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Exploring the Psychological Mechanism Linking Nightmares to Increased Self-Harm Risk

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Abstract

Nightmares, a common sleep disturbance which provoke fearful awakening, have been found to be a significant predictor of suicidal thoughts and behaviours. The research presented in this thesis aims to firstly examine if nightmares are predictive of self-harm regardless of suicidal intent or motivation, and secondly to explore the psychological mechanism linking the occurrence of nightmares to self-harm.

Chapter 2, an online survey, revealed that nightmares were a significant predictor of self-harm regardless of suicidal intent or motivation and that this relationship remained when controlling for the effects of depressive symptoms. High levels of nightmares were also associated with elevated levels of negative affect and defeat.

Chapter 3 prospectively examined the direction of the predictive relationship between nightmares and self-injurious thoughts and behaviours (SITBs) through a 5-day diary study of undergraduate students. Nightmares unidirectionally predicted SITBs when controlling for depressive symptoms and negative affect. Mediation analysis revealed negative affect to be a partial mediator between nightmares and post-sleep SITBs.

Chapter 4 explored differences in the linguistic content of nightmares in individuals with and without a history of self-harm, using nightmare reports prospectively obtained from participants taking part in the diary study. Contrary to the literature, participants with a history of self-harm did not report more words pertaining to death. Exploratory analysis investigating self-harm
recency indicated a higher frequency of perceptual words such as ‘feel’ and body words such as ‘arm’ in participant with current self-harm (< 1 month) compared to those with a history of self-harm (> 1 month) and those without.

Chapter 5 modelled the psychological mechanism linking nightmares to increased self-harm risk via structural equation modeling from survey data. This model incorporated negative affect, hyperarousal and a latent variable ‘self-harm cues’ building on our previous findings and the literature. Our retained model indicated that a 1 standard deviation increase in nightmare score increased the probability of participants having recently (< 1 month) engaged in self-harm.

Chapter 6 tested the predictions of the model computed in the previous chapter using behavioural and psycho-physiological methodology. Psycho-physiological measures when exposed to negatively valenced stimuli did not reveal any differences between high and low nightmare participants, nor were differences observed in self-harm cue sensitivity. However, a medium effect was observed indicating the high nightmare group to be more sensitive to stressors.

These findings are discussed in the context of the literature in Chapter 7. They provide novel insights into the relationship between nightmares and self-harm, and highlight the importance of negative affect and hyperarousal as reducing stress resilience in individuals at risk of deliberate self-injury.

*Key words: Self-harm, Nightmares, Negative affect, Hyperarousal, Mechanism*
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Thesis Communications

The research reported in this thesis has been subject to several oral and poster presentations.

From Chapter 3:

From Chapter 4:

In addition, the data and findings reported in Chapter 3 have been submitted for peer-reviewed publication:
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Chapter 1: Suicidality, Nightmares, and their Association – An Introduction and Review.

1.1. Introduction

Suicide prevention efforts have aimed to establish clear predictive risk factors for suicidal behaviours to identify vulnerable individuals and groups in the hope of effectively intervening. Concurrently, research has established theoretical models of suicide using correlates with the hope of establishing clear causation to further improve early detection and intervention. Such research has established a large list of correlates, risk and protective factors which in the majority of cases can be transposed from one country to another (Nock et al., 2008).

This thesis is concerned with sleep disturbances, more precisely nightmares and their relationship with self-harming behaviour regardless of suicidal intent. The literature supports nightmares as a suicide risk factor independent of depression (Bernert & Joiner, 2007; Sjöström, Hetta, & Waern, 2009; Sjöström, Waern, & Hetta, 2007). A potential explanatory mechanism linking nightmares to suicidal ideation has been detailed in Cukrowicz et al. (2006) and Bernert & Joiner (2007), which follows current theories from the dreaming literature indicating nightmares to be affect deregulators. That is, the majority of dreams have been reported to contain mildly negative or dysphoric themes which over the course of the sleep period (Cartwright, 2010), desensitise the sleeper to that negative content resulting in reduced negative affectivity. However, nightmares are seen as a dysfunction of the regulatory process whereby the usually mild content becomes too intense, forcing awakening and increasing post-sleep negative affect (Cartwright, 2010; Nielsen
Cukrowicz and colleagues propose that post-sleep negative affect induced by nightmares in turn increases sensitivity to stressors and suicidal cues. While promising, this mechanism needs empirical validation which to date, has been lacking. Moreover, the mechanism detailed focuses on suicidal ideation rather than suicidal behaviours such as self-harm.

The present thesis focuses on empirical validation of a mechanism linking nightmares to self-harm regardless of intent or motivation to die and takes into consideration existing theories and research from the fields of suicide and sleep. This is done in the hope of providing an explanation which may inform future suicide prevention and intervention efforts.
1.2. Suicidality

Approximately one million people worldwide die by suicide over the course of a year, while a further 10 to 20 million globally attempt suicide (Bertolote & Fleischmann, 2002). Although suicide statistically remains a rare event, the World Health Organisation lists suicide as the 3rd leading cause of death for those aged 15 to 44 (World Health Organisation, 2010). The UK suicide rate has remained relatively stable between 2001 and 2011, fluctuating from 12.4 to 11.8 per 100000. Recent trends are worrying due to the significant increase from 11.1 to 11.8 per 100000 between 2010 and 2011 (Office for National Statistics, 2013).

Moreover, in light of the current global recession and harsh economic climate, suicide rates are likely to be impacted upon, as with previous recessions where rates increased (Gunnell, 2005). This is reflected by the significant increase in hospital admissions (0.4%, 110490 to 110960 cases) throughout England between 2010-2011 and 2011-2012 which continues the trend of increasing hospital admissions since 2006 (Health & Social Care Information Centre, 2012).

The majority (50% to 66%) of completed suicides occur at the first attempt (Mann, 2002; Rudd, Joiner, & Rajab, 1996). Thus, suicide prevention efforts have aimed to establish clear predictive risk factors for suicide and suicidal behaviours to identify vulnerable individuals and groups to more effectively intervene. Concurrently, drawing theoretical models of suicide using correlates has been performed with the hope of establishing clear causation.
Such research has established a large list of correlates, risk and protective factors. A few of which are demographically or culturally dependent; however, as noted by Nock et al. (2008) the vast majority of factors found in one country can be transposed to another. These risks factors have been broadly categorised as demographic, situational (events), biological, psychiatric and, psychological. Other authors such as Lonnqvist (2009) have used variations in categorisation. However, due to the high interrelation between these risk factors, the application of a categorisation system serves only to ease the explanation of risk factors. The categories themselves do not imply additional connotations. Nock et al. (2008) provide a good overview of risk and protective factors for suicidal behaviour and encourage further research into independent suicide risk factors for the establishment of intervention and prevention models. Similarly, Lau, Segal, & Williams (2004) highlight the need for research into additional risk factors, yet state that many of these factors are socio-demographic; such as gender and age or historical such as prior psychiatric episode or prior self-harm. These factors while important in identifying at risk individuals, do not lend themselves well to intervention efforts.

High risk factors such as mental illness (psychiatric factor) have been widely and consistently linked to increased suicidal risk (Nock et al., 2008). While the strongest and most consistent predictive risk factor for suicide was a previous suicide attempt, increasing suicide risks 40-fold (Harris & Barraclough, 1997), major depression and bipolar disorder follow the trend of prior attempts by increasing suicide risk between 5 to 20 times compared to the general population. Moreover, bipolar disorder is associated with higher lethality employed in suicide attempts (Raja & Azzoni, 2004). Suicide autopsy
studies have supported these findings showing that approximately 91% of suicide completers suffered from a psychiatric disorder (Cavanagh, Carson et al. 2003), while 87.3% had a history of mental disorder with 43% being mood disorders (Arsenault-Lapierre, Kim, & Turecki, 2004). Similarly, in suicide attempters, Suominen, Isometsä, Ostamo, & Lönnqvist (2004) have shown mood disorders to be present in 75% of cases, with alcohol dependence and abuse in 53%, and other substance abuse reported in 12% of attempters. Heavy episodic drinking has been positively associated with increased suicide risk in a large cross-sectional suicide prevention screening and has shown to be independent from depression (Aseltine, Schilling, James, Glanovsky, & Jacobs, 2009). Psychotic spectrum disorders and personality disorders, particularly borderline personality disorder, carry a high risk of suicidal behaviour (Linehan, Rizvi, Welch, & Page, 2000) and co-morbidity of psychiatric disorders elevate suicide risks further (Hawton, Houston, Haw, Townsend, & Harriss, 2003).

As noted by Nock et al. (2008) and Lau et al. (2004), although psychiatric factors have a very high positive association with increased suicide and suicidal behaviour risk, researchers are increasingly questioning the focus on psychiatric factors. Nock et al. (2008) suggests moving the focus of research away from DSM Axis- I and Axis- II diagnoses to more easily modifiable psychological factors. Reviewing the predictive power of risk factors in psychiatric in-patients, Powell, Geddes, Deeks, Goldacre, & Hawton (2000) commented on these factors’ lack of sensitivity and specificity. Psychiatric risk factors were low in accuracy when positively identifying suicidal risk and also low in their ability to demarcate between those at risk and not at risk,
particularly in a clinical context where suicide thankfully remains a statistical rarity.

However, Cavanagh, Carson, Sharpe, & Lawrie (2003) regard the detection and effective treatment of psychiatric disorders to be of greater relevance for the time being. They justify this view by stating effective treatments for key suicidal risk factors with good detection predictability; hopelessness and impulsivity were lacking and needing further development. While this argument is valid and of particular importance with regards to primary care patients, it would excessively focus prevention resources impacting greatly on the general public and sub-clinical populations. Supporting the suggestions of Powell and colleagues, Arria et al., (2009) sampled university students for suicidal ideation and its correlates, finding 6% of first year students to have suicidal ideation. While those suffering from suicidal ideation did show depressive symptoms, only 40% of this group meet clinical criteria for depression. Furthermore, Nock (2009) reports on summarised findings indicating that only 52.7% of black adolescent suicide attempters report meeting clinical criteria for mental disorders, once more highlighting the lack of sensitivity and specificity of psychiatric factors. Thus, research needs to control for depressive symptoms due to large amount of variance in predicting suicidal behaviour for which depressive symptoms explains.

For the outlined reasons, research into psychological risk factors have been of particular interest and helped in the creation of psychological models of suicidal behaviour, such as the Cry of Pain model (Williams, 1997), which are not-overly reliant on mental illness, making them more broadly applicable
to the general population. It must also be noted that there is continued efforts in the development of effective interventions on psychological variables (van Spijker, van Straten, & Kerkhof, 2010). Thus, research into psychological risk factors offers a promising avenue for effective intervention and prevention strategies.

1.2.1. Suicide terminology

Suicide and the associated terminology used in the literature are varied and plentiful, reflecting the wide range of behaviours and constructs scrutinised by researchers. Suicide is defined as the killing of oneself, which is deliberately initiated and performed by the individual with the full knowledge or expectation of a fatal outcome (World Health Organisation, 1998). Completed suicide is the term used in reference to acts with fatal outcomes compared to suicide attempts; acts with non-fatal outcomes.

Suicidal ideations are idiopathic thoughts of taking one’s life, such as imagining the act or actively planning it. Beck, Kovacs, & Weissman (1979) argue that logically, suicidal thoughts must precede attempted or completed suicide. Indeed multiple studies have shown suicidal ideation to be a significant correlate and predictor of suicidal behaviour (Brent et al., 1993; Gili-Planas, Roca-Bennasar, Ferrer-Perez, & Bernardo-Arroyo, 2001; Lewinsohn, Rohde, & Seeley, 1996; Reinherz et al., 1995). Moreover, as the majority (50% to 66%) of completed suicides occur at the first attempt (Mann, 2002; Rudd et al., 1996), suicidal ideations when communicated are an important factor to consider when assessing suicidal risk.
Difficulties have arisen in the systematic categorisation of suicidal behaviours. The term suicide attempt is problematic as deciphering intent at the time of the act is often inaccurate, prone to memory biases by the surviving individual and social stigma, making people reluctant to divulge intentions.

The term “Parasuicide” was coined by Kreitman (1977) in order to include all suicide like acts, due to the over general use of the term ‘attempted suicide’, with which people were branded if presenting with a self-injurious behaviour. As such, the term includes all acts with a non-fatal outcome where one initiates a non-habitual behaviour which would cause self-harm if uninterrupted by another person. While the term parasuicide hoped to alleviate the controversy of labelling acts of individuals presenting an unclear wish to die or level of ambivalence, it has been criticised for implying suicidal intent (Hawton & Catalan, 1987) which may or may not be present during the act.

As parasuicide was deemed an unsatisfactory term, suicidal intent has been used to delineate between suicidal behaviours. The range of suicidal behaviours can be categorised by considering if suicidal intent is present at the time of the act. Suicidal intent referring to an individual’s mind set at the time of the act. Based on the presence of intent, three broad categories of behaviour can be identified within the literature. Firstly, suicide attempts which are defined as an individual carrying out the act with the wish to die and the belief that the action will bring about death. This is contrasted with non-suicidal self-injury (NSSI) whereby an act is performed without the wish to die or in the belief that the act will not cause death. Lastly, self-harm, also referred to as self-injurious behaviours are described as behaviours whereby an individual is unsure or ambivalent about their wish to die or the lethality of the act.
Much of this terminology has arisen due to changes in understanding of suicidal behaviours and our perception of the role of suicidal intent. Intent has been used to refine constructs, delineating between behaviours and lead to the generating of differing models of behaviour which aim to inform prevention and intervention efforts. However, the use of intent to demarcate between behaviours has opened a debate within suicidology (Andriessen, 2006; De Leo, Burgis, Bertolote, Kerkhof, & Bille-Brahe, 2004; Kapur, Cooper, O’Connor, & Hawton, 2013), namely if suicidal intent is a clear dichotomy or if can be viewed as a spectrum.

1.2.1. (i) Role of intent in defining suicidal behaviours

Hawton et al. (2011) define self-harm as the deliberate hurting of one’s body by deliberate self-injury or self-poisoning which takes no account of motivation or suicidal intent. This definition of self-harm which is widely adopted therefore refers to all behaviours including suicide attempts and self-injury without intent to die. However, other studies have demonstrated that self-injury often has no associated suicidal intent and can be used as a coping strategy to reduce distressing negative affect (Chapman, Gratz, & Brown, 2006). Such behaviour would be classified as non-suicidal self-injury (NSSI). Increasingly, NSSI is distinguished from suicidal acts within the literature and described as distinct yet clinically related (Lofthouse & Yager-Schweller, 2009).

Studies have shown that individuals presenting to hospital following acts of self-injury were 66 times more likely to die by suicide compared to the general population within the first year after the act. This increased risk was 64 times greater for males and 90 times greater for females (Hawton, Zahl, &
Moreover, research looking at correlates of NSSI in adolescents and young adults showed 70% of those reporting a recent episode of NSSI had a lifetime history of a minimum of 1 suicide attempt, while 55% reported multiple attempts (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006).

Much of the issue on classifying self-harm behaviours lie with the assessment of suicidal intent as categorical (present or absent). Researchers have used intent to delineate between behaviours and this has been useful in developing theoretical approaches. However, its practical implications have been criticised due to the difficulty of measuring suicidal intent (De Leo et al., 2004). There are difficulties in obtaining a clear ‘yes’ or ‘no’ in relation to suicidal intent due to the persisting social stigma associated to self-harm (Law, Rostill-Brookes, & Goodman, 2009) and the memory biases which are prevalent in high risk individuals (e.g. depressed individuals; Williams & Scott, 1988). Moreover, the frequent reports of ambivalence or uncertainty regarding intent expressed by patients reporting in hospitals following self-harm (Skegg, 2005) makes the categorising of self-harm based on intent as either suicidal or non-suicidal contentious.

