Does Prominent Hyperarousal Explain REM Disruption and Nightmares in Posttraumatic Stress Disorder?

Tyler Phillips

UCT Sleep Sciences Laboratory

Department of Psychology

University of Cape Town

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Abstract

A growing body of research suggests that nightmares are associated with the development, maintenance, and exacerbation of posttraumatic stress disorder (PTSD). It has also been proposed that prominent hyperarousal symptoms in PTSD are a mechanism underlying nightmare production. These symptoms are additionally implicated in the disruption of rapid eye movement (REM) sleep – the sleep stage commonly associated with nightmares. However, there are currently no published studies that have investigated the role of prominent hyperarousal symptoms in the relationship between nightmares and REM sleep disruption in PTSD. I used data from a larger previously-conducted study to investigate this picture. This study was quasi-experimental in design, and collected data using the Most Recent Dream report (Domhoff & Shneider, 1998) and 2 nights of laboratory-based polysomnography sleep monitoring. The sample comprised 41 females aged between 18 and 36 years. I allocated participants to one of three groups: PTSD-diagnosed individuals with prominent hyperarousal symptoms (PTSD HYP+), PTSD-diagnosed individuals without prominent hyperarousal symptoms (PTSD HYP-), and healthy controls (HC). A series of one-way ANOVAs revealed no significant between-group differences in REM-related sleep disruptions in line with the pattern: PTSD HYP+ > PTSD HYP- > HC. Another series of one-way ANOVAs only demonstrated one significant between-group difference in dream report emotionality, which followed the pattern: PTSD overall > HC. Finally, linear regression modelling did not reveal that group status and degree of REM-related sleep disruptions jointly predicted negative emotional intensity of dream reports. These findings do not support the hypothesis that prominent hyperarousal symptoms result in greater REM-related sleep disruptions and nightmare severity in PTSD. Future research is urged to investigate this relationship with a larger sample size, with a depression comparison group, and with more contextually-relevant dream emotionally recall procedures.

Keywords: Posttraumatic stress disorder (PTSD); polysomnography; nightmares; dreams; rapid eye movement (REM) sleep; hyperarousal.

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Table of Contents

Introduction

Given that more than a third of the South African population has experienced a traumatic event (Kaminer, Grimsrud, Myer, Stein, & Williams, 2008), investigations into the factors which influence the development, maintenance, and exacerbation of posttraumatic stress disorder (henceforth PTSD) are crucial. Because only a minor fraction of those exposed to trauma go on to develop PTSD, there must be factors other than trauma exposure determining the pathogenesis of the disorder (Pace-Schott, Germain, & Milad, 2015). One of the most commonly proposed such factors is sleep disturbances (Breslau et al., 2004; Gerhart, Hall, Russ, Canetti, & Hobfoll, 2013; Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002; Spoormaker & Montgomery, 2008).

Nightmares in PTSD

A particularly salient sleep disturbance in PTSD is recurrent, trauma-related nightmares, which are characterised as an "intrusion symptom" in the diagnostic criteria for the disorder (American Psychiatric Association [APA], 2013, p. 271). There is growing consensus that nightmares and frequency of dream recall are associated with severity of waking distress and overall PTSD psychopathology (Levin & Nielsen, 2007; Mellman, David, Bustamante, Torres, & Fins, 2001). Moreover, the presence of nightmares in the aftermath of trauma is correlated with the later development of psychological distress in trauma-exposed individuals (Gerhart et al., 2013). Therefore, trauma-related nightmares appear to be not only a risk factor for the pathogenesis of PTSD, but also a core contributor to the consequent maintenance and exacerbation of PTSD symptoms (Germain, 2013; Levin & Nielsen, 2007; Pace-Schott et al., 2015; Spoormaker & Montgomery, 2008; Yetkin, Aydin, & Özgen, 2010).

An emotion-regulation hypothesis of dream function has often been proposed to explain the importance of nightmares in relation to PTSD psychopathology (Cartwright, Young, Mercer, & Bears, 1998; Levin & Nielsen, 2007; Walker, 2009). Cartwright et al. (1998) postulated that dreaming serves to improve next-day negative mood by promoting mediatory processing of waking emotional conflict. In essence, dreaming is theorised to represent continued processing of emotional experience which, when effective, results in resolutions of emotional distress (Cartwright et al., 1998; Walker, 2009). Indeed, there is considerable evidence that dream content is "consistently and powerfully modulated" by particular facets of waking life (Revonsuo, 2000, p. 877). Therefore, actual dream content may facilitate recovery (or a lack thereof) from emotional trauma (Cartwright et al., 1998;

Walker, 2009). Walker (2009) suggests that the recurrent nightmares which are key to PTSD symptomatology are an indication that the emotional intensity of traumatic memories in dreams is too high for effective resolution to be accomplished in the dreamscape. As such, poor waking affect and high psychological distress persist with the perpetuation of trauma-related nightmares (Levin & Nielsen, 2007; Spoormaker & Montgomery, 2008). Spoormaker and Montgomery (2008) describe several studies confirming that treatment of nightmares with Prazosin, a noradrenergic antagonist, not only reduces the extent of trauma-related content in dreams (and relatedly, frequency of nightmares), but consequently improves waking psychological and emotional distress. Treating nightmares in individuals with PTSD therefore ameliorates the severity of other PTSD symptoms, seemingly by restoring effective emotional processing in the dreamscape (Germain, 2013; Spoormaker & Montgomery, 2008). It is thus important to identify those trauma-exposed or PTSD-diagnosed individuals who experience nightmares in order to explore effective overall PTSD intervention.

Some PTSD-diagnosed individuals experience nightmares more than others (Levin & Nielsen, 2007; Spoormaker & Montgomery, 2008). Importantly, however, the prevalence of nightmares in PTSD-diagnosed individuals has been estimated to range from between 50 and 70% (Spoormaker & Montgomery, 2008) to as much as 90% of those diagnosed (Levin & Nielsen, 2007). Additionally, PTSD-diagnosed individuals tend to experience nightmares regardless of the kind of trauma event endured (Levin & Nielsen, 2007). This rather high prevalence is rendered all the more pertinent given the considerable impact that nightmares have on PTSD psychopathology, as outlined above. Therefore, it is important to comprehend the underlying mechanism(s) determining which individuals proceed to develop nightmares in PTSD.

Hyperarousal May Underlie PTSD Nightmares

One frequently proposed mechanism for the development of nightmares in PTSD is hyperarousal – or the "alteration in arousal and reactivity" symptom cluster (APA, 2013, p. 272). Features of this symptom cluster include hypervigilance, an exaggerated startle response, insomnia-like sleep problems, and irascibility (APA, 2013). Individuals with PTSD with prominent hyperarousal symptoms have more severe overall sleep disturbances than those without (van Wyk, 2013; van Wyk, Thomas, Solms, & Lipinska, 2015). Some researchers (e.g. Levin & Neilsen, 2007; van Wyk, 2013) have established a tentative association between nightmare severity and more prominent hyperarousal-related distress, but more research is needed to investigate this correlation. For example, van Wyk (2013) did not

confirm the hypothesis that PTSD-diagnosed individuals with prominent hyperarousal symptoms would have more negative and distressing dream content than their counterparts without such symptoms. This null finding is partly attributed to the small sample in the study (van Wyk, 2013). However, the association between hyperarousal and worse dream content approached significance (van Wyk, 2013), which favourably indicates that the hypothesis could be confirmed upon replication of the study, with improvements in sample size.

Nevertheless, a promising explanation of hyperarousal as a mechanism of nightmare production is implicated in the current understanding of noradrenergic activity and its relation to experiences of emotional distress. Poignant emotional stimuli prompt activation of noradrenergic pathways in the amygdala (Spoormaker & Montgomery, 2008; van Wyk, 2013). Continued reactive fixation on an emotional stressor, such as trauma, could result in an overproduction of noradrenaline, which consequently leads to amygdala over-reactivity (Spoormaker & Montgomery, 2008; van Wyk, 2013). As it happens, PTSD is associated with raised noradrenaline levels (Insana, Kolko, & Germain, 2012; Pace-Schott et al., 2015), and neuroimaging has confirmed that individuals with PTSD exhibit hyperactivity of the amygdala (Karl et al., 2006; Pace-Schott et al., 2015). Thus, noradrenergic/ amygdala hyperactivity may be a neurological marker of the chronic stress state of hyperarousal in PTSD (Pace-Schott et al., 2015; Spoormaker & Montgomery, 2008). These neurological abnormalities associated with hyperarousal may have a significant bearing on sleep disturbances, such as nightmares, as excessive noradrenaline and amygdala hyperactivity are also postulated to result in disruptions of rapid eye movement (henceforth REM) sleep (Insana et al., 2012; Mellman et al., 2002; Pace-Schott et al., 2015; Spoormaker & Montgomery, 2008). It is therefore important to examine REM sleep to clarify hyperarousal as a mechanism underlying nightmares.

