD-diagnosed participants scored higher than TE participants on measures of stress, anxiety, depression, PTSD symptoms and traumatic experiences (all  $p < .001$ , AUDIT  $p = .018$ ) and than NTE participants on stress, anxiety, depression, PTSD symptoms, AUDIT and trauma exposure (all  $p < .001$ , AUDIT  $p = .005$ ). PTSD-diagnosed participants were significantly older than NTE participants ( $p < .001$ ) and TE participants ( $p = .040$ ). TE participants scored higher on measures of stress ( $p = .021$ ), PCL ( $p = .003$ ) and TEQ scores ( $p < .001$ ) than NTE participants.

**Table 1.** Demographic and Clinical Data for PTSD and Control Groups.

	NTE ( <i>n</i> =42)	TE ( <i>n</i> =52)	PTSD ( <i>n</i> =30)	Total ( <i>N</i> =120)	Test Statistic (df)	<i>p</i>	Effect size
Age	22.81(7.35)	26.42(8.86)	31.40(14.56)	26.40 (10.54)	$F(2,121) = 6.32$	.002	$\eta_p^2 = .11$
Gender	26F, 16M	29F, 23M	15F, 15M	70F, 54M	$\chi^2(2) = .43$	.807	$\phi_c = .06$
DASS^ -depress	2.36 (2.32)	3.17 (3.52)	7.57(4.98)	3.96 (4.36)	$F(2,121) = 17.76$	<.001	$\eta_p^2 = .24$
-anxiety	1.45 (1.95)	2.71 (2.99)	6.60(4.20)	3.23 (3.62)	$F(2,121) = 26.23$	<.001	$\eta_p^2 = .34$
-stress	3.86 (3.55)	6.31(4.20)	11.27(5.30)	6.68 (5.11)	$F(2,121) = 26.37$	<.001	$\eta_p^2 = .33$
PCL^	21.40(4.87)	26.96(6.85)	48.63(12.08)	30.35(13.28)	$F(2,121) = 110.97$	<.001	$\eta_p^2 = .67$
TEQ	.45(.85)	2.83(1.64)	4.42(1.86)	2.48(2.18)	$F(2,121) = 66.52$	<.001	$\eta_p^2 = .54$
AUDIT	5.45(3.85)	6.04(4.28)	9.07(6.33)	6.57(4.91)	$F(2,121) = 5.68$	.004	$\eta_p^2 = .09$
Medication <sup>#</sup>	3	7	4	14			

*Note:* *n* = number of group participants, *N* = number of total participants. ^Means and standard deviations reflect untransformed data. #Medications included either antidepressants, PPIs or pain medications (eg. Lyrica). DASS=Depression, Anxiety and Stress Scales, PCL=PTSD CheckList, TEQ=Traumatic Events Questionnaire

**Sleep Data**

Table 2 contains central tendency data and univariate ANOVA test statistics for PSQI, hours slept before the testing sessions and self-reported RBD symptomology before the testing sessions for each group. There was a significant main effect of group on all sleep measures.

**Table 2.** Summary of Sleep Data on the Pittsburgh Sleep Quality Index, Hours of Sleep and Self-Reported RBD Symptomology

	NTE ( <i>n</i> =42)	TE ( <i>n</i> =52)	PTSD ( <i>n</i> =30)	Total ( <i>N</i> =124)	<i>F</i> ( <i>df</i> )	<i>p</i>	Effect size $\eta_p^2$
<b>PSQI</b>							
-Total	4.48 (2.33)	6.17 (3.24)	9.23 (4.17)	6.34(3.67)	19.11(2,12 1)	<.00 1	.24
<b>Hrs Sleep</b>							
-Session 1	7.51 (1.42)	7.69 (1.15)	6.63 (2.14)	7.37(1.58)	4.82(2,120)	.010	.07
-Session 2	7.65 (1.40)	7.69(1.24)	6.62 (1.99)	7.42(1.56)	5.61(2,121)	.005	.09
<b>REM</b>							
disturbance <sup>^</sup>							
-Session 1	3.64 (4.56)	6.10 (5.81)	12.10 (9.82)	6.72(7.37)	14.46(2,11 9)	<.00 1	.19
-Session 2	3.36 (4.35)	5.10 (5.95)	10.67 (9.72)	5.85(7.16)	11.21(2,12 0)	<.00 1	.16
Nightmares <sup>^</sup>	1.81(3.46)	3.25(5.03)	6.13(6.53)	3.46(5.22)	5.84(2,121)	.004	.09
*							
Behaviour <sup>^*</sup>	.86(1.60)	1.08(1.78)	2.90(3.24)	1.44(2.31)	6.82(2,121)	.002	.10