Due to the aforementioned problems in measuring intent, clinicians and researchers have adopted a convention of focusing on obtaining a description of the behaviour. Clarifying issues of intent are secondary to the description (Skegg, 2005). A description of the behaviour whereby any potential sign of intent is indicated is deemed suicidal in order to avoid underestimating potential suicide risk (Nock, 2010). The demarcation of behaviours between suicide and self-harm thus becomes based on fatality of outcome.
Therefore, throughout this thesis, self-harm will be operationalised as intentional self-injury or self-poisoning, regardless of motivation or suicidal intent and with a non-fatal outcome (Hawton et al., 2011). The above definition of self-harm assumes self-injury to occur with suicidal intent on a continuum. This conceptualisation was selected over one which dichotomises suicidal intent such as NSSI (non-suicidal self-injury) and suicide attempts due to the changing motivations of self-harm within and between episodes, frequent reports of ambivalence associated to self-harm, and the relatively weak evidence base for the dichotomy of suicidal intent (Kapur et al., 2013).

The term “suicidality” will refers to the range of non-fatal suicidal behaviours and cognitions, thus encompassing all self-injurious acts regardless of motivation or intent and thoughts of killing or deliberately injuring oneself. When referring to fatal behaviours the term completed suicide will be applied.

1.2.2. Models of suicidal behaviour

Several models of suicidal behaviour have been specifically tested with individuals who engage in self-harming behaviours. However, the Cry of Pain model (CoP; Williams, 1997) has explicitly been validated for its predictive ability of self-harmful behaviours (Rasmussen et al., 2010), while the Experiential Avoidance Model (EAM; Chapman, Gratz, & Brown, 2006) provides a dynamic representation of self-harm in detail, beyond that of other models. Moreover, these two models show a degree of overlap (detailed in section 1.2.2. (i)). As such, they have been chosen as the theoretical framework relating to self-harmful behaviours throughout this thesis.
The Cry of Pain (CoP) model of suicidal behaviour (Williams, 1997, 2001) successfully combines concepts from the suicide field, notably the theory of Suicide as Escape From Self (Baumeister, 1990) with evidence from the animal literature related to the concepts of Arrested Flight put forth by Gilbert & Allan (1998) regarding the development of depression.

The Cry of Pain model (see Figure 1 for diagram) proposes that suicide is a response to experiencing 3 key factors: defeat, such stress from a loss of status (real or perceived); entrapment, that is a lack of escape from the defeat phenomenon and low rescue potential such as low social support to help or intervene in the situation. The factors are themselves influenced by an individual’s memory deficits and biases leading to hopelessness and suicidal behaviour. Williams (2001) states that self-harm low in suicidal intent represents the early active protest stage in response to defeat and entrapment, while high intent self-harm is a sign of more severe reaction to the defeating and entrapping event which have triggered a ‘conservation-withdrawal’ (Goldney, 1980) behaviour. Self-harm therefore may not come about as a result of suicidal intent; rather it is motivated by a desire to escape unbearable situations. Rasumussen et al. (2010) elaborate that the key component of self-harm is a result of mental anguish and that the motive for the behaviour is secondary. Thus, some self-harming behaviour may not be motivated by suicidal intent, but most are driven by a desire to escape unbearably negative situations.
Williams (1997, 2001) makes a point of explaining the role of emotional experiences in suicidal behaviour. He makes a distinction in the emotional states of suicide attempters and those who have completed suicide. While both attempts and completion are associated with depression, Williams demarcates attempters as having greater levels of anger and irritability compared to non-suicidal psychiatric controls, while completers distinguish themselves by a lack of strong emotions or a sense of apathy. Moreover, Williams argues that suicidal individuals seem to be unable to regulate their emotion and the way in which they experience emotional pain. In certain cases, self-harmful behaviours can provide a mechanism through which an individual can regulate their emotions. Thus, a person’s response to stressors, internal (e.g. painful emotions) or external (e.g. work stress) in origin is important to understand suicidal behaviour.
The CoP model has been empirically supported by testing on self-harm patients admitted for overnight hospitalisation (O'Connor, 2003) and importantly, it has been applied to the study of self-harm (regardless of suicidal intent) on first time and repeat self-harming individuals (Rasmussen et al., 2010). These studies validated the concepts of defeat and entrapment; the mediating role of entrapment between defeat and suicidality and the moderating role of rescue on suicidality.

The Experiential Avoidance Model (EAM, reviewed in Chapman, Gratz, & Brown, 2006) provides an explanatory framework through which self-harming behaviour leads to emotion regulation. Furthermore, it explains how self-harm can become a conditioned maladaptive coping strategy among non-psychotic and cognitively able adults. The focus of this model is on non-suicidal self-injurious behaviour (NSSI). However, as discussed previously, the delineation of different types of self-harm congregates around the issue of intent and is currently a contentious issue. Moreover, our definition of self-harm, that is, self-injury regardless of motivation or intent to die, is all encompassing making this model appropriate to inform our understanding of self-harmful behaviours.

The EAM (see Figure 2) frames self-harm as a negatively reinforcing strategy which provides temporary relief from the experiencing of aversive emotions caused by stressors. This temporary relief from unwanted emotions and experiences is obtained by escape or avoidance, thus regulating affect. Experiences are said to be avoided due to the concurrent negative emotions which they elicit. Following the model, individuals faced with a stressor which elicits a negative emotion, such as anger, shame, sadness or frustration and
who suffer from deficits in affect regulation actively seek to avoid these negative internal experiences (i.e. emotions and related cognitions). Self-harm is used to express in physical terms the internal turmoil felt by the individual, this allows the negative internal experience to become concrete and controllable. Klonsky (2007) provides a clear review of the evidence linking negative affect regulation and self-harm. Emotional relief is hypothesised to occur due to one or a combination of:

(i) Endogenous opioid release resulting in analgesia (Coid, Allolio, & Rees, 1983; Roth, Ostroff, & Hoffman, 1996; Russ, 1992).

(ii) Physical pain distracting from emotional pain (Gottman & Katz, 1989; Gross, 1998).

(iii) Self-punishment used to alleviate stress caused by cognitive dissonance relating to individual’s beliefs about his/herself (e.g. I deserve to be punished, however, I have not been. Therefore, I must punish myself.) (Swann, Hixon, Stein-Seroussi, & Gilbert, 1990).

Self-harm as an experiential avoidant strategy is further negatively reinforced due to the temporary affect regulation and overtime becomes a conditioned response. This regulation strategy is deemed maladaptive not only due to the immediate body damage which it causes, but also because of the paradoxical effect of using avoidant behaviours. The paradoxical effect of emotional avoidant strategies has been reviewed by Abramowitz, Tolin, & Street (2001). This paradoxical effect can be seen by the increased levels of distress brought on by chronic avoidant strategies. This means using self-harm to avoid negative experience will in turn increase distress felt by the individual.
Moreover, Hawton, Zahl, et al. (2003) have shown that engaging in self-harming behaviour greatly increase subsequent risk of suicide attempt and completed suicide.

![Experiential Avoidance Model](image)

**Figure 2 - The Experiential Avoidance Model (adapted from Chapman et al., 2006)**

Research by Gratz (2004) comparing female students who had repeatedly self-harmed to females with no history of self-harm found that females who engaged in self-harm had higher levels of experiential avoidance than matched controls. A similar association was found in male college students showing increased self-harm frequency to be positively correlated with emotional non-acceptance (Gratz & Roemer, 2004). Meanwhile, Chapman (2004) examined the emotional states of participants following an episode of self-harm and found reports of relief as the predominant emotion felt. This is backed by the findings of Rodham & Hawton (2004); in a large community sample of adolescents where motivation for self-harming behaviour was reported as
‘relief from a terrible state of mind’. Psycho-physiological evidence has also been reported by Sachsse, Von Der Heyde & Huether (2002), who using ambulatory monitoring recorded levels of cortisol, episodes of self-harm and self-reported mood of female participants over 86 days. High cortisol levels were associated with strong negative emotions. Moreover, self-harm episodes were immediately followed by a significant drop in cortisol levels. This low level remained for several days. Psycho-physiological evidence of poor distress tolerance has been published by Nock & Mendes (2008) who found higher skin conductance response in adolescents who engaged in self-harm compared to controls when exposed to a distressing task.

1.2.2. (i) Similarities between the Experiential Avoidance and Cry of Pain models

Both the EAM and CoP explain suicidal behaviour. However, the EAM focuses on self-harm (non-suicidal self-injury to be precise), while the Cry of Pain is directed at suicidality’s broad spectrum. Indeed the major difference between these two models is their approach to suicidal intent. The EAM follows the dichotomous perspective which dictates that suicidal intent is either present or is not during self-injury. Having delineated between different types of self-harm as either NSSI or suicide attempt, the EAM focuses on NSSI as a maladaptive negatively reinforced emotion regulation strategy. Moreover, the EAM expresses the dynamic nature of self-harm by including a feedback mechanism to explain the pervasiveness of the behaviour – self-harm being negatively reinforced by the reduction in negative internal experience. As such, the EAM is more explicit about the mechanism through which emotion regulation via self-harm is attained.
The Cry of Pain as described by Williams (1997, 2001), however, assumes suicidal intent to be a spectrum and views self-harm as an early stage reaction prior to the later high intent stage whereby “conservation-withdrawal” (giving up) (Williams, 2001, pp.149) behaviour sets in when the individual has become hopeless. Although these two models differ on their approach to intent, striking similarities between the CoP and EAM do exist.

The models mutually view emotion regulation to be a prominent component of self-harmful behaviours. Moreover, both models expressly focus on a negative starting event. The CoP terms this as a defeat, while the EAM mentions a stressor and its subsequent negative emotional reaction. In both cases, the individual’s perception of the event and their coping abilities moderate the event’s psychological impact.

Moreover, both models frame suicidal behaviour as providing escape from negative experiences with entrapment (CoP) and avoidance (EAM) mediating the relationship between negative event and suicidal behaviour. The EAM states avoidance from negative experience provides temporary relief. The CoP goes further stating that entrapment, an inability to escape the negative event, will result in an individual shutting down and conserving resources (conservation-withdrawal); a sign of hopelessness whereby suicidal behaviour becomes the solution to escape the negative experience. These two accounts differ in the severity of suicidal intent as the CoP describes the process when avoidance is deemed impossible by the individual. The EAM also shows a clear moderating effect of coping skills (distress tolerance, regulation skills) between the stressor and engaging in the avoidant behaviour. Judgements of ‘how stressful’ or ‘how escapable’ and ‘how much affected by social support’
also impact on variables of the CoP. Such judgements are impacted by memory biases and problem solving skills deficit. Additionally, the CoP shows a clear moderating effect of rescue on the relationship between entrapment and hopelessness, which attenuate perception of entrapment felt and thus reduces hopelessness. Both models encompass a mechanism, individual judgement based on individual differences, which delineates the relationship between aversive response and engagement in suicidal behaviour.

Issues of intent aside, the EAM and CoP are congruent in their accounts of self-harm and its role in emotion regulation. It is arguable, therefore, that the EAM is a detailed account of the early stages of suicidality; the self-harming behaviour or “reactance” which precedes the helplessness, as put by Williams (2001). In contrast, the CoP is an overarching linear account of the suicidal process and as such the models may be viewed as complementary.

1.2.3. Nightmares
This thesis focuses on nightmares due to the increasing body of evidence linking the phenomenon to suicidal behaviours (Bernert & Joiner, 2007; McCall & Black, 2013). Moreover, due to the relatively wide array of potential interventions targeting nightmares, ranging from behavioural to pharmacological treatment (Aurora et al., 2010), they appear to be a risk factor amenable to intervention efforts. The following section details the conceptualisation and aetiology of nightmares, and reviews the evidence linking them to suicidal behaviour.
1.2.3. (i) Defining sleep disorders

Sleep disorders can be broadly split into three categories: dyssomnias, parasomnias and sleep disorders associated with medical, neurological or mental illness (American Academy of Sleep Medicine, 2001).

Dyssomnias are disorders characterised by difficulties attaining or remaining in a sleep state or trouble with excessive sleepiness during waking hours. Dyssomnias can be further divided into intrinsic disorders, which are disorders emanating from within the individual’s body (e.g. insomnias and sleep apnea). Extrinsic disorders on the other hand are dependent on environmental cues, hence extrinsic to the individual and often resolved by removing or altering the problematic cue (e.g. alcohol-dependent sleep disorder and inadequate sleep hygiene). Circadian rhythm sleep disorders relate to deregulation of the internal body clock within the normal 24hr per day period. This particular subsection of dyssomnias can be intrinsic; such as neurological irregularities affecting sleep-wake patterns, or extrinsic; such as jetlag. However, their main characteristics remain; the altering of the regular chronobiological pattern.

Parasomnias differ from dyssomnias in that their primary symptomology is increased arousal during sleep and transition of sleep stages and do not impact directly on sleep-wake states. Increases in central nervous system activity characterise parasomnias which consist of arousal disorders (e.g. sleep walking and sleep terrors), disorders impacting on sleep-wake transition (e.g. sleep talking and sleep starts), Rapid Eye Movement (REM) parasomnias (e.g. nightmares and sleep paralysis) and other parasomnias (e.g. sleep bruxism and sleep enuresis). Sleep disorders associated with medical, neurological or
mental illnesses are not primarily sleep disorders, they are sleep disorders which manifest themselves as a major symptom of an illness.

The focus of this thesis, nightmares, are very common parasomnias reported in up to 85% of the population within a year (Levin & Fireman, 2002; Nielsen & Levin, 2007; Nielsen & Levin, 2009). Studies have shown consistently across cultures that 2% to 6% of people report weekly nightmare episodes (Levin & Fireman, 2002; Nielsen & Levin, 2007, 2009). However, this number is believed to be greatly underestimated (Nielsen & Levin, 2007, 2009). This is in great part due to the retrospective nature of nightmare measurement methods used in population surveys (Zadra & Donderi, 2000). The psychometric scales used underestimate nightmare frequency compared to prospective dream logs (Zadra & Donderi, 2000; Lancee, Spoormaker, Peterse, & van den Bout, 2008) due to the attribution of waking events to one particular sleep disturbance over another. For instance, attributing awakening as being due to insomnia rather than nightmares. However, Balgrove & Haywood (2006) make an interesting point when evaluating the awakening criterion which define nightmares, present as an item of frequently used scales (e.g. the Disturbing Dreams and Nightmare Severity Index - DDNSI used by Bernert, Joiner, Cukrowicz, Schmidt, & Krakow (2005) which will be used throughout this thesis). Participants report being able to judge if the emotions associated with the disturbing dream woke them up with increasing certainty, proportional to their judgement in the severity of the disturbing and fearful emotions. Thus, individuals experiencing more intense negative dream affect will be better at indicating if the emotion was the cause of the awakening than individuals experiencing mild negative dream affect.
1.2.3. (ii) Nightmare aetiology and models

Nightmares are REM-related parasomnias comprised of two key characteristics; (i) the dream provokes awaking of the sleeper and (ii) the sleeper has clear recall as to the content of the dream\(^1\). Nightmares are usually long dreams which gradually degenerate with increasingly fearful content, sensations of fear and anxiety or other dysphoric emotions (American Academy of Sleep Medicine, 2001; Nielsen & Levin, 2009).

Nightmares are reported in up to 85% of the population over the course of a year. Studies have shown consistently across cultures that 2% to 6% of people report weekly nightmare episodes. However, this number is believed to be greatly underestimated (Nielsen & Levin, 2007).

Nightmares have been associated with greater psychological disturbances; reduced wakeful psychological functioning such as feeling of fatigue and positively correlated to psychiatric disorders in both adults and children (Hublin, Kaprio, Partinen, & Koskenvuo, 1999). Moreover, the literature has demonstrated clear negative associations between nightmares and emotion regulation (Nielsen & Lara-Carrasco, 2007). In addition, associations between nightmares and hyperarousal (Kramer, Schoen, & Kinney, 1984) and maladaptive coping (Kothe & Pietrowsky, 2001) have been found.

The function of dreaming and the role of nightmares has been the focus of psychological and neurophysiological models such as Fisher, Byrne, Edwards, & Kahn’s (1970) REM sleep desomatisation, Kramer's (1993) mood

\(^1\) Nightmares are distinct different from sleep terrors, parasomnias which causes the sleeper to wake with associated fear and sensations of dread. Nightmares occur during REM sleep as opposed to stage 4 – the stage of the sleep cycle during which sleep terrors occur. Moreover, nightmare content is recalled upon waking. This is in stark contrast to sleep terrors when the sleeper wakes unable to recall the cause of fear.
regulatory hypothesis and the more recent Affective Network Dysfunction (AND) model of Nielsen & Levin (2007, 2009).

