

The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.

The Relationship between PTSD, Hypervigilance, and Disordered Sleep

Mariza van Wyk

VWYMAR015

A minor dissertation submitted in partial fulfillment of the requirements for the award of the degree of Master of Arts in Psychological Research

Department of Psychology

Faculty of the Humanities

University of Cape Town

COMPULSORY DECLARATION:

This work has not been previously submitted in whole, or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work, or works, of other people has been attributed, and has been cited and referenced.

Signature:

Date:

## ACKNOWLEDGEMENTS

I want to extend a note of thanks to my supervisor, Professor Mark Solms, for all his invaluable input and continuing support. I would also like to thank my research partner, Gosia Lipinska, for her innumerable contributions to this project, Dr Kevin Thomas for his conceptual input, and Dr Debbie Kaminer for generously providing supervision and support. To Marlene Gounder, Jan Top, and Ridwana Timol who taught me the principles of sleep research. Also a sincere thanks to the team at Rape Crisis who played a major part in making this research possible, and to all the women who participated in this research whose courage and endurance touched me deeply.

University of Cape Town

## Table of Contents

Acknowledgements-----	2
List of figures-----	6
List of Tables-----	7
Abstract-----	8
Introduction-----	10
PTSD in South Africa-----	10
Disordered Sleep in PTSD-----	10
Findings of Objective Measures of Sleep Quality-----	11
Findings of Subjective Measures of Sleep Quality-----	12
The Neurobiology of the Development of PTSD-----	13
Hypervigilance and Hyperarousal in PTSD-----	14
Nightmares in PTSD-----	17
Methodological Constraints evident in the PTSD Literature-----	18
Specific Aims and Hypotheses-----	21
Methods-----	23
Design and Setting-----	23
Participants-----	23
Exclusion Criteria-----	23
Materials and Apparatus-----	28
Diagnostic and Screening Instruments-----	28
Experimental Measures-----	30
Sleep Equipment-----	31
Procedure-----	32

Ethical Considerations-----	33
Statistical Analyses-----	34
Psychiatric Characteristics of the Sample-----	34
Hypothesis 1-----	34
Hypothesis 2-----	36
Results-----	37
Psychiatric Characteristics of the Sample-----	37
Testing Hypothesis 1: Between-group Differences in Objective and Subjective Measures of Sleep Quality-----	39
Trends in Sleep Data: Cell Mean Plots-----	41
Inferential Statistical Analyses of Objective Measures of Sleep Quality-----	48
Inferential Statistical Analyses of Subjective Measures of Sleep Quality-----	51
Testing Hypothesis 2: Dream Content and Scores-----	52
Discussion-----	55
Sleep-related Variables and Sleep Parameters Included in the Study-----	57
Objective Measures of Sleep Quality-----	59
Trends in Cell Mean Plots-----	59
Parametric Testing-----	61
Planned Comparison 1-----	61
Planned Comparison 2-----	62
Planned Comparison 3-----	63
Subjective Measure of Sleep Quality: The PSQI-----	63
Dream Content Scores-----	65
Limitations and Directions for Future Research-----	68
Conclusion-----	70

References-----	72
Appendix A: DSM-IV Criteria for PTSD-----	79
Appendix B: Most Recent Dream Form-----	81
Appendix C: Dream Rating Instructions-----	82
Appendix D: Socio-economic and Demographic Questionnaire-----	83
Appendix E: Informed Consent Form-----	85
Appendix F: PSQI-----	90
Appendix G: Sleep-related Variables: Results of the Kolmogorov-Smirnov Test of Normality-----	94

University of Cape Town

## List of figures

Figure 1: Exclusions-----	23
Figure 2: The Logic Behind Conducting Orthogonal Planned Comparisons-----	33
Figure 3: Group Mean Data for the Sleep-related Variable Sleep Efficiency-----	39
Figure 4: Group Mean Data for the Sleep-related Variable Sleep Latency-----	40
Figure 5: Group Mean Data for the Sleep-related Variable Awakenings-----	41
Figure 6: Group Mean Data for the Sleep-related Variable Wake After Sleep Onset-----	42
Figure 7: Group Mean Data for the Sleep-related Variable REM%-----	43
Figure 8: Group Mean Data for the Sleep-related Variable REM Latency-----	44
Figure 9: Group Mean Data for the Sleep-related Variable SWS%-----	45
Figure 10: Group Mean Data for the Subjective Measure of Sleep Quality-----	49
Figure 11: Group Mean Data for Dream Content Scores-----	51

University of Cape Town

## List of Tables

Table 1: Demographic Characteristics of the Sample-----	26
Table 2: Results of Between-group analyses of CAPS scores-----	35
Table 3: Independent t-tests for BDI-II Scores-----	37
Table 4: Sleep-related Variables: Descriptive Statistics and Results from Between-group comparisons-----	46
Table 5: Results of Orthogonal Planned Comparisons for Objective Measures of Sleep Quality-----	48

University of Cape Town

## Abstract

Disordered sleep in PTSD constitutes a major component of the presenting symptomatology. However, the literature on PTSD and sleep is characterized by discrepancies across studies, especially due to the fact that some use objective and some use subjective measures of sleep quality. As a result, disordered sleep and its underlying mechanism have been ambiguously characterized in PTSD. Our research focused on the link between PTSD and disordered sleep, using both objective and subjective measures of sleep quality. Specifically, we investigated hypervigilance (one of the three symptom clusters in the PTSD diagnosis) as an underlying mechanism of this link. We also investigated whether hypervigilance affects dream content and themes in individuals with PTSD. We recruited four groups of participants: 9 individuals diagnosed with PTSD with prominent hypervigilance symptoms (HYP+); 10 individuals diagnosed with PTSD without prominent hypervigilance symptoms (HYP-); 14 individuals diagnosed with depression (DEP), and 16 healthy controls (CON). (The DEP group controlled for the frequent comorbidity of depression in PTSD). Each individual spent 1 night in our sleep laboratory undergoing sleep-adapted EEG recordings. We measured sleep latency, awakenings, time spent awake after sleep onset, sleep efficiency, REM%, REM latency and SWS%. We also obtained self-reports of general quality of sleep and two reports of most recent dreams (one preceding the experiment and one upon conclusion of the experiment). Results were analyzed using one-way ANOVA for the objective and subjective measures of sleep quality, as well as for the dream content scores, while orthogonal planned comparisons were run in order to elucidate the nature and extent of differences detected by the overall ANOVA model. Data from objective measures of sleep quality did not statistically confirm our hypothesis, whereas data from subjective measures of sleep quality yielded significant results. Data from the dream content scores approached significance.

## INTRODUCTION

Post-traumatic stress disorder (PTSD) is a highly prevalent anxiety disorder in South African society. This may be due to individuals being exposed to racial abuse and political violence, gender inequality, criminal violence, including rape and murder, and extreme poverty (Stein et al., 2008). PTSD is likely to be a psychiatric consequence of potentially traumatizing events for a significant portion of the at-risk population (Edwards, 2005).

According to the American Psychiatric Association (2000), PTSD is defined as a psychopathological reaction to a traumatic event in which a person experienced or witnessed actual or threatened death, serious injury, or a threat to the physical integrity of self or others. The individual's response may include fear, helplessness, or horror. The formal diagnostic criteria for PTSD, as established by the fourth revision of the Diagnostic and Statistical Manual (DSM-IV-TR; American Psychiatric Association [APA], 2000) are presented in Appendix A. As can be seen, the criteria of the formal diagnosis indicate that the symptoms of PTSD are grouped into three major clusters: re-experiencing symptoms, avoidance symptoms, and hyperarousal symptoms.

Symptom cluster B, the re-experiencing or intrusive recollection symptom cluster, involves distressing images, thoughts, or perceptions related to the traumatic event. This includes feeling as if the traumatic event were reoccurring in the form of hallucinations or illusions, for example. Extreme psychological distress or physiological reactivity to internal or external cues that remind the individual of the event may possibly be present.

Cluster C includes the numbing of general responsiveness, not present before the trauma, and is related to symptoms that include efforts to avoid thoughts, feelings, or conversations associated with the trauma. In addition, people, places, or activities that are reminders of the trauma are also avoided. The recollection of important aspects of the trauma may possibly be impaired as well as noticeably decreased interest or participation in previously pleasurable activities. Furthermore, prominent feelings of detachment or estrangement from others may also be present.

The hyperarousal symptom cluster, cluster D, is characterised by persistent symptoms of increased arousal that typically includes difficulty falling or staying asleep, an increase in irritability or outbursts of anger, difficulty concentrating, accentuated startle response, and the

presence of hypervigilance. It is required that these symptoms must not be present before the experienced trauma.

### **PTSD in South Africa**

PTSD is a highly prevalent disorder in South Africa that most frequently manifests after the experience of physical, and especially, sexual trauma (Carey, Stein, Zungu-Dirwayi, & Seedat, 2003). Individuals in South Africa are particularly vulnerable to being exposed to such a traumatic event due mainly to an entrenched culture of pervasive violence, racial discrimination, and socio-economic and gender inequality. It is estimated that over a third of all South Africans have been exposed to a violent traumatic event in their lifetime (Kaminer, Grimsrud, Myer, Stein, & Williams, 2008). The results of the most comprehensive psychiatric epidemiological study to date, the South Africa Stress and Health (SASH) study, show that anxiety disorders, including PTSD, were the most prevalent class disorder at 15.8% with the average age of onset at 32 years-old. Women showed a significantly higher rate of anxiety disorders when compared to men. The associations with gender, such as the higher rate of anxiety disorders, are consistent with results of other countries. Other studies have further supported, through the use of discriminant function analysis, that the female gender, in addition to a history of sexual violence, are significantly associated with the development of PTSD (Olley, Zeier, Seedat, & Stein, 2005). In addition to the SASH study, a study conducted from a township primary healthcare clinic found that PTSD was one of the most common diagnoses in South Africa with a prevalence rate of 20% (Carey, Stein, Zungu-Dirwayi, & Seedat, 2003). Physical assault and rape serves as some of the most prominent predictors of PTSD onset.

### **Disordered Sleep in PTSD**

A major feature of PTSD is disordered sleep. The relationship between the experience of trauma and fluctuations in sleep architecture suggests that sleep disturbances constitute a normal initial reaction to traumatic experiences. If these disturbances become entrenched, however, a psychopathological stress disorder may develop (Harvey, Jones, & Schmidt, 2003). In general, the term “disordered sleep” refers to a broad range of disrupted sleep behaviour occurring on a regular basis, and causing daytime dysfunction or distress (Roth, 2007).

**Findings of objective measures of sleep quality.** The literature on PTSD and disordered sleep is characterized by discrepant findings across studies in terms of utilizing either objective or subjective measures of sleep quality. Pillar, Malhotra, & Lavie (2000) in a review article propose that when sleep quality is measured objectively via polysomnography (PSG), one of the sleep parameters affected is non-rapid eye movement (NREM) sleep. More specifically, they argue that disruptions in sleep architecture of individuals with PTSD relate to disturbances in slow-wave sleep (SWS)<sup>1</sup>. However, findings are divided in terms of the exact nature of these SWS disturbances. Some studies report no changes in SWS percentage when compared to controls according to these authors, while other studies have indeed detected abnormalities in SWS percentage when compared to controls (Hefez et al., 1987; Glaubman et al., 1990). With regard to rapid eye-movement (REM) sleep abnormalities, studies have inconsistently found changes in these sleep parameters – there seems to be some evidence in the literature that is indicative of an increase in REM sleep latency<sup>2</sup> and REM sleep density<sup>3</sup> (Kramer, & Kinney, 1988), with variably decreased or increased total REM sleep time in the PTSD population (Pillar, Malhorta, & Lavie, 2000). However, the discrepancies in results of the presence or absence of NREM sleep and REM sleep abnormalities can be ascribed to studies being confounded by small sample sizes, insufficient data, proximity to the traumatic event and the sleep data being greatly affected by comorbid psychiatric diagnoses (Dow, Kelsoe, & Gillin, 1996). Major depression is especially known to exert a prominent influence on sleep architecture in the context of PTSD, as it reliably results in changes in both NREM and REM sleep.

Boarts & Delahanty (2007) conducted a meta-analysis in an attempt to elucidate the kinds of sleep disturbances implicated in the sleep architecture of individuals with PTSD when compared to healthy controls. The meta-analysis consisted of 20 studies using PSG to measure sleep architecture. They also investigated the effect of possible moderating variables that have been reported as confounds in many studies. The analyses revealed that individuals with PTSD have more stage 1 (light) sleep and less SWS. Furthermore, results also revealed that PTSD individuals have greater REM density. The study also found a moderating effect on sleep architecture of different variables such as age, gender, comorbid depression and substance abuse

---

<sup>1</sup> SWS consists of stage 3 and stage 4 sleep, also known as deep sleep. Non-rapid eye movement (NREM) sleep consists of SWS along with sleep stages 1 and 2.

<sup>2</sup> The period between sleep onset and the onset of the first REM sleep period.

<sup>3</sup> The frequency of eye movements during REM sleep.

disorders. Therefore it provides evidence for the presence of certain kinds of NREM sleep and REM sleep disruptions. It also highlights the importance of conducting studies that control for confounds in order to obtain an accurate representation of sleep architecture in PTSD individuals.

In terms of abnormalities of other sleep parameters using objective measures, the most consistently reported abnormalities relate to reduced total sleep time (TST) and reduced sleep efficiency (SE) (Dow, Kelsoe, & Gillin, 1996), as well as time spent awake after sleep onset (WASO) (Mellman, 1997). However, other studies have failed to reliably detect objective evidence of significant differences in the sleep architecture of individuals with PTSD (Ross et al., 1994; Klein, Koren, Arnon, & Lavie, 2002; Breslau, Roth, Burduvali, Kapke, Schultz, & Roehrs, 2004).

**Findings of subjective measures of sleep quality.** With regard to utilizing subjective measures to measure differences in sleep quality, there seems to be greater consensus concerning the presence and nature of sleep disturbances as opposed to the findings generated by objective measures of sleep quality. Findings consistently indicate that the sleep quality of individuals with PTSD differs markedly in terms of SE, sleep latency<sup>4</sup>, sleep maintenance and number of awakenings when compared to healthy controls (Engdahl, Eberly, Hurwitz, Mahowald, & Blake, 2000). As such, sleep in individuals diagnosed with PTSD is often characterized variably by objective measures as containing both NREM and REM abnormalities, while subjective measures have characterized the sleep of individuals with PTSD as being much more fragmented and less restorative than normal sleep in healthy individuals.

From the evidence presented above, it is clear that reliably identifying the specific changes that take place in the sleep architecture of individuals with PTSD using PSG is a contentious issue in the literature. There is little agreement on the issue of characterizing the typical sleep architecture associated with PTSD, possibly due to several methodological constraints across studies. Therefore it is important to design a study that addresses the major confounds evident in the PTSD literature, in order to be able to potentially identify the underlying mechanism mediating the relationship between PTSD and disordered sleep. One way of delineating a possible underlying mechanism in this regard entails looking more closely at the specific symptom clusters that form part of a PTSD diagnosis. Individuals with PTSD present

---

<sup>4</sup> The time that elapses from wakefulness to sleep onset

with varying degrees of symptom severity across the three different clusters. These clusters include the re-experiencing and intrusive recollection cluster, the avoidance and numbing cluster, and the hyperarousal cluster. We propose that the hyperarousal cluster, that includes hypervigilance, may be of particular importance in mediating the relationship between PTSD and disordered sleep. Furthermore, to the knowledge of the author, hypervigilance as a possible causative mechanism underlying disordered sleep in PTSD has not been investigated before. The hyperarousal cluster (that includes hypervigilance symptoms) was identified because it is believed that hyperarousal is caused by dysfunctional neurobiological structures and associated neurotransmitters that are also influential in the workings of the sleep/wake cycle. In order to investigate this relationship properly, studies indicate that the physiological processes at work since exposure to the trauma must be taken into account (Mellman, Knorr, Pigeon, Leiter, & Akay, 2004).

### **The Neurobiology of the Development of PTSD**

The occurrence of an environmental stressor has prominent consequences on a physiological level. The cognitive assessment of a real or imagined threat orchestrates the physiological and behavioural responses to this potential threat. The brain plays an especially important role as it releases certain hormones and neurotransmitters in an attempt to adapt to the stressful circumstances (Vanlallie, 2002). Certain brain structures are implicated in this process of adaptation; these include the sympathoadrenal system (SAS) and the hypothalamic-pituitary-adrenocortical (HPA) axis, where the hypothalamic paraventricular nucleus (PVN) plays an important role in the central regulation of the HPA axis. In the face of danger, an acute activation of the SAS occurs, which results in increased production of epinephrine and norepinephrine (also known as adrenaline and noradrenaline). Acute activation of the HPA axis results in the increased secretion of corticotropin-releasing hormone (CRH) and arginine vasopressin (AVP). This secretion suppresses urine production; influences cardiovascular function; and reportedly elevates mood, memory, and selective attention. CRH also stimulates the secretion of adrenocorticotrophic hormone (ACTH), stimulating the adrenal cortex to release glucocorticoids.

Glucocorticoids play an imperative role in the modulation of the normal stress response in the HPA axis. However, the excessive and prolonged release of glucocorticoids in the face of a persistent stressor (as in the case of individuals with PTSD), often leads to damaging effects on

the HPA axis and its related structures (Bremmer, 1999). It is especially the hippocampus, as one such related structure, that is susceptible to these damaging effects, as it has a rich concentration of glucocorticoid receptors. Long-term stress can lead to hippocampal atrophy, with consequent effects on the HPA axis, as the hippocampus plays an important inhibitory role on the workings of the HPA axis. Along with the hippocampus being affected by environmental stressors, the amygdala is also an important structure that is involved in neuroendocrine abnormalities in the face of trauma (Yehuda, 2002). The amygdala plays a critical role in terms of regulating stress, anxiety, and the fear response. Furthermore, it also plays a prominent role in emotional processing and memory consolidation. In contrast to the hippocampus that functions as an inhibitory mechanism on the HPA axis, the amygdala channels HPA axis-excitatory information as it instigates behavioural and cardiovascular responses to stress via different pathways. In other words, the glucocorticoid receptors potentiate rather than inhibit HPA axis responses (Herman & Cullinan, 1997; Herman, Ostrander, Mueller, & Figueiredo, 2005). One pathway of activation, as mentioned before, relates to the activation of the sympathetic nervous system leading to the secretion of norepinephrine from an extensive network of synapses and epinephrine from the adrenal medulla (van Stegeren et al., 2007). It is especially emotional stimuli that exert a pertinent influence on the norepinephrine pathway of activation in the amygdala. Therefore, prolonged activation from an emotional stressor could lead to the excessive release of norepinephrine, which could result in the amygdala becoming impaired and therefore interfering with the processes it is involved with.

The development of PTSD is facilitated by an inability to contain this physiological stress response. This defective stress response is mediated by the increased workings of the SAS, HPA axis, and the damaged hippocampus and amygdala. The combined effects of these systems, in particular the defective amygdala, lead to a state of hypervigilance in individuals with PTSD.

### **Hypervigilance and Hyperarousal in PTSD**

Hypervigilance in the context of PTSD refers to two related physiological conditions: the fear response and the physiological state of hyperarousal. The former is associated with the previously mentioned functions of the amygdala relating to the regulation of stress and anxiety. When this structure is damaged, for example by the presence of intense chronic psychological stress as in the case of PTSD, the threshold of activation of the startle response is decreased. This

decreased threshold leads to a situation where perpetual fear of benign stimuli can become cognitively conditioned and result in the physiological state of hyperarousal (Kim & Gorman, 2005). This state features higher respiratory rates, tachycardia, increased movement, and heightened muscle tension (Fuller et al., 1994). Hypervigilance symptoms, including symptoms influenced by these physiological processes, are reflected in the diagnostic criteria for PTSD, which include irritability and angry outbursts, problems with concentration, and an augmented startle response (DSM-IV-TR; APA, 2000).