*Note:* <sup>^</sup> *F* Statistic, Significance Level and Effect Size reflect transformed data. \*only reported for session 2. HRS=hours, REM=RBDSQ score for the night before the session, PSQI=Pittsburgh Sleep Quality Inventory score. Higher PSQI scores indicate poorer sleep quality

Post-hoc analyses revealed that PTSD-diagnosed participants scored poorer sleep quality on the PSQI than NTE ( $p < .001$ ) and TE groups ( $p = .039$ ); than TE ( $p = .009$ ) and NTE ( $p = .050$ ) on hours of sleep before session 1; than NTE ( $p = .014$ ) and TE participants ( $p = .007$ ) on hours of sleep before the second session; than NTE ( $p < .001$ ) and TE participants ( $p < .001$ ) on RBD symptomology before session 1; and, than NTE ( $p < .001$ ) and TE participants on RBD symptomology before the second session ( $p < .001$ ). PTSD-diagnosed participants also had higher nightmare scores than TE ( $p = .039$ ) and NTE participants ( $p = .001$ ), and higher REM behaviour disturbance scores than TE ( $p = .001$ ) and NTE participants ( $p < .001$ ). TE participants had higher PSQI scores than NTE participants ( $p = .037$ ), indicative of poorer sleep, though no other significant comparisons were found. Controlling for depression, AUDIT and removing medicated participants did not substantially change the sleep results, except for nightmares with depression scores accounting for a significant amount of the variance explained by PTSD status (see Supplementary material).

### Deliberate Memory Recall

Figure 1 shows the mean positive, neutral and negative images explicitly recalled by each group. A 3 (Group) x 3 (Valence) repeated measures ANOVA showed a significant main effect of image valence, GG-corrected  $F(2,241)=121.40$ ,  $p<.001$ ,  $\eta_p^2=.50$ , such that negative images were recalled more than positive and neutral images, and positive images were recalled more than neutral images (all  $p<.001$ ). There was no main effect of group,  $F(2,121)=.071$ ,  $p=.931$ ,  $\eta_p^2<.01$ , and no valence x group interaction,  $F(4,241)=1.39$ ,  $p=.239$ ,  $\eta_p^2=.02$ . Controlling for AUDIT and DASS did not alter these findings.

### Intrusive Memory Data

Figure 2 illustrates the mean intrusive memories for NTE, TE and PTSD groups for positive, negative and neutral image valences. A 3 (Group) x 3 (Valence) repeated measures ANOVA showed a significant main effect of image valence, GG-corrected  $F(2,200)=38.72$ ,  $p<.001$ ,  $\eta_p^2=.24$ , indicating that participants had different numbers of intrusive memories of positive, negative and neutral images. Post-hoc analyses showed there were significantly more negative intrusive memories of IAPS images reported than positive,  $p<.001$ , 95% CI[.25,.59] and neutral,  $p<.001$ , 95% CI[.36,.71] intrusive memories of IAPS images. There was also a significant difference between number of positive and neutral intrusive memories,  $p=.045$ , 95% CI[-.23,-.002]. There was also a significant main effect of group,  $F(2,121)=8.74$ ,  $p<.001$ ,  $\eta_p^2=.13$ , suggesting that groups differed in number of reported intrusions.

