

Brevity and Emotionality of Autobiographical Memories in Post-traumatic
Stress Disorder: Associations with disrupted sleep architecture

Hollie Bradley

Arthur Chen

ACSENT Laboratory

Department of Psychology

University of Cape Town

Supervisor: Kevin Thomas

Co-Supervisor: Gosia Lipinska

Word Count:

Abstract: 269

Main Body: 9983

PLAGIARISM DECLARATION

PLAGIARISM

This means that you present substantial portions or elements of another's work, ideas or data as your own, even if the original author is cited occasionally. A signed photocopy or other copy of the Declaration below must accompany every piece of work that you hand in.

DECLARATION

1. We know that Plagiarism is wrong. Plagiarism is to use another's work and pretend that it is one's own.
2. We have used the American Psychological Association formatting for citation and referencing. Each significant contribution to, and quotation in, this essay/report/project from the work or works, of other people has been attributed, cited and referenced.
3. This essay/report/project is our own work.
4. We have not allowed, and will not allow anyone to copy our work with the intention of passing it off as his or her own work.

NAME:

Hollie Bradley

Arthur Chen

SIGNATURE:

STUDENT NUMBER:

BRDHOL002

CHNSSU001

Acknowledgements

Firstly, we would like to extend a huge note of thanks to our supervisor Kevin Thomas and our co-supervisor Gosia Lipinska for the support, guidance and time invested throughout the project. Both have made significant contributions to this thesis for which we are extremely grateful. We would also like to thank the A W Mellon Foundation for financial assistance.

Abstract

Previously published research suggests that individuals diagnosed with post-traumatic stress disorder (PTSD) have difficulty initiating and/or maintaining sleep. A separate line of research suggests that PTSD-diagnosed individuals have less specific and less emotion-laden autobiographical memories than individuals without PTSD. A third line of research suggests that, in healthy adults, disrupted sleep affects memory processing negatively. However, there are no published studies investigating whether there is an association between sleep disruption and autobiographical memory in PTSD. We used a quasi-experimental design to investigate this possible association, using the Autobiographical Memory Test (AMT) and 1 night of polysomnographic sleep data collected in a sleep research laboratory. Our sample consisted of 40 females aged between 25 and 40 years. Each was assigned to one of four groups: PTSD-diagnosed individuals, non-PTSD trauma-exposed individuals, individuals diagnosed with major depressive disorder but not PTSD, and healthy individuals. A series of linear contrast analyses testing the prediction that PTSD < trauma-exposed = depression < healthy control did not reveal any significant between-group differences with regard to sleep parameters. However, a similar series of linear contrasts showed that PTSD-diagnosed individuals responded with fewer words to all autobiographical memory cues, and generated more emotionally-laden memories in response to those cues. Finally, general linear modelling revealed that the number of minutes spent awake after sleep onset in the second half of the night, as well as group status, accounted for a significant amount of the variance in AMT word count and emotionality for negative cues. These results suggest that disrupted sleep is a mechanism that might help explain the presence of brief and emotionally-laden autobiographical memory in PTSD-diagnosed individuals.

Keywords: Autobiographical Memory Test; overgeneral autobiographical memory; sleep; PTSD; trauma; polysomnography

Brevity and emotionality of autobiographical memory deviations in Post-traumatic Stress Disorder: Associations with disrupted sleep architecture

Each individual is unique in preferences, reactions to different situations, and personality. This uniqueness of individuals is facilitated by a specific type of cognitive process called autobiographical memory (AM). AM can be defined as “the aspect of memory concerned with the recollection of personally experienced past events” (Williams et al., 2007, p. 122). Intact AM is an essential part of what is considered normal functioning in many everyday situations: It contributes to a sense of self, guides social behaviour and problem solving, and facilitates the ability to envision yourself in the future (Brown et al., 2013), making it an important phenomenon for psychologists to understand. How and why AM functioning goes wrong, and the consequences of these deficits, are necessary aspects of such understanding. To this end, studying the clinical population of individuals diagnosed with posttraumatic stress disorder (PTSD) holds key knowledge due to the fact that AM deficits are associated with the diagnosis.

To be diagnosed with PTSD, one must have experienced, directly or indirectly, a trauma in the form of “actual or threatened death, serious injury or sexual violation” (American Psychiatric Association [APA], 2013, p. 271). Other diagnostic criteria include symptoms such as re-experiencing, avoidance, negative cognitions, and altered arousal signs (APA, 2013). Individuals diagnosed with PTSD often experience difficulties with memory (Brewin, 2011). Specifically, these individuals tend to experience intrusive automatic memories as part of the re-experiencing cluster of symptoms (Moradi et al., 2008), and they also show poor episodic memory performance (Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001). They also tend to experience a different quality of AM to that experienced by healthy adults: Autobiographical memories in PTSD tend to be less specific, a quality often referred to as *overgeneral autobiographical memory* (Dalgleish, Rolfe, Golden, Dunn, & Barnard, 2008; McNally, Lasko, Macklin, & Pitman, 1995; Williams et al., 2007).

Sleep is also disrupted in individuals diagnosed with PTSD (Babson & Feldner, 2010), and healthy sleep is important for intact memory functioning (Diekelmann & Born, 2010). Therefore, in order to fully understand the relationship PTSD has to memory, one must examine, firstly, the sleep and memory relationship broadly, and then look more specifically at PTSD’s link to sleep and memory respectively.

Sleep and Declarative Memory

There is an undisputed link between healthy sleep and unimpaired memory performance. One of the critical functions of sleep, particularly with regard to cognition, is to improve memory through its supporting role in memory consolidation (Fogel & Smith, 2011; Marshall & Born, 2007). Several studies show that adequate sleep improves declarative memory, which is a form of long-term memory (Diekelmann & Born, 2010; Ellenbogen, Hulbert, Stickgold, Dinges, & Thompson-Schill, 2006). Declarative memory comprises two subtypes, episodic and semantic. *Episodic memory* refers to remembering past events that are specific in time and place; it typically involves the recollection of vivid sensory, perceptual, and emotional detail. *Semantic memory*, in contrast, refers to the recollection of facts, and concepts (Tulving, 1972). Autobiographical memory is thus a type of episodic memory in that it has a temporal dimension. (Semantic memory, in contrast, has a factual but not a temporal dimension).

The consolidation of declarative memory occurs throughout the night in healthy individuals. Sleep is dominated by two stages most closely associated with memory consolidation, namely slow wave sleep (SWS) and rapid eye movement (REM) sleep. These occur predominantly in the first and second half of the night, respectively. Both SWS and REM have supportive roles in declarative memory processing (Goerke et al., 2013). During SWS, information is distributed from the hippocampus to the neocortex, where some memory traces are processed based on the strength of their potentiation at synapses; weak connections are eliminated (Diekelmann & Born, 2010). During REM, these memory traces are strengthened so as to make them more durable (Diekelmann & Born, 2010).

Other factors involved in declarative memory consolidation include the emotional valence of to-be-remembered material and the integrity of sleep architecture (Atienza & Cantero, 2008). Empirical studies suggest that highly emotional stimuli are recalled better than neutral stimuli, especially after REM sleep (Ellenbogen, Payne, & Stickgold, 2006; Goerke et al., 2013; McGaugh, 2013). One such study confirmed that memory for emotional material, but not neutral material, was more likely to be retained when the individual was afforded the opportunity to enter the later stages of sleep, where the majority of REM sleep occurs (Wagner, Gais, & Born (2001). In that study, participants who stayed awake during the delay between learning material and being asked to recall it showed little enhancement of memory for emotional material.

In summary, both SWS and REM sleep play critical roles in the consolidation of declarative memories. By inference, each has a critical association with the formation of autobiographical memories.

PTSD and Sleep

Sleep disturbances (e.g., insomnia and awakenings) are commonly reported by individuals diagnosed with PTSD. However, studies of PTSD-diagnosed individuals that examine objective disturbances in sleep architecture as measured by polysomnography (sleep-adapted electroencephalograph, or EEG) show inconsistent results (Spoomaker & Montgomery, 2008). For instance, Mellman, Nolan, Hebding, Kulick-Bell and Dominguez (1997) reported that Vietnam War veterans diagnosed with PTSD showed, relative to depressed and healthy participants, more awakenings and increased night-time wakefulness, as well as slightly decreased overall sleep efficiency (amount of time spent in sleep). In contrast, Hurwitz, Mahowald, Kuskowski and Engdahl (1998) found no significant difference in sleep architecture between Vietnam veterans with and without PTSD.

A meta-analysis of polysomnographic studies relating to PTSD and disruption at particular sleep stages attempted to resolve these contrasting accounts (Kobayashi, Boarts, & Delahanty, 2007). That quantitative review found that, relative to healthy controls and to individuals who had experienced trauma but who were not diagnosed with PTSD, individuals diagnosed with PTSD had longer stage 1 Non-REM sleep, higher REM density (frequency of eye movements during REM), and less SWS.

The meta-analysis suggests that the higher REM density indicated altered REM functions in PTSD diagnosed groups (Kobayashi et al., 2007), which is a more specific sleep disruption investigated with regards to PTSD in the literature. This REM fragmentation has been found to predict PTSD development, making it an important focal point (Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002). There are numerous REM changes reported in the literature regarding the PTSD population, these include: more transitions from REM to waking and from REM to stage 1, higher level of brief arousal in REM sleep (Breslau et al., 2004); decreased REM percentage and increased REM latency in the second half of the night (Lipinska, Timol, Kaminer, & Thomas, 2014); and less time spent in a single REM period but more frequent REM occurrences (Engdahl, Eberly, Hurwitz, Mahowald & Blake 2000).

In summary, previously published studies suggest that PTSD-diagnosed individuals have sleep disruptions at both REM and SWS stages, with more significant disruptions during REM.

PTSD and Autobiographical Memory

Several studies suggest that overgeneral AM is characteristic of many types of trauma survivors, including combat veterans (Brown et al., 2013; Moradi, Abdi, Fathi-Ashtiani, Dalglish, & Jobson, 2012), assault victims (Kleim & Ehlers, 2008), and cancer-diagnosed patients (Moradi et al., 2008). AM in PTSD-diagnosed and trauma-exposed individuals is often measured using the Autobiographical Memory Test (AMT). The AMT is a well-established instrument that examines the specificity and quality of autobiographical memories (Williams & Broadbent, 1986). The test proceeds as follows: the examiner provides the participant with a cue word (e.g., *party*) and asks him/her to recall a specific AM related to the word. This administration is repeated across a number of trials, often featuring words that differ in emotional valence (e.g., the positively-valenced word *proud* might be presented, as might the negatively-valenced word *guilt*). So, for example, a trauma survivor might, in response to the cue word *party*, recall a general memory such as disliking most parties. In contrast, a healthy individual may recall disliking a specific person's party last week.

Of note is that there is controversy regarding whether overgeneral memories are valence-free. Some researchers argue that PTSD-diagnosed individuals show a lack of specificity when responding to both negatively- and positively-valenced cue words (Williams et al., 2007). Others, however, argue that PTSD is associated with reduced specificity to positive cues only (Kleim, Graham, Fihosy, Stott, & Ehlers, 2014; Sutherland & Bryant, 2007), and still others have found reduced specificity to negative cues only (Williams, Eelene, Raes & Hermans., 2006).