Hypervigilance and the physiological mechanisms involved in hyperarousal further influence sleep. Hypervigilance involves the inability to adjust arousal levels, which may result in difficulty falling asleep as well as recurrent awakenings (Fuller et al., 1994). This relates to a dysregulation of arousal levels in terms of non-diminished noradrenergic activity (stimulated by or secreting norepinephrine) at night (Mellman, Kumar, Kulick-Bell, Kumar, & Nolan, 1995). In support of the influential properties of noradrenergic activity on sleep architecture, Raskind et al. (2003) conducted a double-blind, placebo controlled trial evaluating the effects of prazosin (an antagonist to the actions of norepinephrine) to 10 individuals with combat-related PTSD over a period of 20 weeks. There were significant improvements in terms of recurrent distressing dreams, as well as an improvement in insomnia symptoms, especially regarding sleep maintenance. These findings were supported by a study conducted by Taylor, & Raskind (2002). These authors reported a moderate to marked improvement in PTSD symptoms, especially in terms of sleep disturbances and nightmares. Clonidine (which inhibits the secretion of norepinephrine), has long been identified as another effective agent in treating symptoms related to hyperarousal, hypervigilance, disordered sleep, an augmented startle response and nightmares in individuals diagnosed with PTSD (Kolb, Burris, & Griffiths, 1984).

In PTSD, there is sometimes a compensatory physiological mechanism present in order to mitigate the relationship between hypervigilance, hyperarousal, and the re-experiencing of trauma through dreaming, which is called the *hyperarousal hypothesis*. The hyperarousal hypothesis relates to what is known as a 'deepened' state of sleep. A deepening of sleep is the opposite of 'light' sleep. Light sleep means that individuals are particularly sensitive to environmental stimuli. Therefore, a deepened sleep state implies that individuals are particularly insensitive to environmental stimuli (Pillar et al., 2000). The hyperarousal hypothesis suggests

that when an individual is hyperaroused, as is the case in PTSD individuals with prominent hypervigilance symptoms, there is an increased pressure to enter REM sleep.

Paradoxically, it appears that a 'deepening' of sleep is able to coexist with hyperarousal in PTSD individuals because when these individuals finally do fall asleep, their sleep is significantly deeper. This deepened sleep state may lead to a higher awakening threshold; that is it is increasingly difficult to wake these individuals once they have fallen asleep (Pillar et al., 2000), while it seems that this is especially relevant in terms of REM sleep.

In testing the hyperarousal hypothesis, 12 veterans diagnosed with PTSD and 12 healthy control veterans were monitored using PSG. Starting from the second entry into REM sleep, gradually increasing clicking sounds were played until the participant woke up. PTSD participants showed higher awakening thresholds. Furthermore, these findings suggest that PTSD participants are harder to wake because they use this deep sleep as a blocking mechanism to suppress anxiety-provoking material, related to the experienced trauma, from penetrating the sleep cycle (Lavie et al., 1998). However, the presence of anxiety-provoking material often manifests as nightmares about the traumatic event in individuals with PTSD. This occurrence is often perpetuated by the over-consolidation of memories and dreaming that occurs during REM sleep via the previously discussed mechanisms of an overactive amygdala and increased noradrenergic activity, for example.

In order to understand the relationship between REM sleep, the functioning of the amygdala and the secretion of norepinephrine, one needs to consider the mechanisms underlying the occurrence of REM sleep. The onset of REM sleep in healthy individuals is identifiable by increased cholinergic activity and diminished aminergic activity (norepinephrine falls in the latter category). Furthermore, there is also increased regional cerebral metabolic activity and blood flow in the amygdala and associated limbic and paralimbic structures (Germain, Buysse, & Nofzinger, 2008). REM sleep is regarded as an endogenous state of heightened activity in the emotional centers of the brain. It is believed that structural and functional abnormalities in the amygdala and other limbic and paralimbic structures might subserve the pathophysiology of PTSD in relation to sleep architecture. More specifically, an overactive amygdala can result in REM sleep interruptions, for instance interfering with the length of REM sleep periods (Spoormaker & Montgomery, 2008). Therefore, it seems feasible that an impaired amygdala could possibly play a causative role in disrupted REM sleep in PTSD (Kim and Hamann, 2007).

REM sleep is the sleep stage in which the majority of dreams occur (Mellman, David, Bustamante, Torres, & Fins, 2001). With emotional processing being disrupted during REM sleep due to an impaired amygdala, memory-consolidation in PTSD individuals is also affected (Bryant, Marosszeky, Crooks, & Gurka, 2000). One proposed mechanism of disruption in this regard relates to the hypothesis that REM sleep accentuates the altered function of the amygdala and interferes with the functioning of the medial frontal cortex in individuals with PTSD. Germain, Buysse, & Nofzinger (2008) propose that this could underlie the occurrence of nightmares. More specifically, neuroimaging studies point to the presence of increased amygdalar activity being especially influential in emotional memory formation during REM sleep (Maquet et al., 1996). Increased levels of norepinephrine can cause the over consolidation of memories during REM sleep, which allows the traumatic event experienced by PTSD individuals to be repeatedly replayed during dreaming (Southwick et al., 1999 ). This over-consolidation of memories can result in persistent flashbacks and repetitive nightmares, for example. A positive feedback loop<sup>5</sup> may be established from the repetition of the traumatic event and the simultaneous release of norepinephrine.

### **Nightmares in PTSD**

The specific features of nightmares include dreams of a terrifying nature accompanied by threats to survival, safety, or self-worth that often result in awakenings. Such dreams frequently produce feelings of anxiety, anger, and grief (Spoormaker, Schredl, & Van den Bout, 2005). According to certain criteria, nightmares are distinguishable from bad dreams in that the content and emotions evoked by bad dreams do not cause an individual to wake up, whereas nightmares do lead to awakenings (Blagrove & Haywood, 2006).

The diagnostic criteria for PTSD state that nightmares are a mechanism of intrusion where the traumatic event is played out (APA, 2000). Re-experiencing a traumatic event through nightmares is considered to be one of the main components of PTSD (Pillar et al., 2000). Neurobiological models of the function of dreaming in the face of trauma propose that dreams fulfill a ‘desomatization’ function. In other words, dreams via the modulation of anxiety lead to a separation of fearful imagery from the expected psychophysiological manifestations of arousal

---

<sup>5</sup> The term ‘positive feedback loop’ in a physiological context refers to “a response mechanism that results in the amplification of an initial change. Positive feedback results in avalanche-like effects, as occurs in the formation of a blood” clot (Fox, p. 6, 2009).

(Nielsen & Levin, 2007). More specifically, the resulting atonia (a lack of muscle tone) from entering REM sleep, could serve as a desensitization mechanism by repeatedly blocking kinesthetic feedback during traumatic dreams in an attempt to eliminate the arousal that is associated with this event. This in turn modulates the self-perpetuation of anxiety and aids in the mastery of a traumatic experience. However, nightmares occur when anxiety levels surpass a specific threshold resulting in autonomic activation via noradrenergic activity and the altered functioning of the amygdala. Furthermore, a cessation of noradrenergic activity is linked to atonia during REM sleep, where elevated levels of norepinephrine might result in an increased frequency of periodic limb movements, as is often seen in PTSD (Spoormaker & Montgomery, 2008).

Empirical studies report that nightmares are experienced by 60% of individuals diagnosed with PTSD. Therefore, it is confirmed in the literature that nightmares are a component of disrupted sleep in PTSD (Fuller et al., 1994). Dream questionnaires, filled out immediately upon waking or when dreams are remembered, have been suggested as a reliable way to further uncover dream content (Domhoff, 2000). When studying dreams, and more specifically, nightmares in individuals with PTSD, it is interesting to note if the dream content is related to the traumatic event. Studies have found that 46% of the remembered dreams concerned related trauma and 6 were exact replications of the traumatic event (Schreuder, Kleijn, & Rooijmans, 2000; van der Kolk, Blitz, Burr, Sherry, & Hartmann, 1984). In terms of themes in dreams, findings suggest that themes of threat predominated in dreams reported by PTSD individuals, while the presentation of trauma was more variably represented. Furthermore, PTSD individuals report dream content to mainly be set in past circumstances and be related to overall distress (Mellman et al., 2001). Dow, Kelsoe, and Gillin (1996) support this finding when comparing dreams of Vietnam War veterans with PTSD to the dreams of healthy controls. Since nightmares containing trauma and threatening material are one of the defining features of PTSD, the evidence suggests that PTSD patients dream about past events.

### **Methodological Constraints Evident in the PTSD Literature**

As previously mentioned, many studies published in the field of PTSD and sleep are identifiable by different methodological constraints. In the literature on PTSD and sleep architecture, there are major discrepancies across studies with regard to findings of disordered

sleep. There are many factors contributing to this phenomenon, but one such prominent factor include the varied use of mostly subjective measures compared to the rather sparse use of objective measures. One major confounding factor of solely relying on subjective measures is the confirmed occurrence of sleep-state misperception when utilizing questionnaires to measure sleep quality. Sleep-state misperception is an occurrence where participants tend to overestimate the severity of their sleep complaints, and this is often associated with a PTSD diagnosis (Caldwell & Redeker, 2005). The occurrence of this phenomenon is well-established in the sleep research field. Therefore, an optimal solution to this misperception is to utilize objective measures, like polysomnography for example, in conjunction with subjective measures in order to obtain a more comprehensive and accurate representation of true sleep architecture.

Another confounding factor that is evident throughout the literature on PTSD and sleep is that most studies choose to include war veterans in their sample. In fact, there are very few studies that have chosen to investigate other forms of traumatic experiences other than war-related trauma. In a comprehensive review article by Harvey, Jones and Schmidt (2003), it is reported that only 3 out of 26 studies included participants that experienced a traumatic event that is not war-related. This narrow focus on one specific type of traumatic experience complicates the generalizability of results to other traumatized populations. Although only focusing on one type of trauma is beneficial in terms of creating a homogenous group, it is also important to investigate other types of trauma that are under-represented in the literature. This complication is of special relevance to a country such as South Africa, where the most prominent predictors for a lifetime diagnosis of PTSD are for women, rape, and for men, political detention and torture (Kaminer, Grimsrud, Myer, Stein, & Williams, 2008). Therefore, even if the focus is still only on one type of trauma, it would contribute significantly to the literature to be able to characterize disordered sleep in PTSD resulting from the occurrence of more common traumatic experiences, like sexual assault for example.

Furthermore, another confounding feature of a big proportion of studies researching PTSD and sleep are the sole inclusion of males, the inclusion of participants over the age of 50, and participants who have typically experienced a traumatic event 15-50 years prior to being enrolled in the study. All of these factors serve as methodological constraints to some degree, especially since the PTSD population in its entirety is very heterogeneous. This complicates the generalizability of results, as mentioned earlier.

In examining the nature of each of these confounds, firstly regarding the sex of participants, clear differences are demonstrable in terms of the female physiological response to stress when compared to males (Kirschbaum, Wust, & Helhammer, 1992). Furthermore, this extends to differences between the different kinds of environmental and psychological stressors to which men and women are typically exposed to (Kaminer et al., 2008). Therefore, an ideal design would include both males and females, and enough of each in order to adequately determine sex-based differences. However, a more practical option relates to choosing either males or females who have been exposed to the same type of trauma in an attempt to create a homogenous group.

With regard to the age of participants, individuals over the age of 40 undergo natural age-related changes with respect to sleep architecture (Blackman, 2000). Furthermore, the sleep architecture of children and adolescents are identifiable by developmental-related changes in their sleep cycle, which includes alterations in sleep stage distribution throughout the night. Therefore, an ideal design would involve only choosing participants who are characterized as young adults.

In terms of the time since trauma, it is apparent that studies in this field rarely exert any between-subjects control over this factor. Harvey et al. (2003) notes that the longer the period between the occurrence of trauma and participation in the study extends, the more likely it is that factors other than PTSD influences sleep architecture. Therefore, an ideal design would consist of participants falling within a narrow range with regard to time since trauma.

Another important factor to consider is the inclusion of a control group that consists only of participants with major depressive disorder (MDD). Recent reviews of sleep studies (Harvey et al., 2003; Pillar et al., 2000) underscore the importance of including such a control group in sleep studies. Depression not only commonly presents comorbidly with PTSD, but exerts a well-defined influence on sleep architecture. The changes in sleep architecture typically include a decrease in SWS, decreased REM latency, and increased REM percentage (Franzen & Buysse, 2009). Furthermore, some studies have indicated that comparing PTSD participants to depressed controls serves as an effective way of differentiating between disordered sleeping in PTSD and that in MDD. For instance, Mellman et al. (1997) found decreased sleep efficiency, decreased total sleep time, an increased number of awakenings and longer sleep latency in PTSD individuals when compared to a depressed and a healthy control group. More recently, Yetkin,

Aydin, & Ozgen (2010) found that individuals diagnosed with PTSD along with depression demonstrated changes in REM latency, whereas individuals diagnosed with only PTSD did not exhibit such changes.

It should be noted, however, that the results of some studies indicate no noteworthy sleep differences between depressed participants and PTSD participants. For example, Woodward, Friedman, & Biliswe (1996) found that the only difference between the two abovementioned groups were significantly reduced SWS in the sleep architecture of depressed individuals. Sleep latency and sleep efficiency were normal in both of these groups. Thus, the relationship between PTSD and depression has not been unambiguously delineated across studies. Nevertheless, definite sleep changes in relation to depression alone have been confirmed convincingly in the literature, and for this reason it is a necessity in a well-designed study of sleep in PTSD to control for this potentially confounding factor given the high rate of comorbidity between PTSD and MDD.

### **Specific Aims and Hypotheses**

In terms of the evidence presented, one might tentatively state that hypervigilance is highly influential in relation to disrupted sleep in individuals with PTSD. The underlying cause for the multiple disruptions in sleep architecture and nightmares relates to the functioning of the amygdala and the excessive release of norepinephrine, which results in a hypervigilant state. There seems to be some association between traumatic stress, hypervigilance, disrupted sleep architecture, the amygdala, and norepinephrine.

Although a relationship between PTSD and disordered sleep has been established, one of the major questions facing the field is what mechanism(s) support the relationship between PTSD and disordered sleep. We investigated hypervigilance as a key factor supporting this relationship. More specifically, the current study tested the following hypotheses:

1. Our research first investigated whether individuals with PTSD with prominent hypervigilance symptoms experience more disordered sleep than PTSD patients without such symptoms, depressed individuals and healthy controls. The specific sleep disturbances include a) reduced sleep efficiency, b) increased sleep latency, c) increased number of awakenings, d) more time spent awake after sleep onset

(WASO), e) changes in REM latency, f) changes in REM% and g) changes in SWS%. The predicted pattern of the severity of sleep disturbances can be outlined as follow: PTSD with prominent hypervigilance symptoms (HYP+) < PTSD without prominent hypervigilance symptoms (HYP-) < depressed individuals (DEP) < healthy controls (CON).

2. Secondly, this research evaluated if PTSD patients with prominent hypervigilance symptoms experience more negative dream themes and content than PTSD patients without hypervigilance symptoms, depressed individuals and healthy controls. As above, the predicted pattern of worse dream content and theme across groups can be summarized in the following way: HYP+ < HYP- < DEP < CON.

University of Cape Town

## METHODS

### Design and Setting

Our research was of a quasi-experimental cross-sectional design and was nested within larger research project that evaluated memory and sleep architecture in PTSD. This study has ethical approval from the Faculty of Health Sciences Research Ethics Committee and the Department of Psychology Research Ethics Committee of the University of Cape Town. The protocol reference number is 363/2009.

Phase 1 of the study, (the *screening phase*), was conducted at the Department of Psychology at the University of Cape Town and the Department of Psychiatry at Groote Schuur Hospital. Phase 2, (*sleep testing night*), was conducted at the sleep laboratory at Vincent Pallotti Hospital in Cape Town.

### Participants

In total 110 participants were screened, and 58 met the criteria to participate in the study. With regard to the exclusion criteria outlined below, 52 participants were excluded. Of the remaining 58 eligible participants, 9 decided to withdraw from the study leaving the final sample at 49.

### Exclusion Criteria

Individuals with any of the following characteristics were excluded from participation:

1. Males
2. Potential participants who are diagnosed with any of the axis I disorders, except for the relevant PTSD and depressive disorders. The sleeping patterns that are prevalent in other axis I disorders might serve as confounds in the current study. Potential participants with no history of any psychiatric disorder were included in the control group.
3. Evidence of a history of alcohol or substance abuse. Alcohol and substance abuse were controlled for because recent studies have found significant differences in the sleep architecture of participants with excessive alcohol consumption. The findings include prolonged sleep latency, decreased delta sleep, and shorter REM latency (Irwin, Miller, Gillan, Demodena, & Ehlers, 2000).

4. Potential participants below the age of 20 years and older than 40. As mentioned earlier, aging is linked to a change in sleep cycles, while the sleep architecture of adolescents displays different characteristics than those of adults (Blackman, 2000).
5. Evidence of the use of sleeping pills, sedative medication or any other psychoactive medication to treat disordered sleep. These different agents alter natural sleep cycles.
6. Potential participants who had experienced trauma more than 5 years ago or fewer than 6 months prior to the screening. The proximity of the trauma exerts an influence on sleep architecture. Furthermore, irrespective of the time since trauma, any potential participant who experienced a traumatic event as a child or teenager was also excluded.
7. Potential participants diagnosed with any neurological condition (e.g. epilepsy or traumatic brain injury), which could potentially influence the outcome of the study. Five potential participants that were screened disclosed that they are HIV-positive, but they did not present with any AIDS-related disorders. Hence, they were regarded as asymptomatic (they had no weight loss, recurrent fever or opportunistic infections). Therefore, they were not excluded based solely on their HIV status.
8. Evidence of receiving any form of pharmacotherapy or psychotherapy for less than 3 months. Participants needed to not be familiar with treatment or have been stable on treatment for a minimum of 3 months.
9. Evidence of not being able to speak, read, or comprehend English sufficiently.
10. Pregnancy after six months, as sleep architecture of pregnant women differs from non-pregnant women (Lee, 1998).