These main effects were superseded by a significant group x valence interaction, GG-corrected  $F(3,200)=8.70$ ,  $p<.001$ ,  $\eta_p^2=.13$ . Pairwise comparisons revealed that PTSD-diagnosed participants reported more negative intrusive memories than NTE participants, PTSD 95% CI[.71,1.29], NTE 95% CI[-.15,.34]. TE participants reported more negative



intrusions than NTE participants, TE 95% CI[.38,.82], and PTSD reported more intrusions than TE at a trend level (see Figure 2). There were not any differences between groups on reported intrusive memories of positive or neutral images. Again, controlling for DASS or AUDIT scores and removing medicated participants did not substantially affect results in intrusive memory scores (Supplementary Data).

## Mediation Analyses

Three separate mediation analyses were conducted using PROCESS v3 with group status as a multi-categorical predictor, negative intrusive memories as the outcome and hours of sleep before session 2, REM2 and PSQI scores as mediating variables. Whilst PTSD status predicted poorer sleep in each of the measures ( $p < .01$  in each model), only the model including REM2 showed a mediation effect (Table 3). A full mediation effect was observed (total effect  $b = .39$ ,  $SE = .19$ ,  $p = .036$ ) but more importantly, the effect sizes for the REM2 indirect effect were by far the largest in comparison to the indirect effects in the other models (Table 3). Effects were retained when medicated participants were removed and when depression or AUDIT were included as covariates in the analysis (Supplementary Material). Breakdown of the REM2 components showed that REM behavioural disturbances contributed largely to the mediation effect ( $b = .17$ ,  $SE = .10$ , 95% CI [.001, .40]), with the contribution of nightmares not reaching significance,  $b = .09$ ,  $SE = .08$ , 95% CI [-.01, .27].

**Table 3.** Summary of direct and indirect effects of PTSD status on number of reported intrusive memories of negative IAPS images

	Effect Size	SE	<i>p</i>	95% CI	
				Lower	Upper
<b>HRS2</b>					
Direct	.32	.19	.096	-.06	.70
Indirect <sup>^</sup>	.10	.07		-.03	.25
<b>REM1</b>					
Direct	.28	.20	.150	-.10	.67
Indirect <sup>^</sup>	.13	.11		-.06	.39
<b>REM2</b>					
Direct	.21	.19	.264	-.16	.58
Indirect <sup>^</sup>	.22	.12		.03	.48
<b>PSQI</b>					
Direct	.26	.19	.185	-.13	.64
Indirect <sup>^</sup>	.17	.10		-.01	.39

<sup>^</sup>Note: indirect effect is calculated as the partially standardized effect. HRS2=hours of sleep before the second session, REM1=RBDSQ score for the night before the first session, REM2= RBDSQ score for the night before the second session, PSQI=Pittsburgh Sleep Quality Inventory score

### Exploratory Moderation Analysis

Moderation analyses using PROCESS were conducted to test the relationship between REM2 score and intrusive memory frequency, as moderated by PTSD status. This yielded a significant interaction between the three groups and REM2 scores,  $\Delta R^2=.04$ ,  $F(2,118)=3.09$ ,  $p=.049$ . Test of simple slopes at each level of the moderator revealed that only PTSD-diagnosed participants reported more intrusive memories if they had higher REM2 scores ( $p<.001$  for PTSD,  $p>.05$  HC and TEC groups). This effect was not changed when PCL score was added as a covariate,  $\Delta R^2=.04$ ,  $F(2,116)=3.03$ ,  $p=.052$ , or when medicated participants were removed,  $\Delta R^2=.04$ ,  $F(2,104)=3.12$ ,  $p=.048$ .

