The relationship between heart rate deceleration during encoding and subsequent sleep-dependent emotional memory consolidation

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Abbreviations

AIDS	Acquired Immune Deficiency Syndrome
ANOVA	Analysis of variance
ANS	Autonomic Nervous System
BDI-II	Beck Depression Inventory – Second Edition
CAPS	Clinician-Administered PTSD Scale
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders – Forth Edition Text Revision
EEG	Electroencephalograph
HC	Healthy control
HIV	Human Immunodeficiency virus
HLOE	Highest level of education
HR	Heart rate
HRD	Heart rate deceleration
IAPS	International Affective Picture System
ICG	Impedance cardiograph
MINI	MINI International Neuropsychiatric Interview
NREM	Non-rapid eye movement sleep
PTSD	Posttraumatic stress disorder
REM	Rapid eye movement
SCL	Skin conductance level
SCR	Skin conductance response
SES	Socioeconomic status
SNS	Sympathetic Nervous System
SWS	Slow wave sleep

TE	Trauma-exposed non-PTSD
VU-AMS	Vrije Universiteit Ambulatory Monitoring System

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Abstract

Emotion and sleep are known to enhance memory, both at encoding and during the consolidation process. Furthermore, those with posttraumatic symptoms have sleep and emotional memory disruption. The interaction of emotion and sleep on subsequent recall is unclear in such a sample. This study examined the relationship between heart rate deceleration (HRD), a physiological autonomic nervous system (ANS) measure of emotion, at encoding, and subsequent sleep-dependent emotional memory consolidation. The study, based on archival data, recruited female participants, living in low-income South Africa characterised by persistent exposure to crime and violence: those with posttraumatic stress disorder (PTSD; n = 21), trauma-exposed (TE; n = 19), and healthy controls (HC; n = 20). Participants viewed highly-arousing positive or negative images and low-arousing neutral images. HRD was measured during picture presentation. Participants spent an 8-hour interval of either sleep or waking, before completing a memory recognition task to identify previously viewed images in contrast to new images.

Results showed no significant differences in HRD at encoding between negative or positively valenced and neutral information, irrespective of between-group differences or condition (sleep or waking). Regarding HRD recorded during encoding and memory performance, there was an association between HRD and recall for positively valenced information rather than negative and neutral information. This association existed for those in the sleep but not the waking condition. The results suggest that psychophysiological reactivity at encoding promotes the consolidation of positive information during sleep, which may be a protective factor for this female cohort, living in an environment dominated by a high degree of negative stimuli in the form of persistent violence and crime. With regards to the group- specific associations between HRD and subsequent recognition of valenced material, there were no significant correlations in the PTSD group. TE participants had

greater psychophysiological reactivity associated with better memory recognition accuracy across intervals of sleep and waking for both valenced and neutral stimuli, with some inconsistencies across sleep and wake conditions. HCs demonstrated better memory recognition with less psychophysiological reactivity for neutral information after sleep. The results tentatively suggested that in participants with PTSD, no association between HRD and memory outcomes is indicative of ANS dysregulation. In TE participants, the finding suggests that autonomic activity plays a key role in promoting memory formation, although indiscriminately of valence or sleep/waking state. In HCs, the result suggests that low physiological reactivity at encoding is necessary for remembering neutral information in those free from trauma. Concerning rapid eye movement (REM) sleep, the results indicated a trend suggesting that as REM fragments, memory recognition accuracy for neutral stimuli decreases. TE participants showed this association, and there was also an association that indicated that as REM percentage increased, memory recognition accuracy for negative stimuli decreased. These findings suggest that REM may be important for ensuring that there is a reasonable distribution of neutral and negative memory traces upon awakening, promoting resilience in the recovery from trauma.

The research highlights that the relationship between HRD is complex and requires further investigation. Future research should compare those living in high and low socio-economic contexts and control for the daily exposure to negative, positive and neutral stimuli in the environment, to better understand how both emotion and sleep contribute to memory processing.

CHAPTER ONE: INTRODUCTION

The relationship between heart rate deceleration during encoding and subsequent sleep-dependent emotional memory consolidation

Emotion is a key feature that enhances memory and experiences with emotional tone are more likely to be remembered (Kensinger, 2009; Zeng et al., 2021). Emotional memories are formed when there is emotional arousal during encoding (Bowen et al., 2017). Another factor that significantly contributes to the strengthening (or consolidation) of memory is sleep. Conversely, sleep deprivation hinders memory consolidation, including memory embedded with emotion, and leads to the deterioration of memory traces or bias for information of only one valence (e.g., negative; Denis et al., 2022; Tempesta et al., 2018). Although there are some inconsistencies across studies (Payne & Kensinger, 2011), the extant literature suggests that there is preferential consolidation of emotional memory over neutral memory during sleep in comparison with waking (Lipinksa & Thomas, 2019; Palmer & Alfano, 2017; Tempesta et al., 2018). Therefore, both emotion and sleep are important for strengthening memory traces.

However, the exact nature of the interaction between the two processes for memory consolidation remains unknown. One possible hypothesis explaining the interaction states that the degree of emotion elicited at encoding, tags memory traces for consolidation during sleep. There is some preliminary evidence for this hypothesis (Cunningham et al., 2014), however, the findings only (a) compare negative and neutral valenced information, and (b) are conducted in healthy participants. What remains unknown, therefore, is (i) whether these findings are robust and replicable, (ii) consistent for positive information, and (iii) carry the same predictive strength in individuals that have documented sleep and emotional memory disruption, such as those diagnosed with posttraumatic stress disorder (PTSD). Individuals with PTSD experience sleep disturbances, including rapid eye movement (REM) disruption

(Ross, 2014; Walker & van der Helm, 2009). Individuals with PTSD also have emotional memory bias, where they tend to have better memory for negatively valenced information in comparison to positively valenced or neutral information (Hayes et al., 2012; Itoh et al., 2019; Zhao et al., 2024). However, the contribution of sleep disturbance to this emotional memory bias in PTSD is unknown.

CHAPTER TWO: LITERATURE REVIEW

Fundamental memory processes

Declarative memories are formed through three processes: encoding, consolidation, and retrieval. While there are other kinds of memories (for review see., Squire & Dede, 2015), this thesis focuses on this aspect of memory. Encoding refers to the process involved in the acquisition of memory, where experiences are first represented in brain structures as a lasting and discernible trace (Hainmeuller & Bartos, 2020; Paller & Wagner, 2002); consolidation transforms initially encoded memory traces into stable long-term representations (Lewis et al., 2011; Tonegawa et al., 2018), while retrieval reactivates previously encoded memory traces in the here-and-now (Buchanan, 2007). Furthermore, Kensinger and Ford (2020) suggest that retrieval, although typically viewed as an endpoint culminating in successful retrieval, can also be understood as a starting point where the memory trace activated influences behaviours and decisions made in the moment – this allows the process of retrieval to refigure the memory trace such as with emotional memories.

Emotion enhances memory

Several factors influence each of these aspects of memory. For example, emotion at the time of encoding enhances the initial strength of the information and, therefore, subsequent memory recall (Abercombie et al., 2008; Jones et al., 2018; Pilarczyk et al., 2022). A wide body of research also shows that sleep enhances the consolidation process (Diekelmann & Born, 2010; Schafer et al., 2020; Tempesta et al., 2018).

Events that trigger an emotional response are better remembered than neutral events (Earles et al., 2016; Squire & Dede, 2015). The valence and arousal components of the

emotional response influence memory, for example, events that are negatively or positively valenced (Tempesta et al., 2015) or which induce high levels of arousal (Hu et al., 2006) are remembered better than neutral or low arousal events (Earles et al., 2016).

Sleep and emotional memory

Although memory is enhanced with the presence of emotion, sleep preferentially consolidates emotional memory. That is, the difference between memory performance for valenced versus neutral information is greater after a period of sleep when compared to a period of waking (Cox et al., 2018; Walker & van der Helm, 2009). For example, Nishida et al. (2009) saw a significant difference in retention of emotional items between nap versus no-nap groups where individuals in the nap condition performed better on recognition of emotional items. Alger et al. (2018) also found that a nap directly after learning, in contrast to a delayed nap some hours post learning and an equivalent period of waking, resulted in a large memory difference between negatively valenced objects and their neutral backgrounds. These findings highlight that emotional memory is enhanced after sleep rather than waking.

However, there are some discrepancies across studies. For example, Atienza and Cantero (2008) presented stimuli which were neutral, negative, or positive to participants who were grouped into control versus sleep-deprived groups. The results showed that emotional memory consolidation was not hindered in the sleep-deprived group compared to the control group. Lewis et al. (2011) found that sleep preserved neutral and emotional memories to the same extent, in other words, there was no preferential consolidation of emotional memories after a full night's sleep in comparison to an equivalent period of waking. Similarly, Ashton et al. (2019) found that there was no significant difference regarding recognition of negative versus neutral stimuli between a group with overnight sleep and a group with daytime wakefulness.

In a meta-analysis by Schafer et al. (2020), the consensus across the literature is that sleep preferentially consolidates emotional memory over neutral memory, but only when taking REM sleep into account. For example, Groch et al. (2013) demonstrated through using a recognition task that memory performance was better for emotional images than for neutral images after a period of REM sleep in contrast to an equivalent period of slow wave sleep (SWS).