The AND model (Nielsen & Levin, 2007, 2009) is a synthesis of the nightmare aetiology literature drawing on empirical evidence from neuroimaging, polysomnographic, behavioural, and psychometric studies. The model puts forth a clear account of nightmare formation rooted in neurophysiology, whereby nightmares are a dysfunction of naturally occurring fear memory extinction provided through normal dreaming. More precisely, the current affective load experienced by an individual is said to dictate the need for the formation of new fear extinction memories. These memories are created during the dream process which dissociate and recombine attributes of fear memories. The memories are then recreated into a new potentially fear extinguishing context. However, nightmares occur due to a failure within this process whereby the recombined memory is consistent to waking-state fear memories akin to phobias or social anxiety.

While the models vary in their explanation of how nightmares arise, the central tenet of these models is that dreams generally have a mood regulatory function via fear extinction or systematic desensitisation, depending on the model of dreaming being used. Research has shown dream affect to be primarily reported as mildly negative by the majority of participants in polysomnographic recording when woken during rapid eye movement (REM) sleep (Hobson, Pace-Schott, & Stickgold, 2003). The sleeper is therefore exposed to this mild negative content over the REM period, with negative affect levels being reduced as a result of the fear extinction or desensitisation. Nightmares, on the other hand, appear to be an abnormality which deregulates
mood and leave the awakened individuals with intense dysphoric feelings such as fear and anxiety. Nightmares’ deregulating effect is supported by a large body of evidence summarised by Cartwright (2010).

1.2.4. Review of evidence linking nightmares to increased suicidality

A multitude of empirical studies have investigated the links between nightmares and suicidality (for an overview see Bernert & Joiner, 2007). The majority of these studies have focused on suicide ideation in clinical populations (Ağargün et al., 1998; Bernert et al., 2005; Chellappa & Araújo, 2007; Krakow et al., 2000; Singareddy & Balon, 2001; Yoshimasu et al., 2006) with nightmares being consistently reported as showing a robust association to suicide ideation. One such study is that of Agargun & Cartwright (2003), who explored the association between REM sleep, suicidal ideation and dream variables (reported quality) in depressed patients by comparing suicide ideating sleepers with control sleepers. They observed that suicide ideating participants achieved REM sleep faster, and had a higher proportion of REM during their sleep cycle than non-suicidal controls. Suicidal ideators also reported negative dream content consistent with reports of nightmares during latter periods of the night and fewer negative dreams during the early stages of the night. The inverse was observed in controls, perhaps indicating easier recall of later dreams influenced suicidal ideation.

While most studies have focused on nightmares in clinical populations, few have actively controlled for the confounding effect of depression. A handful of key studies have sought to control for the effects of depression in order to ascertain if nightmares were independent risk factors for suicidal
ideation. Bernert et al. (2005) was the first study to do so, investigating nightmares and their relation to suicidal ideation in a sample of clinical outpatients. The analysis found a significant association between nightmares and suicidal ideation in females which remained upon controlling for depressive symptoms. Subsequently, Cukrowicz et al. (2006) expanded this research by investigating nightmares and its relationship with suicidal ideation in a student sample. A significant association between nightmares and suicidal ideation was found in females and remained upon controlling for self-reported depressive symptoms. Krakow et al. (2011) repeated these findings in a sample of sleep clinic patients, while Nadorff et al. (2011) investigated nightmares and suicidal ideation in a student population while further controlling for anxiety and post-traumatic stress disorder (PTSD) symptoms in addition to depressive symptoms. Nadorff et al and Krakow et al.’s results supported earlier findings by Bernert et al. and Cukrowicz et al., indicating that the link between nightmares and suicide ideation could be generalised to non-clinical populations. Moreover, by controlling for PTSD symptoms, Nadorff and colleagues established that the nightmare-suicide ideation link extends to idiopathic nightmares and that idiopathic nightmares are independent of PTSD symptomology – itself co-morbid with suicidal behaviour.

A limited number of studies have expanded the literature by looking at suicide attempts and completed suicide. The largest longitudinal population survey (n=36211) focusing on the link between nightmares and suicidality Tanskanen et al. (2001) revealed having nightmares to be a potent predictor of completed suicide at 14 years follow-up. Using self-reported questionnaire data, regression analysis showed members of the general population who
reported occasional nightmares were at a 57% increased risk of suicide. More alarmingly, the risk of suicide for those reporting frequent nightmares jumped 105% higher than those who reported no nightmares. Moreover, Sjöström et al. (2007) studied the occurrence of nightmares in suicide attempters to see if the nightmare could directly predict suicidal behaviour. The study found suicide attempters reported a number of sleep complaints pertaining to trouble attaining and maintaining sleep and early morning waking. However, nightmares were indicative of a five-fold increased risk of suicide. Further research (Sjöström et al., 2009) revealed that persistent nightmares were predictive of repeat suicide attempts when controlling for sex, DSM axis-I diagnosis, depression and anxiety. However, the sleep complaints reported in the previous study, that is troubles attaining or maintaining sleep, were not indicative of repeat attempts. In addition, suicide autopsies examining sleep disturbances in adolescent suicide completers (Goldstein, Bridge, & Brent, 2008) have also shown completers to have higher rates of disturbances (e.g. insomnia and a range of parasomnias) compared to matched controls within their last week of life. Variations in the severity of disturbed sleep symptoms were exhibited by a minority of the sample. However, a higher proportion of this sample was comprised of suicide completers.

1.2.5. Why focus on nightmares?

Due to mounting research and interest in the fields of sleep, death, and suicide Schneidman (1964) disseminated a speculative article proposing a reassessment of the aforementioned concepts. It was proposed that they be reconceptualised as varying degrees of “orientations toward Cessation” (p.96). This new conceptualisation breaks down the penchant of using overt
experiential states when referring to modes of death (homicide, suicide, accidental and natural) and reorganises it towards psychological states (Cessation, Interruption, Continuation and, Termination), more precisely states of consciousness focusing on the individual’s intent towards their own death be it conscious or unconscious.

Cessation is described as the halting of existing and potential future conscious experiences. This concept is exclusively relevant to taking ones’ life as it refers to the final act of introspection by the individual prior to termination. Termination is seen as an end result, that is, the ending of physiological functioning which would classically be referred to as death. Termination does not exclude cessation. In fact, cessation may potentially precede termination by hours or days. Continuation on the other hand is the persistence of experiencing conscious events without breaks or ‘interruptions’; it can be characterised as wakeful everyday existence. If continuation is experiential wakeful consciousness, then interruption is the temporary halting of this experiencing. Loss of consciousness or sleep is interruptive and has been described by Schneidman as ‘temporary cessation’. Following the conceptual paradigm proposed, one’s life becomes a string of fluctuating states of consciousness flowing from continuation to interruption and back until inevitable result of termination. However, the conscious state can end with cessation. Thus, the metaphenomena of sleep and suicide are closely linked in their ability to terminate consciousness and sleep maybe looked at in the terms of temporary respite or temporary death; an escape from the conscious world. For the temporary relief afforded by peaceful sleep to then be brutally negated by a nightmare would therefore reduce one’s ability to obtain said relief via
interruptions and force the individual to remain in the continuation state despite their efforts.

More recently and with the aim of developing prevention and intervention models, general somatic symptoms (Bohman et al., 2012; Medina, Jegannathan, Dahlblom, & Kullgren, 2012) and in particular sleep related somatic symptoms (Tanskanen et al., 2001; Agargun & Cartwright, 2003; Agargun & Beşiroğlu, 2005; Bernert & Joiner, 2007; Goldstein, Bridge, & Brent, 2008; Sjöström et al., 2007, 2009; Nadorff, Nazem, & Fiske, 2011; Susánszky, Hajnal, & Kopp, 2011; Ribeiro et al., 2012) have been deemed increasingly useful predictors of mental illness such as depression, anxiety and suicidal spectrum behaviours.

Studies that have focused on nightmares as a somatic symptom have found them to significantly predict suicide ideation (Bernert & Joiner, 2007; Krakow, Ribeiro, Ulibarri, Krakow, & Joiner, 2011; Nadorff, Nazem, & Fiske, 2011), suicide attempts (Sjöström et al., 2007) and repeat attempts (Sjöström et al., 2009) whilst controlling for depressive symptoms. A number of other studies have investigated sleep related somatic symptoms (Ribeiro et al., 2012; Susánszky et al., 2011) but have not been rigorous in their methodology of assessing the sleep disorder. For instance, Ribeiro and colleagues’ article assess sleep somatic symptoms by using a single item Beck’s depression inventory item regarding sleep difficulties, sleeplessness. As such, issues of reliability and validity of the construct being measured are likely particularly when considering psychometric instruments measuring insomnia and nightmare symptoms both share an item relating awakening, to which a somatic symptoms such as sleeplessness could be attributed to.
Nonetheless, empirical evidence from Ribeiro et al., (2012) indicates that the single item sleeplessness measure to explain greater levels of variance in suicidal ideation than depressive symptoms and hopelessness. Sleeplessness also outperformed depression and hopelessness in predicting suicidal ideation both cross-sectionally and longitudinally. This evidence emanates from a sample of young adult in the military, predominantly males; and as such caution must be taken when generalising to the wider population. However, such evidence is all the more important as it displays that a somatic symptom can be an important predictor. This is all the more relevant in cultures where suicidal behaviour is deemed taboo or not widely discussed whilst help seeking for somatic symptoms such as sleep troubles are more acceptable. Thus, Ribeiro et al.’s work and that of others serve to highlight the importance of sleep related somatic symptoms as a tangible risk of suicidal spectrum behaviours.

Furthermore, Nock, Prinstein, & Sterba, (2009) interviewed individuals who engaged in non-suicidal self-injury about potential alternative strategies used for affect regulation. Much as Schneidman (1964) speculated with regards to interruption, some of the individuals interviewed by Nock and colleagues reported to use sleep as an alternative strategy to self-injury.

With the aforementioned in mind, and evidence from the dreaming and nightmare literature which indicate nightmare to be a dysfunction of mood regulation, nightmares appear to be a potential risk factor for self-harm as they hinder effective mood regulation. Moreover, as increasingly effective treatments are available for nightmares such as Imagery Rehearsal Therapy
(IRT; Barry Krakow & Zadra, 2010), nightmares appear to be a sensitive and specific risk factor amenable to intervention.

1.2.6. Proposed psychological mechanism linking nightmares and suicidality

Krakow et al. (2000) proposed that sleep disturbances fractured their female sexual assault survivors sample’s sleep. This fracturing lead to emotional exhaustion and low energy further burdening the patients’ fragile coping skills. Krakow’s explanation for the effect of sleep disorders follows a diathesis stress model where by the existing suicidal ideation reduces the effect of coping strategies. This reduced effectiveness allows for sleep disturbances to impact on the sufferer further exhausting their coping capacity and creating a vicious circle of increasing suicidality.

Agargun et al.’s (2007) proposed explanation followed similar lines. Namely, that sleep’s function as a mood regulator was not effectively carried out thus augmenting suicidality. The role of sleep as mood regulatory has been supported by REM research by Agargun & Cartwright (2003) suggesting that mood was not regulated during sleep by participants suffering from nightmares, who instead committed affect to long term memory during sleep further depressing them upon waking. Increased negative affect particularly during the early morning, as shown by the negative mood exhibited by major depression patients early in the day compared to later would support this view (Agargun et al., 2007).

In their review of sleep disturbances and suicidality, Bernert & Joiner (2007) provide a more detailed account of the potential mechanisms stating mood regulation to be a primary factor in the association between suicidality
and nightmares. They explain that sleep could provide emotional refuge for those feeling anguish and that frequent disturbances would render this regulation ineffective. This explanatory mechanism had been suggested in previous publication by this research team (Bernert et al., 2005; Cukrowicz et al., 2006) and concurrently to suggestions by Ağargün et al. (1998; Ağargün et al., 2007).

The mechanisms accounting for the relationship between nightmares and increased suicidality by Krakow et al., Agargun et al., and Bernert et al. follow similar explanatory trends, is best summarised in Cukrowicz et al. (2006): nightmares deregulate affect resulting in high levels of negative affectivity upon waking. In turn, the negative affect reduces distress tolerance for stressors and suicidal cues which increase the risk of suicidal behaviours.

This explanation is very descriptive. Beyond findings relating to early higher reported morning negative affect in those suffering from high levels of nightmares established by Antunes-Alves & de Koninck, (2012) there is a lack of supporting evidence for the current flow of cognitive processes, their link to self-harm, and their predictive ability. Importantly, specific definitions of suicidal cues are lacking. Moreover, the correlational data used to support the proposed mechanism does not allow for causality to be established, as such a nightmare could be symptomatic of increased suicidality rather than an independent event which exacerbates suicidal behaviour. As remarked by Nock (2009), the lack of causational evidence with regards to risk factors greatly hinders the creation of effective prevention and intervention programmes.
In addition, much of the empirical evidence from which the proposed mechanism linking nightmares to increased suicidality is derived have not controlled for the effects of depressive symptoms; with the exception of a few key studies (Cukrowicz et al., 2006, Sjostrom et al., 2007, 2009). This is noted in Bernert & Joiner (2007) who call for further research with systematic controls for depressive symptomology. The reasons for increased control of depressive symptoms are two-fold. Firstly, 90% of individuals suffering from depression complain of co-morbid low sleep quality (Hamilton, 1989). Secondly, reviewing the predictive power of psychiatric disorders such as depression in predicting suicide, Powell et al. (2000) indicate psychiatric risk factors lack sensitivity and specificity, that is their accuracy in positively identifying suicidal risk and their ability to demarcate between those at risk and not at risk. Providing supporting evidence for this, Arria et al. (2009) found that in a sample of university students, 6% of first year students indicated suffering from suicidal ideation. However, of this subsample only 40% meet the clinical criteria for depression. Thus, controlling for the variance in suicidality explained by depressive symptoms is of great importance as the latter occurs co-morbidly with sleep disturbances and lacks the sensitivity and specificity of a risk factor for accurate prevention models. Clearly establishing nightmares as an independent risk factor for suicidality and empirically validating the mechanism via which this relationship occurs would be highly beneficial as research shows increasing evidence that nightmares are amenable to intervention (Pigeon & Caine, 2010), such as IRT which reduces nightmare frequency and intensity (Karkow & Zarda, 2010).
A focus on suicidal ideation as opposed to behaviour is also apparent within the literature. Only a few studies have focused on completed, attempted or repeated suicide attempts (Tanskanen et al., 2001; Sjostrom et al., 2007, 2009) and the majority of studies describing potential mechanisms measure suicidal ideation specifically. It is currently not clear whether the psychological mechanism as previously summarised can be successfully applied to self-harm regardless of suicidal intent.

Thus, this thesis explores if the effect observed between nightmares and suicidal ideation and suicide attempts applies to self-harm regardless of intent in view of uncovering the psychological mechanism linking nightmares to self-harm. Moreover, while the aforementioned explanation follows theoretical and empirical findings from the nightmare literature, it overlooks existing models explaining suicidal behaviour such as the Cry of Pain and Experiential Avoidance Model which specifically focus on behaviour rather than suicidal thinking. For instance, the mechanism proposed by Cukrowicz et al. (2006) has omitted key variables, which have empirically been validated and explain large amounts of variance in suicidal spectrum behaviours, such as defeat, entrapment, avoidance and hopelessness. The relationship between nightmares and these variables in relation to suicidal spectrum behaviour, specifically self-harm regardless of intent, will be explored throughout this thesis.
1.3. Ethical issues

The studies in the present thesis have a number of potential ethical implications. The effect of participating in suicide and self-harm research and its potential impacts on mood is deemed key. Empirical exploration of the potential iatrogenic effects of suicide screening on teenagers (Gould et al., 2005) has revealed participants do not suffer from increased distress compared to controls participants. Furthermore, the study indicates participation to be beneficial for those at greatest risk, lowering suicidal ideation levels in participants with a history of suicide attempt. More extensive research protocol where participants are extensively asked about psychiatric conditions and suicidality have also been found to have no negative effects and some benefits in reducing suicidal ideation (Mathias et al., 2012; Smith, Poindexter, & Cukrowicz, 2010).