### Discussion

This study examined the relationship between PTSD status, sleep quality and intrusive memories. As hypothesised, PTSD-diagnosed participants had poorer sleep quality on the PSQI, less total sleep duration and higher levels of RBD symptomology, including both nightmares and REM behaviours, than trauma exposed and non-trauma exposed controls. PTSD-diagnosed participants also reported more negative intrusive memories than the control groups. We also found that self-reported RBD symptomology before the second session positively mediated the relationship between PTSD status and negative intrusive memories. This effect was absent for all other measures of sleep quality and seemed to be driven largely by REM behaviours rather than nightmares. A follow up, exploratory moderation analysis revealed that the effect of self-reported RBD symptomology prior to the second session on reported negative intrusive memories was significantly moderated by group status, such that higher RBD symptomology was associated with higher number of intrusions in PTSD-diagnosed participants but not participants of other groups. These findings have implications for how we interpret the effect of sleep on memory consolidation in PTSD.

PTSD onset is associated with discrete sleep disorders, such as Trauma-Associated Sleep Disorder, which is proposed to be similar to RBD (Mysliweic, et al. 2014; Rackakonda, et al. 2018; Zhang, et al. 2017). This study used the PSQI, a self-report of RBD symptomology (the RDBSQ) and self-reported total hours of sleep prior to the testing sessions to measure different aspects of sleep relevant to emotional memory consolidation. PTSD-diagnosed participants in the present study scored significantly poorer general sleep quality over the past month, higher RBD symptomology, more nightmares, more REM behaviours and less total sleep duration on the nights immediately preceding each testing session. These findings are consistent with previous research that has found poor overall sleep quality (Germain, 2013; Kobayashi, et al., 2007; Maher, et al., 2006), abnormal REM sleep (Germain, 2013; Kobayashi, et al, 2007; Mellman, et al., 2014) and difficulties sleeping (Harvey, Jones, & Schmidt, 2003; Kobayashi, et al., 2007) in PTSD. Our findings also support the similarity of PTSD sleep disturbance to RBD, with sleep quality in PTSD-diagnosed participants being well characterised by RBD scores.

Our finding that the PTSD group reported significantly more negative intrusions than the NTE group, and more negative intrusions than TE participants at a trend level, was consistent with existing literature on PTSD and intrusive memories. Firstly, the main effect of image valence, such that intrusive memories of negative images were reported significantly more frequently than positive and neutral images, confirms the expectation that negative stimuli are more likely to produce intrusions (Brewin, et al. 2010; Ehlers & Clark, 2000). This replicates earlier studies (Kleim, et al. 2016; Nicholson, et al. 2014). Secondly, that the PTSD group experienced more of these negative intrusions than controls reflects trauma-related sensitisation of the memory consolidation system in PTSD. Evidence of dysregulation in neural networks involved in memory consolidation in patients with PTSD change the way that memories are processed (Brewin, et al., 2010; Ehlers & Clark, 2000).

These underlying circuitries include hippocampal volume reduction (Acheson, et al., 2012), amygdala hyperactivity (O'Doherty, Chitty, Saddiqui, Bennett, & Lagopoulos, 2015) and reduced amygdala inhibition by the vmPFC (Pitman, et al., 2012). TE participants also reported more negative intrusions than NTE participants, which accords with recent evidence that trauma exposure that does not result in clinical psychopathology can also impact on neural networks involved in emotional memory consolidation (Stark, et al., 2015) and memory more generally (Chou, La Marca, Steptoe, & Brewin, 2014). Our findings therefore support a dimensional approach to PTSD and trauma exposure (Stark, et al., 2015).