REM Sleep and PTSD

Normally, noradrenergic activity is absent during REM sleep (Germain, 2013; Pace-Schott et al., 2015). Thus, the elevated noradrenaline levels observed in individuals with PTSD may be the cause of disrupted REM (Germain, 2013; Pace-Schott et al., 2015). This postulation is strengthened by the finding that treatment with Prazosin, a noradrenergic antagonist, improves REM sleep consolidation (Germain, 2013). REM sleep disruption is particularly significant in PTSD because, like nightmares, REM disturbances in the aftermath of trauma predict the pathogenesis of PTSD (Germain, 2013; Mellman et al., 2002). In fact, the presence of REM fragmentation following trauma exposure in childhood may not only

persistently affect self-reported sleep quality in later life, but may also increase adult susceptibility to trauma-related psychopathology (Insana et al., 2012). A meta-analytic review by Kobayashi, Boarts, and Delahanty (2007) has established that hyperarousal symptoms, for example, have been positively correlated with REM abnormalities. Indeed, REM disruption is more strongly associated with PTSD patients who have more pronounced hyperarousal symptoms (Lipinska, Timol, Kaminer, & Thomas, 2014; Pace-Schott et al., 2015). Furthermore, REM sleep is known to be meaningfully related to dreaming (Breslau et al., 2004; Levin & Neilsen, 2007; Perogamvros, Dang-Tu, Desseilles, & Schwartz, 2013; Solms, 2000).

Although dreaming does not occur exclusively during REM sleep (Solms, 2000), dreams in REM tend to be more emotionally intense, especially in terms of negativity and aggression (Perogamvros et al., 2013). Indeed, nightmares are conceptualised as primarily a phenomenon of REM sleep (Germain, 2013). Several researchers therefore advocate that nightmares and accompanying REM dysfunction constitute the hallmark of PTSD (Germain, 2013; Ross, Ball, Sullivan, & Caroff, 1989; Spoormaker & Montgomery, 2008). However, the association between REM fragmentation and nightmares in PTSD-diagnosed individuals, especially with regard to prominent hyperarousal symptoms, has not been tested (Mellman et al., 2001; Mellman, Pigeon, Nowell, & Nolan, 2007). Where certain aspects of this picture have been examined, results have been equivocal: Mellman et al. (2007) reported a negative relationship between cortical arousal and nightmare severity, and Germain and Nielsen (2003) found no REM-particular differences between varying intensities of nightmare content. However, disparate findings have arisen mainly because of methodological shortcomings, such as small sample size and sample heterogeneity (Kobayashi et al., 2007; van Wyk, 2013), both of which were limitations in the studies by Mellman et al. (2007) and Germain and Nielsen (2003).

While methodological improvements are necessary to clarify aspects of this picture, what is also clearly required of future research is to investigate the association between REM disruption and nightmares in PTSD-diagnosed individuals with prominent hyperarousal symptoms. Because treatment for sleep disruptions in PTSD has been shown to ameliorate other PTSD symptoms (Spoormaker & Montgomery, 2008), this kind of investigation would allow for more specific and directed treatment recommendations if research can establish early on which individuals – such as those with hyperarousal symptoms – are likely to suffer from trauma-related nightmares.

Rationale, Specific Aims, and Hypotheses

The foregoing literature review suggests that (a) nightmares significantly impact the overall psychopathology of PTSD, (b) hyperarousal appears to disrupt REM sleep, and (c) disrupted REM sleep is associated with nightmares in PTSD-diagnosed individuals. However, no published studies to date have investigated the relationship between REM disruption and nightmares in PTSD-diagnosed individuals with particular emphasis on the degree of prominent hyperarousal symptoms.

In this study, I aimed to address this gap in the literature by analysing polysomnographic sleep data and emotional dream content data across three groups: PTSD-diagnosed individuals with prominent hyperarousal symptoms (or the PTSD hyperarousal group), PTSD-diagnosed individuals without prominent hyperarousal symptoms (or the PTSD non-hyperarousal group), and healthy individuals. I hypothesised that, compared to participants in the other two groups, the PTSD hyperarousal group would have (1) a higher degree of REM sleep disruption and (2) more emotionally negative dream content. I additionally hypothesised that (3) the degree of hyperarousal and REM disruption would jointly predict negative dream content.

Methods

Design and Setting

The current study entailed an analysis of certain data collected through a larger research project by the UCT Sleep Sciences Team over the past four years. The objectives of this larger project related to the contribution of sleep disturbance to memory and emotional functioning in PTSD. Its design was quasi-experimental and cross-sectional.

The key predictor variable in the analysis was group status, and consisted of three levels: PTSD hyperarousal (PTSD HYP+), PTSD non-hyperarousal (PTSD HYP-), and healthy control (HC). Outcome variables will be (1) REM disruption indices, including measures of REM fragmentation (i.e. REM arousals, REM-to-stage-1-NREM and REM-to-wake transitions, and REM density; Breslau et al., 2004; Pace-Schott et al., 2015) and more general REM-related variables (i.e. REM latency and REM%), and (2) degree of negative emotional content in dream reports. Study procedures took place in the Sleep Sciences laboratory at the University of Cape Town (UCT) Department of Psychology.

Participants

A total of 60 participants, all female and between the ages of 18 and 40, comprised the final sample in the previously conducted research. Of these 60 participants, 19 trauma-exposed individuals were excluded from the current analysis for not meeting the diagnostic criteria for PTSD (see Materials). As such, the final sample utilised in the current study was 41 participants. In terms of a power analysis, an effect size of Cohen's $d = 1.04$ was utilised, as this represented the weighted effect size in research regarding nightmare incidence and severity and/ or REM disruption in PTSD (Germain & Nielsen, 2003; Kobayashi et al., 2007; van Wyk, 2013). This power analysis determined that the optimum sample size (at alpha = .05 for a power of .8) should be 39 participants.

Each of the participants was assigned to one of the three groups based on (a) the eligibility criteria below and (b) their aggregated hyperarousal cluster score on criterion D of the *Clinician-Administered PTSD Scale* (see Materials). The groups are as follows: PTSD HYP+ ($n = 11$), PTSD HYP- ($n = 10$), and HC ($n = 20$).

An exclusively female sample is beneficial according to several recommendations from the extant literature on PTSD and sleep. Women are an understudied population in terms of PTSD (Kobayashi et al., 2007; van Wyk, 2013). Moreover, women experience a higher prevalence of nightmares, recall dreams more frequently, assess dreams as more vivid, and report a more significant influence of dreams on waking behaviour than men (Levin & Nielsen, 2007).

Participants who were assigned to the PTSD groups were recruited from branches of the Rape Crisis Centre in Cape Town. Participants who were assigned to the healthy control group were recruited through advertisements in the local newspapers.

Eligibility criteria. The following eligibility criteria were astringently enforced:

1. Potential participants diagnosed with any DSM-IV-TR (Diagnostic and Statistical Manual of Mental Disorders, 4th ed., text revised; APA, 2000) disorders other than PTSD, including a history of alcohol and other substance abuse, were excluded. Disruptions in sleep patterns, including that of REM sleep, that are associated with other psychiatric disorders may have obscuring effects on the results (Benca, 1996). However, potential participants in the PTSD groups who presented with anxiety or mood disorders secondary to trauma were not excluded.
2. Potential participants who experienced trauma more than 5 years or fewer than 6 months prior to recruitment were excluded. Propinquity to trauma has an influence on the functioning of sleep (see, e.g., Insana et al., 2012).

3. Potential participants younger than 18 years and older than 40 years of age were excluded. The sleep patterns of adults differ from those of both elderly individuals and adolescents and children (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004).
4. Potential participants who were taking sedative or psychoactive medications were excluded, as these medications may alter natural sleep cycles (see, e.g., Lund, Reider, Whiting, & Prichard, 2010).
5. Potential participants who carried potentially confounding neurological conditions (e.g. epilepsy) were excluded.

Materials and Apparatus

Diagnostic and screening instruments. The *Clinician-Administered PTSD Scale* (*CAPS*; Blake et al., 1995) is a structured interview designed to detect the presence, frequency, and intensity of PTSD symptoms, using an overt behaviour-based rating scale. Its developers assert that it has excellent reliability and validity, and this has been confirmed where it has been used in South African studies (see, e.g., Stein et al., 2013). In the current study, the CAPS was used, firstly, as the primary diagnostic instrument for the detection of PTSD in trauma-exposed participants. Arriving at a PTSD diagnosis through the CAPS can take nine different scoring routes (Weathers, Keane, & Davidson, 2001). Because it was necessary to obtain as large a sample for the PTSD groups as possible, the lenient *Frequency ≥ 1/ Severity ≥ 2 (F1/I2)* rule was utilised to determine PTSD diagnoses in this study. This scoring method has good reliability (with a kappa coefficient of .81; Weathers et al., 2001). According to the F1/I2 rule, a PTSD symptom exists if it has a frequency score of at least 1 as well as a severity score of at least 2. The detected symptoms must be appropriately distributed across the symptom clusters as outlined in the DSM-IV-TR diagnostic criteria in order to arrive at a PTSD diagnosis (Blake et al., 1995; Weathers et al., 2001).

Once PTSD diagnoses were confirmed, criterion D of the CAPS was used to determine the prominence of hyperarousal symptoms. PTSD-diagnosed individuals were allocated to their respective groups on the basis of the median hyperarousal score. Those who scored below 21 on the CAPS were allocated to the PTSD HYP- group, while those who scored 21 and over were allocated to the PTSD HYP+ group. This decision was informed by the fact that once scores for a symptom cluster are derived, their indications of symptom intensity are reliable enough to enable such an ordering for group allocation (Weathers et al., 2001).