Of note, all the studies presented thus far, except Atienza and Cantero (2008), only compared negative and neutral information. A handful of papers have examined sleep-dependent emotional memory consolidation for both negatively and positively valenced information and compared participants' memory for material of both valences to memory for neutral information. However, these papers have contrasting results, and there is no clear consensus describing how sleep consolidates negative versus positive information. For example, Cellini et al. (2016) used a nap paradigm to investigate memory consolidation of unpleasant, pleasant, and neutral pictures in healthy participants assigned to either a nap or no-nap condition. They found that after a sleep period, neutral pictures were remembered better than pleasant pictures, but no specific effect was found when comparing neutral and unpleasant pictures. This study, therefore, did not show preferential emotional memory consolidation after sleep in comparison to waking. Another study by Wagner et al. (2007) compared memory recognition for angry, happy, and neutral faces after either a full night's sleep or a full night of sleep deprivation. These authors found that after sleep, all faces were recognised with greater accuracy, but that there was no specific recognition advantage for valenced faces (either angry or happy) rather than neutral faces. In summary, these studies have found contrasting results to (a) the overall thrust of the literature, which shows that negative information is consolidated over neutral information after sleep, and (b) also do not

show any clear results regarding the contribution of sleep to the consolidation of negative versus positive information.

The role of emotion at encoding

While the role of emotion and sleep in memory consolidation has been widely studied, the role of emotion at encoding of emotional memory and its subsequent role in sleep-dependent consolidation is less clear.

Emotional elicitation during encoding strengthens memory, which results in enhanced memory performance at retrieval (Abercombie et al., 2008; Berres & Erdfelder, 2021). The process of memory enhancement associated with paired emotion elicitation is thought to be done via a "tagging" process where stimuli that evoke an emotional response are identified and "tagged" for subsequent consolidation, including consolidation during sleep (Cunningham et al., 2014; Jones et al., 2018). Via this "tagging" process, emotion enhances a memory which then becomes (i) less affected by disruptions, (ii) longer-lasting, and (iii) has better accuracy or potentially a combination of the three (Richter-Levin & Akirav, 2003).

However, it is unclear how the sleep-related consolidation process and emotion elicited during encoding interact regarding subsequent memory performance. In other words, does emotion at encoding allow for tagging of memory traces for offline sleep-specific consolidation, rather than daytime consolidation? This would result in better memory retrieval post-sleep than after an equivalent wakeful period.

To study this interaction, Cunningham et al. (2014) examined whether sleep plays a role in modulating changes in heart rate (specifically heart rate deceleration [HRD]) and skin conductance response (SCR) in response to negative and neutral stimuli. HRD is a parasympathetic, autonomic function, which precedes the activation of the sympathetic branch of the autonomic nervous system characterised by heart acceleration. In response to

stimuli, and especially in response to arousing stimuli, there is an initial decrease in heart rate in the first three seconds (Abercombie et al., 2008; Bolinger et al., 2019; Cunningham et al., 2014; Jones & Spencer, 2019). The response is more pronounced in response to stimuli that carry higher rather than lower arousal (Cunningham et al., 2014), and represents the process of orienting to the stimulus.

Cunningham and colleagues (2014) used HRD at encoding to examine how this psychophysiological variable influenced memory following a sleep or wake-filled delay. Their results showed that there was an overall decrease in HRD in the sleep group in response to both negative and neutral images. Moreover, participants who had greater changes in physiological arousal to negative stimuli at encoding and who were part of the sleep group displayed better memory for these negative stimuli. Cunningham et al. (2014), therefore, proposed that the greater the physiological reaction at encoding, the stronger the "tag" which in turn allows for sleep to better consolidate the memory.

When emotional memory and sleep are disrupted: Associations in PTSD

Although the result described by Cunningham et al. (2014) provides some useful insight regarding the interaction of emotion and sleep on subsequent recall, it is unclear whether this relationship carries the same predictive strength in individuals that have documented sleep and emotional memory disruption, such as those diagnosed with PTSD.

Individuals with PTSD suffer from various symptoms including sleep disruption (i.e., interrupted sleep or problems with falling and/or staying asleep) and a memory bias in favour of negative information. That is, they tend to remember negative information more readily than positive or neutral information (American Psychiatric Association, 2013). Regarding the former, Pace-Schott et al. (2015) note that those with PTSD have greater Stage 1 non-REM (NREM) sleep and diminished SWS. Moreover, they (a) spend less time in REM sleep

(Germain, 2013), (b) experience REM fragmentation (the number of awakenings or arousals from REM sleep specifically; Lipinska & Thomas, 2019), (c) higher REM density (the amount of recurring rapid eye movements during REM sleep; van Wyk et al., 2019), (d) REM-associated nightmares not involving the traumatic event, (e) REM-related disordered behaviours (e.g. acting out dreams), and (f) sleep-disordered breathing from that stage of sleep (Germain et al., 2013).

Regarding the latter, those with PTSD may also experience emotional memory bias for negatively valenced information (Itoh et al., 2019). For example, participants were exposed to negative, neutral, or positive words as stimuli and asked to recognize the original words after a ten-minute delay (Itoh et al., 2019). On the recognition memory task, there was a significant difference between PTSD patients versus healthy controls where participants showed significant bias towards negative information (Itoh et al., 2019). Moreover, Itoh et al. (2019) provided evidence for a significant relationship between increased negative memory bias and diminished overall memory function in patients with PTSD. In other words, those with PTSD and low general memory performance demonstrated a significantly greater memory bias for negative stimuli than PTSD patients with normal memory function (Itoh et al., 2019), suggesting that compromised memory functioning is associated with the bias towards negative information. Similarly, Lin et al. (2015) found that participants with greater PTSD symptoms were more likely to recognise words that were perceptual-trauma paired (i.e., one word was perceptual, such as green, and the other was trauma-related) than those who had fewer PTSD symptoms. In addition, the trauma group with greater symptoms recognised significantly more trauma words compared to neutral words (Lin et al., 2015).

Since REM sleep is implicated in the preferential consolidation of emotional memory over neutral memory (Wiesner et al., 2015), there are a number of factors to consider when proposing that those with dysregulated REM sleep, such as those with PTSD, experience a

bias towards negative emotional memories. In terms of emotional memory, while research studies show that negative memory bias is present in those with PTSD (Hayes et al., 2012; Itoh et al., 2019) it seems contradictory to say that this is present in those with REM sleep dysregulation since REM sleep preferentially consolidates emotional memory over neutral memory (for example, negative memory over neutral memory; Wiesner et al., 2015). However, one proposition is that in PTSD-diagnosed individuals, REM dysregulation influences negative information that has been 'tagged' at encoding more strongly than positive or neutral information, so that this category of information is consolidated maximally over the other categories. In healthy controls, the expectation is that individuals experience enhanced emotion at encoding for *both* negative and positive information, which 'tags' this information for subsequent consolidation and relatively equivalent recall/recognition of both these categories at subsequent retrieval.

Rationale

The literature suggests that both emotion and sleep influence memory performance. However, it is not known how these two variables interact to determine the strength of subsequent memory performance. One hypothesis states that emotion at encoding 'tags' memories for consolidation and that this consolidation process occurs primarily during sleep. I aim to determine whether (a) there is evidence to support this hypothesis, (b) this hypothesis is consistent for memory performance for *both positive* and *negative* information (since studies have focused on the comparison of memory for negative and neutral information), and (c) whether participants with PTSD, who have known alterations in emotional memory and sleep, show predictable sleep-dependent emotional memory disruption. To this end, I will test the following hypotheses, using HRD as a measure of autonomically mediated emotional change:

Hypotheses:

1. HRD recorded during encoding of valenced and neutral information will be higher for valenced information than for neutral information.
2. HRD recorded during encoding of valenced and neutral information will be associated with subsequent recall of each respective category of information, with a stronger relationship demonstrated for (a) valenced (positive = negative) rather than neutral information, and (b) sleep rather than waking.
3. Regarding between-group differences:
    a. Negative stimuli: PTSD-diagnosed individuals, in comparison to trauma-exposed (TE) individuals and healthy controls (HCs) will have increased HRD at encoding in response to negative stimuli (PTSD > TE > HC).
    b. Positive stimuli: HCs, in comparison to TE and PTSD-diagnosed participants will have an increase in HRD at encoding in response to positive stimuli (HCs > TE > PTSD).
    c. The relationships described in a and b will be strongest after sleep, rather than waking.
4. The associations between HRD and subsequent recognition of valenced material are group-specific:
    a. negative stimuli, HRD at encoding is associated with better memory recognition accuracy for PTSD participants in comparison to TE and HC individuals (PTSD > TE > HC);

b. positive stimuli, HRD at encoding is associated with better memory recognition accuracy for healthy controls in comparison to TE and PTSD individuals (HC > TE > PTSD), and;
c. these results are more evident in the sleep condition rather than the wake condition.

5. REM disruption will also be associated with memory performance related to negative information more strongly than that of positive or neutral information and this association is more likely in PTSD-diagnosed rather than control participants.

CHAPTER THREE: METHOD

The current study is an extension of a larger study conducted in the UCT Sleep Sciences laboratory, examining the role of sleep in processing memory and emotion in PTSD (Lipinska, 2017). Therefore, the methods described here were carried out by Associate Professor Lipinska, although the rationale and analysis of the data as described in this section are novel.