Figure 1 outlines the reasons for excluding 52 participants from the sample:

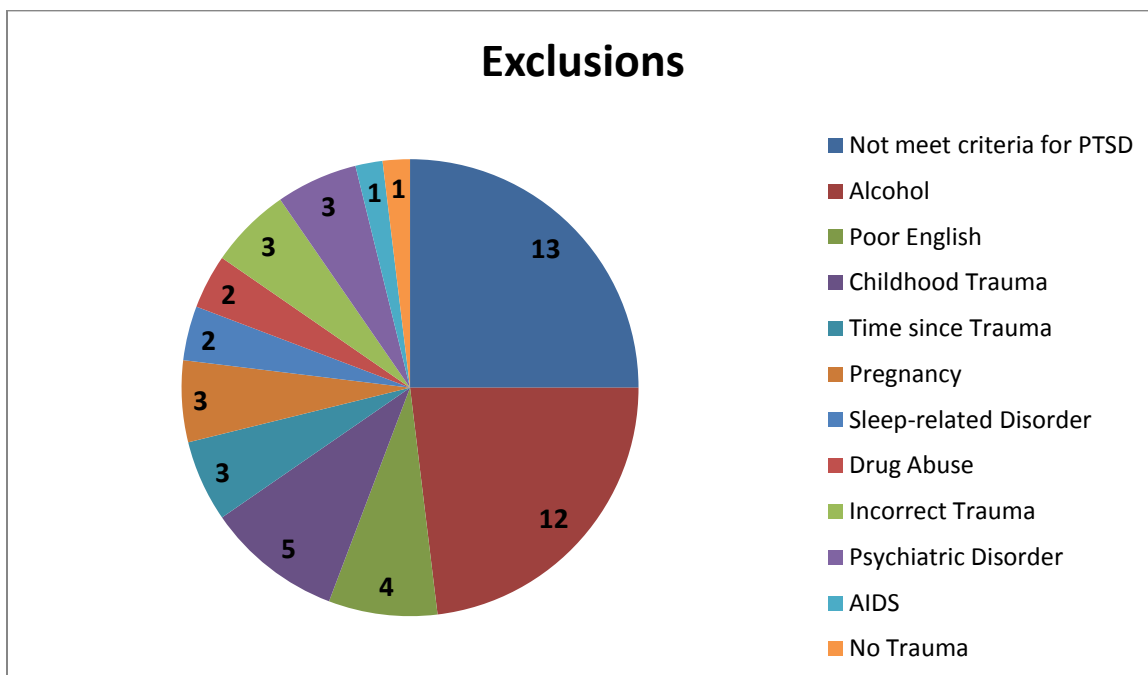


Figure 1. Reasons for excluding 46 participants who were screened. Alcohol = participant met the DSM-IV TR criteria for an alcohol use disorder. Time since trauma = participant experienced the traumatic event either longer than 5 years, or shorter than 6 months prior to the screening. Sleep-related disorder = participant experienced such a disorder other than insomnia. Psychiatric Disorder = participant met the DSM-IV TR criteria for an axis I or axis II disorder other than PTSD, MDD or a related anxiety disorder.

Participants were allocated to the different groups according to the following criteria: a) the presence of PTSD with prominent hypervigilance symptoms (HYP+ group), b) the presence of PTSD without prominent hypervigilance symptoms (HYP- group), c) the presence of depression without any other form of psychopathology (DEP group), and d) the absence of any form of psychopathology (CON group). The number of participants assigned to the four different groups was as follow: HYP+ ( $n = 9$ ), HYP- ( $n = 10$ ), DEP ( $n = 14$ ), and CON ( $n = 16$ ).

Due to the lack of research concerning what constitutes prominent hypervigilance symptoms in an individual with PTSD, group allocation was based on the median hyperarousal cluster scores of all PTSD participants. A score of 25 or below was classified as PTSD without prominent hypervigilance symptoms, where a score of above 25 was classified as PTSD with prominent hypervigilance symptoms.

As can be seen from outlining the sample size of the different groups, it is apparent that the small sample size of the two PTSD groups imposes certain limitations on this study. There

are several reasons for the small sample size, and this will be discussed in more detail in subsequent sections, but can be summarized as follow. Firstly, this study implemented very rigorous exclusion criteria in an attempt to create a homogeneous group and to overcome several methodological limitations of previous studies. On average, more than half of the participants that were screened were excluded from participation. Secondly, all participants were from a very low socio-economic status and this posed problems in terms of being able to attend screenings, because many participants were unable to afford public transport costs, even though they were reimbursed at the conclusion of the screening. Thirdly, this study was conducted over three years, and it became increasingly difficult to recruit participants from communities and trauma organizations due to saturation of these recruitment sources. Fourthly, one of the main criterion of this study stated that only individuals who have been raped *for the first time* as an adult can be included in the study. This is problematic in a country such as South Africa with very high rates of sexual assault as most participants who responded telephonically have been raped more than once, and very often as toddlers or teenagers. On average, only about 16 participants out of a 100 who responded telephonically were not excluded by the criterion above. Furthermore, on average, almost half of participants who made it to the screening interview were excluded due to the other exclusion criteria.

With regard to the demographic characteristics of the sample, we recruited an all-female sample between the ages of 20-40 years who are sexual assault survivors. This narrow age range further restricted sample size, but is imperative that only younger adults be included in the sample as sleep architecture undergo natural changes after the age of 40, while the sleep architecture of teenagers display different characteristics than that of adults (Blackman, 2000).

Although the inclusion of men would have resulted in a much larger sample, they were excluded in an attempt to keep the sample as homogenous as possible. This is true for both sex and type of trauma. In terms of sex, women are under-represented in the PTSD literature even though the prevalence rate of PTSD is not lower for women. The majority of studies in the literature focus on male war veterans. This leads to the second reason in terms of type of trauma. Not only is sexual assault more prevalent among women, it is also under-represented in the literature as a traumatic event leading to the development of PTSD. In summary, the two main reasons for excluding men include firstly, an attempt to keep the sample as homogenous as possible, and secondly, attempting to characterize the sleep architecture of an under-represented

group in the literature who have suffered a traumatic event that has not been extensively studied before. Furthermore, a restricted age range, between 20 to 40 years-old, was employed because (a) aging is linked to a change in sleep cycles (Landolt, Dijk, Achermann, & Borbély, 1996), and (b) the sleep cycles of adolescents display different properties than those of adults (Kales et al., 1970).

All participants were recruited from trauma organizations, for example Rape Crisis and the Trauma Centre, and neighboring communities. Advertisements were placed in newspapers and posters were put up at community police stations, trauma organizations, and clinics. Recruitment began in February 2010 for this study and the larger study. Based on the age and the nature of the trauma, potential participants were identified from past records. Following this process, counselors contacted each potential participant in order to obtain consent for the researcher to contact them. Potential participants were only contacted once consent was obtained.

With regard to individuals who responded telephonically to the advertisements and posters, a number of short questionnaires were administered over the phone in order to ascertain basic demographic information (e.g. age) as well as a previous history of trauma.

In terms of the language of participants, all participants who were eventually included in the study were predominantly Xhosa- and Afrikaans-first language speakers, but all participants were fluent in English.

Participants in the two PTSD groups experienced trauma between 6 months to 5 years prior to enrollment in the study. This inclusion criterion was implemented not only to make the groups as homogenous as possible, but also because time since trauma is influential in determining sleeping patterns; that is to say, the immediate manifestations of PTSD in sleep may differ from later manifestations. Immediate responses to trauma, for example recurrent awakenings, might decline over time (Engdahl, Eberly, Hurwitz, Mahowald, & Blake, 2000). In addition, dream content is suggested to differ in chronic stages of PTSD. Furthermore, the more recent occurrence of a traumatic event is also under-represented in the literature. Most studies in previous years focus on war veterans who typically recruited older adults as part of their sample.

As mentioned earlier, each participant was assigned to one of four groups: HYP+ ( $n = 9$ ), HYP- ( $n = 10$ ), DEP ( $n = 14$ ), and CON ( $n = 16$ ). Although groups are unequal in terms of their sample sizes for reasons stipulated above, they are perfectly matched on all demographic

variables. More specifically, groups were matched in terms of age, IQ, socio-economic status (as determined by income), and time since trauma Results can be seen below:

Table 1  
*Demographic Characteristics of the Sample*

Variable	HYP+ <i>n</i> = 9	HYP- <i>n</i> = 10	DEP <i>n</i> = 14	CON <i>n</i> = 16	<i>F</i> / $\chi^2$	<i>p</i> value
Age (years)	27.56(6.91)	27.36(6.91)	27.14(3.96)	27.31(5.67)	.010	.99
WASI PIQ	83.11(12.11)	77.91(8.23)	82.00(12.48)	83.75(13.36)	.569	.638
Time since trauma	2.38(1.85)	1.91(1.64)	-	-	.337	.569
Income					12.37 <sup>a</sup>	.65

*Note.*

\**p* < .05. The test statistic was ANOVA. WASI PIQ = Weschler Abbreviated Scale of Intelligence Performance IQ score. Time since trauma is presented in years. For Age, WASI PIQ, and Time since trauma means are presented with standard deviations in parentheses. Mean values for Time since trauma are not presented for the DEP and CON groups, as these groups did not experience a traumatic event. Degrees of freedom are (3, 49). For Income, chi-squared statistic is presented as values were recorded as a range and coded as a categorical variable. <sup>a</sup> $\chi^2(18)$  statistic reported.

## Materials and Apparatus

**Diagnostic and screening instruments.** *The Mini International Neuropsychiatric Interview*, (English version 5.0.0; MINI; Sheehan et al., 1998), a structured diagnostic interview, was used to screen for the presence of major DSM-IV Axis I psychiatric disorders. According to the MINI's developer's, the tool has good psychometric properties and can be administered within approximately 15 minutes by a clinician or by a lay person who has undergone appropriate training. In the current study this instrument was used to confirm diagnoses of PTSD and MDD, as well as to exclude any other Axis I psychiatric conditions across all groups. The use of this instrument was also implemented in order to aid in the selection of the healthy control group. These participants were required to carry no MINI-assessed psychiatric diagnoses, while they were also carefully screened in order to ensure that they have not previously experienced a traumatic event under DSM-IV-TR PTSD criterion A. In terms of the depression group, the MINI was also used to exclude the presence of PTSD or other forms of psychopathology. In terms of using the MINI in a South African sample, studies have shown that the MINI has high reliability and validity scores when used with South African populations (Olley et al., 2005).

*The Beck Depression Inventory – Second Edition* (BDI-II; Beck, Steer, & Brown, 1996) consists of 21 standardised self-report questionnaire items that evaluate the severity of

depression in adults. The instrument is reported as valid and reliable by the developers and can be implemented in a clinical setting and as a research tool. The instrument has been successfully used with research conducted in South Africa (Seedat, Nyamai, Njenga, Vythilingum, & Stein, 2004). In this study, the BDI II was used to screen for depression in order to evaluate if one group showed higher depression rates in comparison with the other groups, as well as allocating participants to the DEP group. Furthermore, it was also used to characterize the depressive symptomatology of the DEP group. Participants with a BDI score of 14 or above and who met the MINI criteria for depression and no other major Axis I disorder, formed the depression group. In general a score of 14 -19 indicate mild depression; 20-28 indicates moderate depression; 29-63 indicates severe depression (Beck, Steer, & Brown, 1996).

*The Clinician Administered PTSD Scale (CAPS; Blake et al., 1995)* is a structured interview developed for the assessment of the presence and the main and associated symptoms of PTSD. The instrument is designed in such a way that individuals with limited clinical knowledge and experience in structured interviews can provide reliable ratings of PTSD symptoms. This structured interview determined the frequency and intensity of symptoms by asking standard questions and providing an explicit, behaviourally-anchored rating scale. Any participant that scored below 45 on the CAPS was classified as PTSD sub-clinical. According to the developers, this scale is a good detector of PTSD severity and displays excellent psychometric reliability and validity for determining a PTSD diagnosis (Blake et al., 1995). This instrument has been used in previous research within South Africa (Martenyi, Brown, Zhang, Koke, & Prakash, 2002). In this study, the CAPS was implemented to confirm a PTSD diagnosis and to measure the extent of hypervigilance symptoms as part of the hyperarousal cluster of symptoms within this diagnosis. Due to the lack of literature on what constitutes prominent hypervigilance symptoms in PTSD, it was decided to allocate individuals with PTSD to the two PTSD groups on the basis of the median of the total hyperarousal cluster score. A hyperarousal score of 25 or below was classified as individuals without prominent hypervigilance symptoms, while a score above 25 classified the participant as having prominent hypervigilance symptoms. The CAPS was also used to exclude the presence of PTSD in the control and depressed group.

In terms of scoring, there are nine different ways of scoring the CAPS. All these methods have demonstrated good to excellent reliability (Weathers et al., 2001). This study adopted the *Frequency  $\geq 1$ /Intensity  $\geq 2$ / Total Severity  $\geq 45$  (F1/I2/TSEV45)* method. This method combines

two rules – the *Frequency  $\geq$  I/Intensity  $\geq$  2 (F1/12)* and the *Total Severity  $\geq$  45 (TSEV45)* rule. According to the first rule (F1/12), a symptom is present if its frequency obtains a score of 1 or higher, while its intensity obtains a score of 2 or higher. In order for an individual to be diagnosed with PTSD, DSM-IV criteria must be met with regard to the correct distribution of symptoms across clusters. This approach is considered lenient (Weathers et al., 2001). The second rule (TSEV45) considers a total score of at least 45 as the basis for a valid diagnosis of PTSD. When taken together the two abovementioned rules specify that a CAPS score of at least 45 must be obtained in order for a PTSD diagnosis to be made, while the appropriate distribution of symptoms across clusters must also be present. The combination of these two rules is considered to be a moderate approach, and its application is recommended for situations where a diagnosis of PTSD needs to be confirmed (Weathers et al., 2001). More specifically, in this study there was no referring clinician so it was imperative to avoid false positives and false negatives at the screening interview, therefore a moderate approach also fit well with the screening objective.

*The Michigan Alcoholism Screening Test (MAST; Selzer, 1971)* is a consistent and quantifiable structured interview used to detect alcoholism and substance abuse. Twenty five quick questions are administered while the questionnaire demonstrates good validity. Alcohol and other drug abuse, as a possible cause of disordered sleep, were screened for using this instrument. Any participant scoring greater than 5 on the MAST was excluded. The MAST has proven to be a useful screening instrument with South African populations (Bekker and van Velden, 2003).

**Experimental Measures.** *The Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989)* is a self-rated questionnaire used to evaluate an individual's sleep quality and sleep disturbances over the past month. Seven 'component' scores are generated that assess subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. The PSQI has both psychometric and clinical properties which make it well suited for use in clinical practice and research activities. The PSQI has been successfully administered to South African populations (Rockwood, Mintzer, Truyen, Wessel, & Wilkinson, 2001).

Participants were asked to complete a *Most Recent Dream* questionnaire (Domhoff and Schneider, 1998) (see Appendix B) that included the date, setting, time of day, and location of recollection. Two dream reports were collected, the first report at the screening interview and the second report the morning following testing at the sleep laboratory. Two dreams were collected in an attempt to obtain as much information on dream content and theme. Participants were asked to describe the content of the dream in as much detail and as accurately as possible. In this study, dream content and dream theme were noted. In general, dream reports are frequently collected outside the laboratory, while it is reported that there is little or no difference between dreams obtained from REM sleep awakenings and ones obtained from outside of the laboratory (Domhoff, 2000). The dream reports were analyzed by three blind independent raters. See Appendix C for dream rating instructions.

**Sleep Laboratory Equipment.** It has been suggested that using subjective measures, such as dream forms, as well as objective measures of sleep quality, yields an accurate association between sleep disturbances and nightmares. Objective measures include a sleep-adapted EEG which categorizes sleep into different stages (Harvey et al., 2003).

The Vincent Pallotti Hospital sleep laboratory in Cape Town was selected because it provided all of the necessary equipment and facilities needed to conduct sleep research. The equipment included a sleep-adapted electroencephalograph (EEG) to measure brain activity during sleep, electrooculograph (EOG) electrodes that monitor eye movement, and electromyograph (EMG) electrodes that monitor muscle tone. The standard measurements of the EEG, EOG, and EMG in terms of sleep stages were classified according to the delineation of the American Academy of Sleep Medicine (2007).

For the sleep-adapted EEG recording, a Nihon Kohden Neurofax EEG9000 with sleep options was used. All the equipment met the requirements of all the digital system regulations (e.g. filters on each channel), the rules for display and display manipulation (e.g. the option of viewing the sleep data in different time frames, from 5 seconds to 2 minutes), as well as the digital analysis specifications (e.g. the option of scoring the data either electronically or manually).

Our montage abided by the most recent recommendations as stipulated in the manual of the American Academy of Sleep Medicine (AASM), which was released in 2007. However,

there were some differences in terms of standard polysomnographic<sup>6</sup> reading in the following respects: Our montage did not record any of the respiration parameters (airflow, oximetry, nasal pressure, esophageal pressure, or rib cage or abdominal movements), leg movement or body position. The reason for these omissions relates to the purpose of this study and other studies conducted in our laboratory, where the focus is on an accurate determination of the various sleep stages, arousals and sleep efficiency. The use of the abovementioned measures that were omitted is time-consuming, while they also do not formally address the essence of our research questions.

With regard to the guidelines for electrode placements, the following differences were noted from the AASM standards: we used one more EEG electrode than the minimum stipulated by the AASM. We referenced<sup>7</sup> all our electrodes placed at the right and left earlobes, while we also included an additional set of eye electrodes to supplement the two specified by the AASM. These changes were recommended to the research team by the equipment specialists in order to focus on accurately measuring the sleep stages, arousals, and overall sleep efficiency.

## Procedure

The procedure for this study included an initial screening phase, followed by a testing phase, which was conducted at the sleep laboratory. Participants only underwent one night of sleep recording. Several other studies adopted the approach of testing participants twice, while they discarded the results of the first testing night due to the first-night effect. Polysomnographic recordings show more awakenings and less REM sleep in the recordings of the first night (Le Bon, & Arpi, 2003). However, more recent studies have demonstrated that no *significant* differences emerged in sleep architecture across two consecutive nights spent in the sleep laboratory (Sforza, Chaptot, Pigeau, & Buguet, 2008). More specific to PTSD, a recent study examining the sleep architecture of individuals with PTSD compared to controls, found no first-night effect for this population group, but did find this effect for the control group (Herbst et al., 2010). Considering more recent results, it is evident that there is little consensus in the literature indicating the necessity of an adaptation night. More recent studies report that if differences in sleep architecture are present, they seem to be minimal. Therefore, in light of the time and cost

---

<sup>6</sup> Polysomnography refers to an EEG adapted to record sleep that contains all the channels and specifications provided by the AASM. Because our montage is based on the AASM, but does not include an exact replica of the channels and specifications, we refer to it as an EEG adapted for sleep research.

<sup>7</sup> All electrodes need a reference – a signal to which the electrode makes reference.

associated with including an adaptation night for each participant, it was decided that participants would not undergo an adaptation night.

At the first phase of the study, the *screening phase*, the participant signed a consent form and was briefed on the procedure to follow. The diagnostic and screening measures were then administered. If the participant was considered to be an eligible candidate, she was assigned to one of the four study groups. At the conclusion of the screening session, the experimenter scheduled an appointment for the sleep testing night.

In the second phase of the study, *the sleep testing night*, the participant arrived at the sleep laboratory at 20h00. The experimenter then prepared the participant for a night's sleep. Participants were attached continuously throughout the night to the sleep-adapted EEG monitor that measures sleep architecture. The instrument also measured brain activity through EEG electrodes, eye movements through EOG, and muscle tone through EMG. Once the sleep equipment was set up, all the channels were tested in order to ascertain whether they were working correctly. This was done by asking the participant to perform simple actions, which included biting and blinking. The impedance (or amount of signal interference), was recorded in order to ensure the measurement of a clear reading. Participants were woken up 7-8 hours after going to bed. They were then asked to complete the "Most Recent Dream" questionnaire one more time (the first questionnaire was completed at the screening interview). In addition, participants completed the PSQI in order to evaluate their perceived quality of sleep. A full debriefing occurred after all questionnaires were administered and the procedure was complete. At this time the participant was compensated with R150 for their participation in the study.

### **Ethical Considerations**

All participants were provided with informed consent forms to complete prior to official enrollment in the study. These forms ensured that participants were fully informed about the nature of the study, including all procedures and risks and benefits, and that participants could withdraw from the study at any point without penalty. Furthermore, the forms explained that the tests would not harm them in any way, and that they would be compensated for their time.

Due to the enrollment of participants who have experienced a traumatic event, verbal assurance was provided at the beginning of the screening session that they were allowed to withdraw from the study at any point, and that they don't have to provide more details about the

experienced trauma than what they felt comfortable with. Furthermore, all participants that were struggling with PTSD and/or depression symptoms were referred to appropriate clinics and counselors in their area. At the conclusion of the study, all participants who exhibited signs of psychopathology were provided with a list of counseling centers where they could seek treatment.