The *Mini International Neuropsychiatric Interview* (*MINI* version 5.0.0; Sheehan et al., 1998) is a short structured interview that determines the presence of any DSM-IV-TR Axis 1 psychiatric disorders. Its developers report that the MINI has strong reliability and validity. It has also been validated as a successful measure in South Africa (see, e.g., Myer et al., 2008). In the conducted study, the MINI was used for diagnostic confirmation of PTSD, to exclude the presence of other psychiatric conditions (except for anxiety and mood disorders secondary to trauma in the PTSD groups), and to identify HC participants.

The *Beck Depression Inventory – Second Edition* (*BDI-II*; Beck, Steer, & Brown, 1996) is a standardised self-report questionnaire for assessing the presence and intensity of current depression in adults. Its developers uphold that it has satisfactory reliability and validity. The BDI-II has been validated as a reliable measure of depression in South Africa (see, e.g., Kagee, 2008). This instrument was used to garner information about depressive characteristics reported by those in the PTSD groups, as well as to exclude those HC participants with BDI-II scores of 14 or higher.

Experimental measures.

***Dream recall form*.** The *Most Recent Dream* recall form (Domhoff & Shneider, 1998; see Appendix A) required participants to detail the date on which their most recent dream occurred, as well as the date on and location at which this dream was first recalled. It then asked participants to freely recall as much detail about that dream as they could remember, including setting, characters involved, events that transpired, and the dreamer's feelings. This form was used both at screening and after the two polysomnography-monitored sleep nights to obtain a report of participants' dreams.

Sleep laboratory equipment. The Sleep Sciences laboratory of the UCT Department of Psychology is equipped with polysomnography (PSG), or electroencephalograph (EEG) equipment adapted for sleep research. In addition to the EEG electrodes that map sleep architecture, PSG also measures eye movements through electrooculography (EOG) electrodes, muscle tone through electromyograph (EMG) electrodes, and heartrate through electrocardiograph (ECG) electrodes. These additional measures are crucial to identify REM sleep, as brain activity alone is not a sufficient indicator thereof (American Academy of Sleep Medicine [AASM], 2007). PSG was therefore used to assess REM sleep disruption. The most recent guidelines provided by the AASM (2007) were used to categorise sleep stages according to the above PSG measures.

Sleep measures were recorded using a Nihon Kohden NeuroFax EEG9000. A bipolar longitudinal montage was used in accordance with the AASM (2007) technical specifications

manual. All electrodes were placed according to the international 10-20 placement system. To ensure signal coherence in each channel, standardised sleep-recording filters were used for the EEG and EOG (0.5-35 Hz), EMG (10-70 Hz) and ECG (1-70 Hz) leads.

Procedure

Potential participants first attended an initial screening. The screening took place in a private room in the UCT Department of Psychology, and began with an explanation of the aims and nature of the research to potential participants. Thereafter, each participant read and signed an informed consent document (see Appendix B), following which the screening measures described above were administered. They were also asked to provide a report of their most recent dream (see Appendix A), in order to obtain a dream report that was not related to the sleep laboratory (see, e.g., Dement, Kahn, & Roffwarg, 1965, for more).

Once the screening session had been completed, participants were debriefed about the study procedures conducted thus far. If they met the eligibility criteria, they were assigned to one of the three groups and enrolled into the study, which entailed two PSG-monitored nights in the sleep laboratory. The first night was merely adaptive, as natural sleep architecture is typically altered by the initial night in the laboratory (Spoormaker & Montgomery, 2008).

The participants arrived at the Sleep Sciences laboratory in the UCT Department of Psychology approximately two hours before their normal bedtime, and were briefed about the procedures for the evening and morning (which entailed various cognitive tests related to the objectives of the larger study). The researchers then proceeded with attaching the PSG equipment to the participants, and ensured that it was providing a clear reading. The participants were then allowed to go to sleep within half an hour of their usual bedtime, to best ensure consistency with their normal sleeping pattern.

Participants were allowed an 8-hour period of sleep. After this period, they were awoken, and all PSG equipment was removed. Each participant was then required to provide a dream report, in which she freely recalled the dream(s) she could remember experiencing from the previous night. These dream reports were collected after both the adaptation and experimental sleep nights, in order to acquire a more representative sample of dream reports (see, e.g., Domhoff, 2000). The researchers did not elicit REM awakenings to obtain dream reports, despite the high rate of dream recall thereafter (Dement et al., 1965), because the objective was to study natural REM disruption not altered by laboratory protocol. Finally, the participants were debriefed regarding the study procedures, provided compensation for their time, and allowed to leave.

Ethical Considerations

The larger conducted research project obtained ethical approval from the Research Ethics Committees of UCT's Department of Psychology and Faculty of Health Sciences (see Appendix C). All participants were provided with written informed consent (see Appendix B) to advise them about the study procedures, risks, and benefits. Participants were assured that they could withdraw from participation, penalty-free, at any stage of the study. They were also guaranteed that confidentiality would be strictly upheld, that the tests would not harm them by any means, and that they would be compensated R150 for their time.

Because participants in the PTSD groups were particularly vulnerable (especially during screening/ interviewing, where the nature of their previous trauma exposure was investigated), participants were assured from the outset that they should provide only as much detail as with which they were comfortable. Furthermore, participants in the PTSD groups were referred to appropriate clinics, counselling centres and therapists at the close of all procedures.

Data Management and Statistical Analyses

Subjective measures of sleep. To assess the emotionality of dream reports, the Linguistic Inquiry and Word Count programme (LIWC; Pennebaker, Booth, Boyd, & Francis, 2015) was used. LIWC is a software programme which analyses the content of transcribed linguistic text files (Pennebaker, Booth, et al., 2015). LIWC 2015 is the latest version of the programme, and contains a dictionary composed of "almost 6400 words, word stems, and select emoticons…which defines one or more categories or subdictionaries" (Pennebaker, Boyd, Jordan, & Blackburn, 2015, p. 2). I transcribed the dream reports, and ran these transcribed dream reports through LIWC in order to ascertain the number of emotional words used by participants. The dictionary category file *negative emotion* and subdictionary files *anxiety*, *anger,* and *sadness* were used specifically, which included words such as *hurt, fearful*, *hate*, and *crying*, respectively (Pennebaker, Boyd, et al., 2015). Additionally, the *positive emotion* dictionary file (which included words such as *happy*, *nice,* and *fun*) as well as the *affective processes* file (which combined the *positive* and *negative* emotional category scores) were used to investigate the possibility of other differences in dream emotionality (Pennebaker, Boyd, et al., 2015). LIWC delivered a percentage of words in each dream report which belonged to each of these dictionary and subdictionary categories.

The percentages of negative emotional dream content delivered by LIWC do not, however, provide an indication of the relative negative emotional intensity of the dreams. As

it stands, the only means to derive an indication of intensity of dream emotions is through subjective rating scales (Schredl & Doll, 1998; Sikka, Valli, Virta, & Revonsuo, 2014). Participants themselves did not rate the emotional intensity of their dreams within the larger study. As such, an external-judge measure was employed to assess emotional intensity of dream content. This required the dream reports to be rated on a scale from negative ten, indicating extremely negative content, to positive ten, indicating extremely positive content (Domhoff, 2000). Emotionally neutral content fell within the range of negative two to positive two (Domhoff, 2000). This global scale of emotional dream intensity has been validated and successfully employed in previous South African studies (e.g. van Wyk, 2013). An external judge was consulted, instructed in how to rate the reports, and asked to provide a second set of ratings. All reports were rated blind to group allocation. A high interrater reliability estimate (intraclass correlation coefficient = .98) was established between my ratings and the external judge's ratings. The averages of these two sets of ratings were used as the final ratings in the statistical analyses.

Objective measures of sleep. I scored the sleep variable data according to the AASM (2007) criteria for REM fragmentation. A REM arousal is characterised by a sudden change of EEG frequency "including alpha, theta, and/or frequencies greater than 16 Hz … that lasts at least 3 seconds" (AASM, 2007, p. 36). REM transitions will include REM-to-wake, in which alpha rhythms begin to dominate over the occipital region, and REM-to-stage-1-NREM, which entails an arousal "followed by low amplitude, mixed frequency EEG and slow eye movements" (AASM, 2007, p. 28). The number of both REM arousals and REM transitions may constitute the degree of REM fragmentation alone (Breslau et al., 2004).

However, elevated REM density is an additional indication of fragmented REM sleep (Mellman et al., 2002; Pace-Schott et al., 2015). REM density is defined as the number of rapid eye movements within a phasic REM period, expressed as a percentage of total REM sleep time (Mellman et al., 2002; Moore et al., 2013). An external programmer was consulted to conduct the REM density analyses in MATLAB, using the Stanford EEG Viewer (SEV) toolbox developed by Moore et al. (2013). The SEV was specifically designed to combine power spectral analysis with algorithms for event classification in PSG (Moore et al., 2013). There are several reliable methods of classifying eye movements (EMs) for REM density analyses. The current study employed the *dual threshold* detection method, as it is neither too lenient nor too conservative (Moore et al., 2013). Moreover, this method was validated on a cohort of PTSD-diagnosed combat veterans (Moore et al., 2013), making it quite relevant to the current study. Appendix D contains an explanation of this dual threshold detection

method, as well as a description of how artefactual EOG waveforms were manually eliminated in accordance with guidelines by Brown, Marmor, Zrenner, Brigell, and Bach (2006).

In order to ensure blind scoring with regards to group status, all identifying record details were recoded. To validate this scoring, one quarter of the records was sent to the Panorama MediClinic, where they were also scored blind to the group categorisation of participants. An interrater reliability estimation of 89 percent was established.