Overgeneral AM is not, however, unique to PTSD-diagnosed individuals. Individuals diagnosed with major depressive disorder (MDD), upon administration of the AMT, also display lack of specificity (Buckley, Blanchard & Neill, 2000; Watson, Berntsen, Kuyken, & Watkins, 2013). The lack of specificity in clinically depressed individuals appears to be in regard to positively-valenced memories (Brewin, 1998; Williams et al., 2006).

Of particular interest here, however, is that there is significant overlap in the clinical presentation of MDD and PTSD, and that the disorders are frequently comorbid (Brewin, Reynolds, & Tata, 1999; Tural, Önder, & Aker, 2012). Some authors go so far as to say that the PTSD diagnosis itself is not associated with overgeneral memories, but that (comorbid) depression is (Conway & Pleydell-Pearce, 2000). In a study disputing that view, McNally and colleagues (1995) showed that deficits in AM retrieval were “more strongly related to psychological disturbances arising from trauma than to depression *per se*” (p. 628).

Another aspect of interest here is the lack of extant research into the quality, beyond specificity, of the memories themselves. For instance, no studies have examined whether the emotional content of the retrieved memories is positively or negatively valenced, or to what extent they are emotional at all. The typical AMT study describes only the specificity of retrieved memory; a more nuanced way to look at the quality of AM in PTSD, or in depression, may be to examine the emotional content of the retrieved memory. Such examination may be particularly interesting because a strong line of literature regarding PTSD shows that diagnosed individuals try to avoid the experience of emotions (Frewen, Dozois, Neufeld, & Lanius, 2012; Julian & Breen, 2010). This avoidance extends beyond the emotions related to the traumatic experience and on to emotions more generally (Tull, Gratz, Salters, & Roemer, 2004). The clinical significance of such avoidance is illustrated by Boden et al. (2013), who found, in a group of PTSD-diagnosed military veterans, that the degree to which emotions were suppressed correlated positively with symptom severity.

Rationale, Specific Aims, and Hypotheses

The literature reviewed above suggests that (a) sleep is important for declarative memory (and therefore, by inference, AM) processing, (b) sleep is disrupted in PTSD, (c) AM is disrupted in PTSD, and (d) PTSD-diagnosed individuals tend toward emotional suppression. There are, however, no published studies that directly test whether disrupted sleep might serve as a mechanism underlying overgeneral or less emotional AM in PTSD, and there are no studies that examine the content of AM in response to different valenced cues.

In the current study, we attempted to address these gaps in the literature by collecting polysomnographic sleep data and AMT data across four groups: individuals diagnosed with PTSD, non-PTSD trauma-exposed individuals (hereinafter referred to as the trauma-exposed group), individuals diagnosed with major depressive disorder but not PTSD (hereinafter referred to as the depression group), and healthy individuals. We hypothesised that, compared to participants in the other three groups, individuals diagnosed with PTSD would have (1) worse sleep quality and less sleep quantity, and (2) less specific and less emotional AM. We also hypothesised that (3) across the sample, poorer sleep quality and quantity would each hold predictive value for less specific and less emotional AM.

METHODS

Design and Setting

The study was quasi-experimental in design. The major predictor variable in the study was group status, with four levels: PTSD, trauma-exposed, depression, and healthy control. Outcome variables were measures of sleep quality and AMT performance. Study procedures took place at the University of Cape (UCT) Department of Psychology and the Vincent Pallotti Hospital sleep laboratory.

Participants

As part of a larger research programme, 60 participants, all female and between the ages of 25 and 40 years, were recruited and screened, and met the criteria for participation. Eight of those individuals chose to withdraw from participation after screening. Of the remaining 52 participants, AMT data were unusable for 12 (10 due to experimenter errors in recording participant responses, and 2 due to participants not completing the test). Hence, 40 participants provided data for the current study. Each of those was assigned to one of four groups, based on the inclusion and exclusion criteria described below: PTSD ($n = 11$); trauma-exposed ($n = 9$); depression ($n = 11$); and healthy control ($n = 9$).

The reason for including a depression group alongside the other two control groups is that, typically, many PTSD-diagnosed and trauma-exposed individuals also have significant levels of depression (Löwe et al., 2011). Hence, depression serves as a major potential confound in studies such as this, particularly because sleep architecture in PTSD and major depressive disorder (MDD) is different. For instance, Srinivasan et al. (2009) found decreased SWS and REM latency, and increased REM percentage, in individuals diagnosed with depression, whereas Mellman and colleagues (1997) found that their PTSD group had decreased total sleep and increased awakenings when compared to depressed and healthy groups. Furthermore, there is controversy in the literature as to whether depression or trauma is the key to overgeneral AM (McNally et al., 1995; Van Vreeswijk & Wilde, 2004). Hence, our inclusion of both PTSD-diagnosed and depressed individuals was in service of attempting to clarify the root of overgeneral AM.

We recruited the 20 participants assigned to the PTSD and trauma groups from Rape Crisis Cape Town Trust, which operates in Cape Town and surrounding communities. This organisation is dedicated primarily to providing counselling services to survivors of sexual violence. The 20 participants in the depression and healthy control groups were recruited via

advertisements in local newspapers. Groups were aggregate matched in terms of sex, age, IQ, and socioeconomic status.

Eligibility criteria. The following list of exclusion criteria is exhaustive, and was applied rigorously.

1. Potential participants diagnosed with any DSM-IV-TR (Diagnostic and Statistical Manual of Mental Disorders, 4th ed., text revised; APA, 2000) Axis I disorder, except for PTSD in the PTSD group and MDD in the depression group, were excluded. Disrupted sleep patterns and memory deficits associated with such disorders are potential confounds to the current research question (Harvey, Jones, & Schmidt, 2003). However, those in the PTSD and trauma-exposed groups who presented with anxiety disorders or depression secondary to trauma were not excluded. Within the PTSD group, 1 participant presented with panic disorder with agoraphobia, 2 with panic disorder, 1 with panic attacks, and 4 with dysthymia. Within the trauma-exposed group, 1 participant presented with panic disorder, 1 with social phobia, 1 with agoraphobia, and 1 with dysthymia. Three participants in the trauma-exposed group did not have threshold depressive symptoms. The remaining participants in the trauma groups did meet the criteria for MDD. Participants with no psychopathology formed the healthy control group.
2. Potential participants with a history of alcohol or other substance abuse were excluded. Alcohol or other substance abuse is related to sleep and memory dysfunction, and may therefore also confound interpretation of the results (Gilbertson et al., 2001).
3. Potential participants below the age of 25 years and above the age of 40 years were excluded. Normal aging is associated with mild memory decline and altered sleep cycles (McEwen, 1999), and the sleep patterns of children and adolescents differ from those of adults (Kales et al., 1970).
4. Potential participants who were taking sedative or psychoactive medication were excluded. Sleep medication changes natural sleeping patterns, and psychoactive medication can influence brain structure and function (see, e.g., Vermetten, Vythilingam, Southwick, Charney, & Bremner, 2003).
5. Participants who experienced trauma more than 5 years or less than 6 months prior to study enrolment were excluded. Proximity to trauma has implications for both memory functioning and sleep architecture. For instance, participants who experienced trauma more than 5 years ago may have experienced childhood trauma,

which can affect the developmental trajectory of memory processing (De Bellis, Hooper, & Sapia, 2005).

6. Participants who carried neurological conditions (e.g., epilepsy, traumatic brain injury) with the potential to influence the outcomes of the study were excluded. Five potential participants revealed at screening that they were HIV-positive, but did not present with any AIDS-related disorders and were regarded as asymptomatic (they had no weight loss, recurrent fever, or opportunistic infections). AIDS is associated with HIV-related dementia, while HIV alone is not (Guillemin, Kerr, & Brew, 2005). These potential participants were thus not excluded based purely on their HIV-positive status.

Materials and Apparatus

Diagnostic and screening instruments. The *Mini International Neuropsychiatric Interview (MINI Version 5.0;* Sheehan et al., 1998) is a short (approximately 15-minute) structured diagnostic interview that is used to determine the presence or absence of major DSM-IV psychiatric disorders. The developers report that it has good psychometric properties. Regarding its appropriateness for use in South Africa, the MINI has been successfully administered before in South African research studies (e.g., Joska et al., 2011). The interview can be administered by a clinician or by a lay interviewer with the appropriate training. In the current study, the MINI was used to confirm diagnoses of PTSD and MDD, and to exclude the presence of other psychiatric conditions.

The *Clinician Administered PTSD Scale (CAPS;* Blake et al., 1995) is a structured interview designed to determine PTSD core and related symptoms. Specifically, the instrument allows measurement of the intensity and frequency of PTSD symptoms using an explicit, behaviourally anchored rating scale. According to its developers, the CAPS has good reliability and validity. In the current study, we used the CAPS to validate the PTSD diagnosis provided by the MINI. The CAPS has been successfully administered in South African research studies (see, e.g., Stein et al., 2013). The manner in which we scored CAPS is described in Appendix A.

The *Beck Depression Inventory – Second Edition (BDI-II;* Beck, Steer, & Brown, 1996) is a 21-item self-report standardized measure of depressive symptomatology in adults. The instrument's developers report that it has good reliability and validity. The test is used globally, and has been employed successfully in South Africa (see, e.g., Kagee & Martin,

2010; Nel & Kagee, 2013). In the current study, the BDI-II helped validate the MDD diagnosis provided by the MINI, and provided a measure of the severity of depressive symptomatology.

The *Michigan Alcoholism Screening Test (MAST; Selzer, 1971)* is a 25-item structured interview designed to identify the participant's degree of alcoholism. Its developers report that it has good validity. In the current study, we used the MAST to identify individuals who were alcohol abusers or who were alcohol-dependent. Potential participants who scored more than 5 were excluded. The MAST has been administered successfully in South African research studies (see, e.g., Myers, Nell, Taylor, & Thompson, 1997).

The *Wechsler Abbreviated Scale of Intelligence (WASI; The Psychological Corporation, 1999)* is a short, four-subtest version of the Wechsler Adult Intelligence Scale (WAIS) that measures general intellectual functioning in adults. The instrument includes both Verbal and Performance subtests, which are combined to give a Full Scale IQ score. In the current study, only the Performance index was used as participants were not first-language English speakers and there is no validated non-English WASI equivalent. Performance IQ (PIQ) correlates well with Full Scale IQ, and hence using only the two Performance subtests does not affect the reliability or validity of the WASI greatly (Wechsler, 1999). We used this instrument to ensure that there were no significant between-group differences with regard to general intellectual functioning. The WASI has been administered successfully before in South African research studies (see, e.g., Ferrett, Carey, Thomas, Tapert, & Fein, 2010).