Study Design

The study is a cross-sectional, quasi-experimental study, following a mixed repeated measures design. The repeated-measures variables will be valence (negative, positive, and neutral) and condition (sleep, waking). Other independent variables include HRD measured at encoding (prior to a sleep or waking interval), group status (PTSD-diagnosed versus TE versus HC), and REM-related variables. The outcome variables will be memory performance for negative, positive, and neutral information. This study has already been completed by Lipinska (2017) where memory and sleep were analysed in depth. The current study is a re-analysis of the data collected. Subsequently, only the methods relevant to the current study are discussed below.

Participants

Lipinska (2017) recruited only female sexual assault survivors since the majority of studies focused on PTSD recruit male war veterans (Breen et al., 2019; Kobayashi et al., 2007; Werner et al., 2016). The current study differs from the original study as Lipinska (2017) examined emotion change or sleep and neutral declarative memory, rather than HRD at encoding. For a comparison, see Appendix A. Advertisements were placed in local newspapers to recruit healthy participants. Those who responded telephonically were asked to

respond to a short demographic questionnaire. Additionally, the study recruited participants from the Rape Crisis Cape Town Trust centres located in Observatory, Athlone and Khayelitsha. Associate Professor Lipinska worked with the counsellors at the Rape Crisis centres to identify potential PTSD and trauma-exposed participants.

One hundred and seven potential participants were recruited for screening. Sixty-six met the inclusion criteria and were asked to participate in the following stage. Post-screening, 60 participants remained.

Three groups were created: PTSD group (n = 21), trauma-exposed non-PTSD group (referred to as TE; n = 19), and healthy control group (referred to HC; n = 20). All participants were females who were fluent in English. In trauma groups (PTSD and TE), all participants were sexual assault survivors. Lipinska (2017) included a trauma-exposed group to differentiate between trauma exposure and the development of PTSD symptomology leading to a diagnosis of PTSD since a small number of trauma-exposed individuals have a PTSD diagnosis (Boscarino & Adams, 2009; Hu et al., 2020). In other words, while trauma exposure is necessary for a PTSD diagnosis, it does not guarantee such a diagnosis. Groups were matched on the following criteria: age, highest level of education, Intelligence Quotient, socioeconomic status (based on income), first language, and smoking status. See Table 1 for the sociodemographic data of the sample.

Regarding the current study, a post-hoc analysis calculated using G*Power (Faul et al., 2009) revealed that using an effect size of f = 0,25 based on a study examining HRD as a measure of emotion at encoding in predicting subsequent memory performance for valenced and neutral information (Abercombie et al., 2008), using mixed design ANOVA for repeated measures, within-between interaction, with alpha set at < .05, revealed that the power is 0.99 for the sample size of 60. Thus, the study is adequately powered for this analysis.

Inclusion and exclusion criteria

1. The diagnosis of any DSM-IV-TR (APA, 2000) Axis I disorders, other than PTSD, excluded potential participants. Axis I disorders feature specific sleep changes and memory deficits which would confound the aim of this study (Baglioni et al., 2014). Those in the PTSD or TE groups with anxiety or other mood disorders secondary to trauma exposure were included in the sample.
2. Potential participants who had abused alcohol or other substances for more than one year were excluded since such substance use negatively affects both sleep and memory functioning (Caetano et al, 2023; Conroy & Arnedt, 2014).
3. The age range of participants was limited to 18 to 40 years old. Children and adolescents under the age of 18 demonstrate different sleep cycles to adults (Patel et al., 2024). Furthermore, normal ageing is associated with a mild decline in memory function as there is hippocampal atrophy (Fjell et al., 2015), moreover, sleeping cycles change with normal ageing (Li et al., 2018).
4. Any potential participant who was prescribed any psychoactive medication (including anti-depressants) and/or sedatives for sleep regulation was excluded from the study. Sedatives are known to alter sleep cycles, in addition, psychoactive drugs have significant effects on brain structure and function as well as normal memory processes (Leong et al., 2022).
5. Time since trauma was also used as an exclusion criterion, i.e., the trauma had to have occurred no longer than five years ago and no sooner than six months before screening. This is because the time since trauma affects both sleep cycles and memory processes (Lipinska, 2017). In addition, potential participants who had experienced trauma as children or adolescents were excluded as childhood trauma impacts the developmental processes of memory (Teicher et al. 2016).

6. Neurological conditions that could potentially influence the results (e.g. epilepsy, traumatic brain injury) were excluded. In the screening phase, seven participants indicated that they were HIV-positive. However, they were asymptomatic as they did not present with any HIV- or AIDS-related symptoms (i.e., recurrent fever, weight loss, cognitive difficulties, and opportunistic infections). Elbirit et al. (2015) showed that HIV-positive individuals who are not substance abusers and have an intact immune system are significantly less likely to have HIV-associated neurocognitive disorders. Thus, HIV-positive participants who were asymptomatic were included in the study.
7. Since many of the measures utilised in this research are exclusively in English, participants had to be fluent in English in order to participate.

Materials and Apparatus

Diagnostic and screening instruments. The *MINI International Neuropsychiatric Interview* (MINI 5.0.0; Sheehan et al., 1998) is a brief, structured diagnostic interview which was used to evaluate the major DSM-IV Axis I disorders (Lipinksa, 2017). The MINI is a frequently used diagnostic tool across the globe and it is known to be reliable and valid (Øhre et al., 2014). The MINI can be administered in approximately 15 minutes and has been used in South African research (e.g. Andersen et al., 2020; Field et al., 2018; Narsi et al. 2021). Lipinska (2017) used the MINI to confirm PTSD diagnosis. In addition, the MINI aided in excluding participants who had met the criteria for any DSM-IV Axis I psychiatric conditions besides PTSD, however, participants in the PTSD and TE groups with depression and anxiety consequent to the trauma were included. However, potential PTSD and TE participants who had psychopathology before the trauma were excluded. Lipinska (2017) excluded potential participants who met the MINI's criteria for alcohol or substance abuse and/or dependence. The MINI was also used to identify suitable HCs: HCs could not meet the MINI criteria for

any psychiatric diagnosis, and they were thoroughly screened to confirm that they had not been exposed to any event that could qualify as a DSM-IV-TR PTSD criterion A traumatic event.

The *Clinician-Administered PTSD Scale* (CAPS; Blake et al., 1995) is a structured interview that is administered in the diagnosis and assessment of symptom severity of PTSD (Weathers et al., 2001). The CAPS is known to have excellent reliability and validity for diagnosing PTSD (Lipinska, 2017; Weathers et al., 2001). Moreover, the CAPS has been employed in research in South Africa (e.g., Suliman et al., 2015). Lipinska (2017) used the CAPS to confirm diagnoses of PTSD as was first indicated by the MINI.

The *Beck Depression Inventory – Second Edition* (BDI-II; Beck et al., 1996) is a self-report measure that assesses depressive symptoms and their severity in patients 13 years and older (Smarr & Keefer, 2020). It consists of 21 standardized questions, and it has been used both in clinical and research settings in South Africa (e.g., Naidoo, 2019; Nkoana et al., 2020). The BDI-II has good reliability and validity (Smarr & Keefer, 2020). Lipinska (2017) used the BDI-II to evaluate depressive symptoms in the PTSD and TE groups. In addition, potential HC participants who scored ≥ 14 on the BDI-II were excluded.

A demographic form documented participant details including age, time of the traumatic event, use of sedative and/or psychoactive drugs, and history of neurological conditions.

Experimental Measures. Lipinska (2017) used a subset of images from the *International Affective Picture System* (IAPS; Lang, Bradley & Cuthbert, 2008). The IAPS is a database of colour photographs which create a set of emotion evoking stimuli which are used in research to evaluate emotion and emotional memory (Lipinska, 2017). Every photograph has a normative rating of emotion including valence (positive, negative or neutral) and arousal (arousing and low-arousing). The valenced images were all rated as

arousing and the neutral images were low-arousing. The valence and arousal properties of all of the trials (pre- and post-interval of sleep or waking) and the two sets used in the *Sleep* and *Waking* conditions were approximately equivalent.

The Vrije Universiteit Ambulatory Monitoring System (VU-AMS; Version 5fs; De Geus, Willemsen, Klaver, & van Doornen, 1995; Willemsen, De Gues, Klaver, Van Doornen, & Carroll, 1996) measured autonomic nervous system activation. The VU-AMS was used to record psychophysiological variables relevant to the parent study including continuous electrocardiogram (ECG) activity as well as impedance cardiogram (ICG) activity and skin conductance level (SCL) data. In this study, the ECG data will be used to calculate HRD. HRD is typically measured using an average heart rate (HR) which is determined in the second/s prior to stimulus presentation, thereafter this average heart rate is subtracted from the HR measured during the time of presentation. In other words, the mean heart rate ($\bar{x}$) before stimulus presentation is subtracted from the HR during presentation (y). Therefore: $y - \bar{x} = HRD$. From Lipinska & Thomas' (2019) procedure, HR was measured for three seconds prior to stimulus presentation, thereafter the target picture was presented for six seconds. Therefore, HRD was calculated by subtracting the HR measured in the two seconds prior to stimulus presentation from the HR measured during the first three seconds of stimulus presentation.