Neural dysregulations involving emotional memory processing are thought to contribute to sleep disturbances, and at least partly contribute to poorly consolidated memories as part of a maladaptive positive feedback loop (Germain, et al., 2008; Goldstein & Walker, 2014). Therefore, our final hypothesis used mediation analyses to measure the influence of three distinct measures of sleep quality on the relationship between PTSD status and intrusive memories. When entered as mediating variables, neither model with PSQI score or hours of sleep before the second session produced a significant indirect effect. Although hours of sleep particularly neared significance, the effect size of the indirect path was negligible. However, RBD symptomology before the second session did produce a significant mediating effect, with pronounced reductions in the direct effect and a much larger indirect effect compared to the other two sleep measures. Importantly, substantial differences were observed in indirect path effect sizes between measures of REM before the two testing sessions, despite the fact that these scores had high inter-individual correlations. Therefore, we can surmise that the mediating effect observed in RBD symptomology before the second session was likely a true mediating effect, given that direction of a mediation model is based on the temporality of the pathways towards the outcome variable (McKinnon, Fairchild, & Fritz, 2007). However, we note that this cannot be considered a confirmatory

finding without stronger evidence of a temporal chain that establishes the direction between the variables (Hayes, 2018b).

Our findings align with recent studies by Kleim and colleagues (2016) and Sopp et al (2019), who found that REM density and REM theta-range frequencies respectively predicted intrusive memory development. Our use of the RBDSQ was a unique feature of this study, and also supports the notion that sleep disturbances in PTSD are similar to those observed in RBD, which is a disorder characterised by nightmares and unusual REM behaviours, such as sudden movements (Mysliweic, et al. 2014). These findings therefore support the conceptualisation of a unique sleep disorder, such as Trauma-Associated Sleep Disorder, with unique characteristics; though, the specifics on this cannot be established based on our study alone. Interestingly, depression accounted for a large amount of variance in the difference between nightmares between groups, suggesting that Trauma-Associated Sleep Disorder might be characterised by behaviours rather than nightmares in PTSD. Regardless, our findings generally support the assertion that poor sleep quality is a potentially etiological factor in PTSD and intrusive memories, as PTSD-diagnosed participants clearly suffered poorer sleep quality in our sample and reported more negative intrusions (Germain, 2013; Spoormaker & Montgomery, 2008).

Our exploratory moderation analysis was undertaken on the basis of finding a significant mediating effect of RBD symptomology before the second session on intrusive memories of negative images. We were curious as to whether reported RBD symptomology had affected intrusions of the trauma exposed group, who did not report significantly fewer intrusions than PTSD-diagnosed participants. This moderation analysis revealed that there was a significant positive relationship between the amount of RBD symptomology preceding the second session and the number of negative intrusive memories reported in PTSD-diagnosed participants only, with high, but not medium or low, levels of RBD symptomology

being associated with intrusions in this group. This effect was absent in our two control groups. We conducted this analysis with PTSD symptom severity as a covariate to ensure that this relationship was not simply explained by higher PTSD symptomology more generally. This finding seems to indicate that the nature of RBD symptomology in PTSD is unique, and that RBD symptomology – such as nightmares and REM disturbance more generally – in the general population may not be sufficient to have harmful effects on memory consolidation. This finding needs to be replicated in future confirmatory research.

We acknowledge that our study had several limitations. Our use of IAPs images as emotional stimuli resulted in a floor effect on intrusive memories in healthy controls and for neutral images. Other authors have discussed this floor effect and it is clear that there is a strong effect of trauma on the propensity to develop intrusive memories (Nicholson, et al., 2014; Werner, et al., 2015; Wiesner, et al., 2015). Further, we had participants report intrusive memories retrospectively, which is a less favourable method than daily reporting (eg. Kleim, et al., 2016). Since our second session was conducted only two days after the first session, however, retrospective reporting will have had less of a negative impact on our study design compared to studies with longer inter-session intervals. Since we also included a surprise explicit memory task, we also did not want to provoke rehearsing of the images over the 48-hours between sessions. There are also reports that momentary reporting of emotional memories produces emotional reactivity that can interfere with laboratory studies; hence, retrospective reporting is generally preferred (Wegerer, Kerschbaum, Blechert, & Wilhelm, 2014; Wilhelm & Grossman, 2010). Importantly, due to our partially cross-sectional research design, we are unable to reject the possibility that participants who reported higher RBD symptomology did so because they experienced intrusive memories. Converging research in this area may aid interpretation of the directionality of our findings, and future researchers may address this problem with a longitudinal design. It should be



mentioned, however, that mediation is considered an appropriate model for exploratory research, though conclusions must be considered tentative before such confirmatory paradigms are completed (Hayes, 2018b). A similar limitation is that we measured RBD symptoms on the night before the second session rather than directly after the first session and can hence only infer association of RBD symptoms with number of reported intrusions. However, it should be noted that in the affective de-coupling model (Goldstein & Walker, 2014), it is suggested that de-coupling of affect occurs over successive nights.