Anonymity and confidentiality of all participants was respected, with all identifiers being removed from the data set prior to analysis. Data was available only to the researcher and supervisors and kept on password protected computers and locked filing cabinets. Computers used for physiological data acquisition remained offline as an additional precaution.

In order to verify all ethical concerns were appropriately met, all studies were subjected to review by the University of Nottingham’s School of Psychology internal ethics committee. Moreover, all study debriefings provided additional information to all participant concerning topics covered and came with positive mood induction.
1.4. Summary of theoretical themes and aims of the thesis

Mounting evidence indicates a link between sleep disturbances and increased risk for suicidal ideation and behaviour (Bernert & Joiner, 2007; McCall & Black, 2013). In particular, nightmares have been shown to be a significant risk factor after controlling for the effects of depressive symptoms, PTSD and state anxiety (Tanskanen et al., 2001; Bernert et al., 2005; Cukrowicz et al., 2006; Sjostrom et al., 2007, 2009; Krakow et al., 2011; Nadorff et al., 2011). However, hypotheses concerning a psychological mechanism linking nightmares to increased suicidal risks have remained untested.

Empirical studies have clearly demonstrated a link between nightmares, suicidal ideation, suicide attempts and completed suicide. However, it is currently unclear if this link can be extended to self-harm regardless of suicidal intent or if nightmares are associated solely to behaviours and cognitions where suicidal intent is present. Nevertheless, empirical evidence linking nightmares to affect deregulation (Agargun & Catwright, 2003; Antunes-Alves & de Koninck, 2012) and, research linking self-harmful behaviours to affect regulation (Klonsky, 2009) would suggest a potential association; as both dreaming and self-harm regardless of intent share similar emotion regulating effects. Conversely, nightmares (dysfunction of normal dreaming), have been demonstrated to deregulate affect (Nielsen & Levin, 2007, 2009). It could be said therefore, that nightmare-less sleep should allow for affect regulation while nightmares would deregulate affect increasing need to regulate via self-harm and increase suicidality.
This must be cautiously assessed as the current literature assumes nightmares to increase suicidality yet directional causality has not been explicitly tested. Additionally, all current explanations regarding a mechanism linking nightmares to increased suicidality have omitted important variables from existing suicidal behaviour models such as defeat, entrapment and, avoidance.

This thesis aims to cover the discussed gap in knowledge by:

(i) Exploring the links between nightmares and self-harm regardless of intent and, to test if nightmares can distinguish between those currently engaging in self-harm and those with a history of self-harm. This investigation is reported in Chapter 2.

(ii) Exploring predictive directional relationship between nightmare and self-injurious thoughts and behaviours using a prospective dream logs. This is detailed in Chapter 3.

(iii) Exploring nightmare content and uncover potential themes which may differentiate those at increased vulnerability for self-harm. This is reported in Chapter 4.

(iv) Modeling a potential mechanism between nightmares and self-harm engagement reflective of the current findings in the literature and this thesis. This is explored in Chapter 5.

(v) Empirically testing the proposed mechanism linking nightmares to self-harm using behavioural and psycho-physiological methods is reported in Chapters 6.
Chapter 2: An Investigation of the Relationship between Nightmares, Self-harm Regardless of Suicidal Intent, Negative Affect, and the Cry of Pain Model.

2.1. Introduction

Longitudinal studies have shown nightmares to predict completed suicide at 14 year follow up (Tanskanen et al., 2001). Moreover, cross-sectional studies have demonstrated a prevalence of nightmares when interviewing suicide attempters about sleeping issues (Sjöström, Waern, & Hetta, 2007). Further investigation by Sjöström, Hetta, & Waern (2009) found nightmares to be associated with repeat suicide attempts, with participants reporting frequent nightmares being 3.15 times more likely to make a repeat attempt within the next two years. These findings remained after controlling for self-reported depression, anxiety and DSM-IV axis 1 diagnosis. By controlling for depressive symptoms and other psychiatric factors, research has shown nightmares to be a unique and independent suicide risk factor making nightmares an unambiguous target for clinical intervention such as Imagery Rehearsal Therapy (Krakow & Zadra, 2010).

Studies linking suicidal behavior and nightmares have so far limited their scope to suicide attempts (Sjöström et al., 2007) or repeat attempts (Sjöström et al., 2009). The findings of these studies, by the nature of the population being investigated and defined behaviours being measured, have implied a link between nightmares and suicidal intent. As frequent ambivalence or uncertainty regarding intent is expressed by patients presenting to hospitals following self-harm is reported (Skegg, 2005), the categorizing of self-harm, based on intent as either suicidal or non-suicidal, is a contentious subject. The
practical implications of such delineation have been criticised due to the difficulty of measuring suicidal intent (De Leo et al., 2004). Thus, obtaining a description of the behaviour first and secondly clarifying issues of intent mirroring the approach of clinicians appears to be a logical approach (Skegg, 2005). However, to the authors’ knowledge, no studies have yet investigated the links between nightmares and self-harming behaviour regardless of suicidal intent. Therefore, the present study will investigate this relationship to clarify if the association of nightmares to suicidal spectrum behaviours is exclusive to behaviours of clear suicidal intent. Self-harm regardless of intent, henceforth referred to simply as self-harm, is operationalised following Hawton and colleague’s (2011) as intentional self-injury or self-poisoning, regardless of motivation or suicidal intent and with a non-fatal outcome (Hawton et al., 2011).

Insomnia and its links to suicidality have also been examined by a multitude of studies (for an overview see McCall et al., 2010). This is perhaps not surprising as insomnia shows high co-morbidity with depression (Hamilton, 1989), an important risk factor for suicide. However, the majority of studies cited by McCall et al. focus on in-patients suffering from depression or do not control for the effects of depression. Study that do control for depressive symptoms have found insomnia to be unable to significantly predict suicidal ideation (Bernert et al., 2005; Cukrowicz et al., 2006). Additionally, insomnia shares a key symptom of nightmares; that of awakening during sleep. Thus, further investigation is required to clarify if insomnia and nightmares should be taken as independent risk factor, particularly when controlling for depressive symptoms.
Unlike insomnia, the relationship between nightmares and suicidality (ideation, attempts and completed suicide) has been consistently supported by the literature. However, the mechanism explaining the link between nightmares and suicidal behaviours remains unclear. Bernert & Joiner (2007) have reviewed the literature and proposed that poor overnight affect regulation and increased negative affect after nightmares contribute to increased suicidality. The association between nightmares and negative affect is supported by the dream and nightmare literature (Agargun & Cartwright, 2003; Nielsen & Levin, 2007, 2009; Spoormaker, 2008). Moreover, Nock, Prinstein, & Sterba, (2009) has shown individuals who engage in self-harm (specifically without intent) use sleep as an alternative affect regulation strategy. It would thus be reasonable to propose that self-harming individuals who seek emotion regulation via sleep, yet suffer from nightmares which increase negative affect, are at increased risk of engaging in self-harm to temporarily improve mood. However, testing of the mechanism reviewed by Bernert & Joiner, (2007) whereby high nightmare participants should have elevated negative affect, now requires investigation whilst controlling for the effects of depressive symptoms.

The literature which has focused on the nightmare-to-suicidality relationship has largely omitted existing suicidal behaviour models when informing research and potential models of underlying mechanisms (see Chapter 1 for an overview of literature). The Cry of Pain model (Williams, 1997, 2001) has been empirically validated and its variables; defeat, entrapment and low rescue have been shown to predict suicidal behaviour (O’Connor, 2003). Moreover, the Cry of Pain model is highly pertinent as it
has also been validated with individuals who self-harm (Rasmussen et al., 2010). However, no research, to the author’s knowledge, has explored the potential relationship between Cry of Pain variables and nightmares in those who self-harm. When describing a potential mechanism linking nightmares-to-suicidality, Cukrowicz et al. (2006) suggest that individuals suffering from nightmares become more sensitive to suicidal cues. Both entrapment and defeat are key psychological factors which could be construed as cues to suicidal behaviour. Moreover, sleep as temporary cessation or temporary escape from consciousness (Schneidman, 1964) described in Chapter 1 appears to be thematically linked to the concepts of entrapment. Thus, the role Cry of Pain variables may play in a potential mechanism linking nightmares-to-suicidality requires further exploration.

2.1.1. Aims & hypotheses

This study’s primary aim is to extend the findings of Sjöström et al. (2009, 2007) who revealed nightmares to predict suicide attempts, by investigating the association between nightmares and self-harm regardless of suicidal intent.

Moreover, the mechanism linking nightmares and suicidal ideation described by Bernert & Joiner (2007) suggests that elevated negative affect is a key component of the model. However, this has not been explicitly tested in self-harming participants. Therefore, this study will explore if levels of negative affect differ between participants with clinically significant levels\(^2\) of

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\(^2\) Assessed using the Disturbing Dreams and Nightmare Severity Index, a psychometric tool measuring nightmare severity with validated cut-off scores indicative of clinical severity (Krakow et al., 2006).
nightmares (high nightmare group) compared to participants with subclinical levels of nightmares (low nightmares group).

Additional exploration of potential links between nightmares, defeat and entrapment (Cry of Pain variables; Williams, 1997, 2001) will also be undertaken by testing if participants in the high nightmare group differ from participants in the low nightmare group on reported levels of defeat and entrapment.

The present study’s hypotheses are as follows:

(i) Nightmares will be predictive of self-harm after controlling for the effects of depressive symptoms and insomnia.

(ii) Participants in the high nightmares group will report elevated negative affect compared to those in the low nightmare group. This effect will remain after controlling for the effects of depressive symptoms.

(iii) Participants in the high nightmare group will report elevated defeat and entrapment compared to low nightmare group participants. This effect will remain after controlling for the effects of depressive symptoms.
2.2. Methodology

2.2.1. Design and procedure

A cross-sectional study with control group design was implemented. Data was collected by means of an online survey created using software integrated into the web survey site www.surveymonkey.net. Participants who completed the online survey (and provided a contact email address) were entered into a £25 lottery. Participants could access the questionnaire by following a web link circulated in the recruitment e-mail or through Facebook.

The questionnaire was advertised to participants as “a survey looking into your sleep trouble, mood and behaviours affecting your well-being, namely self-harm”. The design of the questionnaire and the order of the scale followed recommendations for internet based surveys by Dillman, Smyth, & Christian (2008). It was thus decided that the order of the measures would follow the general order advertised; with the survey following a multiple page design where by each scale would be fitted onto a single page. Furthermore, as recommended by Dillman et al. the most engaging and relevant questions to the wider population (sleep trouble, i.e. nightmares) were presented first to encourage continued participation. The most sensitive measure (self-harm) was presented at the end of the questionnaire. Such a layout is argued to give participants more time to engage in the questionnaire; increase the likelihood of continued participation after having already answered the majority of the survey and avoids interrupting the flow of the questionnaire with potentially shocking questions.
Montag & Reuter (2008) have shown that speed in answering incentivised online questionnaires does not affect the scales reliability with Cronbach’s alpha remaining stable. The response format for each item on the survey was therefore chosen to lower survey completion time and avoided high repetition of instructions. Questions with similar instructions were grouped together. To increase participants’ attention and obtain more reliable answers, selectable responses were randomised for each participant, for example, the order of the 4 potential responses on an item from the Beck Depression Inventory would appear in a random order.

Ethical approval for this study was sought and granted by the University Of Nottingham School Of Psychology Ethics Committee prior to data collection. The survey was piloted on a small sample derived from postgraduate students from the School of Psychology at the University of Nottingham prior to full deployment to verify layout and proof read instruction texts as well as the checking the functionality of the survey’s “exit from questionnaire”. This link allowed participant to withdraw from the study.

Participants were required to complete the survey in one sitting and could take as long as they wanted to answer questions. However, they were instructed not to over think the answers and give their initial responses. The survey consisted of 12 individual pages. Participants were first given instructions and an electronic consent form. The Insomnia Severity Index (ISI; Morin, 1993), the Disturbing Dream and Nightmare Severity Index (DDNSI; Krakow, 2006) Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988), the Entrapment and Defeat scale (Gilbert & Allan, 1998), and the
Beck’s Depression Inventory II (BDI-II; Beck, Steer & Brown, 1996) followed in the given order.

Following the BDI, an instruction page appeared reminding participants that their answers would be anonymous and the importance of their honest answers for the purposes of the study. These instructions were followed by the modified Deliberate Self-harm Inventory (DSHI; Gratz, 2001).

Upon submission of their responses, participants were immediately presented with debriefing information which included positive mood induction and contact numbers for support services. Closing the survey redirected participants to the University of Nottingham’s Personality Social Psychology and Health research group website where they could obtain further information on the group’s research activities and information on the researchers responsible for the study. Piloting of the survey revealed the average completion time for the survey to be approximately 20 mins.

2.2.2 Participants

Participants were recruited through convenience sampling. The questionnaire was advertised online (Facebook – University of Nottingham group pages) and by e-mail throughout the University of Nottingham schools and departments. The study was also advertised via email to the Personality, Social Psychology and Health (PSPH) research group participant pool.

A total of 708 participants attempted the questionnaire. Participants were excluded from the analysis due to improper completion (i.e. they did not attempt all question sets) or omitted demographic information (Age, Gender). Participants who had attempted all question set, yet who had small amounts of
missing data (no more than 1 incomplete item per scale) were included. Means were calculated and listwise deletion was used to remove participants with missing mean scores. One hundred and sixty-eight participants withdrew or did not meet inclusion criteria. However, 540 participants aged 18-65 (M= 24.2 years old, SD= 7.9) completed the questionnaire and thus were included in the analysis. The sample was composed of 139 males (M= 25.1 years old, SD= 8.9) and 401 females (M= 23.8 years old, SD= 7.6).

2.2.3. Measurements

The survey was composed of seven scales measuring the key constructs of interest for this study. Additional demographic questions were included at the end of the survey. Scales and the rationale for their selection are detailed below:

The *Insomnia Severity Index* (ISI; Morin, 1993) is a 7 items scale measuring participants’ subjective symptoms and the perceived impact of insomnia on their daily functioning and quality of life. It was selected due to its high validity and reliability (Bastien, Vallières, & Morin, 2001). The scale is composed of 7 items scored on a 0 to 4 scale. The scale has been designed for scores of 0–7 to represent no clinically significant insomnia, a score of 8–14 demonstrating sub-clinical threshold insomnia and 15–21 to show clinical level insomnia of moderate severity. Score of 22-28 are indicative of severe levels of clinical insomnia. The Cronbach’s alpha of the ISI for this sample was α= .87. See Appendix A for a copy of the ISI.

The *Disturbing Dream and Nightmare Severity Index* (DDNSI; Krakow, 2006), chosen due to its brevity and ability to predict clinically salient
nightmare complaints, assesses frequency and severity of participants’ disturbing dreams and nightmares. It is a modified version of the Nightmare Frequency Questionnaire (Krakow et al., 2002). The scale is composed of 7 items. Scores on this scale range from 0-37. Items measure frequency of nights of nightmare per week (0-7), the number of nightmares experience per week (0-14), frequency of nightmare related awakening (0= never to 4= always), the perceived severity of the problem (0= no problem to 6= very severe) and the experienced intensity of the nightmare (0 = not intense to 6 = extremely severe intensity). Scores of 11 and above are indicative of clinical levels of disturbing dreams and nightmares (Krakow et al., 2002). This cut-off was selected to delineate between high nightmare participants and controls. The Cronbach’s alpha of the DDNSI for this sample was α= .86. See Appendix B for a copy of the DDNSI.