Procedure

The greater study (Lipinska, 2017) comprised the initial screening, whereafter there were three nights of testing in UCT's sleep laboratory. Transport was provided as necessary. The initial screening was conducted in a private, quiet space in the UCT Department of Psychology. The screening began with an explanation of the aims of the research and what it entailed. Before the screening methods were administered, every participant read and signed

a document detailing their informed consent. PTSD and TE participants were asked to provide a thorough clinical history in addition to the screening to determine that any other psychopathologies (e.g. panic disorder) developed secondary to the trauma and to ensure that they did not precede the trauma.

After the screening and diagnostic assessment, Lipinska (2017) debriefed the participants. If the participant met the criteria for the study, an appointment was made for the first study session and the participant was allocated to the appropriate group. Study sessions were made up of a *Waking* condition, an adaptation night, and a *Sleep* condition.

Although participants slept two nights in the laboratory, only one was used for statistical analysis, and the other, the adaptation night, allowed participants to become used to the laboratory settings and allowed for more accurate readings on the test night as the literature provides evidence for the first-night effect. The first-night effect refers to the first night's sleep comprising of increased sleep onset latency and decreased sleep efficiency as well as longer REM latency, lowered REM and elevated alpha while the reverse first night effect observes participants who have better sleep quality than usual (Byun et al., 2019). The adaptation night included electrode placement and the participants slept a full 8-hour night in the laboratory.

Participants were requested to abstain from excessive exercise, daytime napping, and alcohol during all of the study sessions. For the *Sleep* condition, participants were requested to only consume caffeinated drinks first thing in the morning and to abstain from them the rest of the day. The *Waking* participants were requested to avoid caffeinated drinks throughout the day. However, baseline measurements of caffeine intake were not collected. Participants who were smokers were asked to smoke their usual amount or less, as the literature has demonstrated that acute withdrawal is more harmful for sleep and cognitive

functions than uninterrupted smoking (Amiri & Behnezhad, 2020; Valentine & Sofuoglu, 2018).

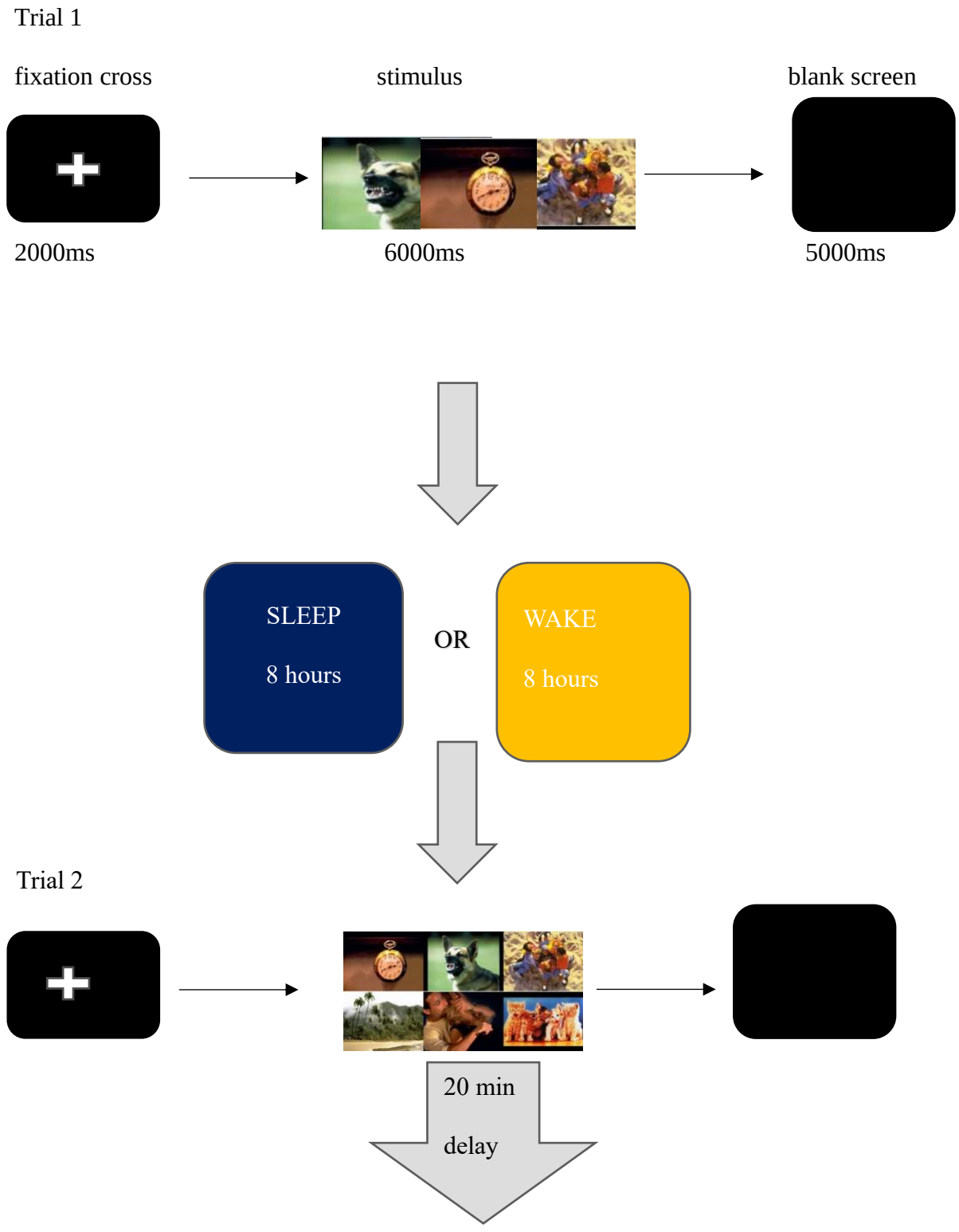
Sleep condition. Before participants slept, they completed *Trial 1* of the emotional memory task. *Trial 1* presented a collection of 90 images which participants were asked to remember. These images were presented with E-Prime software (Schneider, Eschmann, & Zuccolotto, 2002). Figure 1 depicts the procedure described here. They were pictured full-size on a standard 19-inch computer monitor. Each stimulus presentation began with a fixation cross which was presented for 2000ms. Thereafter, the target image was shown for 6000ms and then a blank screen was shown for 5000ms. Thus, the inter-stimulus period was 7000ms. These are recommended durations following other studies using the IAPS (Balconi, Brambilla, & Falbo, 2009; Palomba, Angrilli, & Mini, 1997).

Before the start of the trial, participants sat quietly so the VU-AMS could record baseline readings for HR. The VU-AMS took ECG recordings for each picture. Participants were required to remain seated throughout the experiment and refrain from large movements since these could affect the ECG readings. Small movements, such as tapping the keyboard to respond, do not affect the readings (Porges et al., 2007). Participants were alone in a sound and light-proof room while responding to the task to avoid social desirability effects. Post-trial, participants slept for 8 hours. After which, *Trial 2* – which contained the same images as *Trial 1* but included 45 new images – was administered in the same manner as *Trial 1*. A recognition trial (*Trial 3*) followed a twenty-minute delay after *Trial 2*. Here, participants viewed all 135 images and were requested to differentiate between "old" pictures presented in *Trial 1* and "new" pictures presented in *Trial 2*.

Waking Condition. The same procedure as the *Sleep* condition was followed using the parallel version of the IAPS. The main difference between the *Sleep* and *Waking* conditions was an 8-hour interval of waking rather than sleep.

Post-study completion, each participant was debriefed on the nature of the study. Every participant was shown their sleeping patterns, and the sleep stages were explained. In addition, every participant was compensated ZAR150 for each of the three study sessions.

Figure 1 *Outline of Procedure*



Statistical Analysis

Before statistical analysis, the data were cleaned and HRD was calculated for each participant as described above. Parametric assumptions were tested and, non-parametric tests were be implemented to examine associations, where parametric assumptions failed.

Hypothesis 1

To examine if HRD increases for valenced over neutral information, I planned to conduct a mixed design analysis of variance (ANOVA) using repeated measures (valence, condition) and between-group factors (group) to test the main effect of valence. In this case, HRD is the outcome variable. If the data did not meet parametric assumptions, I planned to run two separate Friedman tests with regards to condition (sleep, wake) to assess whether HRD increased for valenced rather than neutral information.

Hypothesis 2

To examine if HRD is associated with memory performance (the outcome variable), I planned to run an analysis of covariance (ANCOVA) where HRD is the covariate that impacts the main effect of the predictors (a) valence and (b) condition**.** If the data did not meet the parametric assumptions, I planned to run multiple correlations using Spearman's rho. The correlations would look at HRD during encoding and each valence category as well as memory performance.

Hypothesis 3

To examine between-group differences in the relationship between HRD at encoding and memory performance in response to valenced stimuli, the ANCOVA described under *hypothesis 2* will be used where the three-way interaction: valence x group x condition will be the analysis of interest. If the data did not meet parametric assumptions, I planned to perform a Kruskal-Wallis test on the three groups (PTSD, TE, HC).