Experimental measures. We assessed AM using a version of the Autobiographical Memory Test (AMT; Williams & Broadbent, 1986). The version used here is reproduced in Appendix B. The participants were given three practise words before the actual AMT began to ensure they understood their task. During the actual task, participants were presented, one by one, with 5 neutral words, 5 positively-valenced words, and 5 negatively-valenced words. In presenting the words, a positively-valenced word was followed by a negatively-valenced word, which was followed by a neutral word. This pattern repeated for all 15 words. After presentation of each word, the participant was asked to retrieve a specific memory linked to the word. All testing procedures were carried out by one of three female researchers (all postgraduate students), and all responses were audiotaped.

Sleep laboratory equipment and measures. We used polysomnography (PSG), i.e., EEG adapted for sleep recordings. EEG electrodes are used to measure brain activity, electrocardiograph (ECG) electrodes are used to monitor heart rate, electrooculograph (EOG)

electrodes are used to monitor eye movements, and electromyograph (EMG) electrodes are used to measure muscle tone. Sleep stages, as determined by the standard measurements of EEG, EOG, and EMG, were classified according to the outline by Rechtschaffen and Kales (1968).

We recorded sleep measures using a 16-channel Nihon Kohden NeuroFax EEG9000. We used a bipolar longitudinal montage, including the following bipolar derivations: F3-C3, C3-P3, P3-O1 and F4-C4, C4-P4, P4-O2. We placed electrodes according to the international 10-20 placement system. Standardized filters for recording sleep were employed for the EEG and EOG (0.5-35 Hz), EMG (10-70 Hz) and ECG (1-70 Hz) leads to ensure signal integrity in each of the channels. All testing procedures were again carried out by one of three female researchers (all postgraduate students).

Procedure

Potential participants underwent screening in a private room at the UCT Department of Psychology. There, they read and signed an informed consent document (see Appendix C), and thereafter the researcher administered the screening instruments listed above. The participants were then debriefed regarding the procedures thus far. If deemed eligible, the participant was assigned to one of the four study groups and a sleep testing night was scheduled.

The second phase of the study consisted of one night of sleep testing which occurred within 2 weeks of their screening. Participants arrived at the Vincent Pallotti Hospital sleep laboratory at 20h00 and completed cognitive testing that formed part of the larger research program within which this study is nested. The researcher then attached the sleep monitoring equipment to the participant. The lights were turned off within half an hour of their usual bedtime to ensure as little disruption to normal sleeping patterns as possible. In the morning, toward the end of a second session of cognitive testing, participants completed the AMT.

The researcher then debriefed the participants regarding the study procedures, compensated them for their time, and allowed them to leave.

Ethical Considerations

We obtained approval for all study procedures from the appropriate ethics committees of our institution, and all participants provided written informed consent for the study.

Because participants in the trauma groups were particularly vulnerable (particularly during the screening/interview session, where they were faced with specific questions about

previous exposure to traumatic events), participants were verbally assured at the beginning of each session that they did not have to give more details than they were comfortable with, and that they could withdraw from the study at any point without penalty.

All participants were assigned a number that accompanied all data collected in relation to them, thus ensuring confidentiality. Participants were compensated R150 for the night spent in the sleep laboratory. Participants were fully debriefed after the study, and were provided with the contact details of counselling centres and trauma counsellors before being leaving. Additionally, a registered clinical psychologist was made available to participants who experienced any distress during the study process. However, the intervention of the psychologist was not necessary.

Data Management and Statistical Analyses

Scoring and deriving outcome variables. First, the participant's responses to each item of the AMT were coded as either *specific*, *extended*, *categoric*, *semantic association*, or *omission*, using these criteria:

1. **Specific:** a memory specific to 1 day or less.
2. **Extended:** a memory that lasted longer than 1 day.
3. **Categoric:** a memory that referred to a type of event (e.g., 'every time I see my Aunt').
4. **Semantic association:** a memory that provided a definition of the cue word, rather than a memory (e.g., 'sadness is when you are not happy').
5. **Omission:** no memory could be recalled within 60 s.

According to Raes, Hermans, de Decker, Eelen, and Williams (2003), this scoring method has good reliability. Three independent raters coded the memories, and inter-rater reliability was as follows: $r_{AB} = .74$; $r_{AC} = .72$; $r_{BC} = .79$. Where there was a two-thirds majority, the majority coding was used, Where there was total disagreement, the specific items were discussed by the three raters until they were in consensus.

We assessed the emotionality of autobiographical memories using the Linguistic Inquiry and Word Count program (LIWC; Pennebaker, Chung, Ireland, Gonzales, & Booth, 2007). LIWC is a text analysis software program that is used to analyse transcribed verbal text that is stored in Microsoft Word or other similar word processing software files (Pennebaker et al., 2007). LIWC 2007 is the latest version of the program, and includes approximately 80 separate dictionary files, each of which contains "the collection of words that define a particular category" (Tausczik & Pennebaker, 2010, p.27). The transcribed AMT

responses were run through this software in order to determine the number of emotional words used by participants. We used the dictionary file titled *affect* for this procedure. The LIWC affect dictionary includes words such as *happy*, *nice*, *hurt*, *worried*, and *sad*.

The sleep data we collected using the PSG readings were used to derive the following outcome measures: REM percentage (percentage of time spent in REM sleep), SWS percentage (percentage of time spent in SWS), WASO (minutes spent awake after sleep onset), sleep efficiency (percentage of time spent asleep during an 8-hour span), spontaneous arousals (a period of abrupt EEG shift during the night, lasting 3 or more seconds), and number of awakenings (a period of waking after sleep onset that is more than 1.5 minutes in duration; Chokroverty, 2009a). Each of these variables was measured for the first half and the second half of the night separately.

Before conducting statistical analyses on these sleep variables, we scored the data according to the criteria of the AASM (2007). All record names and identification numbers were re-coded so that the sleep data were scored blind to the group allocation of each participant. The UCT sleep research team attended numerous training sessions with Jan Top, a sleep technologist located at Panorama MediClinic, and with Marlene Gounder, the sleep technologist at Vincent Pallotti Hospital, to ensure reliable scoring of data. In addition, 25% of the records scored were sent for validation to the Panorama MediClinic. These records were scored blind, with no knowledge of the participants' group allocation. An inter-rater reliability of 88.5% was calculated between the principal investigator of the larger research programme and the sleep technologist at Panorama MediClinic.

Inferential statistical analyses. To examine between-group differences with regards to sleep and AMT outcome variables, we conducted a series of linear contrast analyses. The decision to use this form of analysis was based on the fact that we had specific predictions about the performance of each group in relation to the others. That is to say, for each outcome variable we predicted that the healthy control group would perform best, followed by the depressed and the trauma-exposed groups (who we predicted would perform equally), followed by the PTSD group. Stated otherwise, $PTSD < T-E = DEP < HC$.

The benefits of using linear contrast analyses are numerous. Perhaps primary among these is that omnibus *F*-tests, such as ANOVAs, do not answer the question of interest directly: They are aimed purely at rejecting the null hypothesis. They also involve a loss of statistical power which may negatively impact the detection of effects, especially when small sample sizes are involved. Using linear contrasts allows for more specific predictions to be

tested with a greater conceptual clarity and a greater power for tests of significance (Rosenthal, Rosnow, & Rubin, 2000).

The logic behind the set of predictions we tested is this: Numerous studies have shown that PTSD is often comorbid with depression, with more severe trauma symptoms associated with greater depression (Araújo et al., 2014). Furthermore, both trauma and depression have been linked to deficient sleep quality (Babson & Feldner, 2010). Hence, to clarify the pure relationship between PTSD and sleep disruptions, it is important to distinguish PTSD-diagnosed individuals from trauma-exposed individuals, and from depressed individuals. Therefore, our inclusion of the trauma-exposed as well as depression group helps determine whether it is trauma or the depression, or the specific symptomatology of PTSD, that is associated with poorer sleep. Finally, the relationship between trauma-exposed and depressed individuals is not yet understood, and that is why we predict, tentatively, that they will perform equally on the outcome measures. However, for our current purposes, it is simply necessary that both groups perform more poorly than the control group but better than the PTSD group.

For all inferential tests, we set the threshold for statistical significance at $\alpha = .05$. We completed all analyses using SPSS version 22.

Testing hypothesis 1. To examine between-group differences with regards to sleep quality, we performed a series of linear contrast analyses, as described above, on the measures of REM percentage, SWS percentage, WASO, sleep efficiency, spontaneous arousals, and the number of awakenings. Note that we analysed data regarding both the number of awakenings and the number of spontaneous arousals as these represent two different kinds of changes in consciousness and may occur with different frequencies between different clinical groups (Chokroverty, 2009a). We recorded data for each of these outcomes for the first and second half of the night separately, and conducted a separate linear contrast analysis (with the prediction PTSD < T-E = DEP < HC) on each.

Testing hypothesis 2. To examine between-group differences with regards to AMT outcomes, we performed a series of linear contrast analyses, as described above, on measures of specificity, word count, number of emotional words used (emotionality), and the proportion of emotional words used relative to the total word count. We recorded data for each of these outcomes on positive, negative, and neutral cue words separately, and conducted a separate linear contrast analysis (with the prediction PTSD < T-E = DEP < HC) on each.

Testing hypothesis 3. We used general linear modelling to examine whether sleep variables could predict, and account for a significant proportion of variance in, AMT outcomes. We built one model for each AMT outcome on which previous analyses had detected significant between-group differences. Only sleep variables from the second half of the night were used. This is because the literature states that sleep disruption in PTSD is characterized by REM disruption (Spoormaker & Montgomery, 2008; Mellman et al., 2002), REM fragmentation in the aftermath of a trauma predicts the development of PTSD (Breslau et al., 2004) and REM occurs predominantly in the second half of the night (Roth, Singh, Steinberg, Waldron, & Moline, 2013).

RESULTS

Sample Characteristics

Regarding sociodemographic variables, Table 1 shows that the four groups were well matched on age, highest level of education, and monthly income. Regarding age, the mean of the entire sample was 27.23 years ($SD = 5.60$, range = 20 - 40). Regarding education, most participants ($n = 39$, 97.5%) had completed at least some high school; a few ($n = 14$, 35%) had completed further educational courses or at least one year of tertiary education. Regarding socioeconomic status, the monthly income of all participants was less than ZAR10 000, suggesting that all could be classified as being of low SES. Regarding WASI PIQ scores, there were no significant between-group differences. The mean of the entire sample was 82.46 ($SD = 13.21$).

Regarding BDI-II scores, Table 1 shows that a one-way ANOVA detected a statistically significant between-group difference, associated with a large effect size. A set of planned orthogonal contrasts investigated the source of that difference. First, data from the healthy control group were contrasted with those from the other three groups, taken together, to confirm that, on average, healthy controls were significantly less depressed than the other participants. As expected, this contrast was statistically significant, $t(36) = -9.55$, $p < .001$, with data trending in the expected direction. In fact, none of the healthy controls scored more than 13 on the BDI-II, indicating that participants in this group were not experiencing even mild levels of depression. The second contrast compared data from the PTSD group to those from the trauma-exposed and depression groups, taken together. This contrast was also statistically significant, $t(36) = -2.77$, $p = .011$. This analysis suggests that individuals diagnosed with PTSD were, on average, more depressed than participants in the trauma-exposed and depression groups. This finding is consistent with previous research showing that more severe trauma symptoms are associated with greater depression (Araújo et al., 2014). The final contrast compared data from the trauma-exposed and depression groups. This time, there was no statistically significant between-group difference, $t(36) = 0.50$, $p = .62$. So, given that the primary clinical characteristic these two groups share is depression, it was of interest to examine whether, and how, they differed with respect to sleep and memory outcome variables. We explored this interest in subsequent analyses.