All study procedures were approved by the Research Ethics Committees of the University of Cape Town's Department of Psychology and Faculty of Health Sciences.

### **Statistical Analyses**

All collected data was analyzed using SPSS software (Howell, 2004). The independent variable was the *group condition*: PTSD group with prominent hypervigilance symptoms (HYP+) ( $n = 9$ ); PTSD group without prominent hypervigilance symptoms (HYP-) ( $n = 10$ ); individuals with depression (DEP) ( $n = 14$ ); and healthy controls (CON) ( $n = 15$ ). The dependent variables included *characteristics of sleep*: sleep latency (time spent falling asleep), awakenings from sleep, WASO, sleep efficiency, REM%, REM latency, and SWS%. The sleep-adapted EEG monitor was used to analyze these characteristics. All analyses were scored based on criteria provided by the American Academy of Sleep Medicine (Kushida et al., 2005). Reports were analyzed by researchers and validated by professionals, while the percentage of agreement on sleep stage scoring between scorers were >85% in all instances.

**Psychiatric characteristics of the sample.** Firstly, several analyses were run in order to determine the psychiatric characteristics of the sample. T-tests were done to determine if there are any significant differences in the total CAPS scores of the two PTSD groups, while one-way ANOVA was run to establish whether these two groups differ significantly in their symptom-distribution across the three different symptom clusters. With regard to characterizing depression across groups, the Kruskal-Wallis test was run on the BDI-II scores because the data violated the assumptions of ANOVA. Furthermore, independent sample t-tests were also run on the BDI-II scores in order to determine if the three clinical groups (HYP+, HYP-, and DEP), are equally matched on depression severity, because it is expected that significant differences will be present if the healthy control group (depression-free) is included in the analysis.

**Hypothesis 1.** The first hypothesis states that hypervigilance could serve as one influential mechanism underlying disrupted sleep architecture in PTSD, and that the pattern of

disturbed sleep across all four groups can be summarized in the following way:  $HYP+ < HYP- < DEP < CON$ . ANOVA was chosen as a first-step analysis in order to detect any between-group differences in objective and subjective measures of sleep quality. Orthogonal planned comparisons were subsequently run in order to confirm or disconfirm the specific and related hypotheses of the study. The logic behind these analyses can be seen in the figure below:

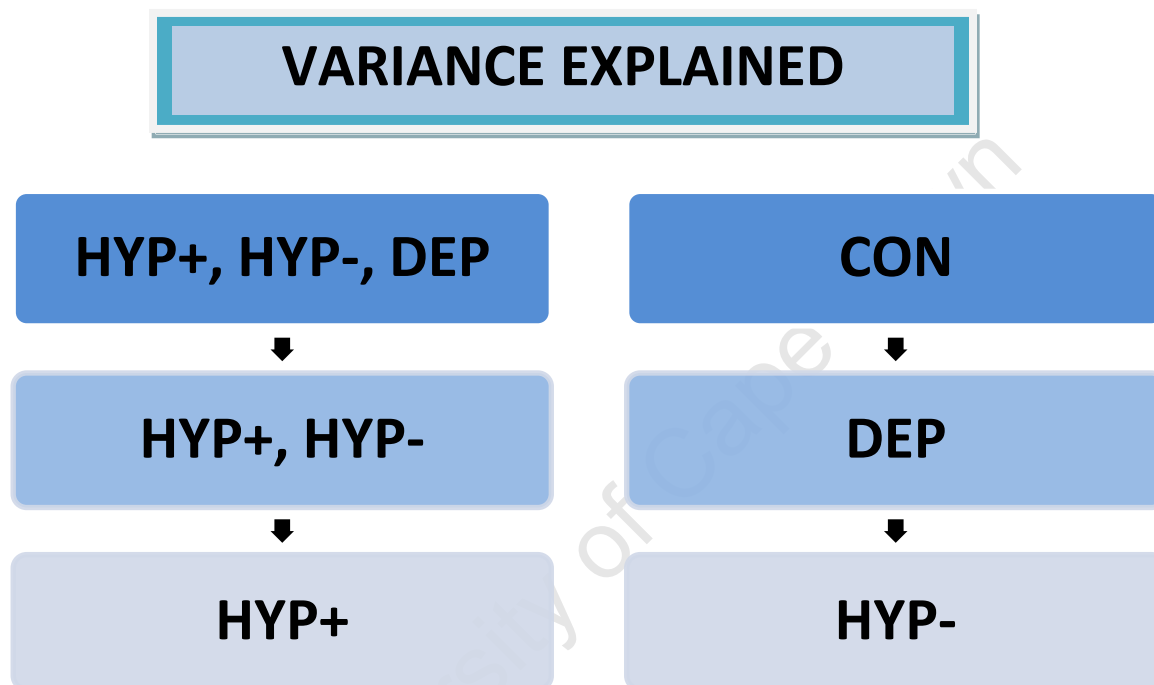


Figure 2. The logic behind conducting orthogonal planned comparisons.

The first analysis entailed comparing the control group with the three clinical groups (HYP+, HYP-, and DEP). This was done in order to establish whether PTSD and major depression exerts a significant influence on sleep architecture when compared to people without any form of psychopathology. The second analysis compared the two PTSD groups with the depression group. This comparison was imperative in terms of delineating the effects of depression, i.e. if significant differences are found in this analysis and groups are matched equally on depression severity, there might be mediating factors other than depression influencing sleep architecture. The third and final analysis compares the HYP+ group with the

HYP- group. This was done in order to address the main hypothesis of this study, i.e. does prominent hypervigilance symptoms result in more disordered sleep when compared to individuals without such symptoms?

**Hypothesis 2.** The second hypothesis states that individuals with prominent hypervigilance symptoms will have more negative dream content and theme compared to the other three groups. This assertion, as above, can be summarized in the following way: HYP+ < HYP- < DEP < CON. With regard to the dream data, all dream reports were randomized by an individual other than the researchers involved in the study in order to establish a blind rating system. First, the individual gave each report a number and recorded that number with the participants name on a master reference sheet. The participants name was then removed from the report. Each report was then typed and a copy was given to three raters. The raters scored the reports for content and theme separately and with no communication. See Appendix C for dream scoring instructions. All scored reports were entered into a spreadsheet using the recorded number as the participants identity. The averaged theme and content score of each person's two reports was used for analysis.

Information provided by the "Most Recent Dream" questionnaire was analyzed for content and theme. The dream was first read in its entirety to rate the overall theme. The theme was classified as negative, neutral, or positive. Dream content was rated using a scale ranging from negative ten, indicating highly negative content, to positive ten, indicating highly positive content (Domhoff, 2000). Spearman's correlation was used to establish inter-rater reliability. A series of statistical analyses followed. Firstly, ANOVA was used to discover any between group differences. Following this, orthogonal planned comparisons were run abiding by the same logic described in the aforementioned section.

## RESULTS

### Psychiatric Characteristics of the Sample

Data obtained from administering the BDI, MINI and the CAPS were used to characterize the presence, the nature, and the severity of psychiatric symptoms in the three clinical groups (HYP+, HYP-, & DEP). The same measures were administered to the healthy control group in order to exclude the presence of Axis I and Axis II diagnoses, and the occurrence of traumatic experiences that would result in participants meeting the DSM-IV-TR criteria and criterion A of a PTSD diagnosis.

A t-test was done in order to determine if there are significant differences in the total CAPS score between the two PTSD groups (a higher score indicates worse PTSD severity). A significant difference in total CAPS score was found between the two PTSD groups  $t(18) = 18.66, p < .05$ . However, this is to be expected, as participants were assigned to the two different trauma groups according to their hyperarousal score. Rather than considering the total CAPS score (that will be inflated by the hyperarousal score), it is more important to look at the distribution of scores across the three different clusters (Re-experiencing, Avoidance and numbing, and Hyperarousal), and determine from this analysis whether groups are adequately matched on PTSD severity. One-way ANOVA was run in order to address this question. All the assumptions of ANOVA were upheld.

Table 2  
*Results of between-group analyses of CAPS cluster scores*

Group	HYP+	HYP-	<i>F</i>	<i>p</i>
Re-experiencing	21.44(8.82)	17.7(5.03)	1.328	.265
Avoidance & Numbing	33.22(9.84)	27.00(6.25)	2.765	.115
Hyperarousal	29.22(3.03)	21.00(4.69)	20.47	.0001**

*Note.* \*\* $p < .01$ . Means are presented with standard deviations in parentheses.  
Degrees of freedom (1, 18)

Results from descriptive statistics indicate higher mean scores for the HYP+ group in terms of the Re-experiencing cluster than the HYP- group. The HYP+ group also scored higher on the Avoidance and numbing cluster than the HYP- group. As expected (because of group

allocation) the HYP+ group also had higher Hyperarousal scores compared to the HYP- group. However, results from between group analyses indicate no significant difference in the distribution of scores for the Re-experiencing and Avoidance and numbing cluster, although there is a significant difference in terms of the hyperarousal cluster scores.

Results from this analysis provide support for the premise that the two PTSD groups were adequately matched on two of the three symptom clusters as determined by the CAPS, as there are no significant differences between groups. The cluster scores were used rather than the total CAPS scores, because it is expected that the hyperarousal scores will inflate the total CAPS score.

In summary, groups were evenly matched on two of the three symptom clusters (Re-experiencing and Avoidance and numbing), therefore indicating equal PTSD severity between groups for these two clusters. The only difference in severity relates to the hyperarousal scores, which fits in with the design of the study. Therefore any differences detected in sleep architecture cannot be ascribed to PTSD severity across 2 of the three clusters, but rather to the difference in severity in terms of the hyperarousal scores. For this reason, PTSD severity was not included as a covariate in subsequent analyses.

With regard to characterizing depression in the clinical groups, all but two participants (from the HYP- group) scored in the moderate to severe range of depression. The remaining two scored in the upper range of mild depression. In terms of determining the severity of depression, the Kruskal-Wallis test was run due to the BDI-II violating the assumptions of ANOVA (normality and homogeneity of variance). Results indicate a significant difference in the rates of depression between the four groups ( $H(3) = 30.73, p < .05$ ). However, this is to be expected, as it was required of the CON group to be depression-free. To investigate whether there is a significant difference in the rates of depression among the clinical groups, independent t-tests were conducted. Results can be seen below:

Table 3  
Independent Sample *t*-tests for BDI-II Scores

Groups	<i>t</i>	<i>p</i>
#1: HYP+ vs. HYP-	1.917	.072
#2: HYP+ vs. DEP	1.849	.272
#3: HYP- vs. DEP	-.317	.754

*Note:*

Degrees of freedom for #1 = 21; #2 = 17; #3 = 22.

These results indicate that there are no significant differences in depression severity between the two PTSD groups, between the HYP+ and DEP groups, or between the HYP- and DEP group. These results indicate that the three clinical groups were matched equally on depression severity. Therefore it seems feasible that there are other influential factors affecting sleep architecture if significant differences do emerge. These variables will be investigated in subsequent analyses.

As noted earlier, many studies are confounded by significant variability in terms of the time since the traumatic event occurred. This study attempted to control for such a confounding factor by implementing a narrow time frame for the occurrence of the traumatic event (6 months-5 years prior to enrollment in the study). One-way ANOVA was conducted to compare the time since trauma in the two PTSD groups (HYP+ and HYP-). The abovementioned strategy was effective as no significant differences between the time since trauma were found between the two PTSD groups,  $F(1,17) = .337$ ,  $p = .569$ . Therefore, time since trauma was not included as a covariate in subsequent analyses.

### **Testing Hypothesis 1: Between-group differences in objective and subjective measures of sleep quality**

Hypothesis 1 state that hypervigilance could serve as one influential mechanism underlying disturbed sleep in PTSD, and that the pattern of disturbed sleep across groups can be summarized in the following way: HYP+ < HYP- < DEP < CON.

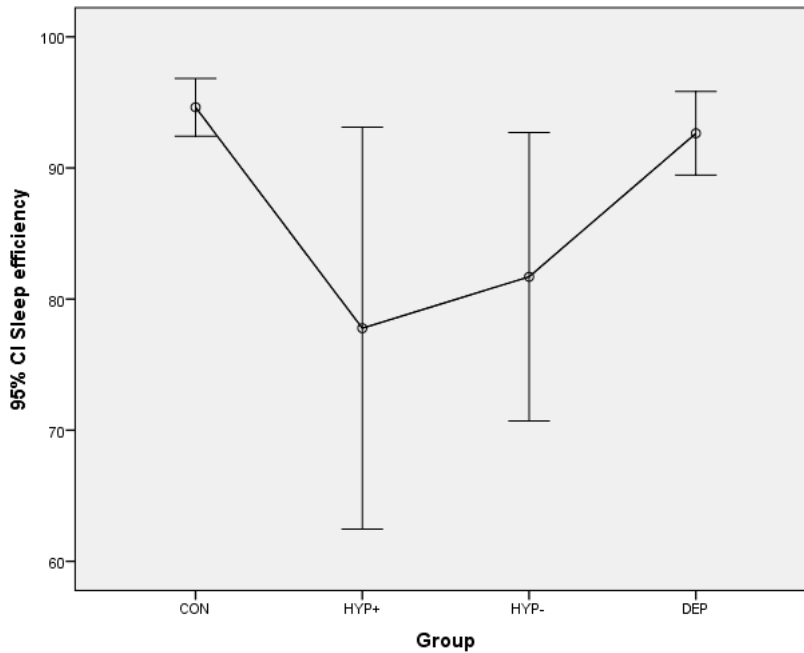
**Sleep data: Testing assumptions.** With the completion of the scoring and validation of the sleep data, the assumptions underlying parametric statistical analyses were tested for all associated sleep variables (sleep efficiency, sleep latency, awakenings, wake after sleep onset,

REM%, REM latency and SWS%). Three of the sleep-related variables violated the assumption of normality (sleep latency, awakenings and REM latency) for the HYP-, HYP+ and DEP group respectively. See Appendix G for results of the Kolmogorov-Smirnov tests.

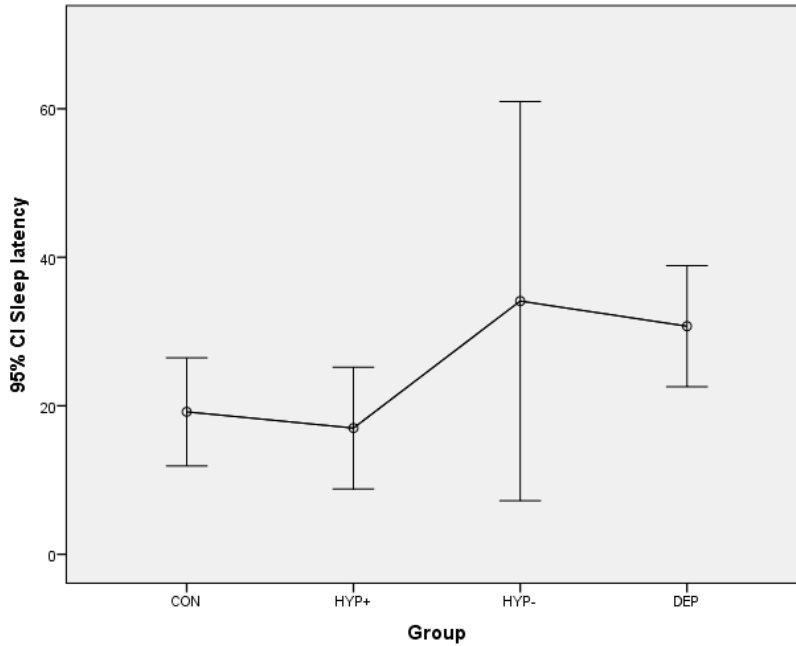
Although these sleep-related variables violate the assumption of normality, it has to be kept in mind that deviations from normal sleep architecture is expected in the three clinical group as it is hypothesized that the sleep architecture of these groups will deviate from the sleep architecture of healthy individuals. Therefore, non-normal distributions of sleep values for the three clinical groups are not an unexpected finding. Further assumption testing revealed that the sleep-related variables sleep efficiency, sleep latency, and REM latency violated the assumption of homogeneity of variance.

Although some variables violated the normality assumption and some the homogeneity of variance assumption, it was still decided to continue with parametric testing for three reasons. Firstly, as explained above one can expect non-normal sleep value distributions for certain sleep-related variables, secondly, the other two assumptions of ANOVA were upheld by all variables (independence of observation and measuring dependent variables on an interval scale), and thirdly, ANOVA is regarded as robust in terms of the violation of assumptions (Field, 2005). However, it has to be stated that all results that follow are interpreted tentatively and with caution because of the reasons stipulated above.

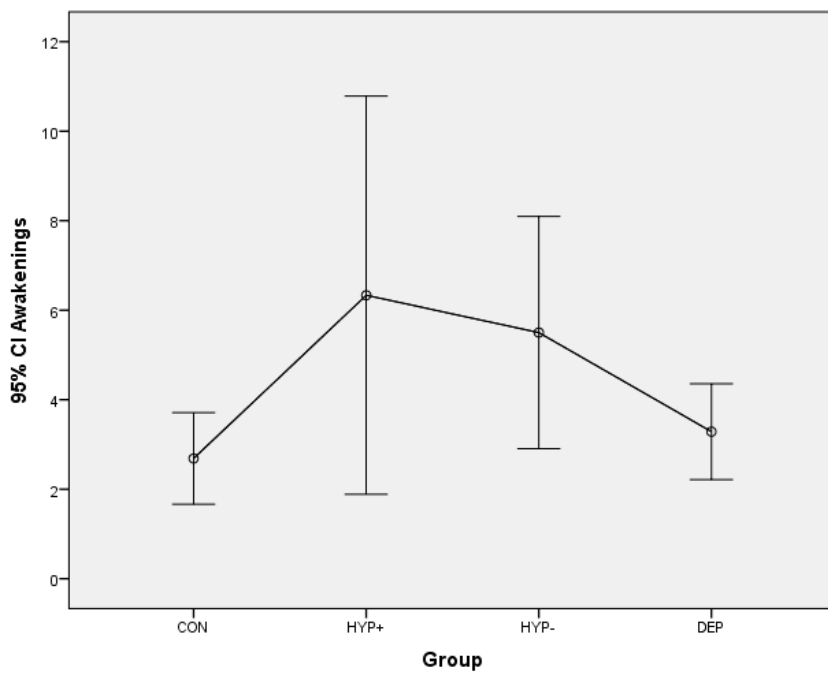
**Trends in sleep data: Cell mean plots.** Figures 3-9 presents cell mean plots for objective measures of sleep quality:



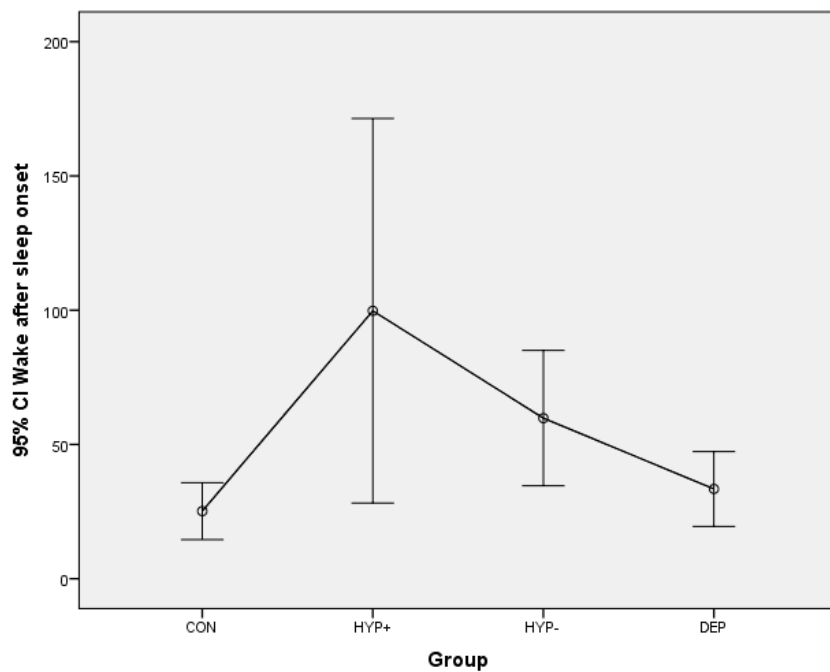
*Figure 3.* Group mean data for the sleep-related variable Sleep efficiency. Error bars represent 95% confidence interval. CON = healthy controls, HYP+ = group with prominent hypervigilance symptoms, HYP- = group without prominent hypervigilance symptoms, DEP = depressed group.