The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) measures state or trait predisposition to positive and negative affect depending on time instructions given to participants. This scale was selected to measure participants’ levels of negative affect. This scale was selected as it has been validated and shows high internal consistency when compared to similar affectivity measures such as the State-trait anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970). Furthermore, it is by comparison brief and easily self-administered, thus was chosen to explore if nightmare sufferers do indeed suffer from increased negative affect as suggested by Bernert & Joiner (2007). The 20 item self-report measure consists of two 10 item subscales – Positive affect (PA) and Negative affect (NA). Items are positive and negative adjectives for PA and NA subscales
respectively and are rated on 5 point Likert like scale ranging from 1 to 5 with higher scores representing participants’ increased identification with the item (‘not at all’, ‘a little’, ‘moderately’, ‘quite a bit’ and ‘extremely’). The two subscales are presented together with the items in a set random order. PA and NA scores are calculated by adding the value rated for each individual item and thus range from 10 to 50 with higher score indicating higher levels of positive or negative affectivity. The time instructions given in this study were given as “Please indicate to what extent you have felt this way during the PAST 2 WEEKS” to reflect the time instructions of other measure within the survey (e.g. BDI, ISI). The Cronbach’s alpha of the PA subscale for this sample was $\alpha= .91$, for the NA subscale $\alpha= .87$. See Appendix C for a copy of the PANAS.

The Entrapment Scale (Gilbert & Allan, 1998), which assesses participants levels of internal (e.g. due to perception of self) and external (e.g. due to perception of life events/situations) entrapment, was selected as it has been validated in both student and depressed samples (Gilbert & Allan, 1998) and used in a study testing the Cry of Pain model of suicidal behaviour (Williams, 1997) on individuals engaging in first time and repeat self-harm (Rasmussen et al., 2010). The 16 items are scored on a 5 point Likert like scale ranging from 0 to 4 (‘not at all like me’ to ‘extremely like me’). Higher scores indicate a greater degree of perceived entrapment. The Cronbach’s alpha for this sample was $\alpha= .96$. See Appendix D for a copy of the Entrapment Scale.

The Defeat Scale (Gilbert & Allan, 1998) assesses feelings of defeat and perceived loss of status. As with the entrapment scale it was selected as it has been validated in both student and depressed samples (Gilbert & Allan, 1998) and used with individuals engaging in first time and repeat self-harm
(Rasmussen et al., 2010). This self-report scale is composed of 16 items scored on a 5 point Likert like scale ranging from 0 to 4 (‘never’ to ‘always/all the time’) with higher ratings being indicative of greater feelings of defeat. Three items (2, 4, and 9) on the scale are reverse scoring items where statements are indicative of success rather than defeat. The Cronbach’s alpha of the defeat scale for this sample was $\alpha = .95$. See Appendix E for a copy of the Defeat Scale.

The *Beck Depression Inventory-II* (BDI-II; Beck, Steer, & Brown, 1996) assesses the presence and severity of depressive symptoms over the previous two weeks to be used as a covariate in our analyses. The BDI-II was selected due to its good validation and high correlation with other depression assessment tools such as the Hamilton depression rating scale (see Beck & Steer, 1987, for a review of BDI validation). The scale is comprised of 21 items scored 0-3. Higher scores are indicative of greater levels of depressive symptoms. The Cronbach’s alpha of the BDI for this sample was $\alpha = .90$. See Appendix F for a copy of the BDI-II.

The *Deliberate Self-Harm Inventory* (DSHI; Gratz, 2001) assesses a range of clinically based self-harm behaviours corroborated by clinical observations (Gratz, 2001) via 17 self-reported items. Each item categorically assesses a particular self-harm behaviour on a yes/no basis. An additional 5 items for each of the behaviours are usually asked as follow-up if participants answer “yes” to one of the 17 behaviours. These items focus on intensity, frequency, time of first onset and total duration of engaging in the behaviour. The DSHI was selected due to its good validation with multiple populations,
showing good test-retest reliability (Gratz, 2001). The Cronbach’s alpha of the DSHI for this sample was $\alpha = .84$.

The DSHI explicitly measures acts of self-harm without conscious intent to die, i.e. non-suicidal self-injury (NSSI). There is debate on whether suicidal intent in self-harm should be measured categorically or on a continuum due to the high levels of ambivalence and varying motives reported by self-harming individuals (McAuliffe, Arensman, Keeley, Corcoran, & Fitzgerald, 2007). For an overview please refer to section 1.2.1. (i). As such, the instructions given to participants were deliberately altered to remove all mentions of suicidal intent, reflecting the aim of this research to test links between nightmares and self-harm regardless of intent to die. For the purpose of brevity in completing the questionnaire, the follow up questions were listed below the 17 items checklist and asked participants who reported having engaged in self-harm to answer in relation to their most recent behaviour. The DSHI is presently used to categorise participants as having a history of self-harm or no self-harm. See Appendix G for a copy of the DSHI (modified).

2.2.5. Data analysis procedure

Initial data screening indicated scores for insomnia, nightmares, negative affect, entrapment, defeat, and depressive symptoms were positively skewed. Insomnia, nightmares, entrapment and depressive symptoms were normalised using square root transformation. Logarithm transformation was used for negative affect and defeat. The data transformations used followed the recommendations of Tabachnick & Fidell (2001) for regression analysis.
To test the associations between nightmares and self-harm, a multivariate hierarchical logistic regression analysis was performed. This regression examined the extent to which nightmare scores could predict self-harm regardless of intent (self-harm/no self-harm) whilst controlling for depressive symptoms and insomnia symptoms. Depressive symptom score and insomnia scores were used at step 1 and nightmares at step 2.

To test if participants in the high nightmare group (DDNSI ≥11) reported greater levels of negative affect than control participants (DDNSI ≤10), ANCOVA was used with nightmare grouping being used as the between group factor, depressive symptoms were entered as a covariate.

To test if participants in the high nightmare group reported higher levels of defeat and entrapment than controls, ANCOVAs were performed with nightmare grouping being used as our fixed factor, and depressive symptoms as a covariate. The dependent variables were defeat followed by entrapment scores.
2.3. Results

2.3.1. Descriptive statistics

Means and standard deviations for our sample and transformed data, and z-score for skew and kurtosis are reported in Table 1. One-way ANOVA $F$ ratio and $p$ values are also given for groups differences. All scale alpha coefficients were acceptable. Insomnia, nightmares, negative affect, entrapment, defeat, and depressive symptoms were positively skewed and normalised via transformation. Rescue was negatively skewed and normalised by transformation. Self-harm was dichotomised into self-harm and no self-harm (over the lifetime).

There were no significant gender differences on age and rescue levels between groups. However, nightmare grouping participants reported significantly higher levels of nightmares, insomnia symptoms, depressive symptoms, defeat, entrapment and negative affect than controls. Pearson’s chi-square was used to check for group differences on dichotomous variables. A 2x2 Chi-square test was used to assess the relationship between nightmare groups (clinical levels/sub-threshold) and self-harm groups (yes/no). There was a significant association between self-harm and reports of nightmares indicative of clinical levels ($\chi^2 (1) = 11.68, p<.001$). Odds ratio indicate that participants suffering from clinically significant levels of nightmares are 2.08 times (1.36 to 3.18, 95% C.I.) more likely to have engaged in self-harm at one point in their lives than participants in the control group. A second 2x2 Chi-square test was used to assess the relationship between nightmare groups and

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3 ANOVA values are based on transformed data. ANOVA was re-run using untransformed data; however no significant changes in $p$-values were detected.
gender (male/female). There was a significant association between gender and reports of nightmares indicative of clinical levels ($\chi^2 (1) = 8.74$, $p=.003$), showing female to be at greater risk of belonging to the nightmare group.
Table 1 - Descriptive statistics for whole sample and split by nightmare grouping

<table>
<thead>
<tr>
<th></th>
<th>Total sample (n=540)</th>
<th>Nightmare (n=109)</th>
<th>Control (n=431)</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Zskew</td>
<td>Zkurt</td>
<td>M-Trsf</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Age</td>
<td>24.16 (7.92)</td>
<td>21.62</td>
<td>25.32</td>
<td>-</td>
<td>23.34 (6.13)</td>
</tr>
<tr>
<td>Gender (females)</td>
<td>401 (74.3%)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>93 (85.3%)</td>
</tr>
<tr>
<td>Nightmares</td>
<td>6.61 (5.79)</td>
<td>12.31</td>
<td>8.98</td>
<td>2.26</td>
<td>15.84 (4.69)</td>
</tr>
<tr>
<td>Insomnia</td>
<td>8.58 (5.49)</td>
<td>6.25</td>
<td>-0.07</td>
<td>2.74</td>
<td>12.59 (3.35)</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>23.18 (8.00)</td>
<td>5.88</td>
<td>-0.53</td>
<td>1.34</td>
<td>28.36 (7.77)</td>
</tr>
<tr>
<td>Defeat</td>
<td>21.83 (12.47)</td>
<td>9.25</td>
<td>2.3</td>
<td>1.27</td>
<td>28.67 (13.38)</td>
</tr>
<tr>
<td>Entrapment</td>
<td>15.03 (15.18)</td>
<td>10.13</td>
<td>-0.04</td>
<td>3.26</td>
<td>22.26 (17.15)</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>14.57 (10.61)</td>
<td>6.75</td>
<td>-0.72</td>
<td>3.5</td>
<td>20.98 (13.38)</td>
</tr>
<tr>
<td>N of self-harming</td>
<td>215 (39.8%)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>59 (54.1%)</td>
</tr>
</tbody>
</table>
2.3.2 Do nightmares predict self-harm beyond the effects of depressive symptoms and insomnia?

A hierarchical logistic regression analyses was used to examine to what extent nightmare scores could predict self-harm regardless of intent (self-harm/no self-harm) whilst controlling for depressive symptoms and insomnia symptoms.

The model (Table 2) significantly predicted participants’ self-harming categorization, $\chi^2 (3) = 72.94, p<.001$, Cox & Snell $R^2 = .13$. Depressive symptoms significantly predicted self-harm (Wald $\chi^2 = 32.39, p<.001$) as did nightmares (Wald $\chi^2 = 6.24, p = .013$), indicating nightmares to be predictive of participants having engaged in self-harm regardless of suicidal intent over their lifetime beyond the variance explained by depressive symptoms. However, insomnia (Wald $\chi^2 = .30, p > .05$) did not significantly predict self-harm within this model.

Table 2 - Logistic regression of nightmares predicting self-harm whilst controlling for the effects of depressive symptoms

<table>
<thead>
<tr>
<th></th>
<th>B (S.E.)</th>
<th>Wald X2</th>
<th>95% C.I. for exp b</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-2.46 (.33)</td>
<td>57.45</td>
<td>0.09</td>
<td></td>
</tr>
<tr>
<td>Depressive Symptoms</td>
<td>.48 (.08)</td>
<td>32.38</td>
<td>1.37</td>
<td>1.9</td>
</tr>
<tr>
<td>Insomnia</td>
<td>-.06 (.15)</td>
<td>0.3</td>
<td>0.75</td>
<td>0.94</td>
</tr>
<tr>
<td>Nightmares</td>
<td>.22 (.09)</td>
<td>6.24</td>
<td>1.05</td>
<td>1.24</td>
</tr>
</tbody>
</table>

Cox & Snell $R^2 = .13$, Nagelkerke $R^2 = .17$
2.3.3. *Does the high nightmare group report higher levels of negative affect than controls?*

To test if participants in the high nightmare group (DDNSI ≥11) report greater levels of negative affect than control participants (DDNSI ≤10), ANCOVA was performed while controlling for depressive symptoms and self-harm history.

The ANCOVA revealed a significant effects of nightmare grouping on negative affect beyond the effect of depressive symptoms\(^4\), \(F(1, 537) = 20.95, p < .001, \eta_p^2 = .038\). Participants in the high nightmare group reported significantly higher levels of negative affect than controls.

2.3.4. *Does the high nightmare group report higher levels of defeat and entrapment than controls?*

To test if participants in the high nightmare group reported higher levels of defeat and entrapment than controls, two ANCOVAs were performed.

The first ANCOVA revealed a significant effect of nightmare grouping on defeat beyond the effect of depressive symptoms\(^5\), \(F(1, 537) = 4.03, p = .045, \eta_p^2 = .007\). Thus nightmare participants suffered from higher levels of defeat than controls.

A second ANCOVA was applied to test for group differences on entrapment after controlling for the effects of depressive symptoms. The

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\(^4\) ANCOVA was repeated for non-transformed data. This revealed a difference in partial eta squared between transformed and non-transformed data analysis of .002. Non-transformed partial eta squared was \(\eta_p^2 = .036, (F(1, 537) = 20.01, p < .001)\)

\(^5\) ANCOVA was repeated for non-transformed defeat data. This revealed a difference in significance level and partial eta squared. Non transformed rescue data did not reveal a significant difference between nightmare and control groups (\(F(1, 537) = 1.80, p = .181\)), \(\eta_p^2 = .003\)
ANCOVA did not reveal a significant effects of nightmare grouping on entrapment beyond the effect of depressive symptoms\(^6\), \(F(1, 537) = .249, p > .05, \eta^2_p = .000\). Thus, indicating there to be a no significant difference on feelings of entrapment between nightmare and control group participants.

\(^6\) ANCOVA was repeated for non-transformed entrapment data. Significance level was not affected, \(F(1, 537) = .01, p > .05\), \(\eta^2_p = .000\)
2.4. Discussion

2.4.1. Do nightmares predict self-harm?

We predicted that nightmares would be predictive of participants who reported having engaged in self-harm after controlling for depressive symptoms and insomnia. Regression analysis showed nightmares did significantly predict self-harm history after controlling for the predictive ability of depressive symptoms and the effects of insomnia thus supporting our research hypothesis. This is consonant with other studies in the literature linking nightmares to increased risk for suicide attempts (Sjöström et al., 2007, 2009). However, importantly the present study specifically included all self-harm regardless of intent. Thus, our analysis shows that nightmares are linked to self-harm regardless of suicidal intent.

Bernert & Joiner (2005) and Cukrowicz et al. (2006) found insomnia to no longer significantly predict suicidal ideation when controlling for depressive symptoms. Our results show a similar effect as insomnia did not significantly predict self-harmful behaviours. This would indicate that rather than self-harm being associated to an inability to achieve sleep or shortened sleep duration which characterises insomnia; it is the dysfunction in emotion regulation which characterises nightmares (Nielsen & Levin, 2007, 2009) that is of importance.

Our findings relating to insomnia show that it could no longer predict self-harm history when controlling for depressive symptoms, much like Bernert & Joiner (2005) and Cukrowicz et al. (2006). Due to mixed findings in the literature, a recent meta-analysis by Pigeon, Pinquart, & Conner (2012)
found that depressive symptoms did not mediate the association between insomnia and suicidal thoughts and behaviours. Thus, our findings are contradictory to this meta-analysis. However, this may be due to our research focussing on self-harm regardless of suicidal intent, while the meta-analysis focussed upon cognitions and behaviours with suicidal intent. Moreover, there is possibility for confusion and the misattribution of awakening between nightmares and insomnia if these concepts are not clearly defined and adequately measured. For instance, Ribeiro et al. (2012) found that sleep disturbances can outperform hopelessness in predicting suicidal ideation. However, this study measures sleep disturbances with a single item from the BDI-II. The present study measured both nightmares and insomnia using psychometrics of high validity and reliability, ensuring participants’ fully understood both constructs of nightmares and insomnia, thus, minimising misattribution of awakening.

One must stress however, that these findings are for self-harm within the lifetime and not recent episodes of self-harm (≤ 1 month). Of the 215 participants reporting a history of self-harm, only 36 had actively engaged in self-harming behaviours up to one month before study participation. The ability of nightmares to make a predictive distinction between individuals who are engaging in or have recently engaged in self-harm from participants with a lifetime history of self-harm would make nightmares a valuable variable to consider in risk assessment tools. Further research with a larger sample is required to obtain the necessary statistical power to test this.