Table 1

Sample Sociodemographic, IQ, and Psychiatric Characteristics (N = 40)

Variable	Group								<i>F</i> / χ^2	<i>p</i>	ESE
	PTSD (<i>n</i> = 11)		Trauma Exposed (<i>n</i> = 9)		Depression (<i>n</i> = 11)		Healthy Control (<i>n</i> = 9)				
Age	28.03	(6.77)	26.67	(6.07)	27.25	(3.62)	26.79	(6.37)	0.12	.95	.01
Education (years)	12.00	(2.37)	11.78	(1.30)	11.91	(1.22)	11.89	(1.05)	0.03	.99	< .01
Monthly Income (ZAR) ^a									12.95 ^b	.16	.33
≥ 10 000	0		0		0		0				
5500 – 9999	0		3		4		0				
2500 – 5499	4		1		1		5				
1000 – 2499	4		3		3		3				
0 – 999	3		2		2		1				
BDI-II total score	31.82	(6.63)	23.00	(9.50)	25.00	(8.06)	5.56	(4.88)	21.59	< .001***	.64
CAPS	71.27	(8.74)	40.11	(11.02)	-	-	-	-	49.87	< .001***	.73
Time since trauma (years)	2.47	(1.90)	1.77	(1.15)	-	-	-	-	0.82	.38	.05
WASI PIQ ^c	84.30	(14.94)	79.67	(13.51)	84.55	(9.77)	80.67	(15.91)	0.33	.81	.03

Note. For all variables except Monthly Income, means are presented with standard deviations in parentheses. For Monthly Income, raw numbers of participants are given. ESE = effect size estimate (in this case, either η^2 (for *F* tests), or Cramer's *V* (for χ^2 tests)); ZAR = South African Rands; BDI-II = Beck Depression Inventory – Second Edition; CAPS = Clinician-Administered PTSD Scale; WASI PIQ = Wechsler Abbreviated Scale of Intelligence Performance IQ Score.

^a*n* = 10 for the Depression group as a result of missing data due to incompleteness of the questionnaire.

^b*df* = 9.

^c*n* = 10 for the PTSD group as a result of missing data due to screening error.

p* < .05, *p* < .01, ****p* < .001.

In summary, the omnibus *F*-test and the set of post-hoc orthogonal contrasts suggested the following order of means with regard to BDI-II scores: PTSD > trauma-exposed = depression > healthy controls. A closer look at the individual BDI-II data indicated that all participants in the PTSD group and almost all participants in the trauma-exposed group met the BDI-II cut-offs for at least mild depression (i.e., a score above 13; $n = 11$ and 6, 100% and 66.6%, respectively).

Regarding trauma symptom severity, Table 1 shows that PTSD-diagnosed participants had statistically significantly higher CAPS scores than participants in trauma-exposed group. This difference was created, at least partly, by recruitment processes and study eligibility criteria, and allowed us to distinguish between the experience of trauma and the development of PTSD subsequent to trauma.

Regarding time since trauma, there was no significant difference between the PTSD and trauma-exposed groups. Hence, this study was not vulnerable to the kind of methodological confound that might have affected previous studies in this field, many of which had large inconsistencies with regards to time since trauma. Furthermore, the analysis shown in Table 1 confirms that time since trauma need not take on covariate status in subsequent analyses.

Hypothesis 1: Between-group comparisons – sleep outcome variables

There are differential distributions of SWS and REM in the two halves of any sleep night, with SWS occurring predominantly in the first half of the night and REM predominantly occurring in the second (Roth et al., 2013). Furthermore, our predicted directions of effect should occur more strongly in the second half of the night than the first, given that disrupted REM in the aftermath of a trauma predicts the development of PTSD and that PTSD-diagnosed individuals have more disrupted REM sleep (e.g., more arousals from REM sleep, more REM-to-wake transitions, and higher REM density) than controls (Kobayashi et al., 2007; Lipinska et al., 2014; Mellman et al., 2002). Hence, to facilitate a clearer investigation we analyzed sleep data for each half of the night separately, but concentrated on the second half of the night in particular.

First, we tested the assumptions underlying parametric statistical analyses (i.e., normality of distribution and homogeneity of variance) for all sleep outcome variables (REM percentage, SWS percentage, WASO, sleep efficiency, spontaneous arousals and number of awakenings), for each half of the night separately. Specifically, we used the Kolmogorov-Smirnov goodness-of-fit test to assess the normality of each data distribution. For this test, a

statistically significant p -value indicates that one can reject the null hypothesis that the data are normally distributed. Appendix D shows that, for data from the first half of the night, only REM percentage and sleep efficiency were normally distributed within all groups. Appendix D also shows that, for data from the second half of the night, only REM percentage was normally distributed within all groups. Therefore, overall only 3 of the 12 sleep variables upheld the assumption of normality.

Analyses of the homogeneity of variance for all sleep variables between groups, using Levene's test, showed that only WASO for the first half of the night violated this assumption (see Appendix D).

Although the majority of the sleep variables violate the assumptions underlying inferential statistical analysis, linear contrasts are a type of ANOVA, and ANOVA is robust to violations of the assumptions of normality (Donaldson, 1968) and homogeneity (Field, 2009) provided sample sizes are equal. Here our sample sizes differend by only 1 participant per group, which is not enough to affect the robustness of the analysis. Furthermore, linear contrast analysis is suited to our data due to the small sample size and the fact that we had specific predictions. Therefore, we proceeded with the analysis as planned.

Table 2 shows the results of linear contrast analyses testing the prediction that, for each sleep outcome variable, this order of means would hold: PTSD < trauma-exposed = depression < healthy controls. The analyses did not detect a statistically significant trend toward this linear pattern of data for any sleep outcome variable, regardless of the half of the night during which it was measured.

Of potential importance for subsequent analyses, however, is that there were variables for which the data showed a trend toward confirming our predicted pattern. Inspection of the group means suggested that second half of the night REM percentage, WASO, and sleep efficiency followed the predicted pattern. The linear contrast analyses for these variables produced relatively smaller p values and relatively larger effect sizes when compared to other sleep outcome variables for the second half of the night.

Table 2

Sleep Outcome Variables: Descriptive statistics and results of linear contrast analyses (N = 40)

Variable	Group								<i>F</i>	<i>p</i>	ESE	
	PTSD (<i>n</i> = 11)		Trauma Exposed (<i>n</i> = 9)		Depression (<i>n</i> = 11)		Healthy Control (<i>n</i> = 9)					
REM %												
First half of the night	11.10	(7.05)	10.02	(5.32)	10.30	(5.70)	8.32	(4.61)	0.78	.38	.15	
Second half of the	21.91	(7.48)	25.81	(6.32)	24.15	(8.29)	26.42	(6.98)	2.02	.16	.23	
SWS %												
First half of the night	22.09	(8.63)	17.23	(11.73)	16.86	(8.35)	27.42	(8.86)	<0.01	.97	.01	
Second half of the	5.15	(6.40)	3.84	(7.54)	5.62	(5.47)	9.57	(6.54)	0.73	.40	.14	
WASO												
First half of the night	8.36	(6.61)	7.39	(7.33)	14.59	(16.44)	17.50	(20.30)	1.38	.25	.19	
Second half of the	31.31	(27.18)	25.67	(28.31)	21.18	(17.20)	18.78	(18.66)	1.46	.23	.20	
Sleep efficiency												
First half of the night	90.21	(6.19)	87.12	(12.31)	81.83	(9.73)	83.87	(8.44)	3.21	.08	.29	
Second half of the	86.75	(11.34)	89.42	(11.59)	91.14	(7.07)	91.16	(8.48)	1.26	.27	.18	
Spontaneous arousals												
First half of the night	15.45	(7.89)	20.56	(7.62)	16.18	(3.79)	19.22	(3.90)	2.26	.14	.24	
Second half of the	23.09	(5.92)	25.67	(9.23)	21.10	(10.39)	21.22	(10.20)	0.06	.81	.04	
Number of awakenings												
First half of the night	2.54	(1.57)	1.78	(0.97)	2.36	(1.50)	2.56	(1.94)	0.17	.68	.07	
Second half of the	2.27	(1.68)	2.22	(2.17)	1.82	(1.17)	2.00	(1.12)	0.21	.65	.08	

Note. Means are presented, with standard deviations in parenthesis. ESE = effect size estimate (in this case, *r*).

WASO = wake after sleep onset.

Hypothesis 2: Between-group comparisons – AMT performance

We began the analysis by testing the assumptions of normality and homogeneity for all AMT outcome variables: specificity, word count, emotionality (the number of emotional words used in the response), and emotionality proportion (the number of emotional words used in relation to the total number of words used) for each of the positive, negative and neutral cue word groups.

Appendix E shows results from Kolmogorov-Smirnov goodness-of-fit tests for normality of distribution. As before, a statistically significant p -value indicates that one can reject the null hypothesis that the data are normally distributed. Only 4 of the 12 AMT outcome variables upheld the assumption of normal distribution.

An analysis of the homogeneity of variance between groups, using Levene's test, showed that word count for positive and negative cues and emotionality proportion for positive and neutral cues, violated this assumption (see Appendix E).

Therefore only four AMT variables upheld both the normality and the homogeneity of variance assumptions. As stated with regards to Hypothesis 1, linear contrast analyses are suited to our data and they are robust to violations of assumptions under conditions of (relatively) equal sample sizes. Therefore, we again proceeded with the analysis.

Table 3 shows the results of the series of linear contrast analyses focused on AMT performance. The analyses detected statistically significant between-group differences for seven of the variables, three of which were in the predicted direct (PTSD < TE = DEP < HC).

Table 3

AMT Outcome Variables: Descriptive statistics and results of linear contrast analyses (N = 40)

Variable	Group								<i>F</i>	<i>p</i>	ESE
	PTSD (<i>n</i> = 11)		Trauma Exposed (<i>n</i> = 9)		Depression (<i>n</i> = 11)		Healthy Control (<i>n</i> = 9)				
Specificity											
Positive ^a	3.09	(1.04)	2.00	(1.50)	3.45	(0.82)	3.78	(0.83)	0.17	.68	.07
Negative ^a	2.73	(1.56)	2.33	(1.32)	3.27	(1.62)	3.44	(1.33)	0.55	.46	.12
Neutral ^a	3.18	(1.25)	2.78	(1.79)	3.18	(1.33)	2.89	(1.96)	0.19	.67	.07
Word Count											
Positive ^b	19.51	(14.08)	31.02	(26.12)	34.16	(17.92)	52.47	(38.49)	6.46	.02*	.39
Negative ^b	22.73	(10.84)	41.00	(27.14)	52.04	(37.19)	59.07	(40.14)	7.35	.01*	.41
Neutral ^b	24.87	(14.36)	50.53	(42.86)	51.25	(34.59)	53.02	(36.05)	5.14	.03*	.35
Emotionality											
Positive ^c	9.94	(3.38)	8.97	(2.73)	7.31	(2.35)	7.08	(2.02)	5.70	.02*	.37
Negative ^c	8.87	(2.73)	7.89	(1.68)	6.84	(1.79)	5.88	(1.27)	9.81	.003**	.46
Neutral ^c	3.14	(1.12)	4.53	(1.70)	2.92	(1.49)	3.70	(1.14)	1.31	.26	.19
Emotionality Proportion											
Positive ^d	.141	(.083)	.094	(.065)	.060	(.049)	.040	(.023)	13.78	< .001***	.53
Negative ^d	.100	(.071)	.065	(.055)	.061	(.087)	.029	(.018)	5.17	.03*	.35
Neutral ^d	.033	(.025)	.046	(.056)	.013	(.009)	.019	(.011)	0.49	.49	.12

Note. For each variable within each group, means are presented with standard deviations in parentheses. ESE = effect size estimate (in this case, *r*).