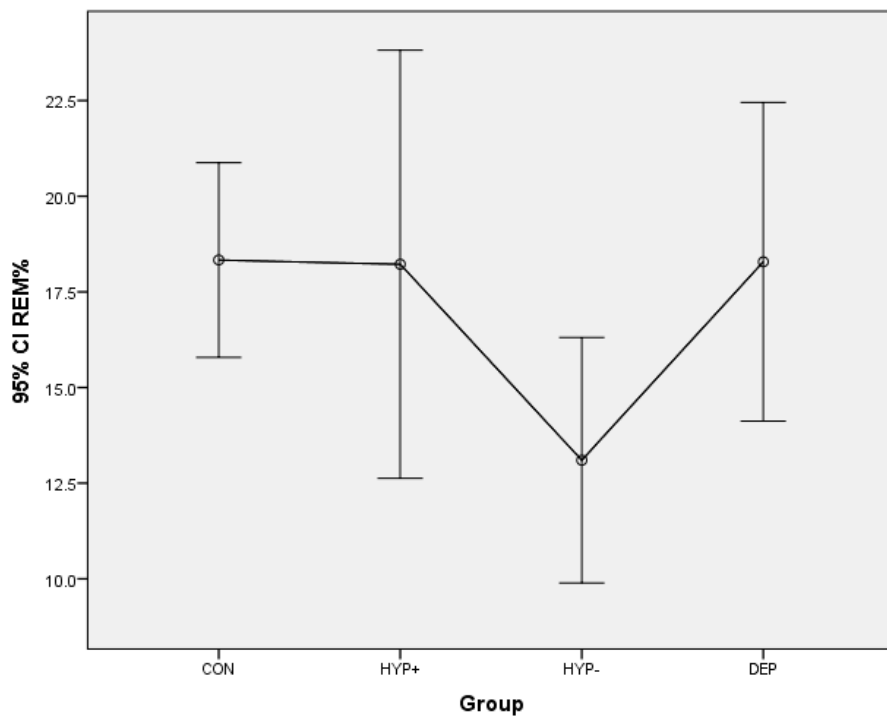
*Figure 4.* Group mean data for the sleep-related variable Sleep latency. Error bars represent 95% confidence interval. CON = healthy controls, HYP+ = group with prominent hypervigilance symptoms, HYP- = group without prominent hypervigilance symptoms, DEP = depressed group.



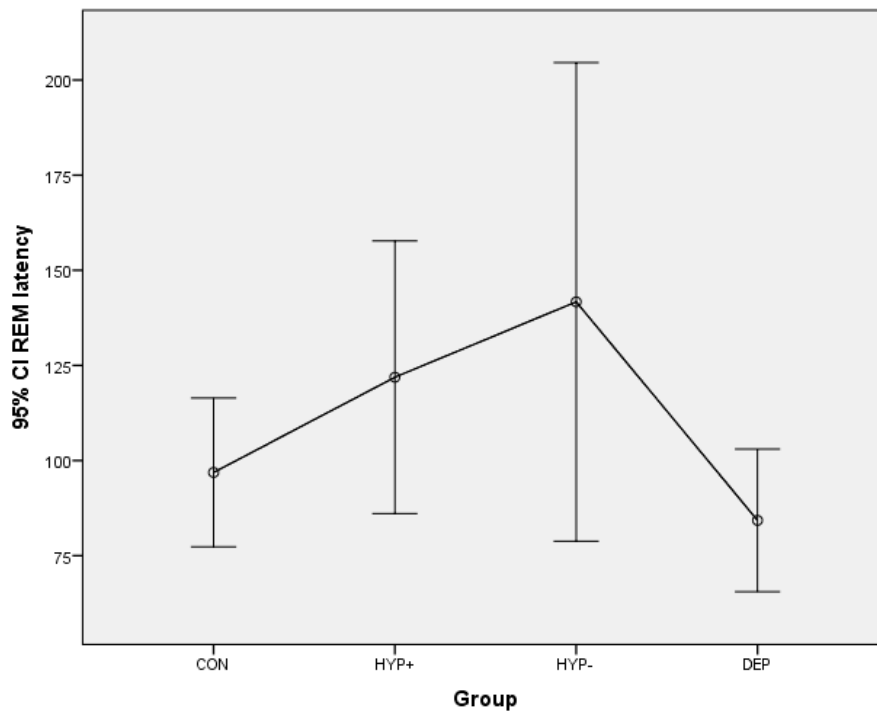
*Figure 5.* Group mean data for the sleep-related variable Awakenings. Error bars represent 95% confidence interval. CON = healthy controls, HYP+ = group with prominent hypervigilance symptoms, HYP- = group without prominent hypervigilance symptoms, DEP = depressed group.



*Figure 6.* Group mean data for the sleep-related variable wake after sleep onset. Error bars represent 95% confidence interval. CON = healthy controls, HYP+ = group with prominent hypervigilance symptoms, HYP- = group without prominent hypervigilance symptoms, DEP = depressed group.



*Figure 7.* Group mean data for the sleep-related variable REM%. Error bars represent 95% confidence interval. CON = healthy controls, HYP+ = group with prominent hypervigilance symptoms, HYP- = group without prominent hypervigilance symptoms, DEP = depressed group.



*Figure 8.* Group mean data for the sleep-related variable REM latency. Error bars represent 95% confidence interval. CON = healthy controls, HYP+ = group with prominent hypervigilance symptoms, HYP- = group without prominent hypervigilance symptoms, DEP = depressed group.

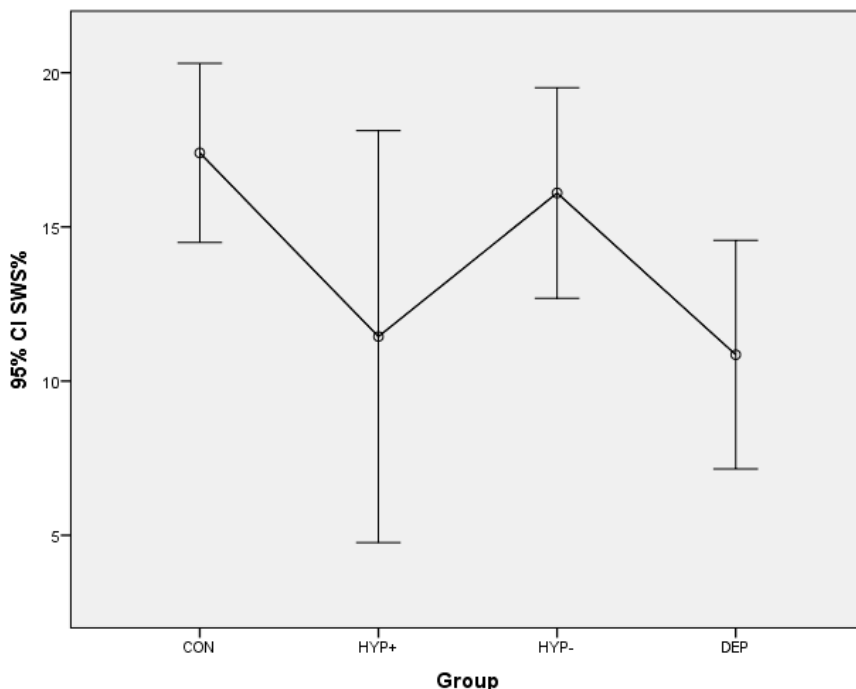


Figure 9. Group mean data for the sleep-related variable SWS%. Error bars represent 95% confidence interval. CON = healthy controls, HYP+ = group with prominent hypervigilance symptoms, HYP- = group without prominent hypervigilance symptoms, DEP = depressed group.

The trends in the figures above are indicative of the HYP+ group experiencing worse sleep quality on three of the seven sleep-related variables when compared to the other three groups. These variables include sleep efficiency (SE), awakenings and wake after sleep onset (WASO). With regard to sleep latency, the HYP+ group fell asleep the quickest. However, their sleep according to these figures are more fragmented, as well as being characterized by being disrupted by periods of prolonged wakefulness when compared to the other three groups.

In terms of the sleep quality of the other three groups, the pattern of disrupted sleep in the HYP+ group is reflected in the sleep data of the HYP- group, although the severity of these disruptions occur to a lesser extent. Sleep quality was reduced on the same three variables as the HYP+ group (SE, awakenings, and WASO), while sleep latency was worse in this group compared to the other three groups. REM latency was also prolonged and there was an overall reduction in REM%.

The sleep quality of the DEP group was better on the three sleep variables mentioned above when compared to the two PTSD groups, however, with regard to sleep latency, the DEP group took the second longest to fall asleep. Overall the sleep quality of the DEP group was worse on all sleep-related variables when compared to the CON group.

In summary, according to the group mean data, there are trends present in the data suggestive of worse sleep quality in the HYP+ group when compared to the other three groups, while the pattern of reduced sleep quality can tentatively be outlined as follow: HYP+ < HYP- < DEP < CON.

**Inferential statistical analyses of objective measures of sleep data.** A series of one-way ANOVAs were conducted, the results of these analyses are presented below:

Table 4

*Sleep-related variables: Descriptive statistics and results from between-group comparisons.*

Variable	HYP+	HYP-	DEP	CON	<i>F</i>	<i>p</i>	ESE
SE	77.78(19.84)	81.7(15.37)	92.64(5.51)	94.63(4.13)	5.88	.002**	.53
SL	17.0(10.66)	34.10(37.57)	30.71(14.12)	19.19(13.67)	1.9	.144	.34
Awakenings	6.33(5.79)	5.50(3.63)	3.29(1.86)	2.7(1.92)	3.26	.030*	.42
WASO	99.78(93.14)	59.80(35.23)	33.43(24.12)	25.19(19.85)	5.87	.002**	.53
REM%	18.22(7.28)	13.10(4.48)	18.29(7.23)	18.33(4.59)	1.98	.130	.35
REM latency	121.89(46.6)	141.70(87.91)	84.29(32.457)	96.87(35.27)	2.83	.049*	.40
SWS%	11.44(8.7)	16.10(4.77)	10.86(6.42)	17.40(5.23)	3.51	.023*	.43

*Note.*

\* $p < .05$ , \*\* $p < .01$ . Means are presented with standard deviations in parentheses. Degrees of freedom in each case were (3, 45). SE = Sleep efficiency. SL = Sleep latency. WASO = Wake after sleep onset. ESE = Effect size estimate, in this case  $\eta^2$

As can be seen above, there are significant between-group differences for five of the seven sleep variables. These variables include SE ( $p = .002$ ), number of awakenings ( $p = .030$ ), WASO ( $p = .002$ ), REM latency ( $p = .049$ ), and SWS% ( $p = .023$ ) with medium to large effect

sizes for these 5 variables. The sleep-related variables sleep latency and REM% did not reach significance in the between-group analysis.

Although these results indicate significant differences in the sleep architecture of the four different groups, further analyses are required to elucidate the nature and direction of the differences that were detected. For this reason, and due to the a priori prediction of disturbed sleep in the different groups (HYP+ < HYP- < DEP < CON), orthogonal planned comparisons were conducted. The first comparison sought to compare the sleep architecture of the three clinical groups with the sleep architecture of the CON group. The second comparison compared the sleep architecture of the two PTSD groups with the DEP group. This analysis is important in terms of being able to tease apart whether the presence of depression significantly influences sleep architecture in PTSD, or whether the changes in sleep architecture can be ascribed to factors unique to the clinical presentation of PTSD. The third and final comparison compared the sleep architecture of the HYP+ and HYP- groups. This last analysis attempts to confirm the first hypothesis in terms of whether hypervigilance serves as one influential mechanism that mediates the relationship between PTSD and disordered sleep. The table below presents the results of the orthogonal planned comparisons on the five sleep variables that differed significantly between groups:

Table 5  
*Results of Orthogonal Planned Comparisons for Objective Measures of Sleep Quality*

Sleep Variable	t	p	ESE
<b>Sleep Efficiency:</b>			
Contrast #1	3.281	.007**	.69
Contrast #2	3.595	.004**	.73
Contrast #3	-.476	.641	.13
<b>Awakenings:</b>			
Contrast #1	-2.384	.032*	.53
Contrast #2	2.797	.018*	.65
Contrast #3	.371	.716	.01
<b>WASO:</b>			
Contrast #1	-2.89	.015*	.67
Contrast #2	2.990	.015*	.70
Contrast #3	1.212	.253	.36
<b>REM latency</b>			
Contrast #1	-1.469	.152	.25
Contrast #2	4.804	.0001**	.71
Contrast #3	.622	.654	.16
<b>SWS%</b>			
Contrast #1	2.393	.025*	.45
Contrast #2	4.2	.024*	.54
Contrast #3	-.81	.179	.38

*Note.*

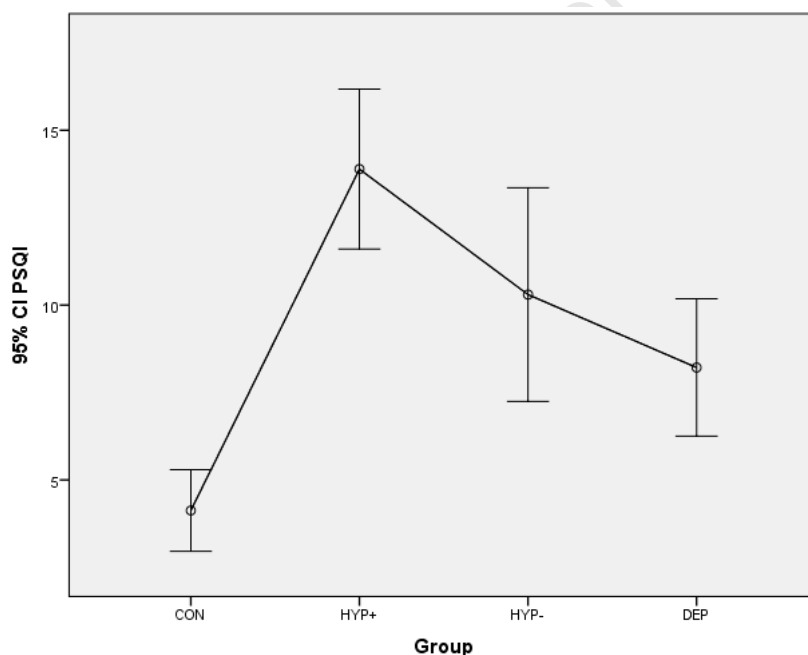
WASO (Wake after sleep onset). HYP+ = group with prominent hypervigilance symptoms, HYP- = group without prominent hypervigilance symptoms, DEP = depressed group, CON = healthy controls. Contrast #1 (HYP+, HYP-, DEP) versus CON; contrast #2 (HYP+, HYP-) versus DEP; contrast #3 HYP+ versus HYP-. \* $p < .05$ . \*\* $p < .01$ . ESE = Effect size estimate

Results of the orthogonal planned comparisons indicate significant differences on four of the five sleep-related variables when the three clinical groups are compared with the CON group with moderate to large effect sizes. Furthermore, the sleep architecture of the two PTSD groups also differs significantly when their sleep architecture is compared to DEP group on the same four sleep-related variables. Lastly, these analyses show a significant difference in the REM latency period between the two PTSD groups and the DEP group, while there are no significant differences evident when the two PTSD groups are compared to the CON group or to each other. Keeping in mind that the three clinical groups were equally matched on depression severity, the

abovementioned differences between the two PTSD groups and the DEP group indicates that there are factors other than depression accounting for significant changes in sleep architecture in the PTSD groups.

The results of the third comparison on all sleep-related variables (HYP+ versus HYP-) revealed no significant differences in sleep architecture between these two groups, therefore the main part of our first hypothesis was not confirmed. However, although it has to be interpreted with caution, there is some indication in the group mean data that the HYP+ group experienced worse sleep quality than the other groups. The orthogonal planned comparisons did however statistically confirm that significant differences in sleep architecture do exist when the three clinical groups are compared to the CON group, and when the two PTSD groups together are compared to the DEP group.

**Inferential statistical analyses of subjective measures of sleep quality.** The PSQI, a self-report questionnaire, was used to obtain subjective ratings of sleep quality in order to be able to compare sleep quality with objective data. Group mean data can be seen below:



*Figure 10.* Group mean data for the subjective measure of sleep quality (PSQI). A high score indicates worse sleep quality.

Descriptive statistics indicate that the HYP+ scored highest on the PSQI (where a high score indicates worse sleep quality) ( $M=13.89$ ;  $SD=2.98$ ). The HYP+ group was followed by the HYP- group with the second highest score on the PSQI ( $M=10.30$ ;  $SD=4.27$ ), with the DEP group scoring third highest ( $M=8.21$ ;  $SD=3.40$ ) followed by the CON group ( $M=4.13$ ;  $SD=2.19$ ). These results reflect similar trends found in the objective data, in terms of the pattern of disturbed sleep described above:  $HYP+ < HYP- < DEP < CON$ . The between-group analyses yielded significant differences between the groups with  $F(3, 45) = 19.69$ ,  $p = .0001$ . The effect size for the PSQI was .51.

Similarly to the analyses conducted on the objective data, it is important to explicate the nature and direction of the between-group differences. Therefore it was decided to run orthogonal planned comparison on the subjective data using the same logic in the aforementioned section. The first comparison (HYP+, HYP-, DEP versus CON) indicated that there is a significant difference in the overall sleep quality generated by the PSQI between the three clinical groups and the CON group ( $p < .0001$ ;  $ESE = .84$ ) With regard to the second comparison (HYP+, HYP- versus DEP), significant differences were detected ( $p = .0001$ ;  $ESE = .88$ ), indicating that the two PTSD groups reported worse sleep quality than the DEP group. Finally the sleep quality scores of the HYP+ and HYP- group were compared. Results revealed a significant difference between these two groups ( $p = .018$ ;  $ESE = .71$ ).

The results obtained from the analyses of the subjective measures of sleep quality mirrors the findings and some of the trends generated from the objective data. The only divergence in the results relates to the detection of a significant difference between the HYP+ and HYP- groups, where the objective data did not reveal any significant difference between these two groups. Possible reasons underlying this finding will be discussed in the Discussion section.