Finally, objective and sensitive measurement of the aspects of sleep quality that we sought to investigate ideally requires polysomnography or other physiological measurements. Our sleep measurements, although obtained using validated questionnaires, therefore represent only rough estimates of the quality of each participant's sleep. Future studies must employ objective and physiological technologies to capture the subtleties of sleep such as REM density, theta rhythm and so on. However, the RBDSQ has been reported recently in a meta-analysis to be an accurate tool for screening for RBD symptomology in the general population (Li et al., 2017), and RBD is increasingly recognised as being analogous to sleep disturbances in many PTSD patients (Feemster, et al. 2019; Husain, et al. 2001; Mysliwiec, et al. 2014; Rachakonda, et al. 2018). It should also be recognised that this is the largest sleep-intrusive memory study to date, and many of our findings were expected based on previous data (such as worse REM disturbance in PTSD patients), suggesting that our measures were sufficiently sensitive to many subtle effects. In this vein, the small effect sizes detected in our mediation and moderation models potentially suggests that the direct influence of sleep quality on the development of intrusive memories may be minimal in the context of other aetiological factors in PTSD. This is particularly probable given that larger sample sizes are most likely to detect true effect sizes, whereas replication of smaller samples necessarily inflates reported effect sizes (Gelman & Carlin, 2014). Replication of this study using

physiological measures in a large sample size is necessary to further gauge the overall impact of sleep quality on intrusive memories.

Overall, the results of this study suggest a role in the development of intrusive memories in PTSD, and RBD symptoms as being characteristic of sleep disturbances in PTSD. However, these results also suggest two divergences from the current literature; the first being that increased RBD symptomology was associated with more reported intrusions in PTSD-diagnosed participants but not trauma-exposed or otherwise healthy participants. Secondly, our effect sizes were small compared to the rest of the literature, suggesting a possible previous inflation of effect sizes in smaller studies. Future studies will need to confirm the directionality of these findings and will need to examine physiological measures of sleep in larger sample sizes.