^aMinimum possible score = 0. Maximum possible score = 5.

^bAverage word count calculated by averaging the word counts for the 5 words of similar valence.

^cThe number of emotional words used in the response to the 5 cue words of similar valence.

^dThe proportion of emotional words used relative to the total number of words used per valence category.

p* < .05, *p* < .01, ****p* < .001.

Hypothesis 3: Associations between sleep and AMT performance in PTSD

General linear models. Although previous analyses detected no significant linear trend toward the predicted between-group differences for sleep outcome variables, we nonetheless proceeded to build general linear models examining the association between sleep quality and AMT performance. We made this decision because three of the sleep variables, WASO, REM percentage and sleep efficiency, showed a trend in their means in the predicted direction, and each of the linear contrast analyses for each of these three variables was associated with an effect size approaching the medium range. Furthermore, these variables all represented sleep from the second half of the night, which is where we would have expected, based on previous literature, the greatest between-group differences in sleep patterns to be.

We built a separate general linear model for each of the seven AMT variables for which the linear contrast analyses showed significant trends. As predictors we entered *group status*, the sleep variables that held the predicted directional pattern (i.e., *WASO*, *REM percentage*, and *sleep efficiency*, all from the second half of the night), as well as the interactions between these sleep variables and group status. For each model we proceeded iteratively, removing variables that contributed the least to the model and working toward a statistically significant model that explained the greatest amount of variance in the AMT outcome variable.

The results of our general linear modelling are as follows. First, for two variables (word count in response to neutral cues, and emotionality in response to positive cues) the set of predictors did not generate a statistically significant model.

Second, for two variables (word count and emotionality proportion, both in response to positive cues) group status was the only significant predictor. For word count in response to positive cues, the model explained 12.8% of variance, $F(3, 36) = 2.91, p = .048$. For emotionality proportion in response to positive cues, the model explained 25.7% of the variance, $F(3, 36) = 5.49, p = .003$.

Third, for three variables (word count, emotionality, and emotionality proportion, all in response to negative cues), the main effects of group status and WASO were significant predictors. For word count in response to negative cues, group status ($p = .027$) and WASO ($p = .117$) combined to account for 15.7% of the variance, $F(3, 35) = 2.811, p = .04$. For emotionality in response to negative cues, group status ($p = .003$) and WASO ($p = .056$) combined to account for 26% of the variance, $F(3, 35) = 4.425, p = .005$. We note that WASO reached trend-level significance as a predictor for this variable. Finally, for

emotionality proportion in response to negative cues, group status ($p = .052$) and WASO ($p = .047$) combined to account for 14.8% of the variance, $F(3, 35) = 2.693$, $p = .047$. Here, WASO featured as a significant predictor while group status reached trend-level significance.

DISCUSSION

The study set out to investigate whether poor sleep quality in PTSD-diagnosed individuals is a mechanism explaining deficits in autobiographical memory. Our interest in this question was based on the knowledge that (a) sleep plays a beneficial role on memory consolidation in healthy individuals, (b) PTSD-diagnosed individuals have disrupted sleep architecture, and (c) PTSD-diagnosed individuals tend to present with AM impairments. We recruited a sample of 40 adult women, assigned each to one of four groups (PTSD, trauma-exposed, depression, and healthy control), and then proceeded to investigate the questions of interest by testing three hypotheses. First, we predicted that PTSD-diagnosed individuals would have poorer objective sleep quality than trauma-exposed, depressed, and healthy participants. Second, we predicted that PTSD-diagnosed individuals would have poorer Autobiographical Memory Test (AMT; Williams & Broadbent, 1986) performance than trauma-exposed, depressed, and healthy participants. Previous studies have shown that PTSD-diagnosed individuals retrieve less specific memories on the AMT (Brown et al., 2013). This is, however, a relatively crude measure of the quality of AM, and therefore we aimed to look at these memories in a more nuanced fashion by examining emotionality as well as specificity. Our third hypothesis was that poorer sleep quality in PTSD would hold predictive value for poorer performance on the AMT.

Hypothesis 1: Findings regarding sleep data

The statistical analysis for sleep-related variables did not confirm hypothesis 1. That is to say, the data did not conform to the predicted pattern which held that PTSD-diagnosed individuals would have the worst sleep outcomes, healthy controls would have the best sleep outcomes, and trauma-exposed and depressed individuals would fall in the middle.

Although there were no statistically significant between-group differences for sleep outcome variables, there were three variables from the second half of the night whose means fit the predicted linear pattern. On these variables (REM percentage, wake after sleep onset, and sleep efficiency), PTSD participants displayed poorer sleep outcomes, followed by trauma-exposed, depression, and then healthy controls. Not only do these variables follow the predicted trend, they also have smaller *p* values and larger effect sizes in comparison to the other sleep variables (REM%, $r = .23$, WASO, $r = .20$ and sleep efficiency, $r = .18$) in the second half of the night. These effect sizes may indicate that a larger sample size would help attain statistical significance as the sample of 40 may not have been large enough to detect a significant difference. A power analysis, with power set at .80 and 4 groups, revealed that a

total sample size of 212,280 and 344 respectively would be needed for these three variables to reach statistical significance.

Specific between-group differences. This study also set out to attempt to distinguish between, in relation to sleep disruptions, trauma exposure per se and trauma exposure with the subsequent development of PTSD. Literature often examines PTSD participants in comparison to healthy controls without taking into account the experience of trauma as a separate factor. Even though some results show trends in our predicted direction unfortunately, as previously stated, there were no statistically significant between-group differences so conclusions cannot be drawn regarding this question.

A similar problem occurs regarding depression as a confound for sleep disruption in PTSD-diagnosed individuals, as depression is associated with its own sleep alterations, yet is often comorbid to PTSD. Our data cannot, however untangle the relative contributions of PTSD and depression to sleep disruption.

Summarizing sleep findings. The study has shown a trend towards worse sleep in PTSD-diagnosed individuals in the second half of the night, particularly for sleep efficiency, time spent awake after sleep onset and REM percentage. However, this trend shows relatively small raw differences between PTSD-diagnosed individuals and individuals in the three control groups, with analyses detecting no between-group differences.

Hypothesis 2: Findings regarding AMT performance

We assessed autobiographical memory using the AMT, which was administered to all participants after each had completed one sleep night in the laboratory. We hypothesised that PTSD-diagnosed individuals would have less specific and less emotional autobiographical memories than trauma-exposed and depressed participants, who in turn would have worse quality memories than healthy controls. The results of the study partially support this hypothesis with statistically significant differences in 7 of the 12 variables measured, three of which were in the predicted direction.

Previous literature regarding the AMT responses of PTSD-diagnosed individuals presents conflicting accounts in terms of the specificity of the memories. Williams et al. (2007) reported that their sample of PTSD participants produced less specific memories to both positively and negatively valenced cues. Williams et al., (2006) reported a similar effect, but only for negative cues, and Kleim et al., (2014) reported the effect was present but only for positive cues.

This study was not able to provide clarity regarding these contradictory findings. Between-group comparisons regarding specificity towards all valenced cues did not reach statistical significance, and the associated effect sizes were negligible (positive cues, $r = .01$, negative cues, $r = .12$, and neutral cues, $r = .07$). This lack of significant effect may again be attributed to small sample size.

In terms of word count, PTSD participants' autobiographical memories consisted of significantly fewer words than the other groups. This relationship was in the predicted direction (PTSD < TE = DEP < HC). The between-group difference was biggest in terms of negative cue words, although both positive and neutral cues also produced medium effect sizes. Therefore, although between-group comparisons of specificity was not significant, the word count analysis shows that there is a distinct difference in the quality of the memories produced by PTSD participants.

For a more in-depth analysis of the quality of the autobiographical memories, we analysed their emotionality. The literature states that patients with PTSD tend to avoid emotions, both broadly and about the trauma they experienced specifically (Tull et al., 2004). However, research specific to the emotional content of autobiographical memories has not been conducted. Contrary to our prediction, emotionality results show that there is a significant between-group difference for positive and negative cue words, in the opposite direction to that predicted (PTSD > TE = DEP > HC). These results indicated that PTSD participants produced significantly *more* emotional words in response to both positive and negative cues. These between-group differences were associated with medium effect sizes. Even when this measure was taken further to take into account the number of emotional words relative to the total number of words used, the pattern remained. This emotionality proportion analysis strengthened the effect size to a large effect size for positive cues ($r = .53$) whereas negative cues remained medium ($r = .35$).

These results suggest that emotionally valenced memories, specifically, are enhanced by the PTSD diagnosis. These findings stand in contrast to what might be predicted given previously published studies in the field. On the basis of those studies, one might predict, as we did, that PTSD-diagnosed individuals would be less emotional in their memories due to a tendency to avoid emotions (Frewen et al., 2012). Instead, we found their memories to be the most emotionally-laden in comparison to the other groups. The contrast between the prediction and the observed data might, be due to the fact that sleep disruptions have been associated with more emotional responses (Zohar, Tzischinsky, Epstein, & Lavie, 2005), a finding we discuss in greater detail below.

Specific between-group differences. Our results show that both depression and trauma may account for a portion of disrupted memory performance in PTSD participants, but the PTSD-diagnosis, over and above trauma and depression, is associated with AM deficits.

Summarizing memory findings. In summary, our results did not support the hypothesis regarding autobiographical memory specificity in itself. They did, however show significant between group differences in the length and emotionality of memories. In the case of length of memories our prediction was confirmed that PTSD-diagnosed individuals had shorter memories. However, regarding the emotionality of memories our hypothesis was disproved with PTSD-diagnosed individuals having more emotional AMT responses.

Hypothesis 3: Associations between sleep and AMT performance

We proceeded to assess whether three of the sleep variables in the second half of the night, together with *group status*, could predict the significant differences in the AMT outcome variables. General linear models were built to investigate this relationship.

We found that for word count in response to neutral cues and emotionality in response to positive cues there was no significant predictive model. For word count in response to positive cues and emotionality proportion for positive cues, *group* was the only significant predictor in the models. And lastly, for word count, emotionality and emotionality proportion all in response to negative cues, *group* and WASO were significant predictors of the outcome.