### **Testing Hypothesis 2: Dream content and theme**

In terms of the second hypothesis of this study, we postulated that the HYP+ group would have more negative dream content and theme when compared to the HYP-, DEP and CON group. Dream content scores were obtained by averaging the mean scores of two reports, the first one filled out at the screening interview, and the second report on the morning following testing at the sleep laboratory. Firstly, a correlation analysis was run in order to determine the inter-rater reliability between the three raters. Pearson's correlation showed a statistically

significant correlation between the three raters,  $r = .85$ ,  $p = < .05$ . Next, one-way ANOVA was run in order to examine the effect of the group condition on dream content and theme. Our hypothesis regarding dream theme was not confirmed as no significant differences were found between groups  $F(3, 47) = 2.429$ ,  $p = 0.78$ . Furthermore, our hypothesis regarding dream content was not confirmed either as no significant differences were found between the four groups  $F(3, 44) = 2.789$ ,  $p = .052$ . However, the data seems to be approaching significance for dream content, which could mean that significant differences might emerge with a bigger sample size. The fact that the dream content scores between the three clinical groups and the CON group do not differ significantly is a very surprising finding, and will be discussed in more detail in the Discussion section. However, the mean trends are supportive of the direction of our hypothesis, i.e. that the HYP+ group will have lower content scores (indicating worse dreams) than the other three groups. When the mean scores of all the groups are considered together, results support the trends demonstrated previously by the objective and subjective sleep data in the following way:  $HYP+ < HYP- < DEP < CON$ . Results of the cell-mean plot can be seen below:

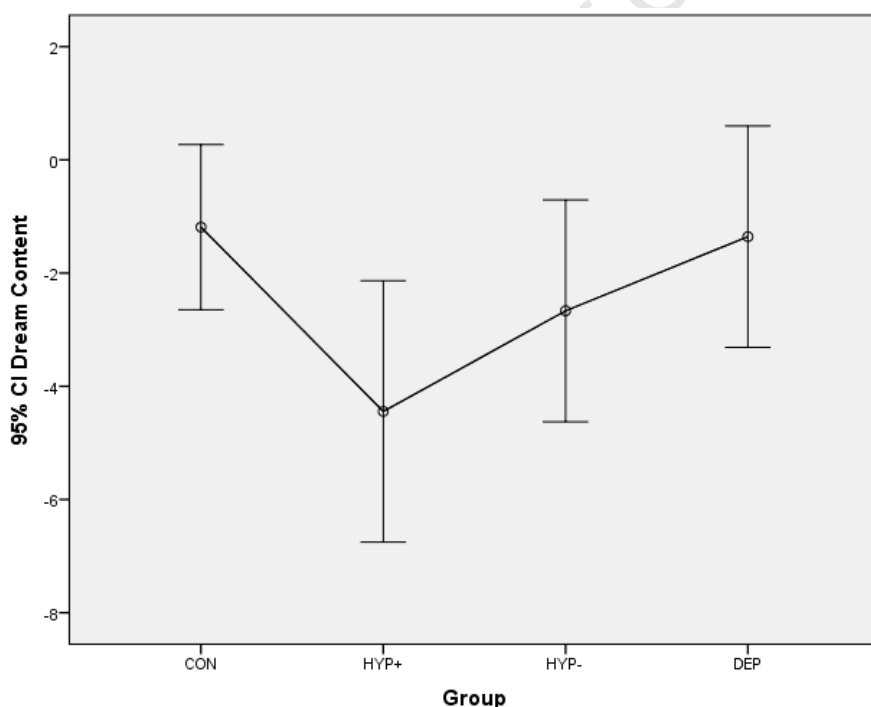


Figure 11. Group mean data for dream content scores. A lower score indicates more negative content.

In light of the data approaching significance the decision was made to proceed with orthogonal planned comparisons using the same logic as in the previous sections. In terms of the first comparison (HYP+, HYP-, DEP versus CON) no significant differences were found ( $p = .08$ ). In the second comparison (HYP+, HYP- versus DEP) a significant difference was found ( $p = .043$ ). However, in the final comparison (HYP+ versus HYP-) no significant differences were detected ( $p = .210$ ).

## DISCUSSION

It is a well-known fact that a major feature of PTSD symptom-manifestation is characterized by sleep disturbances. There is a vast literature supporting this premise, while disturbed sleep is also reflected in the diagnostic clusters associated with PTSD. However, the literature is often marked by discrepant findings regarding the existence, the nature, as well as the extent of sleep disturbances in PTSD. As mentioned in previous sections, some studies claim to have found difference in the sleep architecture of individuals with PTSD when compared to healthy controls, while others have not. There is also little agreement on the specific characteristics of disturbed sleep in these individuals when differences were detected. Furthermore, many studies are confounded by different methodological constraints, which include the use of heterogeneous groups in terms of age, sex, type of trauma, time since trauma, comorbid psychiatric conditions, as well as discrepancies across other demographic factors.

This study attempted to overcome many of the confounding factors mentioned above and address some of the main and often contentious issues still evident in the PTSD and sleep literature. Although the main hypothesis of this study was not statistically confirmed (that prominent hypervigilance symptoms results in more disturbed sleep compared to individuals with PTSD without such symptoms), it did yield significant results in terms of two contentious issues in the literature. Firstly, this study provided strong evidence for the premise that individuals with PTSD experience significantly worse sleep quality than healthy controls. Secondly, when the sleep architecture of individuals with PTSD is compared to individuals with depression, PTSD individuals exhibit significantly worse sleep quality. Therefore, there are factors unique to the clinical presentation of PTSD that results in disturbed sleep independent of the effects of depression. These findings can be stated with reasonable confidence due to this study overcoming many methodological constraints making results more generalizable. These methodological factors are discussed below.

One of the main methodological aims, that also constitute a major contributing factor of this study, was to create a very tightly controlled homogeneous sample. This was achieved by implementing very rigorous exclusion criteria. However, the downfall of such a strategy is inevitably ending up with a much smaller sample size due to high exclusion rates. However, the literature on PTSD and sleep is often characterized by many studies exerting little control over

various demographic and psychiatric factors, and for this reason it was decided to proceed with the abovementioned strategy.

Not only are groups frequently not homogenous, but some PTSD populations are under-represented in the literature. Harvey, Jones and Schmidt (2003), reported that only 3 out of 26 studies included participants that experienced a traumatic event that is not war-related. Furthermore, there is not only a lack of studies investigating other kinds of traumatic events, but more specifically, traumatic events that have a much higher prevalence rate in the general population. Sexual assault is an example of such a traumatic event. Creating a homogeneous sample and investigating a PTSD population other than war veterans, played a major role in the overall design of the study. More specific methodological considerations will be discussed below.

With regard to deciding on an age range for a study, it is imperative to consider the natural changes in sleep architecture that take place after the age of 40. The literature indicates that older adults tend to have a longer sleep latency, lower sleep efficiency, more frequent awakenings and decreased SWS and REM sleep (Blackman, 2000). Therefore including participants over the age 40 might influence the validity of conclusions drawn regarding disturbed sleep architecture, i.e. are the changes in sleep architecture due to the influence of PTSD symptomatology or due to age-related changes?

In terms of the sex of participants, this study decided to focus solely on woman. Although an ideal design would entail including men and women as the influence of sex differences on sleep architecture has not been fully explored. However, only woman were included in this study mainly for two reasons. Firstly, including only women fits in well with one of the main methodological objectives of this study in terms of creating a tightly controlled homogenous sample. Another main reason relates to the fact that the PTSD and sleep literature is identifiable mostly by studies focusing on men who have suffered war-related trauma. Choosing to study women who are sexual assault survivors is important in terms of delineating influential factors unique to women, and unique to sexual assault as a traumatic experience. In addition, this population group is under-represented in the literature, even though sexual assault occurs more frequently than war-related trauma in the general population. For instance, South Africa has one of the highest incidence rates of rape in the world, making this type of trauma a worthwhile and relevant avenue of investigation.

In terms of creating a homogenous group in relation to time since trauma is important, because the later manifestations of disturbed sleep in PTSD might be different from the earlier manifestations. Also measuring sleep architecture relatively early in the manifestation of PTSD (between 6 months and 5 years), reduces the likelihood of other covariant factors influencing results (Harvey et al., 2003).

In addition to these factors, studies are also confounded by the presence of comorbid psychiatric diagnoses, especially in terms of depression and substance abuse, for example. It is especially depression as a possible influential factor that needs to be controlled for. Firstly, depression frequently presents comorbidly with PTSD, and for this reason a depression-only group was included in order to control for the effects of depression on sleep architecture. Secondly, depression exerts a well-characterized influence on sleep architecture. The changes in sleep architecture typically include a decrease in SWS, decreased REM latency, and increased REM percentage (Franzen & Buysse, 2009). It is also worth mentioning that almost all of the PTSD participants included in this study presented with moderate to severe depression. It is possible that samples from developing countries like South Africa might present with higher rates of depression due to the prevalence of high rates of poverty and unemployment. Therefore, it might be difficult to recruit a large enough sample of individuals without depression symptomatology, however, including individuals with high rates of depression is certainly more clinically relevant to PTSD as a clinical syndrome.

A very significant contribution of this study relates to the use of objective measures of sleep quality in conjunction with subjective measures. To the knowledge of the author, very few studies have adopted this approach, where studies frequently rely solely on subjective reports of sleep quality. The combination of these two methods of data collection is imperative, as it enables one to view the nature of sleep disturbances in a holistic fashion. It also enables one to draw more accurate conclusions regarding the overall phenomena of what constitutes sleep disturbances across objective and subjective measures of sleep quality that are specific to PTSD. This is important not only to see if findings of subjective measures of sleep quality are consistent with objective measures of sleep quality, but it is also important to investigate if, and in what way results from subjective measures digress from results of objective measures.

In summary, the design of this study attempted to overcome many of the confounding factors evident in the PTSD literature by creating a homogenous group in terms of various

demographic factors, controlling for comorbid depression by including a group of depressed individuals, as well as implementing rigorous exclusion criteria to control for other psychiatric conditions and other factors that might adversely affect the results obtained from the different measures. Even though this strategy resulted in a much smaller sample size, exerting rigorous control over group characteristics is a major contributing factor of this study.

### **Sleep-related variables and sleep parameters included in the study**

In order to characterize the typical sleep architecture associated with PTSD, this study attempted to look at various sleep parameters through objective measures that might form part of disturbed sleep architecture in PTSD. More specifically, in the context of this study that aims to identify one influential mechanism underlying disturbed sleep in PTSD, the sleep variables considered for inclusion relates to its relationship with hypervigilance symptoms. Firstly, in terms of the sleep variable sleep latency, to the knowledge of the author, previous studies have not investigated this variable in relation to individuals with prominent hypervigilance symptoms. Therefore it was included in order to determine whether individuals with such symptoms do in fact take longer to fall asleep compared to the HYP-, the DEP and the CON groups. Secondly, although awakenings in PTSD (without regard to the presence or absence of prominent hypervigilance symptoms) have been compared with controls, this study aimed to determine whether hypervigilance plays a particularly important role in the frequency of awakenings by comparing the number of awakenings with the other three groups. Thirdly, WASO has not been thoroughly investigated in relation to prominent hypervigilance symptoms in PTSD, and for this reason it was also included. These three sleep variables were considered important for inclusion as they are reflected in the diagnostic criteria of the symptom cluster 'hyperarousal' in the instrument used to diagnose PTSD in this study (the CAPS). In this instrument, hyperarousal is characterized by an inability to fall asleep (sleep latency) as well as problems with sleep maintenance (awakenings and WASO). Finally, in terms of the overall lack of research conducted with specific reference to prominent hypervigilance symptoms, the sleep variable sleep efficiency was also included in order to assess overall sleep quality across the four different groups.

With regard to other kinds of sleep parameters, specifically related to the different sleep stages, SWS%, REM sleep% as well as REM latency were included to provide a more detailed

characterization of the typical sleep architecture associated with PTSD. There is little agreement on the specific distribution of the various sleep stages and related variables that are unique to PTSD, but it is also important to be able to delineate the possible influence of depression on sleep architecture. As mentioned in previous sections, depression is associated with well-defined changes in sleep stage distribution and associated variables, as it typically results reduced SWS%, increased REM sleep% and a shortened REM sleep latency period, i.e. the first onset to REM sleep is shortened in these individuals. Therefore, these variables were also included in the objective analyses of sleep architecture in PTSD.

A self-report instrument, in this case the PSQI was used to compare the findings of objective measures with the findings of subjective measures. Although using objective measures constitutes the best way to accurately depict sleep architecture, the approach of using subjective measures in conjunction with objective measures is integral to the design of the study, as mentioned earlier. It is important not only to obtain a holistic perspective on the sleep architecture of PTSD individuals, but it is also imperative in terms of detecting any patterns that are consistent with, or deviate from the findings of the objective measures.

### **Objective measures of sleep quality: Sleep-adapted EEG**

**Trends in cell-mean plots.** The first analyses on the sleep architecture of the four groups were done on the objective measure of sleep quality. The mean scores on the cell-mean plots outlined a clear trend of worse sleep quality for sleep efficiency, number of awakenings and WASO in the following way: HYP+ < HYP- < DEP < CON. Trends that deviated from this pattern of disturbed sleep include much longer sleep latency for the HYP- group, while this group also had a longer REM latency, more SWS and less REM% when compared to the other groups. One way of interpreting the findings related to sleep latency, SWS and REM latency, is considering the principles that generally govern sleep in humans. These governing forces include the homeostatic and circadian processes of sleep (Borbély, Achermann, Trachsel, & Tobler, 1989). SWS is believed to be governed by the homeostatic process and REM sleep by the circadian process. The pressure to enter SWS is increased as time of wakefulness increases. In other words, the longer the period of wakefulness, the sooner SWS will occur once the person has fallen asleep, while there will also be an increase in the amount of SWS. Therefore if a

prolonged period of wakefulness occurs there will be more SWS during the night while REM latency will be increased. This could possibly serve as one explanation for the trends observed in the cell mean plots for the HYP- group, i.e. it took them longer to fall asleep (increased wakefulness), they had more SWS (homeostatic pressure) with a subsequent increase in REM latency.

Another interesting finding in terms of sleep latency is that the HYP+ group fell asleep the quickest. This is unexpected given their symptomatology of increased arousal. An important consideration to keep in mind when conducting research on this population group in developing countries is that the possibility exists that participants might in fact sleep better in the laboratory environment. Almost all participants live in townships in and around Cape Town. These residential regions are typically characterized as unsafe environments due to high levels of violent crime, while other factors could influence sleep quality such as high levels of noise and overcrowding in shacks and bedrooms. The participants themselves described their living conditions as less than ideal. With the conclusion of the sleep testing night, all participants were asked if they slept better worse than usual. Almost all participants answered that they slept much better than usual due to feeling safer, having adequate temperature control, and being able to sleep alone in a bed. This is a very interesting finding, and somewhat paradoxical in terms of the expected influence that the laboratory environment exerts on sleep architecture. There is a phenomenon that has been noted when conducting sleep research called the 'first night effect'. The first night effect is based on some studies reporting worse sleep quality on the first night in the laboratory due to being in a novel environment and not being used to the PSG equipment (Lorenzo, & Barbanj, 2002). Our study found the opposite to be true for our participants, i.e. they all reported that they slept much better in the laboratory environment. The extent of perceived improved sleep quality was not anticipated in this study, and certainly raises some interesting questions regarding conducting sleep studies in developing countries with a low SES population.

The notion of feeling safe is especially relevant to the two PTSD groups due their history of experiencing a traumatic event (Spoomaker & Montgomery, 2008). For example, these authors also report a decrease in the frequency of nightmares and disconcerting dreams in the laboratory environment. This is even more applicable to the hypervigilant group, because hypervigilance is characterized by an over-concern for safety, increased watchfulness and an

exaggerated startle response. This could possibly be one reason why sleep latency was reduced in the HYP+ group individuals in the laboratory setting. Furthermore if one keeps the trends of disturbed sleep in mind that have been observed thus far ( $HYP+ < HYP- < DEP < CON$ ), one could tentatively assume that the HYP+ group is possibly more sleep deprived to a certain extent than the other three groups. Therefore it is possible that a combination of feeling safer in the laboratory and increased homeostatic pressure due to prolonged wakefulness, could have contributed to these individuals falling asleep much quicker. However, these propositions are only speculative at this stage.

In terms of the SWS% cell mean plots, the DEP group had the least amount of SWS followed by the HYP+ group. It is a well-established fact that depression results in decreased SWS. Participants in the HYP+ and DEP group all had moderate to severe depression, therefore it seems feasible that depression could be an influential factor in this regard.

Findings more specific to the DEP group include that this group had the same amount of REM% as the HYP+ and CON group, but a much shortened REM latency. This is interesting because the literature on sleep architecture of depressed individuals consistently show a decrease in SWS, shortened REM latency (both of these findings are consistent with our data) and an increase in REM percentage (which we did not find) (Franzen & Buysse, 2009).

**Parametric testing.** With regard to parametric testing, between-groups comparisons indicated significantly worse sleep quality across groups for 5 of the sleep-related variables (sleep efficiency, awakenings, WASO, SWS% and REM latency). Although these results are significant, it does not in itself confirm our first hypothesis, i.e. that the HYP+ group will experience significantly worse sleep quality compared to all other groups, and the related hypothesis in terms of the pattern of disturbed sleep being distributed in the following way:  $HYP+ < HYP- < DEP < CON$ .

**Planned comparison 1: Clinical groups vs. control group.** In order to identify an underlying mechanism that mediates the relationships between disordered sleep and PTSD, further analyses were conducted to elucidate the nature of the differences that were suggested by the overall model. The first thing that the planned comparisons revealed was that there are indeed significant differences in the sleep architecture of individuals with PTSD (HYP+ and HYP-) and individuals with depression are compared with controls on four of the five sleep-related variables that were significant in the previous analysis (sleep efficiency, awakenings, WASO, REM

latency and SWS%). Although it was not the main aim of the study, these findings are still noteworthy as differences in sleep architecture between PTSD individuals and healthy controls still serve as a contentious issue in the literature.

Pillar, Malhotra, & Lavie (2000) in their comprehensive review article used pooled standard PSG outcomes of studies comparing the sleep architecture of PTSD individuals with healthy controls. They note that PTSD is not associated with any deviations in sleep architecture in terms of standard outcomes of polysomnographic measurement. However, Kobayashi, Boarts, & Delahanty (2007) in their meta-analysis of 20 PSG studies demonstrated that differences are indeed present in terms of more stage 1 sleep, less SWS and increased REM density. These authors also note the influential role of moderating variables when sleep data is collected. In summary, there is little agreement in the literature in terms of the existence of significant differences in sleep architecture when PTSD individuals are compared with controls, with some studies detecting a difference, while others detect no differences. It is believed that different methodological constraints play a prominent role in this regard. Results from this study strongly supports the premise that there are significant differences in the sleep architecture of PTSD individuals (HYP+ and HYP- grouped together) when compared to healthy controls.

**Planned comparison 2: PTSD groups vs. depressed group.** The second planned comparison revealed another important finding, and clarifies an important issue that often confounds the findings in some studies. It showed that when the sleep architecture of the two PTSD groups is compared with the DEP group, significant differences are present for the same sleep-related variables mentioned above. It is important to remember at this stage that the groups were equally matched on depression severity. In other words, when depression is controlled for, it is clear that the two PTSD groups exhibit significantly worse sleep quality than the depressed individuals.

It has to be noted that differences in sleep architecture between individuals with PTSD and individuals diagnosed with depression also serve as a contentious issue in the literature. Some studies have found no noteworthy differences in the sleep architecture of PTSD individuals compared with depressed individuals (Woodward, Friedman, & Biliswe, 1996), while other studies have detected differences in sleep architecture when individuals with PTSD are compared to depressed individuals and healthy controls (Mellman et al., 1997), Furthermore, Yetkin, Aydin, & Ozgen (2010) found that the sleep architecture of individuals with PTSD who

are also diagnosed with depression exhibit different properties than the sleep architecture of individuals who are diagnosed with PTSD only. These differences include decreased sleep efficiency, decreased total sleep time, increased number of awakenings, and longer sleep latency. Our study also found significant differences in some of these parameters (sleep efficiency, number of awakenings, for example), in the PTSD groups (who are all also diagnosed with major depression), however, the disturbances were significantly worse when compared to the depression-only group. Therefore our results indicate that there are factors over and above depressive symptomatology that lead to significant changes in sleep architecture.