While further research is needed to elucidate causality, our results clearly show an association between nightmares and self-harm nonetheless. This
indicates that self-harming individuals suffer from deficits in affect regulation throughout the diurnal cycle and not just during their waking life. Previous findings from (Nock et al., 2009) who found participants who engaged in self-harm to use sleep as an alternative affect regulation strategy, in combination with the present results have implications for treatment of self-harming individuals. Nightmare reducing treatment (e.g. IRT, Krakow & Zadra, 2010) may help in increasing affect regulation during sleep. If nightmares were to exhibit a causal effect then a reduction in nightmare levels would theoretically reduce the need to regulate affect through self-harm. Alternatively, if nightmares are symptomatic of self-harm then at the very least, nightmare reduction treatment would enable those who use sleep as an alternative emotion regulation strategy to do so more effectively, hopefully improving quality of life.

2.4.2. Do individuals with high nightmare levels have elevated negative affect?

The between groups analysis supported our research hypothesis that predicted negative affect to be greater in participants suffering from elevated nightmare scores. Moreover, these findings remained after controlling for the effect of depressive symptoms indicating nightmares to be independently related to increased negative affect.

This would consequently support the overall premise of this thesis, that nightmares are associated with self-harm and that nightmares sufferers have increased levels of negative affectivity above and beyond levels brought about by depressive symptoms. However, further research is needed to ascertain if, as proposed by Cukrowicz et al. (2006) and, Bernert & Joiner (2007), negative
affect is associated to a reduced tolerance to stressful stimuli in turn associated to self-harm.

As discussed previously, the lack of established directional causality in the literature is problematic as the assumptions of our analysis and conclusions drawn from them in relation to causality may be violated. This would give rise to errors and misattribution due to potential reverse causal effects where by negative affect may increase nightmares rather than nightmares increasing negative affectivity. Moreover, while our Chi-squared test shows participants suffering from clinical levels of nightmares to be at 2.08 time increased risk of having self-harm, the interpretation of this result should be done with caution with regards to causality which may be misattributed. Further research is needed to elucidate this issue.

In addition, it would be more rigorous for affect to be measured prior and post sleep, as close to sleep and of nightmares as possible. The present results utilise retrospective data and likely to comprise a degree of inaccuracy. Zadra & Donderi (2000) have shown when comparing retrospective measurement with prospective dream logs that retrospective measures such as the DDNSI used in this study lead to nightmare report inaccuracies such as false negatives and the underestimation of nightmare frequency. This is due to the nature of nightmares as a phenomenon. As with most dreams, they can be easily forgotten or altered during re-interpretations after waking. Therefore, measurements as temporally close to a nightmare would yield more accurate data. Pre-sleep and post-sleep measurement of affect have been undertaken and reported in the literature by Agargun & Cartwright (2003) and informs the currently proposed mechanism linking nightmares to suicidality summarised in
Bernert & Joiner (2007). However, measurements of suicidality, such as ‘presence self-injurious thoughts’ or ‘engagement in self-injurious behaviour’ during the day following measurements of nightmares and negative affect post-sleep have not been carried out. While such a protocol would not provide support for causality (due to lack of experimental manipulation and control allowing for potential latent variables); it would yield more accurate data and provide an indication of the direction of the relationship between nightmares and self-harm.

2.4.3. *Do individuals with high nightmare levels have elevated defeat and entrapment?*

Investigating the effects of nightmares on defeat and entrapment revealed our high nightmare group reported from significantly higher levels of defeat than controls when controlling for depressive symptoms. However, this difference was small. Entrapment, on the other hand, was not significantly different between the high nightmare and control groups.

While significant, the amount of variance explained and between groups effect size of defeat is relatively small. That is not to say that it should be dismissed as it the discrepancy between our results and, findings from the literature which prove to be of interest. Taylor, Wood, Gooding, Johnson, & Tarrier (2009) have shown defeat and entrapment to follow a single factor structure within non-clinical student samples similar to the sample of the present study. Yet, results obtained from our sample show nightmares to be associated only to defeat when controlling for depressive symptoms but not entrapment. Moreover, from a theoretical perspective such as that of
Schneidman (1964), one would expect nightmares to be more closely related to entrapment as nightmares halt the ‘temporary cessation’ obtained from sleep. One must carefully look at the construct of defeat and the nightmare aetiology literature to make inference from these results. Williams, (1997) and O’Connor (2003) clearly use definitions of defeat where by defeat can be deemed to relate to a stressful event, where one can lose status; be it real or perceived. As dreams regulate affect and nightmares are dysfunctions of the normal regulatory process (Nielson & Levin, 2007), it is reasonable to assume nightmares themselves could be deemed to be a stressful and negative experience eliciting unpleasant emotions needing further regulation. Alternatively, nightmares could be seen as a defeating event in themselves, as a failure to achieve the temporary relief normally offered by undisturbed sleep, although unlikely as status is not impacted upon. It is also possible that defeat increase nightmare likelihood since daily stress and state anxiety have been found to impact on idiopathic nightmare formation (Spoormaker, 2008; Nielsen & Levin, 2007, 2009). Further investigation is required to replicate the group differences for defeat and to elaborate on the role defeat may play in explaining the relationship between nightmares and self-harm.

2.4.5. Further research

A randomised controlled trial utilising interventions aimed at reducing nightmares such as Imagery Rehearsal Therapy (IRT; Krakow & Zadra, 2010) provided to samples of self-harming individuals could be used to lower nightmare levels. By subsequently measuring changes in self-harm rates, potential causal relationships between nightmares and self-harm could be verified. However, such an approach would be difficult within our timeframe.
and require resources beyond the scope of this thesis. As such, more feasible longitudinal research is recommended such as a diary study which could track daily changes in nightmares, negative affect and their impact on self-harming behaviour. Additional benefits of longitudinal diary methodology have been highlighted by Zadra & Donderi (2000) who have shown prospective measurement methods such dream logs decrease inaccuracies such as false negatives and underestimations of nightmare frequency. Such a longitudinal study which will prospectively explore the role of negative affect is reported in Chapter 3.

Moreover, a replication of this study with a larger sub-sample of individuals recently or currently (≤ 1 month) engaging in self-harmful behaviours would allow us to establish if nightmare levels can differentiate between current self-harming individuals and those with a history of self-harm yet who no longer participate in such behaviours. If such findings were obtained this would be highly relevant for prevention efforts and further cement nightmare’s role as a significant risk factor for self-harm. Negative affect is a key variable in the Experiential Avoidance Model (Chapman et al., 2006) whereby a negative internal experience, such as elevated negative affect, is avoided by engaging in self-harm. Our results support a relationship between nightmares and self-harm, and nightmare and elevated negative affect. However, experiential avoidance has not been explored. A large scale psychometric study which will further explore defeat, negative affect, experiential avoidance and other psychological variables of interest is reported in chapter 5. This chapter will collect data in order to model a mechanism
linking nightmares to self-harm using the aforementioned constructs via structural equations.

2.4.6. Key points from Chapter 2

- Past research has shown a link between nightmares and suicidal behaviour. Research has not explicitly explored if this link persist in behaviours regardless of suicidal intent.
- This study investigates if this links applies to self-harm regardless of suicidal intent.
- Nightmares significantly predicted self-harm history beyond the effects of depressive symptoms. Insomnia did not predict self-harm history.
- Individuals with clinically significant levels of nightmares report higher levels of negative affect and slightly higher levels defeat.

2.4.7. Implications for the next chapters

- The next chapter aims to explore the direction of the predictive relationship between nightmares and self-harm to uncover which occurs first, nightmares or self-injurious thoughts and behaviours.
- This information would provide the basis for an empirically driven model of a mechanism linking nightmares and self-harm.
- Methods providing insight into causality are beyond the scope of this thesis due to logistical restrictions. A longitudinal diary study will be implemented instead.
- The chapter will also explore the role of negative affect as potential mediator in this relationship.
3.1. Introduction

Nightmares have been identified as an independent risk factor above and beyond the effects of depressive symptoms for suicidal ideation (Bernert & Joiner, 2007), suicide attempts and repeat suicide attempts (Sjöström, Waern, & Hetta, 2007; Sjöström, Hetta, & Waern, 2009), and completed suicide (Tanskanen et al., 2001). The findings of Chapter 2 have additionally shown nightmares to be predictive of self-harm regardless of suicidal intent and that those suffering from clinical levels of nightmares (≥11 on DDNSI) to be 2.08 times more likely to report having engaged in self-harm during their lifetime. However, the majority of studies investigate the link between nightmares and suicidality have used cross-sectional designs (Bernert et al., 2005; Cukrowicz et al., 2006; Krakow et al., 2011; Nadorff et al., 2011). As such, it is not clear whether nightmares preceded or followed existing self-injurious thoughts and behaviours (SITBs). Moreover, the retrospective assessment of nightmare occurrence over a certain time period in such studies is prone to underestimations. This is because nightmares, as with most dreams, are easily forgotten or altered during re-interpretations, which may lead to false negatives. This has been verified by comparing retrospective measurement with prospective dream logs (Zadra & Donderi, 2000). In order to establish a robust model, the key assumptions about the direction of the predictive relationship between nightmares and SITBs must be established.
While it is ethically difficult to clearly validate the causal relationship between nightmares and suicidality by experimentally manipulating one of these variables, longitudinal studies can provide a preliminary indication of the direction of this relationship. Indeed, there are some studies that have used longitudinal designs (Sjöström, Hetta, & Waern, 2009; Tanskanen et al., 2001); however, these have focused on establishing nightmares as a significant risk factor but did not explicitly investigate the direction of the relationship. Therefore, the aim of the present study is to investigate the direction of the predictive relationship between nightmares and SITBs by using longitudinal prospective diary study in a student population while controlling for baseline levels of depressive symptoms.

The findings of studies exploring nightmares and suicidal behaviours (Bernert et al., 2005; Cukrowicz et al., 2006; Sjöström et al., 2007, 2009; Krakow et al, 2011; Nadorff et al., 2011) have implied a link between nightmares and suicidal intent. This is due to the populations being investigated and the definitions of the behaviors under scrutiny. However, to the authors’ knowledge, no studies have yet investigated the links between nightmares and SITBs in a population with a history of self-harm behaviors regardless of suicidal intent. The present study will therefore operationalise self-harm as intentional self-injury or self-poisoning, regardless of motivation or suicidal intent and with a non-fatal outcome (Hawton, Harriss, et al., 2003).

Further, whilst nightmares have been shown to be a robust independent risk factor linked to SITBs (Bernert & Joiner, 2007; McCall & Black, 2013), little is known about the mechanisms underlying this association. A recent review by McCall and Black (2013) has set out a model through which sleep
disturbances, insomnia and nightmares, can lead to increased suicidal thoughts and behaviours, but these assumptions have not been empirically tested. Negative affect has been described as playing a pivotal role in the association between nightmares and suicidal behaviours (Cukrowicz et al., 2006; Bernert & Joiner, 2007). It has been suggested that nightmares disrupt the normal emotion regulatory process of dreaming (Nielsen & Levin, 2007, 2009), and thus leads to negative affect. In line with this, research has demonstrated that nightmares are associated with increased negative affect post sleep (Antunes-Alves & De Koninck, 2012); however, these studies have not yet linked process to the development of SITBs. Therefore, the present study investigates prospectively the role of negative affect as a pivotal underlying mechanism in the association between nightmares and SITBs.

3.1.1. Aims & hypotheses

Taken together, the present study investigates the (i) direction of the predictive relationship between nightmares and SITBs, and (ii) the effects of negative affect on this association by longitudinally tracking the occurrence of nightmares, and pre- and post-sleep negative affect and SITBs.

Based on previous research and proposed theoretical models (Bernert & Joiner, 2007; Anutnes-Alves & de Koninck, 2012) it is expected that:

(i) Controlling for depressive symptoms, pre- and post-sleep negative affect and pre-sleep SITBs, the occurrence of nightmares significantly increases the likelihood of SITBs post-sleep.
(ii) Controlling for depressive symptoms, pre-sleep negative affect and pre-sleep SITBs, post-sleep negative affect mediates the association between nightmares and post-sleep SITBs.
3.2. Methodology

3.2.1. Design and procedure

A 5 day fixed interval diary study design was implemented. Participants completed daily pre-sleep and post-sleep measures relating to SITBs, negative affect and the occurrence of nightmares using paper & pencil diaries. This study utilises daily prospective dream logs to reduce recall biases associated with retrospective designs (e.g., underestimation of nightmares; Zadra & Donderi, 2000). The study protocol was augmented by daily automatic Short Message Service (SMS) text message reminders pre-sleep (prior to the average bed-time indicated on the PSQI) and post-sleep at an agreed wake up time. Reminders were sent to participants for the duration of their participation in the study. Moreover, given that participants with an existing history of self-harm are more likely to exhibit SITBs, participants’ history of engagement in self-harm regardless of intent is obtained to distinguish and model responses according to self-harm history.

This study was approved by the University of Nottingham School of Psychology ethics committee. Written consent was obtained from all participants prior to participation. Written debriefing information and contact details for the researcher and relevant support groups dealing with issues explored in the present study were provided to all participants.

Participants’ self-harm history, depressive symptoms, and sleep quality prior to diary completion were assessed no more than one week before starting the diary study protocol. Participants attended a thorough compulsory briefing on diary completion, stressing the importance of accurate timely responses.
Participants were requested to complete the diary for 5 consecutive weekdays, providing 5 pre-sleep and 5 post-sleep entries per participant. They were instructed to complete the pre-sleep section of the diary immediately (or no more than 1 hour) prior to going to sleep; and the post-sleep section as soon as they woke up (or no more than one hour after waking). Participants were required to date and time the completion of individual pre- and post-sleep diary entry sheets and submit those daily using the provided researcher-addressed internal mail envelopes. These were to be handed in directly to a collection box by 1pm on the day of ‘post-sleep section’ completion. If participants could not hand their entries at the collection box, they were to notify the researcher via text message by 1pm having submitted their entry via the university internal mailing system. Entries which did not meet the above criteria were excluded.

3.2.2. Participants

Three hundred and ninety-nine (64 males) university students completed the initial screening questionnaires online via the institution’s research participation scheme website. Of those, 286 participants did not respond to invitations for the diary briefing. Of the 113 who attended the briefing, 36 participants were excluded as they did not return any diary entry within the time delays set out in the briefing. A further 5 participants were excluded as they reported the current use of antidepressant medication or sleeping issues more than ‘Once or twice a week’ other than nightmares on the PSQI. This was to control for artificial conflation of nightmare occurrences (Pagel & Helfter, 2003) and substantial levels of confounding sleep co-morbidities.
A total of 72 participants (8 males) aged between 18-32 years ($M = 21.04$, $SD = 3.40$) fully completed at least one diary entry (pre and post sleep) and met the inclusion criteria. Of those, 43 participants (5 males) reported a history of self-harm engagement. Participants were naïve to the hypothesis of this study. Research credits were granted to participants in exchange for participation.

3.2.3. Measurements

Screening Measures:

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) was used to assess potential co-morbid sleep disturbances which could confound findings relating to nightmares. The PSQI comprises 10 questions with open ended items and frequency of sleep disturbances (e.g. inability to fall sleep within 30mins, difficulties breathing, pain during sleep) rated on a 4-point rating scale (from 0=not during the past month to 3=three or more times a week). Question 5 assessed the frequency and type of sleep disturbances (using 10 items) experienced over the last month. Responses on these items of more than ‘once or twice a week’ (>2) were used as exclusion criterion for the current study.

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) was used to assess the presence and severity of depressive symptoms over the last two weeks. The BDI-II has previously shown good reliability (Cronbach’s alphas = .91) and validity (A. T. Beck, Steer, Ball, & Ranieri, 1996) and was used to control for the impact of depressive symptoms in the current study.
The *Deliberate Self-Harm Inventory* (DSHI; Gratz, 2001) was used to assess participants’ self-harm history over the lifetime. Seventeen items dichotomously assessed self-harm behaviours (No/Yes). Participants responding positively to any of the 17 items were categorised as having a history of self-harm (SH group) while those reporting no self-harm behaviours were categorised as the no history of self-harm group (no SH group). The DSHI is a well-validated tool assessing self-harm behaviors corroborated by clinical observations. The DSHI explicitly measures acts of self-harm without conscious intent to die, also known as non-suicidal self-injury. As this study investigates the link between nightmares and SITBs in individuals with a history of self-harm regardless of intent to die or motivation, the instructions to participants were modified to encompass all acts of self-harm by removing any mention of suicidal intent. The DSHI shows good reliability (Cronbach’s alphas = .80).