Hypothesis 4

To examine whether the associations between HRD and subsequent recognition of valenced material are group specific, with the prediction that for (a) negative stimuli, HRD at encoding is associated with better memory recognition accuracy for PTSD participants in comparison to TE and HC individuals (PTSD > TE > HC) and that for (b) positive stimuli, HRD at encoding is associated with better memory recognition accuracy for healthy controls in comparison to TE and PTSD individuals (HC > TE > PTSD) and (c) these results were more evident in the sleep condition rather than the wake condition, I first conducted correlation analyses using Spearman's rho. If the data met parametric assumptions, I planned to follow up on the correlational analyses, using regression analysis, with HRD, group, and HRD x group as predictors, with the negative, positive, and neutral recognition as outcomes (1 model per valence category).

Hypothesis 5

To examine whether features of sleep, and specifically REM sleep, contribute to the relationship between HRD at encoding and memory recognition for valenced and neutral information, I first aimed to determine basic associations using correlation analyses, to see whether there were associations between REM sleep parameters and memory outcomes in each valence category. The REM parameters included in the analyses were REM Percentage, REM Fragmentation, and REM Fragmentation to wake/NREM1. If these associations existed and parametric assumptions were met, I planned to perform regression analysis with HRD, group, and REM parameters as predictors.

CHAPTER FOUR: RESULTS

Table 1

Sociodemographic Data for the Current Sample (N = 60)

	Group				
Variable	PTSD (n = 21)	TE (n = 19)	HC (n = 20)	F/X^2	p
Age	25.52 (4.36)	24.42 (4.53)	25.30 (4.62)	0.33	.721
Years of Education	11.67 (1.59)	12.53 (2.09)	12.90 (2.00)	2.28	.111
Home Language				7.77	.412
isiXhosa	15	16	16		
Afrikaans	3	0	2		
English	2	1	2		
Other	1	2	0		
Employment				9.64	.206
Unemployed	7	8	10		
Employed	11	6	3		
Student	3	5	7		
Household Income				19.34	.057
R0-R999	1	1	1		
R1000-R2499	3	6	5		
R2500-R5499	12	7	5		
R5500-R9999	0	4	5		
R10 000+	5	1	4		
Smoking					
Yes	7	1	5	5.01	.088
No	14	17	15		
Drug Use				14.67	.442
Historically yes	5	4	0		
No	16	15	20	3.80	.194
HIV+					
Yes	5	1	1		
No	16	18	19		
BDI-II	30.14 (6.23)	17.72 (7.98)	6.2 (3.4)	78.864	<.001

Note. For Age, Years of Education, and BDI-II, means are presented with standard deviations in parentheses. ANOVAs were run for Age, Years of Education and BDI-II scores. For

Income, Employment, Language, Smoking and Drug Use, Fisher's exact test static is presented.

Hypothesis 1

I aimed to examine if the HRD response at encoding was more pronounced (more negative) for valenced over neutral information. Since the data did not meet the parametric assumption to run a repeated-measures 2 x 3 ANOVA, two separate Friedman tests were run with regards to the sleep and wake conditions respectively, to assess whether HRD increases for valenced rather than neutral information in each condition.

There was no statistically significant difference in HRD for valenced information over neutral information in the sleep condition, $\chi^2(2) = 3.100$, $p = .212$. Similarly, in the wake condition there was no statistically significant difference in HRD for valenced information over neutral information, $\chi^2(2) = 2.475$, $p = .290$. Therefore, contrary to the prediction, HRD was not more pronounced in response to valenced, rather than neutral stimuli.

Hypothesis 2

I aimed to evaluate whether HRD recorded during encoding of valenced and neutral information would be associated with subsequent recall of each respective category of information, with a stronger relationship demonstrated for (a) valenced (positive = negative) rather than neutral information and (b) sleep rather than waking. However, the data did not meet the parametric assumptions and I, therefore, performed correlations using Spearman's rho. Moreover, to account for inflation in Type 1 Error associated with multiple correlations, the *p*-value was adjusted using the Benjamini-Hochberg procedure.

There was no relationship demonstrated between HRD during encoding and subsequent memory recall for the neutral sleep and wake conditions as expected. Contrary to the prediction, there were also no associations between HRD and memory recognition in

response to negative stimuli after an interval of sleep and wake. Regarding the emotional memory performance in response to positive stimuli after an interval of sleep: there was a significant negative correlation between hits and HRD, r_s = -.382, p = .002, adjusted p-value = .009 as well as a significant positive correlation between misses and HRD r_s = .371, p = .002, adjusted p-value = .009. These results demonstrate that as correctly identified images (hits) increase, HRD decreases, in other words, there are a greater number of hits when there is greater psychophysiological arousal (larger negative HRD). In addition, misses decrease as HRD decreases, in other words, there are a smaller number of incorrectly identified images (misses), when there is increased psychophysiological arousal (large negative HRD). Furthermore, there was a significant negative correlation between accuracy (*d*') and HRD, r_s = -.328, p = -.006, adjusted p-value = .018. This can be interpreted similarly to the results for hits: with more psychophysiological arousal (large negative HRD), there is greater recognition. There were no significant correlations between HRD and memory performance in response to positive stimuli after an interval of wake. The findings partly confirm the hypothesis, since the association between HRD and hits, misses and accuracy occurs with regards to the sleep but not waking condition and in response to one aspect of valenced stimuli (positive stimuli only), in comparison to neutral stimuli.

Table 2

Hypothesis 2: HRD Association with Memory Performance Across Valence and Condition

	Sleep	Wake
	HRD	
	Negative	
Hits	.136	-.068
Misses	-.136	.068
d'	-.032	-.023
	Positive	
Hits	-.382** (*p*=.002)	-.046
Misses	.371** (*p*=.002)	.046
d'	-.328** (*p*=.006)	-.109
	Neutral	
Hits	-.067	.047
Misses	.067	-.050
d'	-.071	.068

Note: Spearman's rho values are given with (*p*-values).

Hypothesis 3

To examine between-group differences in the relationship between HRD at encoding in response to valenced stimuli, a Kruskal-Wallis test was performed on the three groups (PTSD, TE, HC). There were no between-group differences with regards to HRD in response to negative or positive stimuli, presented either prior to sleep or waking (see Table 2).

Table 3

Heart Rate Deceleration by Group Across Sleep and Wake Conditions

Sleep	PTSD	TE	HC	$H(2, n=60)$	*p*-value
Negative	-2.77 (-4.14; -1.73)	-2.81 (-3.67; -2.37)	-3.17 (-4.43; -2.11)	.641	.726
Neutral	-2.40 (-3.48; -1.50)	-3.07 (-4.29; -2.16)	-2.58 (-3.62; -2.05)	3.063	.216
Positive	-2.50 (-3.85; -1.61)	-3.20 (-3.99; -2.03)	-2.61 (-3.40; -1.76)	1.761	.415
Wake	PTSD	TE	HC	*H(2,n=60)*	*p*-value
Negative	-2.04 (-3.88; -1.13)	-2.62 (-3.32; -1.48)	-2.22 (-4.01; -1.54)	.672	.715
Neutral	-2.63 (-3.81; -1.58)	-2.62 (-3.98; -1.59)	-2.16 (-3.48; -.93)	2.554	.279
Positive	-2.13 (-3.6; -1.145)	-3.18 (-4.26; -2.32)	-2.18 (-3.56; -1.71)	3.362	.186

**.Correlation is significant at the 0.01 level (1-tailed).
*.Correlation is significant at the 0.05 level (1-tailed).
Note: Values are presented as median (interquartile range).

Hypothesis 4

In addition, I ran analyses to examine associations between HRD and emotional memory recognition per group using correlation analyses (Spearman's rho). To account for inflation in Type 1 Error associated with multiple correlations, the p-value was adjusted using the Benjamini-Hochberg procedure. Only some of the per-group correlations survived the Benjamini-Hochberg procedure.

In response to negative stimuli after an interval of sleep, there was a significant inverse correlation between HRD at encoding and *d'* for the TE group, r_s = -.499, p = .015, adjusted p = .045. This indicates that as HRD decreases, or becomes increasingly negative, then *d'* or accuracy increases. In other words, the more psychophysiological reactivity at encoding, the more accurate the memory recognition performance in the TE group. There were no associations for the PTSD and HC groups after an interval of sleep, in response to negative stimuli.

In response to positive stimuli after an interval of sleep, there were no significant results for any of the groups.

In response to neutral stimuli after an interval of sleep, there was a significant inverse correlation between HRD at encoding and *d'* for the TE group, r_s = -.504, p = .014, adjusted p = .045. This indicates that as HRD decreases, or becomes increasingly negative, then *d'*, or accuracy, increases. In other words, the more psychophysiological reactivity at encoding in response to neutral stimuli, the more accurate the memory recognition performance in the TE group. There was a significant positive correlation between HRD at encoding in response to neutral stimuli and *d'* for the HC group, r_s = .518, p = .010, adjusted p = .045. This indicates that as HRD increases, or becomes more positive, then *d'* also increases. In other words, the less psychophysiological reaction at encoding in response to neutral stimuli, the more accurate the memory recognition performance in the HC group.

Overall, these results show that the TE group have inverse associations across valences between HRD and *d'* which may imply that greater psychophysiological reactivity is associated with better memory recognition accuracy after an interval of sleep, irrespective of the valence of the stimuli. On the other hand, the HC group displays the opposite effect where lower psychophysiological reactivity at encoding is associated with better memory recognition accuracy after an interval of sleep, in response to neutral stimuli. These results show that TE participants have opposing reactions to the HC group. In addition, there are no observed significant correlations in the PTSD group in response to negative and neutral stimuli.