Inferential statistical analyses were conducted using SPSS version 23. Preliminary analyses of statistical assumptions for the inferential techniques were conducted. I employed the following statistical methods to test the three hypotheses:

Hypothesis 1. I first hypothesised that prominence of hyperarousal symptoms would result in greater disruption to REM sleep. I ran a series of one-way ANOVAs to determine between-group differences with respect to the number of REM arousals alone, the number of REM transitions alone, as well as their combination. I additionally ran a one-way ANOVA to examine between-group differences in REM density. Supplementary comparisons of general REM sleep characteristics (i.e. percentage of time spent in REM sleep, or REM%, and time before the initiation of the first REM period, or REM latency) were also conducted. I expected the following pattern between the groups for indices of REM fragmentation: PTSD HYP+ > PTSD HYP- > HC. I expected to find the same pattern for the REM latency variable, but the opposite pattern for the REM % variable (i.e. PTSD HYP+ < PTSD HYP- < HC).

Hypothesis 2. Here I hypothesised that prominence of hyperarousal symptoms may result in more emotionally negative and intense dream content. I similarly ran one-way ANOVAs to examine between-group differences in emotionality of dream reports as defined by (1) percentages of emotional content derived from the LIWC analysis and (2) ratings of emotional intensity using the Domhoff (2000) scale. The LIWC variables included percentages of “anxiety” emotions, “anger” emotions, “sadness” emotions, negative emotions overall, positive emotions overall, and overall affective processes. I expected between-group differences for negative emotional dream content, as well as for (negative) emotional intensity and overall affective processes, to be patterned as follows: PTSD HYP+ > PTSD HYP- > HC. I expected the opposite pattern for positive emotional dream content.

Hypothesis 3. Finally, I hypothesised that prominence of hyperarousal symptoms, combined with degree of REM disruption, is predictive of negative emotional dream severity. I began by examining correlations between the REM sleep variables and dream emotionality variables which, in the foregoing analyses, were found to demonstrate significant between-

group differences. Where I found higher correlations, I used the particular REM variable as a predictor of that particular dream emotionality variable. I set out to explore a linear model of the relationship between hyperarousal/ group status, REM sleep variables, and dream content using hierarchical multiple regression analyses.

Results

Sample Characteristics

In terms of sociodemographic variables, participants were suitably matched on age, level of education, and monthly household income, as indicated in Table 1. The mean age of the entire sample was 25.34 (*SD* = 4.28, range = 18 – 36). In terms of level of education, all participants had acquired at least some level of high school education, with a modest majority of participants (*n* = 26, 63.42%) having actually completed high school. Several participants (*n* = 16, 39.02%) had completed some form of tertiary education. There is somewhat less homogeneity for monthly household income, with about a quarter (*n* = 10, 24.39%) of participants exceeding an income of ZAR10 000 per month. This suggests that these participants might be in a higher socioeconomic bracket than the remaining 31 (75.61% of) participants. However, these potentially higher earners were more or less evenly distributed across the groups. Hence, no significant differences for monthly household income were observed between them.

In terms of depression, a one-way ANOVA revealed a statistically significant between-group difference in BDI-II scores with a large effect size. To examine the source of this difference, a pair of planned orthogonal contrasts was conducted. Firstly, HCs were contrasted with the combination of PTSD groups, and a statistically significant difference was found, as expected from inspection of the descriptive statistics, $t(37) = 16.23$, $p < .001$. This revealed that HCs had considerably lower depression levels than the PTSD groups (indeed, no control participant scored higher than 12 on the BDI-II). The second contrast was between the PTSD groups, and this did not reveal a statistically significant difference, $t(37) = 1.22$, $p = .229$. Thus, the PTSD HYP+ and PTSD HYP- groups did not differ in their levels of depression. As such, I expected with reasonable confidence that further differences between these two groups were not attributable to levels of comorbid depression.

Regarding total CAPS scores, Table 1 indicates that an independent samples t-test detected a statistically significant difference between the PTSD groups at an alpha threshold of .05. I followed up the CAPS total score analysis to see whether this significant difference

Table 1

Sample Sociodemographic & Psychiatric Characteristics

	Group								
	PTSD HYP+		PTSD HYP-		HC				
Variable	(*n* = 11)		(*n* = 10)		(*n* = 20)		F / t / X^2	*p*	EES
Age	25.18	(4.14)	25.60	(4.12)	25.30	(4.62)	.03	.975	<.01
Education (years)	12.09	(2.02)	11.90	(1.73)	12.90	(2.00)	1.12	.336	.06
Monthly Income (ZAR)							13.99	.082	.41
≥ 10 000	4		2		4				
5500 – 9999	0		0		5				
2500 – 5499	5		6		5				
1000 – 2499	2		0		5				
0 – 999	0		2		1				
BDI-II total	31.82	(5.33)	29.22	(6.34)	6.20	(3.41)	134.57	< .001**	.88
CAPS total	73.73	(12.84)	56.20	(17.12)	-	-	2.67	.015*	1.23
CAPS Hyperarousal	26.09	(3.67)	14.80	(3.94)	-	-	6.78	< .001**	3.12
CAPS Re-experiencing	18.18	(4.45)	15.20	(7.94)	-	-	1.08	.296	.49
CAPS Avoidance & numbing	29.45	(9.05)	26.20	(7.16)	-	-	.91	.376	.42
Time since trauma (years)	1.21	(0.74)	1.46	(1.26)	-	-	.57	.578	.26

Note. Means are presented with standard deviations in parentheses for all variables except Monthly Income. For Monthly Income, raw numbers of participants are given. ZAR = South African Rand; EES = estimate of effect size (i.e. η^2 for F tests, Cramer's V for the χ^2 test, and Cohen's d for t tests). For all F tests, df = 2, 39; for the X^2 test, df = 9; for all t tests, df = 19.

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

was attributable only to differences in hyperarousal cluster scores, or also to differences in scores for CAPS clusters other than hyperarousal symptoms. I conducted additional independent samples t-tests on the CAPS score totals for the other symptom clusters. No statistically significant between-group difference was found for CAPS re-experiencing cluster total scores or for CAPS avoidance and numbing cluster total scores. Therefore, the only difference in PTSD symptom severity between the HYP+ and HYP- groups was for the symptom cluster by which they were definitively differentiated. I thus expected that further statistical differences between the two PTSD groups would be attributable only to their different levels of hyperarousal symptom severity, and not to other features of their PTSD. This postulation is strengthened by the fact that there was no statistically significant between-group difference for time since trauma, which is known to affect sleep in PTSD (Insana et al., 2012).

In summary, all participants were adequately matched on sociodemographic characteristics. In terms of BDI-II scores, planned orthogonal contrasts suggested the following relationship: PTSD HYP+ = PTSD HYP- > HC. While depression may be responsible for differences between the PTSD groups and HC group, it is unlikely to result in differences between the PTSD groups themselves. Regarding CAPS scores, omnibus ANOVAs revealed that PTSD groups differ only in their severity of hyperarousal cluster scores. Taken together, these findings suggest that the differences in sleep variables between the PTSD groups may be attributable to their defining characteristic – differing hyperarousal levels – and not to any other psychiatric or sociodemographic features.

Testing Hypothesis 1: Between-Group Differences in REM Disruption

Here I hypothesised that individuals with prominent hyperarousal symptoms would demonstrate the greatest REM-related sleep disruptions in comparison with participants in other groups. As such, it was hypothesised that indices of REM disruption would have the following pattern: PTSD HYP+ > PTSD HYP- > HC.

Preliminary statistical analyses were conducted for the REM disruption variables regarding the assumptions underlying one-way ANOVA (which include normality of data distribution and homogeneity of variance). Normality of data distribution was assessed using the Kolmogorov-Smirnov goodness-of-fit test. If this test delivers a statistically significant *p*-value, one can consider the data distribution significantly different from normal. Results of the Kolmogorov-Smirnov test revealed that only REM latency ($p < .001$) and REM transitions ($p = .038$ at $\alpha < .05$) were non-normally distributed. Although two of the five

REM disruption variables were not normally distributed, a non-normal distribution is relatively common for small sample sizes (Field, 2009). Moreover, ANOVA is generally robust to violations of normality (Field, 2009). These two violations were thus deemed unproblematic in terms of further analyses.

Analyses of homogeneity of variance, using Levene's test, revealed that all 5 REM disruption variables met the parametric assumption of homogeneous variances (i.e. no *p*-value was less than .05). I therefore proceeded with the ANOVA analyses as intended.

The results of the series of one-way ANOVAs testing the above hypothesis for each REM sleep variable are displayed in Table 2. No statistically significant between-group differences for any REM sleep variables were detected. Moreover, in terms of group means, no REM fragmentation variables (i.e. REM density, REM transitions, and REM arousals) followed the predicted pattern of PTSD HYP+ > PTSD HYP- > HC. The largest effect size was evidenced with respect to REM transitions, but group status only accounted for a moderate 11% of the variance in this variable.

In terms of REM%, however, it appears from the group means that the PTSD HYP+ group had the least amount of REM sleep, followed by the PTSD HYP- group, with the HC group having the largest amount of REM sleep. This may be of interest for subsequent modelling, as it seemed to reflect a pattern in line with my hypotheses regarding group differences for REM functioning, although the between-group differences in means are small.