Of note here is that the only sleep variable to feature as a predictor, WASO, was only predictive of negative cue but not positive cue outcomes. A number of studies have indicated that sleep disruption causes individuals to be more emotionally reactive when shown negative rather than neutral stimuli (Yoo, Gujar, Hu, Jolesz, & Walker, 2007; Vandekerckhove & Cluydts, 2010). This is due to the greater activation of amygdala under conditions of sleep deprivation. This brain structure is responsible for processing emotions such as fear, anger and pleasure (Sotres-Bayon, Bush, & LeDoux, 2004). In fact the amygdala is known to be over reactive in PTSD-diagnosed individuals in comparison with controls (Shin, Rauch, & Pitman, 2006). Our findings support the results of this study as we found that increases in WASO (i.e. sleep disruption) predicted more emotionality for negative information in PTSD diagnosed individuals in comparison with control individuals. Speculatively sleep disruption in PTSD-diagnosed individuals may contribute to increased amygdala activation in this population with the observed effect of an increase in emotionality

for negative cues. A factor other than sleep may be influencing the observed difference for positive cues as only group status predicted that outcome variable.

Strengths and Limitations

The study had many methodological strengths, especially in the exclusion criteria to attempt to control for confounding variables related to sleep and memory. For example the exclusion of certain age ranges, those with a history of alcohol abuse, those with other psychiatric diagnoses or on psychoactive medication, help the study eliminate these as explanations for the data. The study also used female rape survivors which is a new source of data as most other studies have used male war veteran populations. Additionally, time since trauma was controlled for, furthering the methodological strength of the study.

The study also addressed a relatively sparse area of research, namely, sleep quality, memory and their interaction. Memory and sleep deficits can have detrimental effects on daily living therefore furthering an understanding of them is worthwhile. Lastly, no other studies have addressed the quality of memory content in response to the AMT which we addressed in this study making our contribution unique.

There were, however, limitations of the current study, the biggest of which is the small sample size. Due to the trends found in the data we suggest that a larger sample size may be needed to detect significant effects, also in order to detect significant interactions that require additional statistical power.

Another limitation is the presence of depression in all groups except the control. Ideally a study would have PTSD and trauma-exposed participants without depression, however this is relatively unrealistic to attain.

Directions for Future Research

We suggest that future research attempts to clarify the relationships described above with a larger sample size. The data also suggested that sleep data be collected over more than one night in order to account for the variability in any one night of sleep in all individuals.

Future studies could also analyse the AMT responses in accordance with the five coding categories (*specific, categoric, extended, semantic association, and omission*) rather than just as specific or non-specific for analyses.

The emotionality findings could be assessed as a linear prediction (PTSD < TE < DEP < HC) as opposed to having DEP and TE in the middle, as it appears that TE perform more poorly on all of the significant outcome variables.

CONCLUSION

In conclusion, the current study did not show support for significant between-group differences in sleep variables, however some trends were observed from the second half of the night in the predicted direction. We have shown support for the hypothesis that autobiographical memories are disrupted in the PTSD population, with PTSD participants recalling significantly shorter memories. Surprisingly, however, the study highlighted new information finding that these shorter memories in PTSD-diagnosed individuals hold more emotional content than for control individuals. We further found that the time spent awake in the second half of the night predicted responses to negative cues in the AMT, suggesting that PTSD-diagnosed individuals that woke for longer periods in the second half of the night had more emotional memory responses to negative cues.

REFERENCES

- AASM (2007). The AASM manual for the scoring of sleep and associated events. In 2007 (Ed.): American Academy of Sleep Medicine
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th edition., text revision). Washington, DC: American Psychiatric Association
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental Disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing
- Araújo, A. X., Berger, W., Coutinho, E. S., Marques-Portella, C., Luz, M. P., Cabizuca, M., . . . Mendlowicz, M. V. (2014). Comorbid depressive symptoms in treatment-seeking PTSD outpatients affect multiple domains of quality of life. *Comprehensive psychiatry*, *55*(1), 56-63
- Atienza, M., & Cantero, J. L. (2008). Modulatory effects of emotion and sleep on recollection and familiarity. *Journal of Sleep Research*, *17*, 285-294
- Babson, K. A., & Feldner, M. T. (2010). Temporal relations between sleep problems and both traumatic event exposure and PTSD: A critical review of the empirical literature. *Journal of Anxiety Disorders*, *24*(1), 1-15
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Manual for the beck depression inventory The Psychological Corporation. *San Antonio, TX*
- Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., & Keane, T. M. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress*, *8*(1), 75-90
- Boden, M. T., Westermann, S., Mcrae, K., Kuo, J., Alvarez, J., Kulkarni, M. R., . . . Bonn-Miller, M. O. (2013). Emotion regulation and Posttraumatic Stress Disorder: A prospective investigation. *Journal of Social and Clinical Psychology*, *32*(3), 296-314

- Breslau, N., Roth, T., Burduvali, E., Kapke, A., Schultz, L., & Roehrs, T. (2004). Sleep in Lifetime Posttraumatic Stress Disorder: A Community-Based Polysomnographic Study. *Archives of General Psychiatry*, *61*, 508-516
- Brewin, C. R. (1998). Intrusive autobiographical memories in depression and post-traumatic stress disorder. *Applied Cognitive Psychology*, *12*(4), 359-370
- Brewin, C. R., Reynolds, M., & Tata, P. (1999). Autobiographical memory processes and the course of depression. *Journal of Abnormal Psychology*, *108*(3), 511-517
- Brewin, C. R. (2011). The Nature and Significance of Memory Disturbance in Posttraumatic Stress Disorder. *Annual Review of Clinical Psychology*, *7*, 203-227
- Brown, A. D., Root, J. C., Romano, T. A., Chang, L. J., Bryant, R. A., & Hirst, W. (2013). Overgeneralized autobiographical memory and future thinking in combat veterans with posttraumatic stress disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *44*(1), 129-134
- Buckley, T. C., Blanchard, E. B., & Neill, W. T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review*, *20*(8), 1041-1065
- Chokroverty, S. (2009a). An overview of normal sleep. In S. Chokroverty (Ed.), *Sleep disorders medicine* (pp. 5-23). Philadelphia, PA Saunders Elsevier
- Conway, M. A., & Pleydell-Pearce, C.W. (2000). The construction of autobiographical memories in the self memory system. *Psychological Review*, *107*, 261-288
- Corporation, T. P. (1999). *Wechsler Abbreviated Scale of Intelligence*. San Antonio, Texas: Harcourt Brace & Company
- De Bellis, M. D., Hooper, S. R., & Sapia, J. L. (2005). Early trauma exposure and the brain. In J. J. Vasterling & C. R. Brewin (Eds.), *Neuropsychology of PTSD* (pp. 153-177). New York, NY: The Guilford Press

Diekelmann, S., & Born, J. (2010). The memory function of sleep. *Nature Reviews Neuroscience*, *11*, 114-126

Donaldson, T. S. (1968). Robustness of the F-test to errors of both kinds and the correlation between the numerator and denominator of the F-ratio. *Journal of the American Statistical Association*, 660-676

Ellenbogen, J. M., Hulbert, J. C., Stickgold, R., Dinges, D. F., & Thompson-Schill, S. L. (2006). Interfering with Theories of Sleep and Memory: Sleep, Declarative Memory, and Associative Interference. *Current Biology*, *16*, 1290-1294

Ellenbogen, J. M., Payne, J. D., & Stickgold, R. (2006). The role of sleep in declarative memory consolidation: passive, permissive, active or none? *Current Opinion in Neurobiology*, *16*, 716-722

Engdahl, B. E., Eberly, R. E., Hurwitz, T. D., Mahowald, M. W., & Blake, J. (2000). Sleep in a community sample of elderly war veterans with and without posttraumatic stress disorder. *Biological Psychiatry*, *47*, 520-525

Ferrett, H. L., Carey, P. D., Thomas, K. G., Tapert, S. F., & Fein, G. (2010). Neuropsychological performance of South African treatment-naïve adolescents with alcohol dependence. *Drug and Alcohol Dependence*, *110*, 8-14

Field, A. (2009). *Discovering statistics using SPSS*. Sage publications

Fogel, S. M., & Smith, C. T. (2011). The function of the sleep spindle: A physiological index of intelligence and a mechanism for sleep-dependent memory consolidation. *Neuroscience and Biobehavioral Reviews*, *35*, 1154-1165

Frewen, P. A., Dozois, D. J., Neufeld, R. W., & Lanius, R. A. (2012). Disturbances of Emotional Awareness and Expression in Posttraumatic Stress Disorder: Meta-Mood, Emotion Regulation, Mindfulness, and Interference of Emotional Expressiveness. *Psychological Trauma: Theory, Research, Practice, and Policy*, *4*(2), 152

- Gilbertson, M.W., Gurvits, T.V., Lasko, N.B. Orr, S.P., & Pitman, R.K. (2001). Multivariate assessment of explicit memory function in combat veterans with posttraumatic stress disorder. *Trauma stress, 14*, 413-420
- Goerke, M., Cohrs, S., Rodenbeck, A., Grittner, U., Sommer, W., & Kunz, D. (2013). Declarative memory consolidation during the first night in a sleep lab: The role of REM sleep and cortisol. *Psychoneuroendocrinology, 38*, 1102-1111
- Guillemin, G. J., Kerr, S. J., & Brew, B. J. (2005). Involvement of quinolinic acid in AIDS dementia complex. *Neurotoxicity Research, 7*, 103-123
- Hurwitz, T. D., Mahowald, M. W., Kuskowski, M., & Engdahl, B. E. (1998). Polysomnographic sleep is not clinically impaired in vietnam combat veterans with chronic posttraumatic stress disorder. *Biological Psychiatry, 44*, 1066-1073
- Joska, J. A., Westgarth-Taylor, J., Myer, L., Hoare, J., Thomas, K. G., Combrinck, M., . . . Flisher, A. J. (2011). Characterization of HIV-Associated Neurocognitive Disorders Among Individuals Starting Antiretroviral Therapy in South Africa. *AIDS and Behavior, 15*(6), 1197-1203
- Julian, T., & Breen, W. E. (2010). Everyday Strivings in War Veterans With Posttraumatic Stress Disorder: Suffering From a Hyper-Focus on Avoidance and Emotion Regulation. *Behavior Therapy, 41*(3), 350-363
- Kagee, A., & Martin, L. (2010). Symptoms of depression and anxiety among a sample of South African patients living with HIV. *AIDS Care, 22*(2), 159-165
- Kales, A., Kales, J. D., Sly, R. M., Scharf, M. B., Tan, T. L., & Preston, T. A. (1970). Sleep patterns of asthmatic children: all-night electroencephalographic studies. *Journal of Allergy, 46*(5), 300-308
- Kleim, B., & Ehlers, A. (2008). Reduced autobiographical memory specificity predicts depression and posttraumatic stress disorder after recent trauma. *Journal of Consulting and Clinical Psychology, 76*(2), 231-242