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## Figures

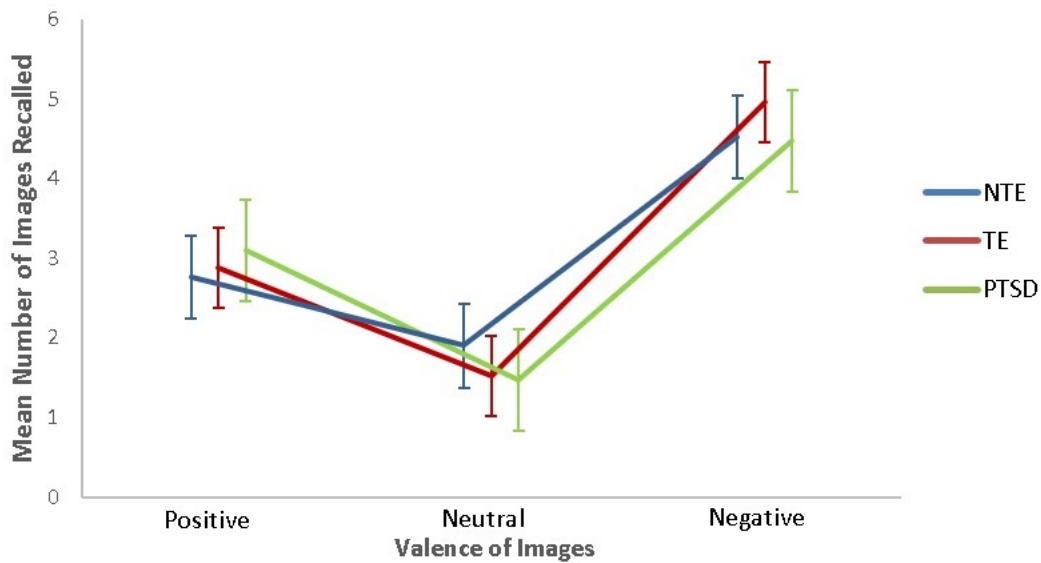
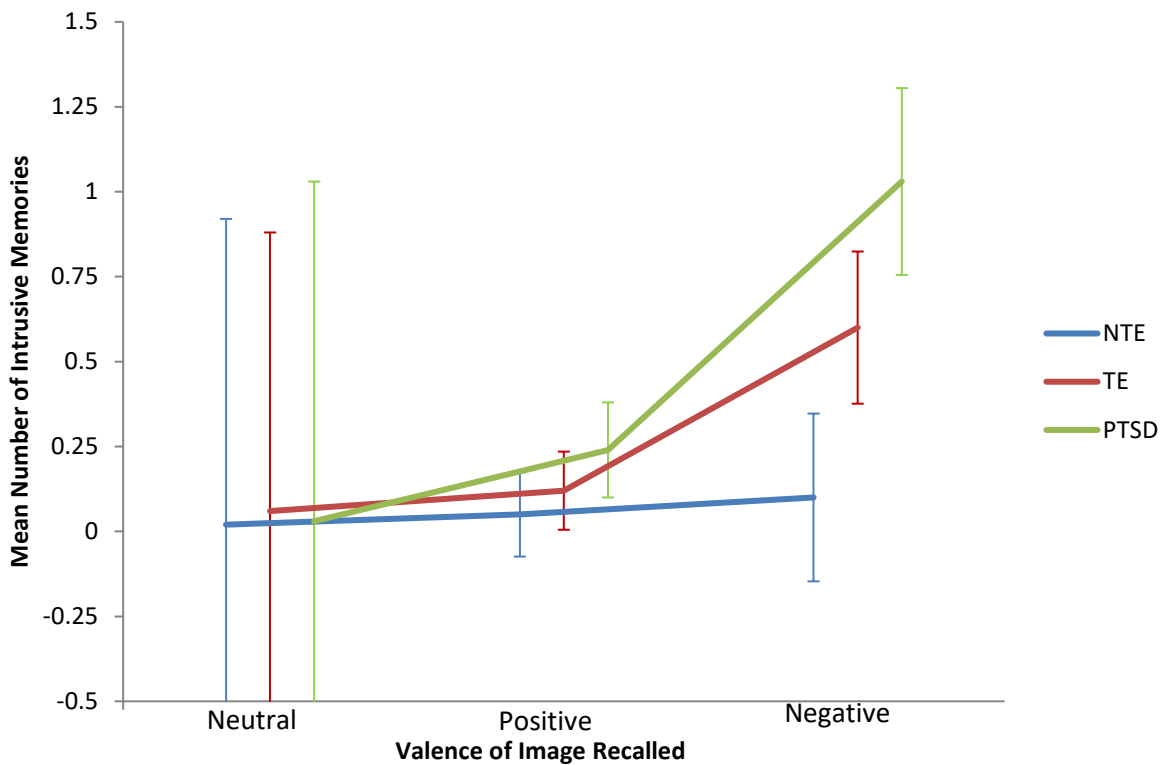


Figure 1. Mean number of neutral, positive and negative memories deliberately recalled for NTE, TE and PTSD groups. Error bars: 95% Confidence Intervals



*Figure 2.* Mean number of neutral, positive and negative intrusive memories for NTE, TE and PTSD groups. Error bars: 95% Confidence Intervals