Testing Hypothesis 2: Between-Group Differences in Dream Report Emotionality

Hypothesis 2 proposed that prominence of hyperarousal symptoms would reflect greater negative emotional dream content. As such, it was proposed that the percentage of negative emotional word content, and (average) intensity of negative dream emotionality, would be greatest for the PTSD HYP+ group, and lowest for the healthy control group. Moreover, positive emotional dream content was expected to exhibit the reverse relationship between the groups.

In terms of the parametric assumptions underlying the ANOVA tests for these variables, the Kolmogorov-Smirnov tests for normality revealed that positive emotional content ($p = .002$) as well as "anxiety", "anger", and "sadness" emotional content (all $p < .001$) were non-normally distributed. Furthermore, negative emotional content overall ($p = .054$) and overall affective processes ($p = .052$) approached non-normality. Thus, it can only be confidently asserted that the emotional intensity variable was normally distributed. However, as stipulated above, omnibus ANOVA is relatively robust against violations of

Table 2

REM Disruption Variables: Descriptive Statistics and Results of One-Way ANOVAs

	Group								
	PTSD HYP+		PTSD HYP-		HC				
Variable	(n = 10[b])		(n = 10)		(n = 20)		F[c]	p	EES
REM arousals	18.00	(8.98)	21.40	(8.10)	21.35	(9.60)	.51	.603	.03
REM transitions[a]	9.20	(3.12)	11.90	(4.15)	8.55	(4.45)	2.28	.116	.11
REM arousals & transitions	27.20	(11.92)	33.30	(11.09)	29.90	(12.95)	.62	.542	.03
Average REM density	32.41	(9.34)	27.99	(14.68)	34.94	(11.78)	1.11	.339	.06
REM sleep %	17.10	(6.99)	18.54	(4.71)	19.31	(4.30)	.63	.537	.03
REM latency	100.15	(38.11)	81.15	(23.03)	104.60	(42.61)	1.33	.278	.07

Note. Means are presented with standard deviations in parentheses for all variables. EES = estimate of effect size (in this case, η^2).

[a]REM transitions include REM-to-NREM-stage-1 and REM-to-wake transitions.

[b]One participant in the HYP+ group did not have any REM sleep and was therefore excluded from all analyses, except REM sleep %.

[c]df = 2, 37 for all F tests, except for REM sleep % (df = 2, 38).

normality, which are likely given this study's small sample size (Field, 2009). In terms of homogeneity of variance, Levene's test did not return significant *p*-values for any of the dream emotionality variables. Thus, all of these variables upheld the parametric assumption of homogeneity of variance. Therefore, I proceeded with the intended analyses.

A total of 70 dream reports were collected from the 41 participants. All participants were given 3 opportunities to provide dream reports. A total of 19 participants (46%) provided only 1 dream report, 16 participants (39%) provided only 2 dream reports while the remaining 6 participants (15%) provided 3 dream reports. Overall, the distribution of dream reports was relatively proportional across the groups: the PTSD groups provided 17 dream reports each, while the healthy control group, which is double the size of the PTSD groups, provided just over double (i.e. 36) the dream reports. Indeed, there were no statistically significant between-group differences in dream report count, $F(2, 38) = .28$, $p = .759$, $\eta^2 = .01$. I averaged the dream emotionality scores across the reports provided by each participant, so that for each dream emotionality variable, each participant had only one score, as opposed to two or three scores. This decision was based on the fact that the scores for each dream report per participant represent dependent data.

Table 3 provides the results of the series of one-way ANOVAs testing the group difference hypothesis for the variables of dream emotionality. The only statistically significant between-group difference was for emotional intensity as assessed by the subjective rating scale. This yielded a large effect size (Cohen, 1988), where group status accounted for 34% of the variance in emotional intensity. Tukey's post-hoc tests revealed that the source of this significant difference was between the PTSD HYP+ and HC groups ($p < .001$) as well as between the PTSD HYP- and HC group ($p = .026$ at $\alpha = .05$). The PTSD groups were not significantly different from each other ($p = .465$). Thus, the PTSD groups as a whole had more intense emotionally negative dream content than the HC group. While the HYP+ group had more intense negative dream content than the HYP- group, this difference was not statistically strong.

It is worth noting, firstly, that the ANOVA for "anger" dream content as assessed by LIWC approached statistical significance, with a large effect size (Cohen, 1988). The biggest difference seemed to be between the PTSD HYP- and HC groups, but the PSTD HYP+ group also had a higher average anger-related word count than the HC group. Although the results for this variable do not confirm the hypothesised pattern, it might nevertheless be suggested that PTSD groups together have more anger-related emotional dream content than the HC group. Secondly, negative emotional content overall also showed trend-level significance.

Table 3

Dream Report Emotionality Variables: Descriptive Statistics and Results of One-Way ANOVAs

	Group								
	PTSD HYP+		PTSD HYP-		HC				
Variable	(*n* = 11)		(*n* = 10)		(*n* = 20)		*F*	*p*	EES
LIWC Anxious Emotions	.50	(.58)	.58	(.83)	.66	(.95)	.13	.882	.07
LIWC Anger Emotions	.72	(.86)	1.49	(1.13)	.56	(.93)	3.22	.051	.15
LIWC Sad Emotions	.56	(.63)	1.21	(1.66)	.56	(.93)	1.33	.276	.07
LIWC Negative Emotions	2.05	(1.37)	4.14	(3.05)	2.37	(2.20)	2.68	.082	.12
LIWC Positive Emotions	1.17	(1.19)	.71	(.82)	1.83	(1.81)	2.09	.138	.10
LIWC Overall Affect	3.21	(2.19)	4.85	(3.06)	4.20	3.01	.90	.416	.05
Emotional Intensity Ratings	-5.09	(2.41)	-3.76	(2.52)	-1.05	(2.67)	9.75	<.001*	.34

Note. Means are presented with standard deviations in parentheses for all variables. All LIWC variables are expressed as percentages. Emotional severity ratings are on a scale from – 10 (very negative) to +10 (very positive). All variables represent mean values across all three dream report opportunities. EES = estimate of effect size (in this case, η^2). *df* = 2, 38 for all *F* tests.

*$p < .001$.

However, the observed pattern of group means (i.e. PTSD HYP- > HC, but HC > PTSD HYP+) did not clearly resemble that of "anger" content (i.e. that the PTSD groups overall had more negative emotional content in dream reports than HCs).

Testing Hypothesis 3: Associations between REM Disruption and Dream Emotionality

Here I hypothesised that prominence of hyperarousal symptoms, in combination with degree of REM-related sleep disruption, would jointly predict negative emotionality in dreams. Despite obtaining no significant between-group differences for any REM-related variables, I nonetheless proceeded to explore a linear model investigating the association between REM-related variables and dream emotionality. This decision was based on the fact that, in the foregoing analyses, at least one dream emotionality variable (i.e. emotional intensity ratings) demonstrated a statistically significant between-group difference.

Before proceeding to the build model, I examined the strengths and significance levels of the correlations between each REM-related variable and emotional intensity ratings. I decided to run these correlations first in order to avoid adding too many variables into the regression model. Given the small sample size, adding too many variables into the model would substantially inflate Type 1 error. No statistically significant correlations were found between any REM-related variables and emotional intensity ratings. In fact, most correlations between REM-related variables and this dream emotionality variable were very weak (i.e. $r < .1$). However, of all the REM-related variables, REM% delivered the strongest correlation with emotional intensity ($r = .22$, $p = .167$). Because REM% was the only variable that had a correlation with emotional intensity ratings above $r = .1$, I decided a further exploratory analysis was warranted. I thus set out to model emotional intensity ratings of dream reports as a function of REM%.

For this model of emotional dream intensity, I included as predictors REM%, group status, as well as their interaction. This model was constructed iteratively using hierarchical regression: I added a predictor that contributed most significantly to the model and removed those that provided negligible contributions, in order to arrive at a statistically significant model that best accounted for the variance in emotional intensity ratings.

The results of the linear modelling are as follows. I found the model using group status and REM% as predictors of emotional intensity to be statistically significant, $F(3, 37) = 6.76$, $p = .001$. The interaction between these two predictors accounted for 35.4% of the variance in emotional intensity. However, group status alone was responsible for this statistical significance ($p < .001$), as adding REM% to the model did not result in a

statistically significant change in R^2 (p = .358). In fact, the addition of REM% into the model only accounted for 1.5% of the variance above the 33.9% explained by group status. Thus, although REM% contributed somewhat to an explanation of the variance in emotional intensity, this slight addition does not warrant its inclusion in an optimal model of emotional dream intensity. I determined that hyperarousal group status alone optimally predicted emotional intensity of dream reports.

Discussion

The extant literature suggests that prominent hyperarousal symptoms may be a mechanism underlying nightmare severity and REM sleep disturbances in PTSD – both of which seem to represent primary contributors to the development and maintenance of the disorder. However, the empirical findings regarding this proposal have so far been inconsistent and equivocal. There are discrepant reports concerning the presence, strength, and direction of the relationship between nightmare intensity and REM sleep disruptions (Germain & Nielsen, 2003; Spoormaker & Montgomery, 2008), and between hyperarousal prominence and nightmare severity in PSTD (Levin & Nielsen, 2007; Mellman et al., 2007). Importantly, no studies to date have focused specifically on the role of hyperarousal symptom prominence in the relationship between nightmare severity and degree of REM abnormalities in PTSD. In this study, I aimed to address the gap and inconsistencies in the literature by adopting this specific investigative focus. I hypothesised that PTSD-diagnosed individuals with prominent hyperarousal symptoms would have (1) the greatest REM-related sleep disruptions and (2) the most negative and intense emotional dream content in comparison to their counterparts without prominent hyperarousal symptoms and healthy controls. Finally, I proposed that (3) hyperarousal group status and REM disruption would together predict the extent and intensity of negative emotional dream content.