*Diary Measures:*

The *Positive Affect and Negative Affect Schedule-Short Form* (PANAS-SF; Thompson, 2007) was used to assess positive and negative affect for the diary protocol. The short form measures mood with 5 items for each scale rated on a 5-point scale (from 1=not at all to 5=extremely). The temporal reference was set to ‘currently’ to assess participants’ mood states at the respective time points of the diary entries. The PANAS-SF has been widely validated and shown to be a reliable (Cronbach’s alphas of .76 for NA and .75 for PA subscale) psychometric assessment of affect (Thompson, 2007). The Negative Affect subscale was used in the present study to measure fluctuation in pre- and post-sleep negative affect.
One Diary Entry consisted of separate pre-sleep and post-sleep sections, printed in booklet format on one A4 page. Each time section was located on one side of the booklet. Both sections included the PANAS-SF to assess pre- and post-sleep mood, respectively, followed by 2 questions relating to presence or absence of self-injurious thoughts (have you had thoughts of deliberately injuring yourself?) and self-injurious acts (Deliberately injured yourself?) with a dichotomous answer format (Yes/No). Pre-sleep question prefixed the SITB questions with “today” while post-sleep questions stipulated “since waking up.” The post-sleep section comprised an additional question asking participants if they had experienced a memorable negative or dysphoric dream eliciting awakening (with dichotomous answer format: Yes/No) and some additional space underneath for verbal descriptions of the content of nightmares (the qualitative data collected here is analysed and presented in chapter 4). See Appendix H for a copy of a Diary Entry.

The variables ‘pre-sleep SITBs’ and ‘post-sleep SITBs’ were computed by pooling together diary items pertaining to presence of self-injurious thoughts and presence of self-injurious acts at their respective time points resulting in binary variables (SIBTs present/not present) for each day of the study resulting in 5 entries of pre-sleep SITBs and 5 post-sleep SITBs per participant. Similarly, occurrence of ‘nightmares’ was computed as a binary (No/Yes) variable. Negative affect (NA) scores for each entry section were computed following PANAS-SF scoring guidelines providing average mood scores for ‘pre-sleep NA’ and ‘post-sleep NA’.
3.2.4. Data analysis procedure

Generalized Estimating Equations (GEE - Liang & Zeger, 1986; Zeger & Liang, 1986) - a subtype of generalized linear modeling - were used to analyse the data. While hierarchical linear models (HLM) are typically preferred for analyses of data with longitudinal clustering, they require assumptions of normality to be met to obtain accurate estimates. GEE produces more efficient and unbiased regression estimates when analysing longitudinal data with non-normal response variables, such as binomial and multinomial data (Ballinger, 2004). In addition, the intended hierarchy for the present models specified originally individual diary entries at level one, clustered within participants at level two, and nested within self-harm history groups at level three which lends itself to HLM analysis.

However, exploration of the data revealed that there were no cases of reported post-sleep SITBs, and only two cases of pre-sleep SITBs in the non-SH group (reported by two independent participants). This created a complete separation of this dependent variable (pre-sleep SITBs) based on self-harm grouping. Therefore, HLM analysis estimating the effect of 3rd level variable (self-harm history) would have been unsuitable due to impaired model convergence. This effectively limited the range of models which could be to be fitted to 2 levels only making the ability of HLM to fit models with more than 2 levels redundant. This in addition to the more efficient estimates provided by GEE indicated GEE to be a more suitable analysis method than HLM in the given circumstances. GEE models were, therefore, only computed on the subsample of participants reporting a history of self-harm to obtain estimates
of the association between nightmares and SITBs. Analyses were performed using IBM SPSS Statistic v.21 (IBM Corporation, 2012).

In order to identify directionality of effects and rule out alternative relationships, three potential pathways (summarised in Figure 3) were tested:

a) the first model examined whether nightmares (X) significantly increased the reporting of SITBs post-sleep (Y);
b) the second model examined whether SITBs pre-sleep (W) significantly increased the likelihood of experiencing nightmares (X);
c) the third model examined whether SITBs pre-sleep (W) significantly increased the reporting of SITBs post-sleep (Y) regardless of nightmares (X).

Pathways ‘a’ and ‘c’ were tested simultaneously within one model. The GEE model was specified to use logit link function with a binomial distribution as the dependent variable (presence of post-sleep SITBs) was binary (SITBs vs no SITBs). The reference category was set to ‘first’ thus providing estimates relating to presence of SITBs. Predictive factors entered
into the model were pre-sleep SITBs and nightmares. Pre-sleep negative affect (pre-sleep NA), post-sleep negative affect (post-sleep NA) and depressive symptoms were entered in the model as covariates. To test pathway ‘b’, the GEE model was specified to use a logit link function with nightmares entered as the dependent variable. Predictive factors entered into the model were pre-sleep SITBs with the covariate being pre-sleep NA and depressive symptoms. For both models the correlation structure was set to auto-regressive AR(1) and hybrid method was used with 95% maximum likelihood confidence interval (CI). Main effects for all variables were sought.

Mediation analysis (A. F. Hayes, 2013) was performed testing the mediating effect of post-sleep negative affect on the relationship between nightmares and post-sleep SITBs while controlling for depressive symptoms, pre-sleep negative affect and pre-sleep SITBS. This method was performed on the full sample and repeated self-harm group subsample.
3.3. Results

3.3.1. Attrition rates and descriptive statistics

A 13.9% attrition rate was observed over the course of the study for the whole sample as participants did not complete all diary entries or their entries did not meet the study inclusion criteria. A total of 328 out of the potential 360 diary entries were obtained over the course of the study. Over the 5 day period, 39 counts (11.9%) of pre-sleep SITBs were recorded, while post-sleep SITBs were reported 19 times (5.8%). Nightmare occurrence was reported 47 times (14.3%).

Table 3 - Frequency of event and negative affect means (standard deviations) for each of the diary days for participants with a history of self-harm

<table>
<thead>
<tr>
<th>Day</th>
<th>Pre-sleep SITBs</th>
<th>Nightmares</th>
<th>Post-sleep SITBs</th>
<th>Pre-sleep NA Mean (SD)</th>
<th>Post-sleep NA Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1 (n=43)</td>
<td>7 (16.3%)</td>
<td>11 (25.6%)</td>
<td>4 (9.3%)</td>
<td>8.35 (3.33)</td>
<td>8.07 (3.04)</td>
</tr>
<tr>
<td>Day 2 (n=40)</td>
<td>10 (24.4%)</td>
<td>5 (12.2%)</td>
<td>5 (12.2%)</td>
<td>8.59 (3.02)</td>
<td>7.88 (3.64)</td>
</tr>
<tr>
<td>Day 3 (n=37)</td>
<td>5 (13.5%)</td>
<td>11 (29.7%)</td>
<td>4 (10.8%)</td>
<td>8.46 (3.61)</td>
<td>7.86 (3.15)</td>
</tr>
<tr>
<td>Day 4 (n=36)</td>
<td>7 (19.4%)</td>
<td>5 (13.9%)</td>
<td>3 (8.3%)</td>
<td>7.47 (2.56)</td>
<td>7.14 (2.44)</td>
</tr>
<tr>
<td>Day 5 (n=36)</td>
<td>8 (22.2%)</td>
<td>3 (8.3%)</td>
<td>3 (8.3%)</td>
<td>8.11 (2.99)</td>
<td>7.00 (2.61)</td>
</tr>
</tbody>
</table>

Descriptive statistics for participants having reported a history of self-harm engagement are reported in Table 3. Means and standard deviations (S.D.) are provided for pre-sleep and post-sleep negative affect, frequency counts are provided for categorical data. Pre-sleep and post-sleep SITBs as well as nightmares were reported by participants with a history of self-harm throughout the 5 days of the study. Two pre-sleep occurrences of pre-sleep SITBs were also reported by two participants without a history of self-harm on day 3 and 4. Participants reporting a history of self-harm ($M= 21.02, SD= 3.43$) and those without ($M= 21.07, SD= 3.35$) did not significantly differ in age ($p>$
.05). However, participants reporting a history of self-harm \((M=18.70, SD=10.24)\) had significantly higher levels of depressive symptoms than participants with no self-harm \((M=10.28, SD=6.88)\), \(t(70)=3.87, p<.001\). The attrition rate for the self-harm subsample was 16.3%. The 43 participants reporting a history of self-harm engagement yielded 193 cases out of the potential 215 cases to use for the models. All reports of post-sleep SITBs were associated with this subsample, as were 73.9% of nightmares and 94.7% of pre-sleep SITBs.

3.3.2. Exploring pathways ‘a’, ‘b’, and ‘c’ within the self-harm history subsample

The models were estimated using the 193 diary entries provided by the self-harm group\(^7\). Estimation parameters are displayed in Table 4. Redundant parameters such as independent variable reference categories have been omitted from the table for both models.

The first model, examining pathways ‘a’ and ‘c’ simultaneously, indicates that as hypothesised, nightmares could significantly predict post-sleep SITBs \((\chi^2 (1) = 4.19, p = .041)\) beyond depressive symptoms, pre-sleep negative affect, and post-sleep negative affect providing support for pathway ‘a’. Nightmares significantly increased the risk of experiencing post-sleep SITBs. However, pre-sleep SITBs \((\chi^2 (1) = 1.11, p > .05)\) did not significantly predict the occurrence of post-sleep SITBs beyond the effects of depressive symptoms, pre-sleep negative affect, and post-sleep negative affect; thus

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\(^7\) Estimates were additionally obtained with the full sample. The estimates for the full sample mirrored those reported above for our subsample. However, effect sizes on the full sample for pathway ‘a’ were slightly conflated due to the increased sample size. I.e. The occurrence of nightmares significantly increased \((\chi^2 (1) = 3.92, p = .048)\) the likelihood of post-sleep SITBs by 4.17 times (95% C.I. [1.02 – 17.11], \(p < .001\)).
failing to support pathway ‘c’. Similarly, model 2, examining pathway ‘b’, indicates that pre-sleep SITBs did not significantly predict nightmares ($\chi^2 (1) = 1.12, p > .05$) beyond the effects of depressive symptoms and pre-sleep negative, thus further failing to support pathway ‘b’.

### Table 4 - GEE model 1 and 2 for our self-harm history subsample testing pathways ‘a & c’, and ‘b’ respectively

<table>
<thead>
<tr>
<th>Parameter*</th>
<th>Beta (S.E.)</th>
<th>Odds Ratio (95% C.I.)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive symptoms</td>
<td>.13 (.03)</td>
<td>1.13 (1.07 to 1.20)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Pre-sleep NA</td>
<td>-.06 (.10)</td>
<td>.94 (.77 to 1.15)</td>
<td>.547</td>
</tr>
<tr>
<td>Post-sleep NA</td>
<td>.23 (.12)</td>
<td>1.26 (1.00 to 1.58)</td>
<td>.050</td>
</tr>
<tr>
<td>Pre-sleep SITBs</td>
<td>.59 (.56)</td>
<td>1.80 (.60 to 5.42)</td>
<td>.291</td>
</tr>
<tr>
<td>Nightmares</td>
<td>1.39 (.68)</td>
<td>4.01 (1.06 to 15.15)</td>
<td></td>
</tr>
</tbody>
</table>

**GEE Model 1 †**

**GEE Model 2 ‡**

<table>
<thead>
<tr>
<th>Parameter*</th>
<th>Beta (S.E.)</th>
<th>Odds Ratio (95% C.I.)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive symptoms</td>
<td>.01 (.02)</td>
<td>.99 (.95 to 1.04)</td>
<td>.782</td>
</tr>
<tr>
<td>Pre-sleep NA</td>
<td>.18 (.07)</td>
<td>1.22 (1.08 to 1.39)</td>
<td>.002</td>
</tr>
<tr>
<td>Pre-sleep SITBs</td>
<td>-.14 (.49)</td>
<td>.65 (.24 to 1.76)</td>
<td>.394</td>
</tr>
</tbody>
</table>

†Dependent variable = post-sleep SITBs, Reference category = no SITBs
‡Dependent variable = nightmares, Reference category = no nightmares

*Redundant parameters (categorical independent variable’s reference category) have been omitted

3.3.3. Mediation of nightmares and post-sleep SITBs by post-sleep negative affect

The mediating effect of post-sleep negative affect on the relationship between nightmares and post-sleep SITBs while controlling for depressive symptoms, pre-sleep negative affect and pre-sleep SITBs is presented in Figure 4. Path A represents the direct effect of nightmares on the mediator (post-sleep NA). Path B represents the direct effect of the mediator on post-sleep SITBs. Path C shows the total effect of nightmares on post-sleep SITBs mediated by post-sleep negative affect. The indirect effect of nightmares on post-sleep SITBs through post-sleep negative affect is represented by $A \times B$. The normal theory test for this indirect effect was significant ($Z = 2.06, p = .039$). Although
a significant reduction of the direct (C) path coefficient was observed, it remained greater than zero indicating this mediation to be partial. Path A was significant and positive as was path B indicating that the occurrence of nightmares were related to increased post-sleep negative affect; this negative affect in turn was associated to increased risk of post-sleep SITBs.

Figure 4 - The mediating effect of post-sleep negative affect on the relationship between nightmares and post-sleep SITBs
3.4. Discussion

3.4.1. Do nightmares predict SITBs?

The present study aimed to clarify the direction of the predictive relationship between nightmares and SITBs. The rationale for such a study was the empirical validation of the assumption that nightmares were predictive of SITBs as has been demonstrated in the literature (Tanskanen et al., 2001; Bernert & Joiner, 2007; Sjöström et al., 2007, 2009; Nadorff et al., 2011, 2013; McCall et al., 2013), rather than SITBs being predictive of the occurrence of nightmares. We did this to in order to establish solid empirical foundations for theoretical models of this association. To achieve this we investigated three potential pathways. Pathway ‘a’ predicted nightmares to be predictive of post-sleep SITBs (as would be expected from existing literature). Pathway ‘b’ predicted the inverse of ‘a’, that is that, pre-sleep SITBs would predict the occurrence of nightmares. Finally pathway ‘c’ predicted that pre-sleep SITBs would predict SITBs post-sleep.

Our findings provide clear empirical support for the first hypothesis: that nightmares predict SITBs; thus supporting pathway ‘a’. Indeed, our model indicated that participants with a history of self-harm experiencing a nightmare were 4.01 times (95% C.I. [1.06 – 15.15]) more likely to experience SITBs upon waking than participants who had not experienced nightmares. Having had a nightmare was the strongest predictor of post-sleep SITBs in a model accounting for depressive symptoms and negative affect. Concurrently, our models did not support alternative pathways ‘b’ and ‘c’, showing the occurrence of pre-sleep SITBs could not predict nor did it increase the risk of nightmares. Likewise, pre-sleep SITBs could not predict nor did it increase the
risk of post-sleep SITBs. This support for pathway ‘a’ and failure to support pathways ‘b’ and ‘c’ provide complimentary support for a unidirectional predictive relationship between nightmare and SITBs.

Nock and colleagues (2009) had previously found individuals who engaged in self-harm to use sleep as an alternative affect regulation strategy when experiencing urges to self-injure. The increased risk of post-sleep SITBs in participants with a history of self-harm following the occurrence of nightmares and high negative affect could indicate that self-harming individuals, who suffer from deficits in affect regulation (Klonsky, 2009) may do so not only during their waking life but throughout the diurnal cycle. Nock and colleagues findings, in combination with those of the present study provide support towards a rationale for the treatment for nightmares in individuals who are experiencing problems with self-harm. This echoes calls from within the literature (Krakow, Ribiero et al., 2011) for nightmare reducing treatment such as Imagery Rehearsal Therapy (Krakow & Zadra, 2010) which may aid in increasing affect regulation during sleep and could potentially reduce SITBs.