In the wake condition, the TE group had inverse significant correlations across the positive r_s = -.596, p = .004, adjusted p = .036 valenced category. There was no observed significance in the other groups across negative, positive, or neutral and there was no observed significance in any group in the neutral category over an interval of wake.

Regarding between-group associations between HRD and memory recognition, participants in the PTSD group showed no relationship between HRD at encoding and subsequent memory recall of either valenced or neutral information, irrespective if the consolidation period was filled with sleep or waking. In contrast, the TE group, had better recognition of material with increased physiological response. This result occurred when the consolidation period was filled with both sleep and waking. For the sleep condition this relationship occurred with regards negative and neutral stimuli, while for the waking condition, it occurred with regards to valenced material only. Notably, with regards to associations with recognition of neutral stimuli after sleep, the TE and HC groups showed opposing results. While TE participants tended to remember more stimuli when their physiological response was strong; HC participants tended to remember neutral material when their physiological response was low.

Table 4

HRD Association with Memory Performance in Response to Valenced Stimuli Across Groups

Sleep	HRD		
	PTSD	TE	HC
Negative *d'*	-.064	-.499* (*p*=.015/.045)	.318 (*p* =.086)
Positive *d'*	-.359 (*p*=.066)	-.369	-.193
Neutral *d'*	-.156	-.504* (*p* = .014/.045)	.518** (*p* = .010/.045)
Wake			
	PTSD	TE	HC
Negative *d'*	-.077	-.401*	.229
Positive *d'*	.059	-.596** (*p* = .004/.036)	-.001
Neutral *d'*	.213	-.153	.159

*. Correlation is significant at the 0.05 level (1-tailed)

**. Correlation is significant at the 0.01 level (1-tailed)

Note: Spearman's rho values are given with (*p*-value/adjusted *p*-value)

Hypothesis 5

Table 4 shows the descriptive statistics regarding REM percentage, REM fragmentation, and REM Fragmentation to Wake/NREM1 for the PTSD, trauma exposed and healthy control groups. To get an impression of the possible associations between parameters related to REM sleep and memory recognition accuracy (*d'*) for each valenced category, I performed correlation analyses, using Spearman's rho, with regards to the whole sample, and taking each group into account. To account for inflation in Type 1 error associated with multiple correlations, the p-value was adjusted using the Benjamini-Hochberg procedure.

Table 5

Descriptive Statistics for REM variables across group (Non-parametric distribution)

	Group		
	PTSD	**Trauma-Exposed**	**Healthy Controls**
Variable	***n=21****	***n=19***	***n=20***
REM Percentage	19.30 (14.75; 22.90)	18.80 (16.50; 21.60)	18.60 (16.79; 23.08)
REM Fragmentation	17.50 (14.00; 27.75)	18.00 (11.00; 25.00)	22.50 (12.00; 31.00)
REM Fragmentation to Wake/NREM1	9.50 (7.00; 14.00)	9.00 (6.777; 15.00)	7.50 (5.00; 11.75)

Note: Values are presented as median (interquartile range).

*REM Fragmentation and REM Fragmentation to Wake/NREM1, n=20

None of the whole sample or per-group correlations survived the Benjamini-Hochberg procedure and uncorrected analyses are given to describe trends in the data.

Uncorrected analysis performed with the whole sample showed a significant inverse correlation between REM fragmentation and *d'* for neutral stimuli, r_s = .-.276, p = .018. This indicates that with increased REM fragmentation, there is a decrease in memory recognition accuracy for neutral stimuli. Similarly, there was a significant inverse correlation between REM Fragmentation to Wake/NREM1 and *d'* for neutral stimuli, r_s = .-.246, p = .032.

When the correlations were performed per group, the only significant associations were found concerning the TE group. There was a significant inverse correlation in the TE group between REM percentage and *d'* for negative stimuli after an interval of sleep, r_s = -.419, p = .037. This indicates that for the TE group, when REM percentage increases, memory recognition performance decreases. With regards to REM fragmentation, there was a significant inverse correlation in the TE group between REM fragmentation and *d'* in

response for neutral stimuli, r_s = -.504, p = .014 . This indicates that with increased REM fragmentation, there is a decrease in memory accuracy for neutral stimuli in the TE group.

CHAPTER 5: DISCUSSION

This research project set out to investigate the role of autonomic nervous system reactivity (ANS; measured using HRD) in subsequent emotional memory recognition after an interval of sleep and waking, to gain insight into sleep-dependent emotional memory consolidation processes. Specifically, I aimed to investigate whether elevated arousal at encoding in response to valenced as opposed to neutral stimuli was associated with better recognition of valenced rather than neutral information after a period of sleep. I wanted to see if this effect was specific to sleep, rather than waking, suggesting that sleep-dependent consolidation processes of emotional content were supported by ANS reactivity at encoding of that information. I also aimed to investigate whether these processes were disrupted in the context of trauma, specifically when trauma resulted in significant psychiatric symptoms (PTSD). I predicted that in PTSD-diagnosed participants, rather than trauma-exposed or healthy controls, increased ANS function at encoding in response to negative rather than positive or neutral information would be associated with a higher memory recall of that category. Lastly, I aimed to see whether features of REM sleep, which previous studies show may modulate emotional memory, are associated with memory for valenced information (negative and positive) and whether those with PTSD have stronger REM and negative emotional memory associations, based on my previous prediction of higher recall of negative information in this group. To achieve these aims I used archival data which was collected from 60 participants: 21 of whom were diagnosed with PTSD, 19 who had been exposed to trauma, and 20 healthy controls.

I first investigated valence-related (valenced versus neutral) differences, and between-group (PTSD versus trauma-exposed versus healthy controls) differences in HRD at encoding. Secondly, I evaluated associations between this aspect of autonomic activation and

memory recognition for valenced information, taking into account whether there had been an interval of sleep or waking and whether participants were PTSD diagnosed, trauma-exposed or healthy controls. Thirdly, I evaluated whether REM disruption would be associated with memory performance.

The results showed that there was no difference in HRD, representing a component of ANS activation, during the encoding phase of valenced and neutral stimuli irrespective of measurement occurring prior to sleep or waking and there were no differences between the PTSD, TE, and HC groups with regards to this measurement.

With regards to associations between HRD at encoding and memory performance for valenced and neutral information after either a period of sleep or waking, the results showed that after a period of sleep, higher HRD reactivity was associated with better recognition of valenced information rather than neutral information. However, this association was only evident in response to positive stimuli. After a period of waking, there were no associations between HRD reactivity and memory performance for valenced or neutral stimuli. This pattern suggests that arousal mechanisms at encoding may influence sleep specific modulation of memory traces, but only concerning positive information.

Further analysis showed no association between HRD reactivity and memory recall accuracy for PTSD participants. However, TE participants displayed increased ANS functioning that is associated with enhanced memory performance for both valenced and neutral information. This applied to both the sleep and waking conditions, although it was marginally stronger for associations related to sleep. HC participants showed a contrasting association in response to neutral information after a period of sleep, in other words, HC participants displayed low arousal with better memory recognition performance, while TE participants displayed higher arousal with better memory recognition performance.

Specifically, with regard to the role of REM sleep and memory performance across the groups, the results only showed trend-level associations, that did not meet statistical requirements. However, the trends indicated that with increased REM fragmentation, irrespective of whether the arousals lightened sleep or did not, there was a decrease in memory recognition accuracy for neutral stimuli. When these results were examined by group, there were only similar associations for those in the TE group that were significant before statistical correction. These indicated that with higher REM percentage, the TE group performed with decreased memory recognition accuracy for negative information. With regards to higher REM fragmentation in the TE group, there was a decrease in memory recognition memory accuracy for neutral stimuli, in other words when there was disruption of REM sleep for TE participants, they had poorer memory recognition accuracy for neutral information.

Autonomic Functioning in Response to Valenced and Neutral Stimuli

The literature suggests that there is a deceleration in heart rate in the first seconds post stimulus response to emotionally (valenced) arousing stimuli (Abercombie et al., 2008; Bolinger et al., 2019; Cunningham et al., 2014; Jones & Spencer, 2019). Thus, the first hypothesis investigated whether this ANS reactivity at encoding is higher for valenced rather than neutral information. The results indicated no significant difference in parasympathetic reactivity between negative or positively valenced and neutral information. This held true for encoding before an interval of sleep and an interval of wake. This finding is contrary to what was predicted in the first hypothesis.

Every participant in the current sample lived in low-SES environments in the Western Cape of South Africa. The majority of the participants in this sample fell into the monthly income bracket of R2500 – R5499 (n = 24) and the average number of years of education was

12.34. South Africa is known to have one of highest incidence rates of gender-based violence (GBV) globally (Hartmann et al., 2023) and the staggering majority of cases of interpersonal violence (IPV) happen in low-and-middle-income countries (LAMICs) (Makanga et al., 2015). While the current study focused on sexual assault survivors, it is important to consider the environments in which these participants lived. It is possible that exposure to community violence could contribute to chronic stress which may result in dampened psychophysiological reactivity (Williamson et al., 2015). This applies to all participants irrespective of group, who were from low SES communities with high rates of violence.