REM Disruption Findings

The first hypothesis was not confirmed by the statistical analyses for group differences in REM-related variables. In other words, it was not statistically established that PTSD-diagnosed individuals with prominent hyperarousal symptoms (PTSD HYP+) had the greatest degree of REM-related sleep disruptions, followed by PTSD-diagnosed individuals without prominent hyperarousal symptoms (PTSD HYP-), with healthy controls (HC) having the lowest degree of REM-related sleep disruptions.

Not only were there no statistically significant between-group differences in REM-related sleep disturbances, but the groups means for the REM fragmentation variables (i.e.

REM transitions, REM arousals, and REM density) and for REM latency were not suggestive of the predicted pattern of PTSD HYP+ > PTSD HYP- > HC. The group means for REM% were suggestive of the hypothesised pattern (PTSD HYP+ < PTSD HYP- < HC), although the effect size for this pattern was small.

These findings (i.e. that differences in PTSD symptomatology did not demonstrate corresponding differences in REM-sleep patterns) mirror those of Mellman et al. (2007), van Wyk (2013), and Yetkin et al. (2010). Based on these studies, a possible reason for the current null findings might be the differential influence of laboratory habituation effects (Mellman et al., 2007; van Wyk, 2013). In the laboratory setting, healthy controls tend to report poorer sleep quality than usual (Le Bon et al., 2001), while PTSD-diagnosed individuals tend to report improved sleep quality, due to feeling safer in this environment (Spoormaker & Montgomery, 2008). These habituation effects may be present up to the fourth night of laboratory-monitored sleep (Le Bon et al., 2001). Given that my PTSD sample was comprised almost entirely of women living in township communities, where poor living conditions and high rates of violence predominate, it is possible that the beneficial effects of the sleep laboratory environment were augmented in this study (van Wyk, 2013). This may be particularly salient for the PTSD hyperarousal group, given their symptomatic preoccupation with threats to safety (Spoormaker & Montgomery, 2008). Thus, all three groups may have had similar levels of REM disruption due to the differential effects of laboratory PSG-monitoring.

An alternative explanation for why group differences in REM-related variables were not detected focuses on the high rate of comorbid depression in PTSD participants (Yetkin et al., 2010). Depression tends to affect REM-related variables in ways opposite to PTSD. For example, PTSD-diagnosed individuals are suggested to exhibit increased REM latency and decreased REM%, while depression-diagnosed individuals tend to demonstrate decreased REM latency and increased REM% (Kobayashi et al., 2007; Yetkin et al., 2010). All but one PTSD-diagnosed participant had at least moderate depression, and around half of the PTSD sample had severe depression. The low socioeconomic status of PTSD participants has also been suggested to exacerbate comorbid depression symptoms (Everson, Maty, Lynch, & Kaplan, 2002; van Wyk, 2013). Thus, the high rate of comorbid depression in the current sample may have reduced or otherwise altered the effects of PTSD (and hyperarousal symptom prominence) on REM-related variables, resulting in no significant between-group differences.

Finally, an important consideration regarding these null findings relates to sample size. An alternative explanation posits that this study's small sample size may mean the data are not representative of the population. It is therefore possible that a larger sample may yield different results which might confirm the hypothesis. Thus, while it is possible that my results serve to disconfirm this hypothesis altogether, the above confounds and caveats preclude confidently assenting to this conclusion.

Dream Emotionality Findings

I hypothesised that the extent and intensity of negative emotional dream content would be highest for the PTSD hyperarousal group, lower for the PTSD non-hyperarousal group, and lowest for the healthy control group. The results of my statistical analyses tentatively confirmed this hypothesis for negative emotional intensity as assessed by the Domhoff (2000) rating scale, but did not confirm the hypothesis for the extent of negative emotional content as assessed by the LIWC programme.

For negative emotional intensity of dream reports, each of the PTSD groups were found to be significantly different from the healthy control group, indicating that PTSD participants as a whole had more intense negative dream content than healthy participants. This makes sense in terms of the literature on dream functionality. Dream content is purported to be contingent on waking emotional experience (Levin & Nielsen, 2007; Walker, 2009). Accordingly, heightened waking emotional distress is proposed to translate into more emotionally intense dream content (Levin & Nielsen, 2007). The fact that PTSD is characterized by considerable waking emotional distress therefore explains why (negative) intensity of dream emotions is higher in PTSD participants than in healthy controls. However, the PTSD groups were not found to the significantly different from each other in this regard. This is counterintuitive in light of the above literature, as prominent hyperarousal symptoms in PTSD represent a condition of further intensified waking emotional distress (Levin & Nielsen, 2007; Spoormaker & Montgomery, 2008). Yet, because the PTSD hyperarousal group had slightly more intense negative dream content than the PTSD non-hyperarousal group, we may expect that this difference might reach statistical significance with improvements in sample size and dream report count.

In contrast, no statistically significant between-group differences were found for dream emotionality content as assessed by the LIWC word-count software. Interestingly, differences in "anger"-related content and negative emotional content overall approached statistical significance. However, the differences in group means for these variables were not

suggestive of the hypothesized pattern. Indeed, for "anger" related content, the group mean for PTSD HYP+ was closer to that for HC than to that for PTSD HYP-, while for negative emotional content overall, PTSD HYP+ had the lowest relevant word count. The LIWC results are thus in stark contrast to the Domhoff (2000) scale results.

The discrepant results between these different measures might be due to issues in dream report provision for this sample. It has been postulated that chronic prominent hyperarousal symptoms may induce emotional numbing, which results in a diminished capacity to explicitly convey the extent of negativity in dreams (Germain, 2013; Levin & Nielsen, 2007). Word-count-focused content-analysis measures, such as the LIWC software, may therefore not reflect the degree of implicit negative emotions in PTSD HYP+ dreams as successfully as an intensity rating scale (Sikka et al., 2014). Additionally, the issue of language proficiency may have confounded dream emotionality analyses (LIWC more so than the intensity rating scale). English was the first language of only 12.2% of participants in this sample. Because participants' capacity for accurate, nuanced and extensive articulation in English may have been compromised, their dream reports may not have provided the sufficiently elaborate description of dream content that is required for accurate quantification of report emotionality (Sikka et al., 2014; van Wyk, 2013).

The Association between Hyperarousal, REM Disruption and Dream Emotionality

The results of the linear modelling did not confirm the third hypothesis. That is, the combination of the degree of prominent hyperarousal symptoms and of REM-related sleep disruptions did not successfully predict dream report emotionality. No REM-related variables were significantly or strongly correlated with negative emotional intensity (the only dream emotionality variable that demonstrated significant between-group differences). For the linear model that was constructed, the vast majority of the variance in negative emotional intensity was explained by group status alone; adding REM% to the model did not result in a significant contribution to predictions about intensity of negative emotions in dreams.

A possible reason for the disconfirmation of this hypothesis is the fact that around one quarter of vivid, emotional dreams do not occur during REM sleep (Solms, 2000). Moreover, the degree of prominent hyperarousal symptoms in PTSD has been correlated with architecture differences in other sleep stages (e.g. stage 1 non-REM) in which REM-like dreams do occur (Kobayashi et al., 2007; Solms, 2000; van Wyk, 2013). Thus, the possibility that some dreams with intense negative emotions may not have occurred during participants' REM sleep periods might account for the insignificant association between these variables.

Strengths, Limitations, and Directions for Future Research

Beyond aiming to address a key gap in the literature concerning sleep disturbances in PTSD, this study sought to improve upon the methodological shortcomings of previous research in this area. A considerable strength in this study was the recruitment of a more homogeneous sample through rigorously-controlled exclusion criteria. The potential confounding influence of other psychiatric and demographic factors in participants were minimised as far as possible. Moreover, the use of an exclusively female sample with a common traumatic experience met a resounding call from the extant literature on PTSD, as (1) women are an understudied PTSD population (Kobayashi et al., 2007), (2) nightmares are purported to occur more commonly and have more intense features and effects in women (Levin & Nielsen, 2007), and (3) sexual assault is the trauma most strongly associated with PTSD in South African women (Kaminer et al., 2008). Thus, this sample's particular composition not only improves this study's generalisability, but steers the investigation of PTSD and sleep disturbances towards a considerably vulnerable and important population.

A drawback of the strict exclusion criteria was the consequential small sample size. This study aimed to improve upon the PTSD group sample size used in van Wyk (2013) by using more lenient CAPS scoring rules, but this only raised the PTSD sample by 2 participants. Although power analyses indicated that my sample size would be sufficient to detect reliable statistical effects, the predominantly small effect sizes of the group comparison results indicate otherwise. Additionally, the sample constitution was limited by the fact that group sizes were unequal, as there were many more control participants than participants in each PTSD group.

Another kind of limitation relates to the possible effects of the laboratory environment on dreaming and dream recall. A reduction in both nightmare episodes and recall of dreams has often been observed for individuals undergoing PSG-monitored sleep in laboratories (Germain, 2013; Levin & Nielsen, 2007; Spoormaker & Montgomery). In this study, only 41.4% of the dream reports were related to dreams recalled after a night of PSG-monitored sleep. Not only does this indicate that laboratory rates of dream recall and nightmare episodes were not high, but it also demonstrates that most of the analysed dream reports were not related to the laboratory setting, and therefore not related to the observed REM sleep patterns therein. Considering this, I re-ran the dream emotionality analyses using only the dream reports related to the laboratory measures, but I found no difference in results (see Appendix E). Thus, although this is an important limitation, it is not the reason for my null findings.