- Kleim, B., Graham, B., Fihosy, S., Stott, R., & Ehlers, A. (2014). Reduced Specificity in Episodic Future Thinking in Posttraumatic Stress Disorder. *Clinical Psychological Science, 2*, 165-173
- Kobayashi, I., Boarts, J. M., & Delahanty, D. L. (2007). Polysomnographically measured sleep abnormalities in PTSD: A meta-analytic review. *Psychophysiology, 44*, 660-669
- Lipnska, M., Timol, R., Kaminer, D., & Thomas, K. G. (2014). Disrupted rapid eye movement sleep predicts poor declarative memory performance in post-traumatic stress disorder. *Journal of sleep research, 23*, 311-319
- Löwe, B., Kroenke, K., Spitzer, R. L., Williams, J. B., Mussell, M., Rose, M., . . . Spitzer, C. (2011). Trauma Exposure and Posttraumatic Stress Disorder in Primary Care Patients: Cross-Sectional Criterion Standard Study. *The Journal of clinical psychiatry, 72*, 304-312
- Marshall, L., & Born, J. (2007). The contribution of sleep to hippocampus-dependent memory consolidation. *Trends in Cognitive Sciences, 11*, 442-450
- McEwen, B. S. (1999). Stress and the aging hippocampus. *Front Neuroendocrinol, 20*(1), 49-70
- McGaugh, J. L. (2013). Making lasting memories: Remembering the significant. *Neuroscience, 110*, 10402-10407
- McNally, R. J., Lasko, N. B., Macklin, M. L., & Pitman, R. K. (1995). Autobiographical memory disturbance in combat-related posttraumatic stress disorder. *Behaviour Research and Therapy, 33*(6), 619-630
- Mellman, T. A., Bustamante, V., Fins, A. I., Pigeon, W. R., & Nolan, B. (2002). REM Sleep and the Early Development of Posttraumatic Stress Disorder. *American Journal of Psychiatry, 159*, 1696-1701
- Mellman, T. A., Nolan, B., Hebding, J., Kulick-Bell, R., & Dominguez, R. A. (1997). Polysomnographic comparison of veterans with combat-related PTSD, depressed men, and non-ill controls. *Sleep, 20*, 46-51

- Moradi, A., Abdi, A., Fathi-Ashtiani, A., Dalgleish, T., & Jobson, L. (2012). Overgeneral autobiographical memory recollection in Iranian combat veterans with posttraumatic stress disorder. *Behaviour Research and Therapy*, *50*(6), 435-441
- Moradi, A. R., Herlihy, J., Yasseri, G., Shahraray, M., Turner, S., & Dalgleish, T. (2008). Specificity of episodic and semantic aspects of autobiographical memory in relation to symptoms of posttraumatic stress disorder (PTSD). *Acta Psychologica*, *127*(3), 645-653
- Myers, J. E., Nell, V., Taylor, T., & Thompson, M. L. (1997). An Investigation into Neurologic and Neurobehavioral Effects of Long-Term Agrichemical Use among Deciduous Fruit Farm Workers in the Western Cape, South Africa. *Environmental Research*, *73*, 132-145
- Nel, A., & Kagee, A. (2013). The relationship between depression, anxiety and medication adherence among patients receiving antiretroviral treatment in South Africa. *AIDS Care*, *25*, 948-955
- Pennebaker, J. W., Chung, C. K., Ireland, M., Gonzales, A., & Booth, R. J. (2007). The Development and Psychometric Properties of LIWC2007. *Austin, TX, LIWC. Net*
- Raes, F., Hermans, D., de Decker, A., Eelen, P., & Williams, J. M. G. (2003). Autobiographical memory specificity and affect regulation: an experimental approach. *Emotion*, *3*(2), 201-206
- Rechtschaffen, A., & Kales, A. (1968). A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. National Institutes of Health. *Washington, DC*
- Rosenthal, R., Rosnow, R. L., & Rubin, D. B. (2000). *Contrasts and effect sizes in behavioral research: A correlational approach*. Cambridge, U.K: Cambridge University Press
- Roth, T., Singh, N., Steinberg, F., Waldron, A., & Moline, M. (2013). Sleep architecture following treatment of patients with middle-of-the-night insomnia with buffered sublingual zolpidem compared to placebo. *Sleep medicine*, *14*, e211-e212

- Selzer, M. L. (1971). The Michigan Alcoholism Screening Test: The quest for a new diagnostic instrument. *American Journal of Psychiatry*, *127*(12), 1653-1658
- Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., ... & Dunbar, G. C. (1998). The Mini-International Neuropsychiatric Interview (MINI): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, *59*, 22-33
- Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, Medial Prefrontal Cortex, and Hippocampal Function in PTSD. *Annals of The New York Academy of Sciences*, *1071*(1), 67-79
- Sotres-Bayon, F., Bush, D. E., & LeDoux, J. E. (2004). Emotional Perseveration: An Update on Prefrontal-Amygdala Interactions in Fear Extinction. *Learning & Memory*, *11*, 525-535
- Spoormaker, V. I., & Montgomery, P. (2008). Disturbed sleep in post-traumatic stress disorder: Secondary symptom or core feature? *Sleep Medicine Reviews*, *12*, 169-184
- Srinivasan, V., Pandi-Perumal, S. R., Trakht, I., Spence, D. W., Hardeland, R., Poeggeler, B., & Cardinali, D. P. (2009). Pathophysiology of depression: role of sleep and the melatonergic system. *Psychiatry Research*, *165*, 201-214
- Stein, D. J., Rothbaum, B. O., Baldwin, D. S., Szumski, A., Pedersen, R., & Davidson, J. R. (2013). A factor analysis of posttraumatic stress disorder symptoms using data pooled from two venlafaxine extended-release clinical trials. *Brain and behavior*, *3*(6), 738-746
- Sutherland, K., & Bryant, R. A. (2007). Rumination and overgeneral autobiographical memory. *Behaviour Research and Therapy*, *45*(10), 2407-2416
- Tausczik, Y. R., & Pennebaker, J. W. (2010). The Psychological Meaning of Words: LIWC and Computerized Text Analysis Methods. *Journal of Language and Social Psychology*, *29*(1), 24-54

- Tull, M. T., Gratz, K. L., Salters, K., & Roemer, L. (2004). The Role of Experiential Avoidance in Posttraumatic Stress Symptoms and Symptoms of Depression, Anxiety, and Somatization. *Journal of Nervous and Mental Disease, 192*, 754-761
- Tulving, E. (1972). Episodic and semantic memory 1. In *Organization of memory*. New York: Academic Press
- Tural, Ü., Önder, E., & Aker, T. (2012). Effect of Depression on Recovery from PTSD. *Community Mental Health Journal, 48*, 161-166
- Van Vreeswijk, M. F., & Wilde, E. J. (2004). Autobiographical memory specificity, psychopathology, depressed mood and the use of the Autobiographical Memory Test: a meta-analysis. *Behaviour Research and Therapy, 42*, 731-743
- Vandekerckhove, M., & Cluydts, R. (2010). The emotional brain and sleep: An intimate relationship. *Sleep Medicine Reviews, 14*(4), 219-226
- Vermetten, E., Vythilingam, M., Southwick, S. M., Charney, D. S., & Bremner, J. D. (2003). Long-term treatment with paroxetine increases verbal declarative memory and hippocampal volume in posttraumatic stress disorder. *Biological Psychiatry, 54*(7), 693-702
- Wagner, U., Gais, S., & Born, J. (2001). Emotional memory formation is enhanced across sleep intervals with high amounts of rapid eye movement sleep. *Learning and Memory, 8*, 112-119
- Watson, L. A., Berntsen, D., Kuyken, W., & Watkins, E. R. (2013). Involuntary and voluntary autobiographical memory specificity as a function of depression. *Journal of Behavior Therapy and Experimental Psychiatry, 44*, 7-13
- Wechsler, D. (1999). WASI: Wechsler abbreviated scale of intelligence. San Antonio, TX: Psychological Corporation
- Williams, J. M., & Broadbent, K. (1986). Autobiographical memory in suicide attempters. *Journal of Abnormal Psychology, 95*(2), 144-149

- Williams, J. M. G., Barnhofer, T., Crane, C., Herman, D., Raes, F., Watkins, E., & Dalgleish, T. (2007). Autobiographical memory specificity and emotional disorder. *Psychological Bulletin*, *133*(1), 122-148
- Williams, J. M. G., Eelen, P., Raes, F., & Hermans, D. (2006). Reduced autobiographical memory specificity and affect regulation. *Cognition & Emotion*, *20*(3-4), 402-429
- Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep — a prefrontal amygdala disconnect. *Current Biology*, *17*(20), R877-R878
- Zohar, D., Tzischinsky, O., Epstein, R., & Lavie, P. (2005). The effects of sleep loss on medical residents' emotional reactions to work events: a cognitiveenergy model. *Sleep*, *28*, 47-54

Appendix A

CAPS Scoring Method

There are nine different ways of scoring the CAPS, all with good to excellent reliability (Weathers, Keane, & Davidson, 2001). We used the *Frequency ≥ 1 / Intensity ≥ 2 / Total Severity ≥ 65 (F1/I2/TSEV65)* method. The first rule (F1/I2) states that a symptom is present if the frequency with which it occurs is scored as 1 or higher and the intensity is scored as 2 or higher. Using this scoring rule, for an individual to be diagnosed with PTSD, DSM-IV criteria must be met in terms of the correct distribution of symptoms across clusters. This rule is considered lenient (Weathers et al., 2001). The second rule (TSEV65) takes a total score of at least 65 as the basis for a valid diagnosis of PTSD and was derived as the optimal score for a PTSD diagnosis based on the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1997). Together, these rules specify that a CAPS score of at least 65 must be reached for a diagnosis of PTSD to be made, and that there must be the appropriate distribution of symptoms across clusters.

Appendix B
Autobiographical Memory Test Scoring Form

1. Happy			
<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

2. Failure			
<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

3. Rhythm

<h3>3. Rhythm</h3>			
<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

4. Relieved

<h3>4. Relieved</h3>			
<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

5. Guilty

5. Guilty			
<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

6. Shoes

6. Shoes			
<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

7. Joy

<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

8. Helpless

<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

9. Tree

<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

10. Devoted

<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

11. Rejected

<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

12. Uncle

<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

13. Tender

13. Tender			
<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

14. Sad

14. Sad			
<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

15. Library

<input type="checkbox"/>	<input type="checkbox"/>		<input type="checkbox"/>
<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response	<input type="checkbox"/> specific <input type="checkbox"/> extended <input type="checkbox"/> categoric <input type="checkbox"/> sem ass <input type="checkbox"/> no response		
Prompt for clarification <input type="checkbox"/>			

Appendix C Informed Consent Document

Informed Consent to Participate in Research and Authorization for Collection, Use, and Disclosure of Sleep Patterns, Performance on Memory tasks and Other Personal Data

You are being asked to take part in a research study. This form provides you with information about the study and seeks your authorization for the collection, use and disclosure of your sleep architecture patterns, cognitive performance data, autonomic arousal data and urine samples as well as other information necessary for the study. The Principal Investigators (the people in charge of this research) or a representative of the Principal Investigator will also describe this study to you and answer all of your questions. Your participation is entirely voluntary. Before you decide whether or not to take part, read the information below and ask questions about anything you do not understand. For your information – this study is covered by UCT's No Fault Insurance Policy.