Autonomic Functioning and Memory Recall Performance

In this study, HRD recorded during encoding was associated with subsequent recall of positively valenced information, where a greater psychophysiological response was associated with better memory recognition accuracy for positive stimuli rather than negative and neutral information, although this association was weak in strength. It was, however, seen when participants completed the task with an interval of sleep and not waking. This result should be interpreted cautiously based on the strength of the relationship; therefore subsequent interpretations are largely speculative.

Regarding valence, it partially aligns with the literature which shows that emotion enhances memory (Earles et al., 2016; Kensinger & Ford, 2020; Squire & Dede, 2015), although the result was only evident for positive and not negative stimuli. The lack of association between HRD at encoding of negatively valenced information and memory recall of those stimuli is surprising. In addition, the stronger association between psychophysiological reactivity and memory recall accuracy for positive information may also be attributed to the low SES environment in which the participants live contributing to the negative stimuli failing to reach the threshold required for psychophysiological response due

to daily exposure to negative scenes in their environment desensitizing the participants. For example, chronic stress exposure can lead to dysregulated cardiac reactivity in response to stress and stress response systems may become depleted resulting in blunted cardiac reactivity, a result which is especially seen in females (Cavanagh & Obasi, 2020). Furthermore, tentatively, the stronger association between psychophysiological reactivity and memory recall accuracy of positive stimuli may serve as a protective factor for participants from these communities.

Regarding sleep and wake comparisons, the data showed that the relationship between HRD at encoding and subsequent memory recall was only evident after a period of sleep and not waking. Past studies have shown that sleep preferentially consolidates emotional memory (Bennion et al., 2015; Walker & van der Helm, 2009). The literature, however, does not demonstrate any clear results regarding the contribution of sleep to the consolidation of negative versus positive information. Moreover, the literature is dominated by studies which compare negative to neutral, while few examine negative to neutral and positive to neutral (Alger et al., 2018; Ashton et al., 2019; Cox et al., 2018). A recent study demonstrated that the preferential effect of sleep on emotional memory is specific to negatively valenced stimuli (Denis et al., 2022). Denis et al. (2022) recruited adults between 18 to 59 years old for their online study. They used self-report measures and a self-paced recognition task after a 12-hour delay filled with either nighttime sleep or daytime wakefulness. Similar to the current study, stimuli presented in their emotional memory trade-off task consisted of scenes which were negative, positive or neutral on neutral backgrounds, and these scenes were later presented amongst new scenes in the recognition task. This study differs from the current study as the task is somewhat different, they used a broad sample of adults as opposed to looking participants with psychopathology compared to HC, their study was conducted online as opposed to in a sleep laboratory and they did not assess psychophysiological reactivity. The

results from the current study would suggest the positive information is associated with greater psychophysiological reactivity and subsequently enhanced memory recall accuracy, at least in this cohort of female trauma survivors and healthy controls.

Glaser et al. (2012) conducted a study examining sex differences in emotional memory using the IAPS. They found that females had better accuracy in memory performance for emotional stimuli when the stimuli were positively valenced images than for negatively valenced images. This contrasted with males who were more accurate in performance for highly arousing, negative images. Thus, it is important to consider the influence of sex differences when comparing the current study to previous studies that have broadly examined male or mixed-sex cohorts.

Autonomic Functioning and Between Group Differences Associated with Sleep and Waking

Regarding group differences in the relationship between HRD at encoding in response to valenced stimuli, the differences between the PTSD, TE, and HC groups were not significant in response to either negative or positive stimuli irrespective of whether it was before an interval of sleep or wake. While the lack of differences in psychophysiological reactivity between sleep and wake conditions is in line with Cunnigham et al. (2014) who found similar results, this study expected that there would be differences between PTSD-diagnosed, trauma exposed and healthy controls with greater reactivity in the two trauma groups (with those carrying a diagnosis more reactive than those who did not) (Iffland et al., 2019).

Some studies demonstrate high HRD in the context of PTSD, for example Chou et al., (2018) found that in a sample of PTSD participants, there was greater HRD during an autobiographical trauma recall compared to recall of a neutral routine. D'Andrea et al. (2013)

had two groups in their study respond to startling sounds which were presented over a period of 15 minutes: a high and low trauma group where the high trauma group had a cluster of characteristics including earlier age at time of trauma, a greater number of types of traumas, a higher sum of current PTSD symptoms and a higher mean of dissociation state compared to the low trauma group. This high trauma group displayed pronounced HRD which diminished in strength throughout the task, with an initial small HR acceleration increased as the task progressed. Participants in the low trauma groups demonstrated high HRD over time in response to this startling sound stimulus, which remains consistent, while HR acceleration decreases throughout the task. D'Andrea et al. (2013) explain that the stress response happens in two stages, the orienting stage and the fight or flight stage according to the defence cascade model. Notably, HRD occurs in the orienting stage, which represents parasympathetically-driven ANS functioning where there is a mild increase in parasympathetic activity resulting in moderate HRD, and concomitant sympathetic nervous system (SNS) activity leading to increased HR, marking the fight or flight phase of the autonomic response, which prepares the individual for action (Beutler et al., 2022). Those with conditions such as PTSD would be expected to exhibit enhanced arousal as a reaction to a threat, marked by their lack of initial HR acceleration and diminishing HRD over the task period, while the low trauma group displayed enduring parasympathetic activity with lessening sympathetic reactivity resulting in an oriented-but-prepared state (D'Andrea et al., 2013).

When compared to the current study, the methodology differed as they used only an audio negative stimulus as in a startling sound whereas the current research utilised IAPS images with negative and positively valenced as well as neutral stimuli. In addition, the current study did not find between group differences in response to valenced and neutral

stimuli, however there is a pattern of association with emotional memory which is discussed below.

Furthermore, some studies have found that other psychophysiological markers best represent changes in ANS activation in PTSD-diagnosed individuals. For example, tonic immobility which is an involuntary reaction or reflex characterised by severe, but reversible, motor inhibition and inability to appropriately respond to environmental stimuli. Tonic immobility has psychophysiological indicators including posture or body sway, elevated heart rate and decreased heart rate variability (Norte et al. 2019) which may be better markers of ANS response in those with PTSD. Norte et al. (2019) demonstrated that individuals with PTSD, when listening to an audio recording of their autobiographical trauma, exhibited a cardiac response that had greater acceleration and prolonged tachycardia indicating enhanced SNS activation. PTSD participants who reported increased tonic immobility upon stimulus exposure, had a greater and sustained HR acceleration compared to PTSD participants who had low or no signs of tonic immobility. This is similar to the previously discussed study by D'Andrea et al. (2013) as both demonstrate a prolonged increase in HR acceleration indicating sustained SNS activation. However, Norte et al. (2019) did not examine HRD, representing the parasympathetic branch of the ANS response.

Group Specific Associations Between Autonomic Functioning and Memory Recall

While there were no between-group differences in HRD in response to valenced and neutral information, there were some group specific associations between ANS reactivity and emotional memory recognition. It is worth noting that the direction of the relationship between HRD and memory recognition accuracy is inverse for the PTSD and TE groups compared to the healthy control group. Although the association between HRD and memory was in the same direction for those with PTSD in comparison to those with trauma exposure,

they were not meaningful for the PTSD group. Overall, this demonstrates opposing reactions in the trauma groups compared to healthy controls.

Regarding those with PTSD, these results may tentatively indicate dysregulation of ANS functioning and memory processes in those with PTSD. For example, Norte et al. (2013) showed that PTSD participants exhibited both an elevated heart rate in response to an autobiographical trauma script and sustained accelerated heart rate post-exposure, this is referred to as failure to recover. These findings in the PTSD population implied diminished vagal tone of the heart (Norte et al., 2013; Williamson et al., 2015), with vagal tone indicative of parasympathetic functioning. It is therefore possible that in this sample, PTSD participants failed to display expected HRD responses, also indicative or parasympathetic functioning, and consequent lack of association with emotional memory, due to failure to recover. The study did not examine heart rate acceleration, as it was beyond the scope of the project.

One possibility, therefore, is that the nature of PTSD, is characterised by disorganised ANS reactivity which includes in some groups numbed emotional response (increased HRD, or orienting/parasympathetic response, and a sustained sympathetic response) and in some, elevated responsivity (increased and sustained parasympathetic response). In this sample, I see no association between HRD and memory in those with PTSD, while there are associations in the other groups, which does imply disorganisation of ANS and emotional memory associations.