A further limitation in this study was the absence of a depression-only comparison group. Because of the high rates of comorbid depression in PTSD-diagnosed individuals, having this group would have helped disentangle the effects of each disorder on sleep disturbances.

While a strength of this study was the use of multiple (even innovative) measures of dream report content, the usefulness of these dream report measures was limited by the language proficiency difficulties of participants. Moreover, the participants themselves did not provide ratings of the intensity of negative emotions in their dream reports, which would have been useful to these analyses (Sikka et al., 2014). Other potential limitations with data collection methods include a possibly insufficient number of dream reports (see, e.g, Domhoff, 2000) and of observation nights in terms of attenuating laboratory-setting effects. However, more dream report provision opportunities and observation nights were implemented than in previous studies (cf. Mellman et al., 2007; van Wyk, 2013).

Future research into the role played by hyperarousal symptom prominence in the relationship between REM sleep disruptions and nightmares in PTSD is urged to improve upon the foregoing shortcomings, while maintaining the methodological strengths of the current sampling process. Particularly important changes include an increase in sample size, the use of a depression-only comparison group, and a more reliable means to assess emotional dream content in non-native English speakers. Ambulatory kinds of PSG and extended habituation periods are recommended (Spoormaker & Montgomery, 2008), but the implementation of the former recommendations alone may provide sufficient clarity to the necessary further investigations into PTSD symptomatology and sleep disturbances.

Conclusion

This study did not lend support to the hypothesis that PTSD-diagnosed individuals with prominent hyperarousal symptoms would have the greatest REM-related sleep disruptions, in comparison to PTSD-diagnosed individuals without prominent hyperarousal symptoms and healthy controls. Additionally, I did not confirm the hypothesis that PTSD-diagnosed individuals with prominent hyperarousal symptoms would have more negative and intense dream content than their counterparts without prominent hyperarousal symptoms. Instead, I found that PTSD-diagnosed individuals as a whole had more negatively intense emotional dream content than healthy individuals – although this was only established with one measure of dream emotionality. Finally, this study did not confirm the hypothesis that the degree of prominent hyperarousal symptoms, combined with the degree of disrupted REM

sleep, would jointly predict the negativity and intensity of dream emotions. Although these hypotheses were disconfirmed, some methodological issues in this study preclude any definitive conclusions about sleep disturbances and PTSD symptomatology. Future research is urged to replicate this investigation with a larger sample, with more contextually-appropriate dream report materials, and with the use of a depression comparison group. Given the high rate of trauma exposure in South Africa (Kaminer et al., 2008), and given that nightmares and REM sleep disturbances significantly influence the pathogenesis and prognosis of PTSD (Insana et al., 2012; Pace-Schott et al., 2015), continued investigations into these psychopathological features are imperative to improve prevention and treatment strategies for posttraumatic stress disorder.

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Appendix A

Dream Recall Form

Age _____

Gender _____ **MOST RECENT DREAM**

Date Today _____

We would like you to write down the last dream you remember having, whether it was last night, last month, or last year. But first please tell us the date this dream occurred: _____. Then tell us what time of day you think you recalled it: _____. Then tell us where you were when you recalled it: _____.

Please describe the dream exactly and as fully as you remember it. Your report should contain, whenever possible: a description of the setting of the dream, whether it was familiar to you or not; a description of the people, their age, sex, and relationship to you; and any animals that appeared in the dream. If possible, describe your feelings during the dream and whether it was pleasant or unpleasant. Be sure to tell exactly what happened during the dream to you and the other characters. Continue your report on the other side and on additional sheets if necessary.

Appendix B
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 Investigator (the person 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
"Neutral and Affective Memory Processing during Sleep in PTSD"

3. Principal Investigator and Telephone Number(s)

Malgorzata (Gosia) Lipinska
University of Cape Town (UCT)
Contact number: 084 621 0683

4. What is the purpose of this research study?

This research aims to investigate the whether disrupted sleep helps to explain memory problems in Post Traumatic Stress Disorder

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

In this experiment, you will be called in for 4 study sessions – 2 during the day and 2 spanning a whole night.

Before commencing the actual study, you will undergo a screening process whereby the Principal Investigator listed in # 3 of this form or her 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 study session will take place during the day. You will be asked to come to UCT (PD Hahn building) at 9.00am in the morning for a study session of approximately 1.5 hours. A urine sample will be taken before the session begins. **This urine sample will measure MHPG (3-Methoxy-4-Hydroxyphenylglycol) as a measure of central nervous system noradrenergic activity. MHPG is a metabolite of noradrenaline which reflects how much noradrenaline is active in your body. Noradrenaline is a neurotransmitter which is implicated in the flight or fight response as well as emotional learning.** You will be asked to void into a plastic container, which will be used for scientific analysis. First you will be presented with some information that is part of a memory task. Secondly, as part of the session you will be asked to view some pictures. During this task a small device will be attached to your finger. This measures minute electrical changes on your skin. Some electrodes will also be placed on your chest to measure heart rate. This will conclude the morning session. You will be asked to return to UCT 8 hours later for a second session, which will follow exactly the same procedure.

The third session will be a sleep adaptation night at UCT's sleep laboratory. This session will be schedules 48 hours after the first session. You will be asked to come in at 10pm. Transport will be provided if you require it. During this session you will simply get used to sleeping at the laboratory attached to all 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. They will be available to you for assistance at any time. In the morning all the equipment will

The fourth session will also take place at the sleep laboratory. It will be scheduled for the night after the adaptation night and will start at 8pm. During this session the testing procedure described in session 1 will be followed. That is firstly a urine sample will be taken, secondly a memory test will be administered, and thirdly some pictures will be presented alongside measures of skin conductance and heart rate. This procedure will also be followed after an 8-hour period of sleep. After testing you will again be hooked up to the polysomnograph. In the morning all the equipment will be removed and the morning testing session will begin (following the same procedure as that of the evening session).

You may also be asked to begin with the adaptation night, followed by the sleep night, followed 48 hours later by the day time testing described as the first session.

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 Investigator 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 daytime session – one in the morning and one 8 hours later in the afternoon (each about 1.5 hours) plus 2 consecutive nights.

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

75

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 (including the 1 day-time session and 2 night-time sessions) will not delve into past memories and traumatic events experienced, 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. 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 3 main study parts (daytime session 1 and 2, the adaptation night and the sleep study night). Thus if you participate in the research for 3 nights you will receive R450.

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 Investigator 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 PhD 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 C
Ethical Approval Documents

UNIVERSITY OF CAPE TOWN

FACULTY OF HUMANITIES

DC: HUM /

PROPOSAL APPROVAL FORM

DOCTORATE (A research proposal must accompany this form) X	RESEARCH MASTERS (A research proposal must accompany this form)	C/W MASTERS

SECTION A: (To be completed by candidate)
Please complete this form and return it to the Faculty Office once you have obtained the signatures of the supervisor(s) and Head of Department.

Surname	Lipinska				First Name(s)	Malgorzata	
Title	Mr.	Ms.	Mrs. x	Miss	Student No	LPNMAL001	
Address	Mount Prospect Farm, Pagasulei Road, Constantia, 7806						
Telephone(Home	0846210683				Work/Cell	← same	

Note: Your UCT Email address is the default email address for all official communication – make sure that you access it regularly. please! send to gosia@runlikeagirl.co.za

Department	Psychology
Title of Dissertation:	Memory Processing in Post-Traumatic Stress Disorder

Qualifications held			
Degree/Diploma	Major(s) &) Subjects	Month/Year awarded	University
Masters in Psychological Research	Psychology	June 2011	University of Cape Town

Signature of candidate: _____ Date: 05.02.2012

SECTION B:

	Name	Signature	Date
Supervisor	Kevin Thomas		FEB 5, 2012
Co-supervisor (if applicable)	Debbie Kaminer		6/2/2013
HOD	Mark Solms		6/12/2013
Deputy-Dean: Research			
Ethics approval obtained where applicable	on behalf of Departmental Ethics Committee		6/2/2013

UNIVERSITY OF CAPE TOWN

FACULTY OF HEALTH SCIENCES
Human Research Ethics Committee

FHS016: Annual Progress Report I Renewal

HREC office use only (FWA00001637; IRB00001938) This serves as notification of annual approval, including any documentation described below.			
☑ Approved	Annual progress report	Approved until/next renewal date	30/03/2016
☐ Not approved	See attached comments		
Signature Chairperson of the HREC		Date Signed	4/2/15

Comments to PI from the HREC

HUMAN RESEARCH ETHICS COMMITTEE
-4 FEB 2015
HEALTH SCIENCES FACULTY
UNIVERSITY OF CAPE TOWN

Principal Investigator to complete the following:

1. Protocol information

Date (when submitting this form)	20.01.2015			
HREC REF Number	428/2013	Current Ethics Approval was granted		2014
Protocol title	Memory Processing During Sleep in posttraumatic Stress Disorder			
Protocol number (if applicable)				
Are there any sub-studies linked to this study?	□ Yes □X No			
If yes, could you please provide the HREC Refs for all sub-studies? Note: A separate FHS016 must be submitted for each sub-study.				
Principal Investigator	Malgorzata Lipinska			
Department / Office Internal Mall Address	Psychology Department/ room 2.04			