This suggests that there must be other mechanisms at work that are unique to the clinical presentation of PTSD that plays a prominent role in disturbed sleep architecture in this population group. This is exactly what the subsequent analysis attempted to delineate. We postulated that one of the main mechanisms in this relationship relates to the presence of prominent hypervigilance symptoms that form part of the state of hyperarousal. This hypothesis was generated because these two related phenomena are underpinned by defective neurobiological structures and processes that influence the stress response and sleep architecture, amongst other things. Therefore it was important to compare the sleep architecture of the hypervigilant group with the sleep architecture of the group without prominent hypervigilance symptoms. This constitutes the third and final comparison.

**Planned comparison 3: Hypervigilant group vs. Non-hypervigilant group.** When these two groups were compared with each other, no significant results were detected on four of the 5 sleep-related variables, except in terms of REM latency. Therefore this analysis did not statistically confirm our main hypothesis, i.e. these results do not provide evidence for the premise that hypervigilance serves as one influential mechanism causing disordered sleep in PTSD. There are a couple of possible reasons for this non-significant finding. Firstly, as mentioned in the previous section, individuals with PTSD reported that they slept much better than usual on the night of testing in the sleep lab. It was hypothesized that this could possibly be due to these individuals feeling much safer in the laboratory environment. This might be particularly applicable to the hypervigilant group as the behavioural manifestations of prominent hypervigilance symptoms include an over-concern for safety, increased watchfulness and an exaggerated startle response. Therefore, the hypervigilant group might possibly respond more positively to a safe environment than the non-hypervigilant group due to the nature of their

symptomatology. Another factor that might possibly have exerted an influence on the results that were obtained relates to the relatively small sample size of the two PTSD groups. Although recruitment for this study was affected by various practical challenges, it is imperative for future studies to recruit a larger sample size in order to acquire more statistical power when investigating this hypothesis.

In summary, several analyses provided strong evidence for significant differences in sleep architecture when the three clinical groups were compared with the CON group, as well as significant differences between the two PTSD groups and the DEP group. Although no significant differences were found between the HYP+ and HYP- , group mean trends are suggestive of worse sleep quality in the HYP+ group on some sleep-related variables. Other things that need to be taken into consideration relates to certain environmental factors exerting an influence on data collected at the sleep testing night, as described above. To conclude, overall, the trend of sleep disturbances as measured by objective measures can tentatively be summarized in the following way: HYP+ < HYP- < DEP < CON.

### **Subjective measures of sleep quality: The PSQI**

The self-report measure, the PSQI was used in order to acquire subjective ratings of overall sleep quality. As mentioned earlier, a major contributing factor of this study relates to the use of objective measures in conjunction with subjective measures of sleep quality. The results of the few sleep studies (not specific to PTSD) that have utilized this approach are often indicative of discrepancies when the objective measures of sleep quality are compared to the subjective ratings of sleep quality. That is to say, people often report worse sleep quality than what was detected by polysomnography.

This phenomenon is known self-report bias. Nofzinger et al. (2004) investigated the prevalence of self-report bias and found that the median absolute magnitude of over-estimation was approximated to be as high as 27%. However, the notion of a self-report bias is influenced by various factors such as age, sex and intelligence level. Furthermore, previous studies have indicated that children and adolescents, males and individuals with a lower level of intelligence, are more biased in terms of providing self-aggrandizing reports on personality inventories when compared to females, older adults, and individuals with a higher level of intelligence. With

regard to this study, discrepancies in such self-report bias were most likely reduced due to the creation of a homogenous group in terms of age, sex, and intelligence level.

In addition to this, the rate of self-report bias may have been reduced due to evidence produced by previous studies that show when a questionnaire is completed in person there are lower tendencies towards self-report bias (Nofzinger et al., 2004). The current study adopted such an approach. Furthermore, Nederhof (1985) propose that when forced-choice items are included in a questionnaire, self-report bias can be decreased, as was the case in the current study.

Overall, the data obtained in this study from using sleep quality scores of the PSQI, tend to be consistent with trends observed in objective measures of sleep quality. That is to say that there was a trend towards worse sleep quality in the same direction mentioned in the previous section:  $HYP+ < HYP- < DEP < CON$ . The overall model of between-group differences were significant for the sleep quality score, while planned comparisons revealed significant differences in sleep quality between the three clinical groups and the CON group, a significant difference between the two PTSD groups and the DEP group, as well as a significant difference between the HYP+ and the HYP- group. This last finding indicating a significant difference in subjective sleep quality between the HYP+ and HYP- group, is the only finding that deviates from the results obtained from the objective measure of sleep quality. Although there were trends suggestive of worse sleep quality when in the HYP+ group in the objective data, this difference did not reach statistical significance.

As mentioned above, the findings from the subjective measure of sleep quality statistically support the findings of sleep disturbances as determined by objective measures of sleep quality in all but one way. And for this reason, one can assume that self-report bias was most likely reduced in this study due to the factors stipulated above. However, the significant difference in sleep quality between the HYP+ and HYP- might to a certain extent be accounted for by what is known as 'sleep-state misperception'.

Sleep-state misperception occurs when an individual reports more severe sleep disruptions than what was detected and verified by objective measures of sleep quality. The notion of sleep-state misperception is grounded in perceptual alterations in terms of perceived sleep quality. One could argue that the sleep-symptoms specific to hypervigilance, as reflected in the diagnostic cluster of hyperarousal found in the CAPS (i.e. frequent awakenings and more time spent awake

after sleep onset), could have inflated the sleep maintenance constructs that form part of the overall sleep quality score as determined by the PSQI. Although objective measures did detect trends in the data indicative of differences regarding these sleep variables between the HYP+ and HYP- groups, these differences might be more pronounced in self-report questionnaires, as there is a well-established tendency of complaints of individuals to be augmented when questionnaires are used to measure different psychological constructs. Furthermore, there might be some validity in the premise that hypervigilant individuals, due to the nature of their symptomatology as reflected in diagnostic instruments (increased sleep latency and problems with sleep maintenance), experience worse sleep quality than individuals without such symptoms. This might be especially true in terms of sleep maintenance, and there might be a higher rate of preoccupation with problems that do exist (due to the physical and behavioral components that form part of the state of hypervigilance), resulting in reports that indicate more severe sleep disturbances.

In summary, results from subjective measures of sleep quality provide statistically significant evidence indicative of more disturbed sleep across the four groups in the following way: HYP+ < HYP- < DEP < CON. These findings are mostly consistent with trends observed in the objective measures of sleep quality.

### **Dream Content Scores**

Our hypothesis regarding more negative dream content and theme in the HYP+ group compared to the other groups was not confirmed. However, the data did approach significance in terms of dream content, but a bigger and more representative sample is needed to substantiate these findings. Furthermore, the cell-mean plot confirmed that the direction of our hypothesis was correct (HYP+ < HYP- < DEP < CON).

The fact that the results obtained from analyzing the dream data did not reach statistical significance was unexpected and not consistent with previous literature. For example, a longitudinal study by Pesant & Zadra (2005) found more negative dream content in individuals who are depressed, where their dreams tend to have increased representations of aggression and negative emotions, for example. In terms of PTSD, nightmares are considered a mechanism of intrusion where the traumatic event is replayed, while it also forms part of the diagnostic criteria of PTSD (Pillar et al., 2000). Therefore the non-significant results were quite unexpected, and possible reasons for this will be discussed subsequent paragraphs.

Not having a representative sample of dreams from all the participants could be a major confounding factor in this regard. The procedure entailed the filling out of two Most Recent Dream Reports on two separate occasions (the 1<sup>st</sup> one at the screening and the 2<sup>nd</sup> one post-testing at the sleep laboratory). Keeping in mind that the requirement for the timing of the trauma is between 6 months and 5 years ago, collecting only two dreams over this time period since the onset of PTSD is not sufficient in order to draw definite conclusions. However, due to practical reasons, it was not possible to obtain a representative sample of dreams since the onset of PTSD in these participants. The filling out of two reports at the stipulated times above was regarded as the most efficient strategy that could be implemented for this study, and future research is needed in terms of determining what constitutes a representative sample of dreams in this population group.

Another confounding factor possibly distorting the dream report data could relate to the language proficiency of the participants. For this study English proficiency was required for participation to take place. Almost all participants reported that English was their second language, with isiXhosa followed by Afrikaans, being listed as their first language. It was often the case that screenings had to be terminated prematurely due to potential participants not meeting the language proficiency requirement. Even those participants that did meet the minimum English proficiency requirements, communication in English between the researcher and the participants was still problematic in some instances. The validity of the dream report relies on the adequate articulation of dream events, dream characters, and thoughts and feelings that were present in the dream. The scoring of the dream content was designed to rely on keywords denoting emotive content and experiences. It is possible that the dream reports (filled out in English) could be less animated and less descriptive than what they would have been if participants filled out the dream reports in their first language, where they presumably would have had access to a more extensive vocabulary.

A possible third confounding factor in terms of the accuracy of the dream reports, relates to the reliance on memory when participants were asked to recall dreams. It was often observed that (a) participants struggled considerably to recall dreams in its entirety, or important aspects of their dreams, and (b) that participants sometimes found the act of remembering their dreams very disconcerting. This was especially true when the dream was a replica of the traumatic event or contained traces of some related or unrelated traumatic/violent experience.

With regard to a possible explanation for (a), it is well known that memory performances in individuals with PTSD are impaired in different ways (Jenkins, Langlais, Delis, & Cohen, 1998) that could all affect the rate and accuracy of dream recall. Verbal memory impairments, word fluency, and associated cognitive performance deficits have been reported in individuals with PTSD when compared to healthy controls (Johnsen & Asbjørnsen, 2008 Uddo, Vasterling, Brailey, & Sutker, 1993).

In terms of a possible explanation for (b) it is important to consider the nature of PTSD as a clinical syndrome and the implications of the different diagnostic clusters on symptom-expression in these individuals. The numbing and avoidance cluster (Cluster C) could have affected how participants engaged with dream content, for example, making attempts to block out disconcerting components of dreams or providing dreams that contain less severe theme and content in an attempt to avoid being confronted with potentially traumatic content of their dreams.

The following case study illustrates the tendency to block out potentially traumatic dream content: With the conclusion of the screening interview, a participant was asked to fill out the first Most Recent Dream questionnaire. The instructions were clearly explained and the participant indicated that she understood. Following this, the participant was provided with as much time as she needed to reflect on her dreams in order for her to provide as much detail in the report as she can. 10 Minutes elapsed and the participant had still not written anything down. The instructions were explained again, and she confirmed for a second time that she understood. About five minutes after explaining the instructions for a second time, the participant started crying hysterically and asked to leave and expressed that she doesn't think that she will be able to participate in the study. She was consoled and given the opportunity to discuss anything that she was feeling, while she was reminded that all conversations are confidential. She reported that in the past she frequently had disconcerting dreams that reflected the traumatic event that she experienced and that it was too unsettling for her to think about them, while she also struggled to remember them clearly. The participant expressed the need to leave due to feeling too distressed after attempting to recall her dreams. However, she returned voluntarily at a later stage and did eventually provide two dream reports. Cases like the abovementioned one support the notion that data obtained from dream reports may have been influenced by the active efforts of PTSD participants to avoid being confronted by traumatic content in their dreams.

### **Limitations and Directions for Future Research**

Probably the most obvious limitation of this research relates to the small sample size. This limitation is due to several reasons. Firstly, in an attempt to overcome many of the methodological constraints evident in other studies, this study had a very high exclusion rate due to the rigorous exclusion criteria that were implemented. On average only 16/100 individuals who responded telephonically were eligible to attend the screening interview (due to experiencing more than one traumatic event, often as toddlers or teenagers), while almost half of participants that were eligible for the screening interview were excluded based on the various exclusion criteria listed in previous sections.

Another important factor influencing sample size can be ascribed to the socio-economic status of the population. It was often the case that participants were unable to afford public transportation costs to participate in screenings, even though they were reimbursed with the completion of the screening interview. It was also on many occasions not deemed safe for the researcher to enter the townships at certain times to provide transport for the participants. For example, there were many instances of periods of prolonged violence and protests in these areas during times of political turmoil in the country. Furthermore, due to high levels of violent crime in these areas, certain times of the day were also regarded as unsafe. Therefore, many eligible participants who responded to the advertisements placed in newspaper never made it to the screening interview.

The sample size was further reduced with the exclusion of males and restricting the type of trauma to sexual assault. Furthermore, substance abuse, especially alcohol abuse also resulted in numerous exclusions. However, it was imperative to exclude these individuals in light of results of previous studies being greatly confounded by including individuals with alcohol abuse problems. As mentioned in previous sections, alcohol abuse exerts a prominent influence on sleep architecture resulting in changes in many sleep parameters (Irwin, Miller, Gillan, Demodena, & Ehlers, 2000).

In summary, although the rigorous exclusion criteria resulted in a very high exclusion rate inevitably leading to a smaller sample size, this study decided proceed with the exclusion strategy in order to create a tightly controlled homogeneous group. Although the small sample

size is a limitation of this study, overcoming several methodological constraints by creating a homogenous group, is a major contributing factor.

Another limitation of this research relates especially to the procedure of the dream reports. Several reasons for this were outlined above, like language proficiency, not enough reports over a large time span and the influence of PTSD symptomatology on the recall and reporting of dreams. Future research should aim firstly to establish how many reports constitute a representative sample in this population group. Domhoff (2000) suggests that no less than 100 dreams should be collected for the sample as a whole. Furthermore, research should also aim to evaluate the efficacy of using dream journals as opposed to single reports in terms of obtaining a larger and more detailed sample of dreams.

This study aimed to create a homogenous group in terms of type of trauma and time since trauma and only looked at interpersonal violence. Although this is regarded as a relevant avenue of research and important to the design of the study, the heterogeneity in terms of type of trauma in PTSD in general should not be discounted. This is important because the nature of sleep disturbances across different types of trauma have not been clearly demarcated. The development of PTSD is associated with a broad range of traumatic events, and although interpersonal violence is understudied in the PTSD literature, it would be interesting if future research could compare the nature of disturbed sleep found in different types of trauma like vehicular accidents or a natural disaster, for example.

## CONCLUSION

The results of this study can address two important issues with relation to PTSD and sleep architecture. Firstly, results demonstrate that there are indeed significant differences in the sleep architecture of individuals with PTSD when compared with healthy controls on subjective measures of sleep quality, as well as a number of sleep variables on objective measures. These sleep variables include sleep efficiency, number of awakenings, WASO, and SWS%. Differences in sleep architecture between these two groups still serve as a contentious issue in the PTSD literature. This could partly be due to various methodological differences. This study attempted to create a homogenous group in terms of sex, age, socio-economic status, performance IQ, type of trauma, proximity to trauma, psychiatric and pharmacological treatment, as well as comorbid psychiatric diagnoses. This was an attempt to overcome many of the methodological constraints prevalent in the PTSD literature. Secondly, as an elaboration of this, by including a group of individuals diagnosed with depression, the results also show that when depression is controlled for, PTSD individuals still exhibit significantly worse sleep quality on both objective and subjective measures of sleep quality.

The main aim of this study was to not only determine the nature and extent of sleep disturbances in individuals with PTSD, but more importantly, to identify a possible influential mechanism that underlies disordered sleep architecture in PTSD. Such a mechanism, to the knowledge of the author, has not been proposed by previous studies investigating sleep in PTSD. Firstly, this study set out to investigate whether the presence of prominent hypervigilance symptoms in PTSD could account for more disturbed sleep in individuals with PTSD compared to individuals without such symptoms, as well as when compared to controls and depressed individuals. Secondly it evaluated whether the presence of prominent hypervigilance symptoms leads to more negative dream content and theme when compared to the other three groups.

Objective and subjective measures were utilized to measure sleep quality. As mentioned above, several analyses revealed statistically significant differences in sleep architecture when the hypervigilant group was compared to the healthy controls and the depressed group. Results from subjective measures overall mirrored findings from objective measures. Our hypothesis regarding identifying hypervigilance as one influential mechanism mediating the relationship between PTSD and sleep was not statistically confirmed. However, although these results should

be interpreted with caution, group mean data was suggestive of a trend of worse sleep quality in the HYP+ group on a number of sleep-related variables when compared to the other groups. Interestingly enough, data from subjective measures revealed a significant difference between the sleep quality of the HYP+ and the HYP- group.

In summary, when all the data of objective and subjective measures of sleep quality is taken into consideration in conjunction with the dream data, one can tentatively state that the overall pattern of the severity of disturbances across the different measures can be outlined in the following way HYP+ < HYP- < DEP < CON. Although this study did not statistically confirm our main hypothesis, it did reveal some interesting trends for future research to build on.

University of Cape Town

## 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 (DSM-IV-TR ed.)*. Arlington: American Psychiatric Association.
- Balgrove, M., & Haywood, S. (2006). Evaluating the awakening criterion in the definition of nightmares: How certain are people in judging whether a nightmare woke them up? *Journal of Sleep Research, 15*, 117-124.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the beck depression inventory-II*. San Antonio: Psychological Corporation.
- Bekker, D., & van Velden, D. P. (2003). Alcohol misuse in patients attending a defense force general medical clinic. *South African Family Practice, 45*, 10-15.
- Blackman, M. R. (2000). Age-related alterations in sleep quality and neuroendocrine function: Interrelationships and implications. *Journal of the American Medical Association, 248*, 879-881.
- Blake, D. D., Weathers, 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*, 75-90.
- Borbély, A. A., Achermann, P., Trachsel, L., & Tobler, I. (1989). Sleep initiation and initial sleep intensity: Interactions of homeostatic and circadian mechanisms. *Journal of Biological Rhythms, 4*, 37-48.
- Bremner, J. D. (1999). *Does stress damage the brain?*. *Biological Psychiatry, 45*, 797-805.
- Bryant, R. A., Marosszeky, J. E., Crooks, J., & Gurka, J. A. (2000). Posttraumatic stress disorder after severe traumatic brain injury. *American Journal of Psychiatry, 157*, 629-631.
- Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). *The pittsburgh sleep quality index (PSQI): A new instrument for psychiatric research and practice*. *Psychiatric Research, 28*, 193-213.
- Caldwell, B. A., & Redeker, N. (2005). Sleep and trauma: An overview. *Issues in Mental Health Nursing, 26*, 721-738.

- Carey, P. D., Stein, D. J., Zungu-Dirwayi, N., & Seedat, S. (2003). Trauma and posttraumatic stress disorder in an urban Xhosa primary care population: Prevalence, comorbidity and service use patterns. *Journal of Nervous Mental Disorders, 191*, 230-236.
- Domhoff, G. W. (2000). Methods and measures for the study of dream content. In M. Kryger, T. Roth & W. Dement (Eds.), *Principles and practice of sleep medicine* (3rd ed., pp. 463-471). Philadelphia: W. B. Saunders.
- Domhoff, G. W., & Schneider, A. (1998). New rationales and methods for quantitative dream research outside the laboratory. *Sleep, 21*, 398-404.
- Dow, B. M., Kelsoe, J. R., & Gillin, J. C. (1996). Sleep and dreams in Vietnam PTSD and depression. *Biological Psychiatry, 39*, 42-50.
- Edwards, D. (2005). Post-traumatic stress disorder as a public health concern in South Africa. *Journal of Psychology in Africa, 15*, 125-134.
- 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.
- Field, A. (2005). *Discovering statistics with SPSS* (3rd ed.). California: SAGE Publishing.
- Franzen, P. L., & Buysse, D. J. (2009). Sleep in psychiatric disorders. In S. Chokroverty (Ed.), *Sleep disorders medicine* (pp. 538-549). Philadelphia: Saunders Elsevier.
- Fuller, K. H., Water, W. F., & Scott, O. (1994). An investigation of slow-wave sleep processes in chronic PTSD patients. *Journal of Anxiety Disorders, 8*, 227-236.
- Germain, A., Buysse, D. J., & Nofzinger, E. (2008). Sleep-specific mechanisms underlying posttraumatic stress disorder: Integrative review and neurobiological hypotheses. *Sleep Medicine Reviews, 12*, 185-195.
- Glaubman, H. M., Miculincer, A., Porat, A., Wasserman, O., & Birger, M. (1990). Sleep of chronic post-traumatic patients. *Journal of Traumatic Stress, 3*, 255-263.
- Harvey, A. G., Jones, C., & Schmidt, A. (2003). Sleep and posttraumatic stress disorder: A review. *Clinical Psychology Review, 23*, 377-407.
- Hefez, A., Metz, L., & Lavie, P. (1987). Long-term effects of extreme situational stress on sleep and dreaming. *American Journal of Psychiatry, 144*, 344-347.