1. Name of Participant

2. Title of Research Study

Overgeneral autobiographical memory in post-traumatic stress disorder: Associations with disrupted sleep architecture

3. Principal Investigators and Telephone Numbers

Hollie Bradley and Arthur Chen

University of Cape Town (UCT)

Contact number: 0787016746 and 0824636416

4. What is the purpose of this research study?

This research aims to investigate the whether PTSD diagnosed individuals report less specific and less emotionally laden autobiographical memories than controls and if autobiographical memory is related to their sleeping patterns.

5. What will be done if you take part in this research study?

In this experiment, you will be called in for 2 study sessions both spanning a whole night.

Before commencing the actual study, you will undergo a screening process whereby the Principal Investigators listed in # 3 of this form or their assistant, will administer a number of short psychiatric questionnaires and an IQ test. The psychiatric questionnaires will ask about your mood, your patterns of behaviour and possible symptoms you may be experiencing. One aspect of the questionnaire may ask about details relating to any traumatic events you may have experienced. These questionnaires are research instruments that allow us to identify certain patterns of interest. During this screening the researcher will also inform you in detail about the design of the study and the research questions we hope to address with this study.

We will also take a comprehensive medical history from you where we will ask you to provide us with details of any medication you are currently on and any other things we should be aware of.

The first session will be a sleep adaptation night at UCT's sleep laboratory. This session will be scheduled at a time convenient to you. You will be asked to come in approximately 2 hours before your normal bedtime. Transport will be provided if you require it. During this session you will simply get used to sleeping at the laboratory whilst attached to the equipment. You will be briefed in detail on the procedure. You will be hooked to a polysomnograph (PSG) which is an EEG machine designed to monitor your sleep pattern. Electrodes will be placed on your head, chest, near your chin and temples; these are completely safe and present no danger whatsoever to your health. They are designed to transmit physiological indications of the stage of sleep you are experiencing at a given point in time, to a computer monitor. Once the electrodes are set up an 8-hour sleep period will follow. The experimenters will be available to you for assistance at any time. In the morning all the equipment will be removed and your skin cleaned to ensure no lasting effects.

The second session will also take place at the sleep laboratory. It will be scheduled for one week after your adaptation night will start approximately 3 hours before your normal bedtime. During this session the testing procedure described in session 1 will be followed again, the only difference being you will be administered a memory test before you go to bed. During the memory test you will be asked to retrieve specific personal memories relating to given words. Again an 8-hour sleep will follow and all equipment will be detached and cleaned in the morning.

After the sleep sessions are over, you will be debriefed about the study. You will also have the opportunity to ask questions and thus learn more about psychological research. If you have any questions now or at any time during the study, you may contact the Principal Investigators listed in #3 of this form.

6. If you choose to participate in this study, how long will you be expected to participate in the research?

Screening and interview session: approximately 2 hours.

Study sessions: 2 whole nights, one week apart.

7. How many people are expected to participate in the research?

60

8. What are the possible discomforts and risks?

During the initial screening you may be faced with fairly specific questions regarding past traumatic events as well as your current psychological functioning. These questions may illicit painful or unpleasant memories or make you aware of various symptoms you are experiencing. Should you experience distress as a result of these memories or symptoms or wish to seek support for the symptoms experienced, the researcher will refer you to trained clinicians who will be able to provide support.

Sleeping in an environment other than your own bedroom might feel strange and uncomfortable at first. Great precautions will be taken to ensure your safety and comfort. The sleep laboratory at UCT is fully equipped with a proper bed, clean bedding, and restrooms. It is situated in a secure building with adequate security. Attempts will be made to familiarise

you with the PSG and the electrodes used will be padded and lubricated so as to be as non-intrusive as possible.

Although the study sessions themselves will not delve into traumatic events experienced specifically, if any difficult memories should arise during the process, you will be referred to trained clinicians for extra guidance.

10a. What are the possible benefits to you?

You may or may not personally benefit from participating in this study. Participation in this study may, however, improve your understanding of some factors that affect sleep and may influence your management of your health generally.

10b. What are the possible benefits to others?

The information from this study may help improve our understanding of the importance of sleep and its link to memory. This study aims to show that symptoms do not exist in isolation but influence each other. If it is indeed the case that difficulties in sleeping are related to difficulties in memory then we know we need to focus more on addressing sleeping patterns. In fact some research has shown that if you improve sleeping patterns other symptoms also improve and this study hopes to elaborate on this.

11. If you choose to take part in this research study, will it cost you anything?

Participating in this study will not cost you anything.

12. Will you receive compensation for taking part in this research study?

You will receive financial compensation of the amount of R150 for each of the 2 sleep laboratory nights.

13a. Can you withdraw from this research study?

You are free to withdraw your consent and to stop participating in this research study at any time. If you do withdraw your consent, there will be no penalty.

If you have any questions regarding your rights as a research subject, you may phone the Psychology Department offices at 021-650-3430. You may also contact the Human Research Ethics Committee at 021-406-6626 or email: shuretta.thomas@uct.ac.za.

13b. If you withdraw, can information about you still be used and/or collected?

Information already collected may be used.

14. Once personal and performance information is collected, how will it be kept secret (confidential) in order to protect your privacy?

Information collected will be stored in locked filing cabinets or in computers with security passwords. Only certain people have the right to review these research records. These people include the researchers for this study and certain University of Cape Town officials. Your

research records will not be released without your permission unless required by law or a court order.

15. What information about you may be collected, used and shared with others?

This information gathered from you will be demographic information, information on a past traumatic event and the related diagnosis of post-traumatic stress disorder and/or depression, records of your sleep architecture, performance on cognitive tests, and scores on the IQ test and psychiatric inventory. If you agree to be in this research study, it is possible that some of the information collected might be copied into a "limited data set" to be used for other research purposes. If so, the limited data set may only include information that does not directly identify you. For example, the limited data set cannot include your name, address, telephone number, ID number, or any other photographs, numbers, codes, or so forth that link you to the information in the limited data set.

16. How will the researcher(s) benefit from your being in the study?

In general, presenting research results helps the career of a scientist. Therefore, the Principal Investigators and others attached to this research project may benefit if the results of this study are presented at scientific meetings or in scientific journals. This study is being undertaken for the Principal Investigator's Honours degree.

17. Signatures

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; and how the participant's performance and other data will be collected, used, and shared with others:

Signature of Person Obtaining Consent and Authorization Date

You have been informed about this study's purpose, procedures, possible benefits, and risks; and how your performance and other data will be collected, used and shared with others. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time.

You voluntarily agree to participate in this study. You hereby authorize the collection, use and sharing of your performance and other data. By signing this form, you are not waiving any of your legal rights.

Signature of Person Consenting and Authorizing

Date

Please indicate below if you would like to be notified of future research projects conducted by our research group:

_____ (initial) Yes, I would like to be added to your research participation pool and be notified of research projects in which I might participate in the future.

Method of contact:

Phone number:

E-mail address:

Mailing address:

Appendix D
Tests of Assumptions Underlying Inferential Statistical Analyses:
Sleep outcome variables

Levene's test for homogeneity must be run between-groups as it is a test for homogeneity between-groups. Kolmogorov-Smirnov, however, was run within groups to analyse normality, this is due to a need to check the distribution of data within groups in order to compare them to one another.

Table C1

Sleep-Related Variables (First Half of the Night): Results for the Kolmogorov-Smirnov test of normality (N = 40)

Variable	Group			
	PTSD (n = 11)	Trauma Exposed (n = 9)	Depression (n = 11)	Healthy control (n = 9)
REM %	.20	.20	.20	.20
SWS %	.20	<.01**	.20	.20
WASO	.20	<.01**	.02*	.06
Sleep efficiency	.20	.20	.20	.20
Number of awakenings	.20	.02*	.19	.04*
Spontaneous arousals	.20	.16	.03*	.14

Note. *p*-values for the K-S test are presented. T-E = trauma-exposed; WASO = wake after sleep onset.

p* < .05, *p* < .01, ****p* < .001.

Table C2

Sleep-Related Variables (Second Half of the Night): Results for the Kolmogorov-Smirnov test of normality (N = 40)

Variable	Group			
	PTSD (n = 11)	Trauma Exposed (n = 9)	Depression (n = 11)	Healthy control (n = 9)
REM %	.20	.20	.20	.20
SWS %	.07	< .01***	.20	.07
WASO	.04*	.07	.07	.03*
Sleep efficiency	.07	.08	.06	.03*
Number of awakenings	.20	.06	.01*	.08
Spontaneous arousals	.20	.20	.03*	.20

Note. *p*-values for the K-S test are presented. T-E = trauma-exposed; WASO = wake after sleep onset.

p* < .05, **p* < .01, **p* < .001

Table C3
Sleep Outcome Variables: Results for Levene's test of homogeneity of variance (N = 40)

Variable	Half of the Night	
	First	Second
REM %	.284	.720
SWS %	.324	.836
WASO	.024	.191
Sleep Efficiency	.308	.295
Number of Awakenings	.293	.159
Spontaneous Arousals	.070	.481

Note. Data presented are *p* values. WASO = wake after sleep onset. Bolded values indicate a significant violation of the assumption of homogeneity of variance.

Appendix E
Tests of Assumptions Underlying Inferential Statistical Analyses:
AMT outcome variables

Levene's test for homogeneity must be run between-groups as it is a test for homogeneity between-groups. Kolmogorov-Smirnov, however, was run within groups to analyse normality, this is due to a need to check the distribution of data within groups in order to compare them to one another.

Table D1
AMT outcome Variables: Results for the Kolmogorov-Smirnov test of normality (N = 40)

Variable	Group			
	PTSD (n = 11)	Trauma Exposed (n = 9)	Depression (n = 11)	Healthy control (n = 9)
Specificity				
Positive	.03*	.20	.04*	.06
Negative	.20	.20	.14	.20
Neutral	.20	.20	.20	.20
Word count				
Positive	.02*	.01*	.20	<.01**
Negative	.20	.20	.20	.01*
Neutral	.20	.20	.20	.16
Emotionality				
Positive	.20	.20	<.01**	.20
Negative	.20	.20	.20	.20
Neutral	.20	.03*	.20	.03*
Emotionality Proportion				
Positive	.20	.20	.03*	.20
Negative	.05	.03*	<.01**	.20
Neutral	.07	.02*	.18	.20

Note. Data are *p*-values for the K-S. T-E = trauma-exposed. WASO = wake after sleep onset.
 p* < .05, *p* < .01, ****p* < .001.

Table D2
AMT Outcome Variables: Results for Levene's test of homogeneity of variance (N = 40)

Variable	Levene's <i>p</i>
Specificity	
Positive	.391
Negative	.687
Neutral	.210
Word count	
Positive	.030
Negative	.050
Neutral	.097
Emotionality	
Positive	.389
Negative	.266
Neutral	.302
Emotionality Proportion	
Positive	.046
Negative	.231
Neutral	.001

Note. Bolded values indicate a significant violation of the assumption of homogeneity of variance.