1.1 Does this protocol receive US Federal funding?	□ Yes	□x No
1.2 If the study receives US Federal Funding, does the annual report require full committee approval?	□ Yes	□X No
1.3 Has sponsorship of this study changed? If yes, please attach a revised summary of the budget.	□ Yes	□x No

Appendix D

EOG Management Processes for REM Density Analyses

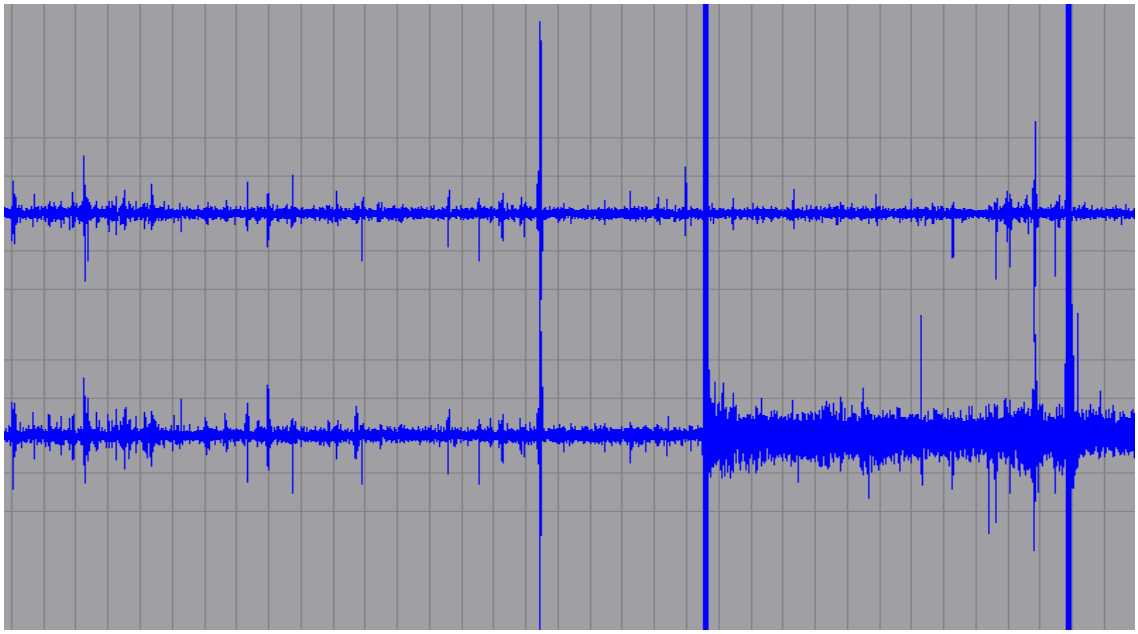
Dual-Threshold Detection Method

The dual-threshold method specifies that an EOG signal must have peak above 30μV and a nadir below 10μV in order for an EM to be detected. Each 2-second period of REM sleep was dichotomously scored as with or without this kind of EM. In order to minimise false-positive detections of EMs, the EOG channels were notch filtered at 50Hz and band pass filtered between 0.3 and 30Hz. These filtering/de-noising techniques reduced power-line interference and minimised (but not eliminated) wavelet artefacts, respectively (Moore et al., 2013). Elimination of artefactual EOG waveforms in REM stages was carried out manually in accordance with Brown et al. (2006) and this process is explained in the following paragraphs. Once these artefacts were eliminated, REM density was calculated for each 30-second epoch as the percentage of 2-second mini-epochs with detected EMs. Thus, the overall REM density value per participant is the average of the REM density percentages across all of their 30-second REM epochs (Moore et al., 2013).

Manual Elimination of EOG Artefacts Procedure

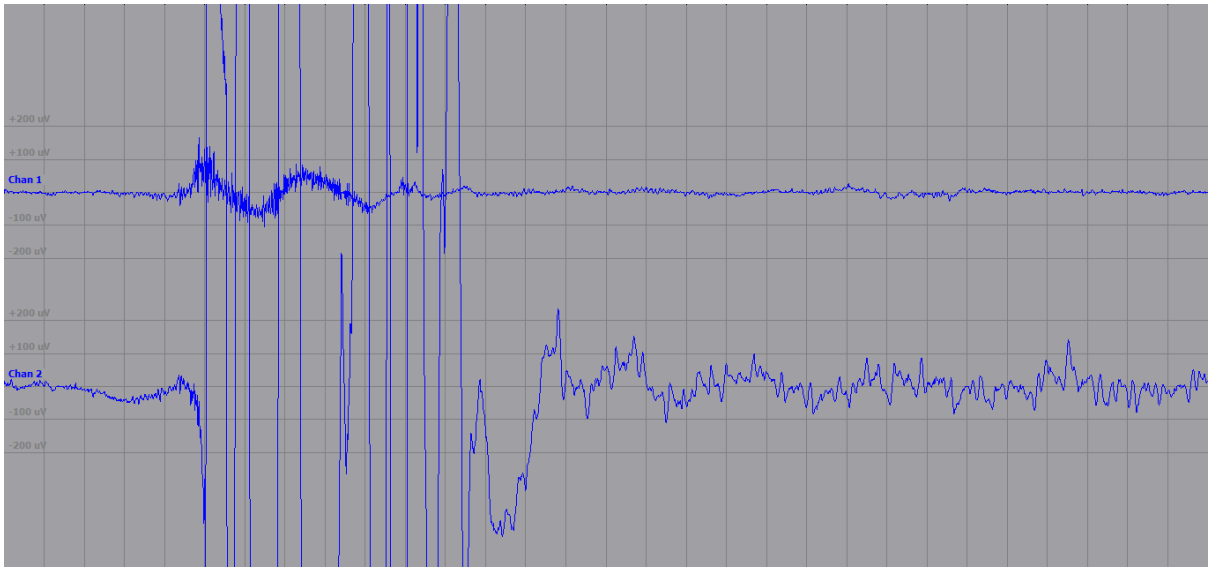
The external programmer produced an EDF (i.e. European Data Format) file each participant's two EOG channels across the night. These channels only represented EOG signals over REM sleep periods. Thus, in each EDF file, we simultaneously observed the two EOG channels, which represented a concatenation of EOG signals across all REM periods for that night's sleep. The EDFs were inspected using EDFBrowser version 1.58.

I visually compared the two EOG channels to inspect if, at any point, either one significantly diverged from the pattern of activity observed in the other. An example of this divergence is shown in the figure below. The activity of the EOGs mirror each other in the first half of this figure, but in the second half, they diverge, with the second (lower) EOG providing an atypically high signal. This is an indication of possible artefact.



Note. The timescale of this EOG record is 45 minutes. Each square block about the channel represents 50µV in amplitude.

If this divergence lasted longer than 30s, then the display was magnified in order to better inspect the difference. If the divergent signal in question was not behaving appropriately (e.g. exceeded 1000µV and/or did not present with the typical appearance of an EOG waveform; Brown et al., 2006), then this was considered an artefact. The figure below represents a scale magnification of the same example artefactual EOG. Evidently, the second EOG is registering signal artefacts in this instance.



Note. The timescale for this figure is 30s. The amplitude is the same as that in the previous figure.

Once these artefactual EOG patterns were detected, the REM density figures for these epochs were deleted, as they were deemed unreliable. These false REM density figures were therefore not included in the averaging out of REM densities across all epochs.

Every participant's REM EOG signals were examined in this way. Artefacts were identified in, and removed from, seven out of the 40 records (one participant had no REM sleep). Artefacts such as those illustrated above are most likely due to a dislodged EOG electrode at the time in question.

Appendix E

Results of Dream Emotionality One-Way ANOVAs without Using the 1st Dream Report

Dream Report Emotionality Excluding 1st Reports: Descriptive Statistics and Results of One-Way ANOVAs

	Group								
	PTSD HYP+		PTSD HYP-		Healthy Control				
Variable	(*n* = 5)		(*n* = 6)		(*n* = 11)		*F*	*p*	EES
LIWC Anxious Emotions	1.18	(1.46)	.00	(.00)	.76	(1.12)	1.70	.210	.15
LIWC Anger Emotions	.40	(.65)	1.53	(1.69)	1.03	(1.53)	.84	.445	.08
LIWC Sad Emotions	.33	(.47)	.32	(.35)	.44	(.87)	.08	.928	.01
LIWC Negative Emotions	2.09	(1.60)	2.86	2.12	2.85	(2.43)	.24	.793	.02
LIWC Positive Emotions	2.32	(1.33)	1.31	(.82)	1.30	(1.41)	1.24	.311	.12
LIWC Overall Affect	4.41	(1.89)	4.17	(2.65)	4.16	(2.93)	.02	.984	<.01
Emotional Intensity Ratings	-6.50	(3.83)	-4.90	(1.52)	-2.68	(3.12)	3.14	.067	.25

Note. Means are presented with standard deviations in parentheses for all variables. All LIWC variables are expressed as percentages. Emotional severity ratings are on a scale from – 10 (very negative) to +10 (very positive). All variables represent mean values across the last 2 dream report opportunities. Group sizes are reduced due to excluding participants who did not provide any laboratory-related reports. EES = estimate of effect size (in this case, η^2). df = 2, 19 for all F tests.