Regarding those with trauma exposure, but not PTSD, the results are an indicator that greater psychophysiological reactivity is associated with enhanced memory recognition accuracy for negative and neutral stimuli after an interval of sleep in the TE group. While the relationship between psychophysiological reactivity and enhanced memory recognition accuracy was not as strong for positive information, it did demonstrate a relationship in the same direction. In addition, in the waking condition, the TE group exhibited a trend towards

valenced (both negative and positive) material having better memory recognition accuracy associated with HRD. However, only the association with regards to positive information was meaningful. The results showed that an increase in orienting towards the stimuli, as demonstrated by HRD is associated with enhanced memory recognition accuracy. Thus, psychophysiological reactivity is associated with enhanced memory performance (Pilarczk et al., 2022). However, this is relatively indiscriminate as it is found across sleep and waking as well as for valenced and neutral information. It may be suggested that trauma experience results in a decreased capacity to discriminate between information that is salient or not. For example, Garfinkel et al. (2014) demonstrated that those exposed to trauma including PTSD diagnosed individuals have deficits in using contextual information which leads to inappropriate memory expression in both safety and danger contexts. Furthermore, this difficulty with utilising contextual cues may result in fear memories that are not adapted to the context and may play a role in other characteristics of PTSD, such as sustaining a state of perceived threat and danger which lends itself to hyperarousal and avoidance (Garfinkel et al., 2014). According to a review by Pace-Schott et al. (2015) studies suggest impaired attainment of extinction, a type of emotional memory integral to healthy emotion regulation where there is inhibition of a conditioned fear response, is impacted by sleep quality and diagnosis of anxiety disorders. An impaired ability to acquire extinction may partly be due to an elevated capacity to attain a conditioned fear which is associated with modulated ANS reactivity. Furthermore, memory consolidation processes allow for extinction to generalise and this is facilitated by healthy sleep (Pace-Schott et al., 2015). Overall, these studies and their suggestions lend themselves to the proposition that trauma experience diminishes the capacity to discriminate salient information from other stimuli, whether this may be due to deficits in using contextual information or due to impaired extinction which does not

generalise and results in hyperarousal which in turn interrupts sleep dependent memory processes.

The PTSD group exhibits a pattern of blunted reactivity as described in the above section (D'Andrea et al., 2013; Norte et al., 2019) resulting in a lack of demonstrated associations.

Results for the HC group indicated that lower psychophysiological reactivity at encoding is associated with better memory recognition accuracy for neutral stimuli after an interval of sleep. These results show opposing associations in the TE and HC groups after an interval of sleep, the TE groups show elevated arousal with enhanced recall while HCs demonstrate decreased arousal with enhanced recall.

These results tentatively demonstrate an expected association between low psychophysiological reactivity and neutral memory recognition in HCs after a period of sleep. ANS-memory associations as observed here could speculatively aid in retaining neutral information for those living in low SES areas with high exposure to crime and subsequent threats to personal safety; this healthy processing may occur during sleep in HCs. On the other hand, the lack of ANS-memory association in response to valenced stimuli in HCs may once again be due to blunted cardiac response (D'Andrea et al., 2013; Norte et al., 2013; Norte et al., 2019).

REM Disruption and Memory Recognition Performance

Sleep, particularly REM sleep, is implicated in memory and ANS regulation (Mukai & Yamanaka, 2023). In the current study, there were tentative relationships between aspects of REM sleep and memory recognition accuracy for valenced or neutral stimuli. This discussion is speculative due to the weak nature of the results.

Across the whole sample, there was an inverse relationship between REM fragmentation and memory recognition accuracy for neutral stimuli. In other words, when there was greater REM fragmentation, there was lower memory recognition accuracy for neutral pictures. Similarly, there was an inverse relationship between REM fragmentation to wake/NREM1 for neutral stimuli. Since REM contributes to memory consolidation (Almeida-Filho et al., 2018), it follows that REM disruption as in REM fragmentation or REM fragmentation to wake/NREM1 would hinder memory recognition performance. It is interesting that this occurs only in the neutral category. Although the overwhelming thrust of earlier literature shows causal connections between slow-wave sleep and neutral declarative memory (Cox et al., 2012; Gais & Born, 2004; Walker, 2009), a number of studies also show that REM sleep is active in consolidating neutral stimuli. For example, Ribeiro et al., (2007) demonstrated that gene expression during REM sleep is crucial for long term potentiation which allows for long term memory formation. More recently, Almeida-Filho et al. (2018) suggest that memory corticalisation, including neutral declarative memory, is activated by REM sleep.

When examining the results in terms of group, the only relationships observed occurred in the TE group. With regards to REM fragmentation, there was an inverse relationship between REM fragmentation and memory recognition accuracy for neutral stimuli. In terms of REM percentage, there was an inverse relationship between REM percentage and memory recognition accuracy for negative stimuli. It could be suggested that for those who are recovering from trauma and living in low SES, crime affected areas such as these participants, REM is involved in the regulation of what is remembered whether it is valenced or neutral. For these participants to remain resilient to their surroundings, they would ideally remember neutral and valenced information equally to avoid being

overwhelmed by negative information, as is observed in psychopathology like PTSD and depression (Itoh et al., 2019; Urban et al., 2018).

With regards to the current results, for TE participants recovering from trauma, the degree to which their REM is fragmented or not may be particularly relevant and hence, if they do not have fragmentation, they recall neutral information adequately. On the other hand, higher amounts of healthy REM may be adaptive (Cowdin et al., 2014) and depress memory performance for negative stimuli which facilitates the individual's resilience.

Limitations

There are several notable limitations to this study.

The sample consisted of only female, sexual assault survivors which is a strength for contributing to research on females with PTSD but limits the generalizability of the findings since the association between emotion and memory performance can be affected by gender differences (Glaser et al., 2012; Pilarczyk et al., 2022). In addition, trauma type may influence the presentation of symptoms or reactivity. For example, Schalinksi et al. (2013) suggest that victims of trauma, where the individual is physically overcome by a perpetrator, such as in instances of rape, show a shutdown defence response. Again, this limits the generalizability of the results.

The statistical analyses employed, because of the highly non-parametric nature of the data, did not allow for the consideration of covariates and the level of depressive symptoms, which differed by group, and should therefore be accounted for. While I took a more conservative approach, also because of the small sample size, future studies should recruit a larger sample size, which allow for better stability in the data.

The use of the IAPS may not be consistently culturally appropriate for the South African context and the original study was conducted before the development of the SA-APS.

Participants who reside in low SES areas in South Africa, such as those in the current sample, are exposed to high rates of crime and violence potentially resulting in desensitization to the images from the IAPS (Nestadt et al., 2022). While HC participants had not personally been exposed to or experienced a traumatic event as outlined by the DSM-IV, these participants are more likely to have heightened sensitivity and awareness of traumatic events. In turn, they are likely to have heightened sensitivity to negative stimuli (Sassenberg et al., 2015) or desensitization (Nestadt et al., 2022) in an effort of resilience or coping with their environment. Therefore, images from the IAPS may not have elicited sufficient emotional response. However, regarding emotion elicitation and in the context of sleep research, the IAPS is considered the gold standard and has been successfully used in other studies (Ashton et al., 2018; Cellini et al., 2019; Tempesta et al., 2015).

Considering the results of the current study, it is suggested that future research can examine the responses to stimuli with regards to both HR acceleration and HRD since trauma participants can present with altered cardiac reactivity including both the parasympathetic and sympathetic components of the ANS (D'Andrea et al., 2013; Norte et al., 2019).

Conclusion

Overall, the study showed tentative associations between HRD and subsequent memory performance post-sleep. The results suggest that psychophysiological reactivity at encoding promotes the consolidation of positive information during sleep, which may be a protective factor for this female cohort.

In terms of memory recall, the lack of association of psychophysiological reactivity with memory performance in the PTSD group may be due to blunted cardiac response and deficits in discriminating salient information.

There was a tentative, expected association between low psychophysiological reactivity and neutral memory recognition performance in HCs after a period of sleep. This could potentially facilitate retaining neutral information for those in high stress environments and this healthy processing may occur during sleep in HCs.

Lastly, results regarding REM disruption, there was tentatively an association with decreased memory recognition for neutral stimuli across the whole sample. The TE group demonstrated that when they experience larger proportions of REM, they are less likely to retain negative information, while neutral memories are susceptible to REM fragmentation.

Overall, the research highlights that the relationship between HRD, a measure of ANS functioning, is complex and requires further investigation. The results indicate that both sleep and emotion influence memory performance and specifically for those with PTSD, the expected relationships are not evident, suggesting either that they do not exist or that there is dysregulation of ANS functioning and memory processing. The latter interpretation is favoured, because HRD and memory associations are present in the other groups.

The tentative associations outlined in this research need to be confirmed in a larger sample which could consider environment, such as comparing those living in high and low socio-economic contexts and control for the daily exposure to negative, positive and neutral

stimuli in the environment in order to better understand how both emotion and sleep contribute to memory processing. The current data indicates that there are possible sleep effects and REM sleep may play a role in the process. Valence specific effects remain to be understood and these should be explored in greater detail in future research.

Ethical Consideration

The current study uses archival data from Dr Lipinska's 2017 PhD. All of these study procedures have been carried out and were approved by Research Ethics Committees of the University of Cape Town's Department of Psychology and Faculty of Health Sciences (HREC 428/2013).

Appendix A

	Lipinska (2017)		Smith
Conceptual questions	Whether and how sleep disruption in PTSD-diagnosed individuals impacted: 1. Neutral declarative memory performance 2. Emotional memory performance 3. Emotional reactivity	Conceptual questions	1. Is there evidence to show that emotion at encoding, measured by HRD, "tags" memories for consolidation during sleep? 2. Is this consistent for negative *and* positive information? 3. Do PTSD participants show predictable sleep-dependent emotional memory disruption?
Psychophysiological Variables	1. HR 2. PEP 3. LVET 4. SCL		1. HRD at encoding
Papers published	Breen et al. (2019) Lipinska & Thomas (2017) Lipinska & Thomas (2017) Lipinska & Thomas (2019)	Contribution to the literature	1. Inclusion of positive stimuli in the encoding phase; answering the question of where it is something about negative emotion or emotion? 2. Contributing to research on women and PTSD

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