- Herbst, E., Metzler, T. J., Lenoci, M., McCaslin, S. E., Inslicht, S., Marmar, C. R., & Neylan, T. C. (2010). Adaptation effects to sleep studies in participants with and without chronic posttraumatic stress disorder. *Psychophysiology*, *47*, 1127-1133.
- Herman, J. P., Ostrander, M. M., Mueller, N. K., & Figuerido, H. (2005). Limbic system mechanisms of stress regulation: Hypothalamo-pituitary-adrenocortical axis. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *29*, 1201-1213.
- Howell, D. (2004). *Fundamental statistics for the behavioral sciences* (5th ed.). Belmont: Thomson Learning.
- Irwin, M., Miller, C., Gillin, J. C., Demodena, A., & Ehlers, C. L. (2000). Polysomnographic and spectral sleep EEG in primary alcoholics: An interaction between alcohol dependence and african-american ethnicity. *Alcoholism: Clinical and Experimental Research*, *24*, 1376-1384.
- 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*, 300-308.
- Kaminer, D., Grimsrud, A., Myer, L., Stein, D. J., & Williams, D. R. (2008). Risk for post-traumatic stress disorder associated with different forms of interpersonal violence in South Africa. *Social Science & Medicine*, *67*, 1589-1595.
- Kim, J., & Gorman, J. (2005). The psychobiology of anxiety. *Clinical Neuroscience Research*, *4*, 335-347.
- Kim, S. H., & Hamann, S. (2007). Neural correlates of positive and negative emotion regulation. *Journal of Cognitive Neuroscience*, *19*, 776-798.
- Kirschbaum, C., Wust, S., & Helhammer, D. (1992). Consistent sex differences in cortisol responses to psychological stress. *Psychosomatic Medicine*, *54*, 648-657.
- Kobayashi, I., Boarts, J. M., & Delahanty, D. L. (2007). Polysomnographically measured sleep abnormalities in PTSD: A meta-analytic review. *Psychophysiology*, *44*, 660-669.
- Kramer, M., & Kinney, L. (1988). Sleep patterns in trauma victims with disturbed dreaming. *Psychiatric Journal of the University of Ottawa*, *13*, 12-16.
- Kushida, C. A., Littner, M. R., Morgenthaler, Y., Alessi, C. A., Bailey, D., & Coleman, J. W., M. (2005). Practice parameters of the indications for polysomnography and related procedures: An update for 2005. *Sleep*, *28*, 499-521.

- Landolt, H., Dijk, D., Achermann, P., & Borbély, H. A. (1996). Effect of age on the sleep EEG: Slow-wave activity and spindle frequency activity in young and middle-aged men. *Brain Research*, 738, 205-212.
- Lavie, P., Katz, N., Pillar, G., & Zinger, Y. (1998). Elevated awakening thresholds during sleep: Characteristics of chronic war-related posttraumatic stress disorder patients. *Biological Psychiatry*, 44, 1060-1065.
- Lee, K. A. (1998). Alterations in sleep during pregnancy and postpartum: A review of 30 years of research. *Sleep Medicine Reviews*, 2, 231-242.
- Lorenzo, J., & Barbanoj, M. (2002). Variability of sleep parameters across multiple laboratory sessions in healthy young subjects: The "very first night effect". *Psychophysiology*, 39, 409-413.
- Maquet, P., Péters, J., Aerts, J., Delfiore, G., Degueldre, C., Luxen, A., & Franck, G. (1996). Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. *Nature*, 383, 163-166.
- Martenyi, F., Brown, E. B., Zhang, H., Koke, S. C., & Prakash, A. (2002). Fluoxetine v. placebo in prevention of relapse in post-traumatic stress disorder. *The British Journal of Psychiatry*, 181, 315-320.
- Mellman, T. A., David, D., Bustamante, V., Torres, J., & Fins, A. (2001). Dreams in the acute aftermath of trauma and their relationship to PTSD. *Journal of Traumatic Stress*, 14, 241-247.
- Mellman, T. A., Knorr, B. R., Pigeon, W. R., Leiter, J. C., & Akay, M. (2004). Heart rate variability during sleep and the early development of posttraumatic stress disorder. *Biological Psychiatry*, 55, 953-956.
- Mellman, T. A., Kumar, A., Kluck-Bell, R., Kumar, M., & Nolan, B. (1995). Nocturnal/daytime urine noradrenergic measures and sleep in combat-related PTSD. *Society of Biological Psychiatry*, 38, 174-179.
- Mellman, T. A., Nolan, B., Hebding, J., Kluck-Bell, R., & Dominguez, R. (1997). A polysomnographic comparison of veterans with combat-related PTSD, depressed men, and non-ill controls. *Sleep*, 20, 46-51.
- Olley, B. O., Zeier, M. D., Seedat, S., & Stein, D. J. (2005). Post-traumatic stress disorder among recently diagnosed patients with HIV/AIDS in South Africa. *Aids Care*, 17, 550-557.

- Pesant N., & Zadra, A. (2005). Dream content and psychological well-being: A longitudinal study of the continuity hypothesis. *Journal of Clinical Psychology, 62*, 111-121.
- Pillar, G., Malhorta, A., & Lavie, P. (2000). Post-traumatic stress disorder and sleep - what a nightmare! *Sleep Medicine Reviews, 4*, 183-200.
- Raskind, M. A., Dobie, D. K., Kanter, E. D., Petrie, E. C., Thompson, C. E., & Peskind, E. R. (2000). The alpha1-adrenergic antagonist prazosin ameliorates combat trauma nightmares in veterans with posttraumatic stress disorder: A report of 4 cases. *Journal of Clinical Psychiatry, 61*, 129-133.
- Rockwood, K., Mintzer, J., Truyen, L., Wessel, T., & Wilkinson, D. (2001). Effects of a flexible galantamine dose in Alzheimer's disease: A randomised controlled trial. *Journal of Neurology, Neurosurgery & Psychiatry, 71*, 589-595.
- Ross, R. J., Ball, W. A., Dinges, D. F., Kribbs, N. B., Morrison, A. R., Silver, S. M., & Mulvaney, F. D. (1994). Rapid eye movement sleep disturbance in posttraumatic stress disorder. *Biological Psychiatry, 35*, 195-202.
- Ross, R. J., Ball, W. A., Sanford, L. D., Morrison, D. F., Silver, S. M., Kribbs, N. B., & McGinnis, D. E. (1999). Rapid eye movement sleep changes during the adaptation night in combat veterans with posttraumatic stress disorder. *Biological Psychiatry, 7*, 938-941.
- Roth, T. (2007). Insomnia: Definition, prevalence, etiology and consequences. *American Academy of Sleep Medicine, 15*, 7-10.
- Schreuder, B. J. N., Kleijn, W. C., & Rooijmans, H. G. M. (2000). Nocturnal re-experiencing more than forty years after war trauma. *Journal of Traumatic Stress, 13*, 453-463.
- Seedat, S., Nyamai, C., Njenga, F., Vythilingum, B., & Stein, D. J. (2004). Trauma exposure and post-traumatic stress symptoms in urban African schools. *The British Journal of Psychiatry, 184*, 169-175.
- Selzer, M. L. (1971). The michigan alcoholism screening test: The quest for a new diagnostic instrument. *American Journal of Psychiatry, 127*, 1653-1658.
- Sforza, E., Chapotot, F., Pigeau, R., & Buguet, A. (2008). Time of night and first night effects on arousal response in healthy adults. *Clinical Neurophysiology, 119*, 1590-1599.
- Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., . . . Dunbar, G. C. (1998). The mini-international neuropsychiatric interview (M.I.N.I.): The

- development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, 59, 22-33.
- Southwick, S. M., Bremner, J. D., Rasmusson, A., Morganill, C. A., & Charney, D. S. (1999). Role of norepinephrine in the pathophysiology and treatment of posttraumatic stress disorder. *Biological Psychiatry*, 46, 1192-1204.
- Spoormaker, V. I., & Montgomery, P. (2008). Disturbed sleep in post-traumatic stress disorder: Secondary symptom or core feature? *Sleep Medicine Reviews*, 12, 169-184.
- Spoormaker, V. I., Schredl, M., & Van den Bout, J. (2005). Nightmares: From anxiety symptom to sleep disorder. *Sleep Medicine Reviews*, 10, 19-31.
- Stein, D. J., Seedat, S., Herman, A., Moomal, H., Heeringa, S. G., & Kessler, R. C. ... Williams, D. R. (2008). Lifetime prevalence of psychiatric disorders in South Africa. *The British Journal of Psychiatry*, 192, 112-117.
- Taylor, F., & Raskind, M. A. (2002). The[alpha]1-adrenergic antagonist improves sleep and nightmares in civilian trauma posttraumatic stress disorder. *Journal of Clinical Psychopharmacology*, 22, 82-85.
- Van der Kolk, B., Blitz, R., Burr, W., Sherry, S., & Hartmann, E. (1984). Nightmares and trauma: A comparison of nightmares after combat with lifelong nightmares in veterans. *American Journal of Psychiatry*, 141, 187-190.
- Van Stegeren, A. H., Oliver, T. W., Everaerd, W., Scheltens, P., Barkhof, F., & Rombouts, S. (2007). Endogenous cortisol level interacts with noradrenergic activation in the human amygdala. *Neurobiology of Learning and Memory*, 87, 57-66.
- Vanltallie, T. B. (2002). Stress: A risk-factor for serious illness. *Metabolism*, 51, 40-45.
- Weathers, F. W., Keane, T. M., & Davidson, J. R. (2001). Clinician-administered PTSD scale: A review of the first ten years of research. *Depression and Anxiety*, 13, 132-156.
- Woodward, S. H., Friedman, M. J., & Biliswe, D. L. (1996). Sleep and depression in combat-related PTSD inpatients. *Biological Psychiatry*, 39, 182-192.
- Yehuda, R. (2002). Post-traumatic stress disorder. *The New England Journal of Medicine*, 346, 108-114.
- Yetkin, S., Aydin, H., & Ozgen, F. (2010). Polysomnography in patients with post-traumatic stress disorder. *Psychiatry and Clinical Neuroscience*, 64, 309-317.

## Appendix A

### DSM-IV-TR criteria for PTSD

In 2000, the American Psychiatric Association revised the PTSD diagnostic criteria in the fourth edition of its Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR). The diagnostic criteria (Criterion A-F) are specified below. Diagnostic criteria for PTSD include a history of exposure to a traumatic event meeting two criteria and symptoms from each of three symptom clusters: intrusive recollections, avoidant/numbing symptoms, and hyper-arousal symptoms. A fifth criterion concerns duration of symptoms and a sixth assesses functioning.

#### *Criterion A: Stressor*

The person has been exposed to a traumatic event in which both of the following have been present:

1. The person has experienced, witnessed, or been confronted with an event or events that involve actual or threatened death or serious injury, or a threat to the physical integrity of oneself or others.
2. The person's response involved intense fear, helplessness, or horror. Note: in children, it may be expressed instead by disorganized or agitated behavior.

#### *Criterion B: Intrusive recollection*

The traumatic event is persistently re-experienced in at least one of the following ways:

1. Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: in young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
2. Recurrent distressing dreams of the event. Note: in children, there may be frightening dreams without recognizable content.
3. Acting or feeling as if the traumatic event were reoccurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur upon awakening or when intoxicated). Note: in children, trauma-specific reenactment may occur.

4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
5. Physiological reactivity upon exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

*Criterion C: Avoidance/numbing*

Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by at least three of the following:

1. Efforts to avoid thoughts, feelings or conversation associated with the trauma.
2. Efforts to avoid activities, places or people that arouse recollections of the trauma.
3. Inability to recall an important aspect of the trauma.
4. Markedly diminished interest or participation of significant activities.
5. Feeling of detachment or estrangement from others.
6. Restricted range of affect (e.g., does not expect to have a career, marriage, children or a normal lifespan).

*Criterion D: Hyperarousal*

Persistent symptoms of increasing arousal (not present before the trauma), indicated by at least **two** of the following:

1. Difficulty falling or staying asleep
2. Irritability or outbursts of anger
3. Difficulty concentrating
4. Hypervigilance
5. Exaggerated startle response

*Criterion E: duration*

Duration of the disturbance (symptoms in B, C, and D) is more than one month.

*Criterion F: functional significance*

The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

*Specify if:*

Acute: if duration of symptoms is less than three months.

Chronic: if duration of symptoms is three months or more.

*Specify if:*

With or Without delay of onset: Onset of symptoms at least six months after the stressor.

**Appendix B**

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.

University of Cape Town

## Appendix C

### DREAM RATING INSTRUCTIONS

Dream reports need to be read carefully and thoroughly for negative, neutral, or positive content. Themes should also be identified in each report. Dream reports should be read at least twice to evaluate content and themes separately. If you feel necessary, you may re-read them.

#### DREAM THEMES:

Read reports carefully for negative, neutral, or positive themes based on the criteria below:

\*Negative content consists of a dream of a threatening nature. For example, violence, aggression, distressing emotions (e.g. sadness) death, or personal harm of any sort.

\*Neutral content consists of a dream of normal occurrences, no extreme emotions of any sort, and nothing harmful or threatening to the individual.

\*Positive content consists of a dream of positive emotions or experiences.

#### CONTENT RATING INSTRUCTIONS:

1. Read reports thoroughly to identify negative, neutral, or positive key words. Tally the amount of positive or negative keywords. In the absence of positive or negative keywords, mark as neutral content. Based on the keywords that are found, give one overall score rating words for positive, negative, or neutral content using the scale below. -10 indicates very negative content and 10 indicates very positive content.

2. Read reports for negative, neutral, or positive content. Use your own discretion to determine how negative, neutral, or positive the overall content of the whole dream is and assign the appropriate number based on the scale below. -10 indicates very negative content and 10 indicates very positive content.

-3 to -10 = negative content

-2 to -2 = neutral

3 to 10 = positive

#### SCORING:

When the dream reports have been successfully rated, please mark all scores on the back of the report. Clearly stipulate which score belongs with which category.

## Appendix D

### Socio- Economic Status and Demographic Questionnaire

1. Age: \_\_\_\_\_
2. Sex (circle one): Male Female
3. What is your home language? (Please circle only *one* option)  
 English Afrikaans Xhosa Zulu Pedi  
 Other (please specify \_\_\_\_\_)
4. What is the total monthly income of the household in which you live? If you are a student please take care to put your immediate caregiver's monthly income, not your own. (Please circle only *one* option):
 

R0 – R499	R500 – R999	R1000 – R2499
R2500 – R5499	R5500 – R9999	R10 000+
5. Occupation (please circle appropriate letter):
  - (a) Unemployed
  - (b) Self-employed
  - (c) Business employed
  - (d) Student/pupil
  - (e) Other (*Specify*) \_\_\_\_\_
6. Education: Highest degree or grade completed: \_\_\_\_\_

## Appendix E

### *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, 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 ("Study Subject")

---

#### 2. Title of Research Study

“The Relationship between PTSD, hypervigilance and Disordered Sleep.”

#### 3. Principal Investigator and Telephone Number(s)

Mariza van Wyk  
University of Cape Town  
Contact number: 083 5658 190

#### 4. What is the purpose of this research study?

This research aims to investigate if symptoms of hyperarousal influences disordered sleep 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 a sleep study on 1 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. They are merely research instruments that allow us to identify certain patterns of interest.

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 sleep study will be arranged at least one week in advance, at a time convenient to you. Transport will be provided. You will retain your routine bedtime and waking time but will be asked to avoid caffeine and sugar in your diet for a few hours before bedtime. You will be required to come to the sleep laboratory based at Vincent Pallotti Private Hospital between 19 30 and 20 00 and will be briefed once more, 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. One or two researchers will be surveilling the monitor in an adjoining room. They will be available to you for assistance at any time. There is a panic button at your bedside should you need assistance at any point during the night.

In the morning the electrodes will be removed and you will be given a lift back home.

After the sleep session is over, you will be informed in detail about the design of the study and the research questions we hope to address with this 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 hrs and sleep study: 1 night

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

60

**8. What are the possible discomforts and risks?**

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 Vincent Pallotti is fully equipped with a proper bed, clean bedding, restrooms and a kitchenette. It is situated in a secure building with adequate surveillance and alarm system. 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 whole process will not delve deep 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. 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 spending a night in the sleep lab.

**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.

**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 masters 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: \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

Unive



d) Cannot breathe comfortably

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

e) Cough or snore loudly

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

f) Feel too cold

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

g) Feel too hot

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

h) Had bad dreams

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

i) Have pain

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

j) Other reason(s), please describe \_\_\_\_\_  
\_\_\_\_\_

How often during the past month have you had trouble sleeping because of this?

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	-------------------------------------

6. During the past month, how would you rate your sleep quality overall?

Very good \_\_\_\_\_

Fairly good \_\_\_\_\_

Fairly bad \_\_\_\_\_

Very bad \_\_\_\_\_

7. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all \_\_\_\_\_

Only a very slight problem \_\_\_\_\_

Somewhat of a problem \_\_\_\_\_

A very big problem \_\_\_\_\_

10. Do you have a bed partner or room mate?

No bed partner or room mate \_\_\_\_\_

Partner/room mate in other room \_\_\_\_\_

Partner in same room, but not same bed \_\_\_\_\_

Partner in same bed \_\_\_\_\_

If you have a room mate or bed partner, ask him/her how often in the past month you have had . . .

- a) Loud snoring

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

- b) Long pauses between breaths while asleep

Not during the past month \_\_\_\_\_ Less than once a week \_\_\_\_\_ Once or twice a week \_\_\_\_\_ Three or more times a week \_\_\_\_\_

c) Legs twitching or jerking while you sleep

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____
-----------------------------------	-------------------------------	------------------------------	------------------------------------

d) Episodes of disorientation or confusion during sleep

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____
-----------------------------------	-------------------------------	------------------------------	------------------------------------

e) Other restlessness while you sleep; please describe\_\_\_\_\_

---

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____
-----------------------------------	-------------------------------	------------------------------	------------------------------------

University of Cape Town

## Appendix G

*Sleep-related variables: Results of the Kolmogorov-Smirnov test of normality*

Variable	HYP+ ( <i>n</i> = 9)	HYP- ( <i>n</i> = 10)	DEP ( <i>n</i> = 14)	CON ( <i>n</i> = 16)
Sleep efficiency	.109	.200	.175	.105
Sleep latency	.200	.0001**	.200	.106
Awakenings	.003**	.178	.105	.141
WASO	.107	.200	.175	.084
REM%	.200	.198	.200	.200
REM latency	.200	.53	.026*	.112
SWS%	.200	.200	.200	.200

*Note.*

*p*-values for the K-S tests are presented. WASO = wake after sleep onset. \**p* < .05. \*\**p* < .01

University of Cape Town