3.4.2. Does negative affect mediate the relationship between nightmares and SITBs?

The mediational role of post-sleep negative affect shown by our mediation analysis suggests that nightmares lead to post-sleep negative affect in turn leading to self-injurious thoughts and behaviour. This resonates with the literature suggesting that nightmares act as emotion dysregulators, hindering the normal mood regulatory process of dreaming leaving the nightmare sufferer with increased affective loading (Nielsen & Levin, 2007,
2009). These findings support notions that nightmares dysregulate mood (Bernert & Joiner, 2007; Nielsen & Levin, 2007, 2009) leading to greater sensitivity to cues and emotions upon waking (Cukrowicz et al., 2006). However, this mediational role is partial. The results of the GEE model which used post sleep negative affect as a covariate additionally indicated that experiencing post-sleep negative affect increases risk of post-sleep SITBs. Concurrently, pre-sleep negative affect increases the risk of nightmares. These results highlight the importance of taking into account negative affect in further models of the association between nightmares and SITBs.

Moreover, our findings support the empirically validated notion that dreaming serves an emotion regulatory process (Cartwright, 2010) and highlight that nightmare-less sleep is protective against SITBs.

3.4.4. Limitations and further research

Although supportive of the literature and of the pathway which hypothesised nightmares to predict SITBs experienced in the post-sleep period, our results must be interpreted with caution for several reasons. Firstly, the observational nature of this study does not allow us to validate any potentially causal relationship between nightmares and SITBs. Whilst they allow us to draw conclusions about the direction of the relationship, the replication and validation of these findings by way of experimental manipulation (reduction) of nightmares further our understanding of the potential causal mechanisms. If an intervention using robust randomised controlled trial methods resulted in a reduction in nightmares and a reduction in SITBs (as compared to a control group with no change in nightmares or SITBs), this would provide strong
evidence for the existence of a causal relationship between nightmares and SITBs. Nevertheless, a strength of the present study was the prospective collection of nightmare data which provides a more accurate estimate of nightmares than those generated in studies which have collected these data retrospectively (Zadra & Donderi, 2000). Moreover, as the mediation of nightmare to post-sleep SITBs by post sleep negative affect is partial, latent variables need to be explored to further our understanding of this mechanism. Hyperarousal as suggested by McCall & Black (2013) should be explored as one of these potential latent variables.

The generalisability of our findings to the wider population must be done with caution due to our sample being comprised predominantly of female undergraduate students. Replication with a larger male cohort would be useful. Moreover, as nightmares are known to fluctuate and generally decrease as a function of ageing (Nielsen, Stenstrom, & Levin, 2006); replications with a variety of ages would be useful to see if the effect of nightmares on SITBs remained. This would allow for potential intervention to be offered to specific target group where treatments impact would be maximised.

Lastly, the current paper and pencil methodology, although augmented by text message reminders, does not guarantee participants completed the diary in the time indicated on the entries. This potential non-compliance and retrospective completion would lead to decreased accuracy of measurements. Augmented paper and pencil method was selected to ease participant compliance and eliminate the pre-requisite of internet connections or computer literacy. This method has been used widely in the psychological literature (Bolger, Davis, & Rafaeli, 2003). Moreover, all efforts were made to reduce
the impact of non-compliance using a variety of techniques. This included clear instructions given during our pre-study briefing, text message prompts, increasing participant engagement by allowing them to describe nightmares and, the omission from analysis of entries submitted past a daily deadline. These efforts were effective as demonstrated by the relatively low attrition rate (13.9%) over the course of the study for the whole sample. Future studies could benefit from using Personal Digital Assistant (PDA) which would provide clear time locking information and improve the validity of findings.

The direction of the predictive relationship between nightmares and SITBs was explored here in order to empirically validate basic theoretical assumptions regarding the association between nightmares and self-injurious thought or acts. While replication with diverse samples and technologically enhanced methodology is recommended, this study provides preliminary evidence supporting the premise that nightmares are potent predictors of SITBs and that this relationship is unidirectional. Cukrowicz et al. (2006) and Bernert & Joiner (2007) suggested that a mechanism associating nightmare to suicidal behavior would incorporate dysregulated mood. Our results support their suggestion showing post-sleep negative affect to predict post-sleep SITBs. Moreover, post-sleep negative affect is a partial mediator in the relationship between nightmares and post-sleep SITBs. As such, we suggest negative affect to be included in future theoretical models linking sleep disturbance and suicidal behavior such as that of McCall & Black (2013), particularly when modeling the relationship of nightmares to self-harm. Nightmares offer a potent predictor of SITBs, which importantly is amenable to interventions. This highlights the potentially useful nature of nightmare reducing treatments.
for individuals with a history of self-harm engagement. This would likely be a promising avenue for future research.

3.4.5. Key points from Chapter 3

- Past research had shown a correlational link between nightmares and self-harm, but causality has not been established.
- This is the first study to the author’s knowledge using diary methodology to study nightmares effect on SITBs.
- This study aimed to explore the predictive direction of this relationship.
- Nightmares unidirectionally predict self-injurious thoughts and behaviours in participants with a history of self-harm.
- Negative affect partially mediates the relationship between nightmares and post-sleep self-injurious thoughts and behaviours.
- This is the first study explicitly testing the direction of the nightmare on self-harm relationship.

3.4.6. Implications for the next chapters

- Future models (Chapter 5) now have an empirical basis for using nightmares as an exogenous variable.
- Future models of a psychological mechanism linking nightmares to self-harm should incorporate the partial mediating role of negative affect.
- The next chapter will investigate if nightmare content differs between participants with and without a history of self-harm as little has been done with regards to nightmare content in recent years.
Chapter 4: Diary Study Part (ii) – Investigating the Content of Nightmares in Self-Harming Participants.

4.1. Introduction

A growing literature (Bernert, Joiner, Cukrowicz, Schmidt, & Krakow, 2005; Cukrowicz et al., 2006; Sjöström, Hetta, & Waern, 2009; Sjöström, Waern, & Hetta, 2007; Nadorff, Nazem, & Fiske, 2011) and our findings from Chapters 2 and 3 empirically supports the association between nightmares and self-harm beyond the effects of depressive symptoms. While the body of evidence linking nightmares to suicidality is growing, much of the recent focus has been on the observable occurrences of nightmares, rather than the content of the experience. In fact, research into dream content and its links to suicidal behaviour (Evans, 1990; Firth, Blouin, Natarajan, & Blouin, 1986; Langs, 1966; Maltsberger, 1993; Raphling, 1970) has been sparse for the last 20 years. Moreover, to the author’s knowledge, no studies have explored the negative dream content of self-harming individuals regardless of suicidal intent. The present study investigates if differences in nightmare content between participants with a lifetime history of self-harm and non-self-harming student controls could serve as a marker of increased vulnerability for self-harm.

In addition, this study aims to address some important methodological and terminology issues such as the lack of clarification regarding suicidal intent of the behaviours being linked to negative dream content; the subjectivity of content analysis methodology employed and the retrospective nature of the nightmare data acquisition. These issues are detailed below.
Earlier research in this field has focused on the reported dream content of suicide attempters who were either depressed or psychiatric in-patients and has shown greater proportions of reported themes pertaining to death, exhaustion, disintegration, annihilation, murder and killing, surrender, peaceful departures and, reunion with the dead compared to controls (Langs, 1966; Ralphing, 1970; Firth et al., 1986; Evans, 1990; Maltsberger, 1993). However, it is not clear whether this finding also pertains to individuals with issues of self-harm regardless of suicidal intent.

Moreover, these dream analysis studies were undertaken using subjective methods, mostly content analysis, which require inter-rater reliability, and thus introduce an element of error. For instance, Firth et al. (1986) comparing dream content of suicidal, depressed and violent inpatients had an inter-rater reliability of 0.83. While this level of reliability is high, it does allow for discrepancies. However, this issue can be overcome through the use of computer software and text analysis packages such as the Linguistic Inquiry and Word Count (LICW; Pennebaker, Francis, & Booth, 2001), which can systematically and objectively summarise a linguistic text in terms of its content. This addition to the toolbox of literary analysis has injected oneirology with a newfound sense of objectivity and convenience. Therefore, the present study utilises the LIWC 2007 (Pennebaker & Chung, 2007) as a method of data analysis to objectively quantify negative dream content utilising the default dictionary included in the LIWC package. This default dictionary is comprised of 32 psychological constructs and 7 personal concern categories. The LIWC has been empirically validated (Tausczik & Pennebaker, 2010) and shown to detect emotionality and thinking style from text extracts,
allowing for their linking to real-world behaviours such as social coordination, honesty or deception (Tausczik & Pennebaker, 2010). Moreover, the speed of text analysis offers a potentially time and cost-effective tool in detecting individuals at risk of self-harm from their negative dream reports. This is of importance to prevention and intervention efforts as studies have shown that individuals presenting to hospital following acts of self-injury were 66 times more likely to die by suicide within the first year after the act compared to the general population (Hawton, Zahl & Weatherall, 2003). As reporting self-harm may be seen as taboo (McAllister, 2003), the ability to detect at risk individuals from more actively disclosed phenomena such as nightmare contents may enable earlier detection.

Another issue of the existing literature pertains to the definition of key terms, particularly related to suicidality. For instance, Raphling (1970) defines “suicide attempt” using the criteria of self-destructive intention, rather than intent to die. Such a definition implies clear suicidal intent, however; as frequent ambivalence or uncertainty regarding intent is expressed by patients reporting in hospitals following self-harm (Skegg, 2005), the categorising of self-harm, based on intent as either suicidal or non-suicidal, is a contentious subject as little evidence has been reported to support the dichotomisation of suicidal intent (Kapur et al., 2013). Therefore, self-harm will be operationalised throughout the present study as intentional self-injury or self-poisoning, regardless of motivation or suicidal intent and with a non-fatal outcome (Hawton et al., 2011).

In addition, the literature is unclear as to the temporal relationship between negative dreams and self-injurious acts. For instance, Firth et al.
(1986) reported participant’s negative dreams that occurred within a window of 6 months prior to the self-injurious act, whereas Raphling (1970) recorded negative dreams between 2-21 days (M= 7.3 days) prior. Moreover, these studies rely on retrospective assessments, and as such dream reports are open to re-interpretation and memory biases. The present study employs a longitudinal design, in which negative dreams and their attributes (vividness, intensity, and distress) are obtained within hours of their occurrence.

4.1.1. Aims & hypothesis

The aim of the present study is to explore differences in negative dream content between self-harm groups and non-self-harm student controls using the LIWC software for systematic and objective content analysis and a longitudinal design to reduce retrospective bias. Moreover, the study assesses dream content specific to self-harm regardless of suicidal intent, and will explore if participants with a history of self-harm and non-self-harm controls differ on linguistic categories.

As previous research revealed suicide attempters to use a greater frequency of death themes in their dream reports than controls, this study extents this research to participants reporting a history of self-harm regardless of suicidal intent. The explicit hypothesis for this exploratory study is therefore as follows:

(i) Participants with a history of self-harm will report significantly more linguistic content pertaining to death themes compared to controls.
In addition, this study also explores differences in dream content related to default LIWC categories, 5 psychological constructs (affective, cognitive, social, perceptual, and biological processes) and 6 personal concern categories (work, achievement, home, leisure activities, money, and religion). Given the lack of prior research related to those aspects, no directional hypotheses are made.
4.2. Methodology

4.2.1. Design and procedure

The design and procedure for this study follows that of the study reported in Chapter 3. A 5 day fixed interval diary study design was implemented using paper & pencil diaries. Participants completed daily post-sleep measures of the occurrence of nightmares and to write as detailed as description as possible if a negative dream was recalled. The study protocol was augmented by daily automatic SMS text message reminders post-sleep at an agreed wake up time to prompt participants to complete their entries. Reminders were sent to participants for the duration of their participation in the study. Moreover, participants’ history of self-harm regardless of intent is obtained to perform between groups analysis.

This study was approved by the University of Nottingham School of Psychology ethics committee. Written consent was obtained from all participants prior to participation. Written debriefing information and contact details for the researcher and relevant support groups dealing with issues explored in the present study were provided to all participants.

Participants’ self-harm history, depressive symptoms, and sleep quality prior to diary completion were assessed no more than one week before starting the diary study protocol. Participants attended a thorough compulsory briefing on diary completion, stressing the importance of accurate timely responses. Participants were requested to complete the diary for 5 consecutive weekdays, providing a potential 5 negative dream entries per participant. They were instructed to complete the post-sleep section as soon as they woke up (or no
more than one hour after waking) to minimise dream re-interpretation. Participants were required to date and time the completion of individual diary entry sheets and submit those daily using the provided researcher-addressed internal mail envelopes. These were to be handed in directly to a collection box by 1pm on the day of ‘post-sleep section’ completion. If participants could not hand their entries at the collection box, they were to notify the researcher via text message by 1pm having submitted their entry via the university internal mailing system. Entries which did not meet the above criteria were excluded.

4.2.2. Participants

Participants in this study are those who were recruited and took part in the diary study reported in Chapter 3 (see Chapter 3 Section 3.2.2. for additional details). A total of 72 participants (8 males) aged between 18-32 years ($M = 21.04$, $SD = 3.40$) provided one complete diary entry which met the inclusion criteria. However, only 47 participants (42 females, 5 males; aged 18-32, $M = 21.60$, $SD = 3.84$) provided negative dream reports during the study (25 participants reported no negative dreams). Of the 47 participants used within our analysis, 14 reported no history of self-harm, while 33 reported a history of self-harm regardless of suicidal intent. Within this subsample of 33 self-harming individuals, 6 participants reported a self-injurious act within 1 month or less of participating in the study while the remaining 27 reported their last self-injurious acts to be more than 1 month ago. Participants were naïve to the hypothesis of this study. Research credits were granted to participants in exchange for participation.
4.2.3. Measurements

Screening Measures:

The *Pittsburgh Sleep Quality Index* (PSQI; Buysse, et al., 1989) was used to assess potential co-morbid sleep disturbances which could confound findings relating to nightmares. As with the study reported in Chapter 3, question 5 was used to assess the frequency and type of sleep disturbances experienced over the last month using 10 items. Responses of more than ‘once or twice a week’ (>2) on any of the 10 items asked in question 5 excluded participants from taking part in the diary study.

The *Beck Depression Inventory-II* (BDI-II; Beck, Steer, & Brown, 1996) was used to assess the presence and severity of depressive symptoms over the last two weeks. This allowed for the statistical control of the impact of depressive symptoms in the analyses.

The *Deliberate Self-Harm Inventory* (DSHI; Gratz, 2001) was used to assess participants’ self-harm history over the lifetime. The 17 item DSHI is a self-report measure dichotomously assesses acts of deliberate self-harm (No/Yes) operationalised as the destruction of or alteration of body tissue, without suicidal intent, resulting in injury severe enough to cause tissue damage such as scarring. As with previous studies the instructions to participants were modified to encompass all acts of self-harm by removing any mention of suicidal intent. This was done to obtain a measure of self-harm history regardless of motivation or intent to die. Participants responding positively to any of the 17 items were categorised as having a history of self-harm (SH group) while those reporting no self-harm behaviours were categorised as
having no history of self-harm (no SH group). Self-harm history (SH group/no SH group) was used as a grouping variable in our analyses. An additional question relating to time of last self-injurious act is asked following the 17 items. This allowed for self-harm recency to be classified as none (no self-harm report), self-harm history (≥1 month) or current self-harm (≤1 month) for the purposes of an exploratory analysis.

*Diary Measures:*

One *Diary Entry* consisted of separate pre-sleep and post-sleep sections, printed in booklet format on one A4 page (Appendix H). Each time section was located on one side of the booklet. The post-sleep section was divided into mood and SITB questions (used in Chapter 3) and an additional question asking participants if they had experienced a memorable negative or dysphoric dream eliciting awakening (with dichotomous answer format: Yes/No) and some additional space underneath for a written descriptions of the content of nightmares. Below the descriptions, participants were asked to rate the negative dream on 3 items; vividness, intensity, and distress. Each of these items were to be rated on a 5-point scale (from 1= not at all to 5= extremely).

*4.2.5. Diary transcription procedure*

Selected dream reports were electronically transcribed (from hard copies to MS Word files) and run through the LIWC 2007 software, which calculated the frequencies and percentage of words used for a given category contained within each diary. The present study utilised default pre-defined categories of meaningful psychological constructs provided with the LIWC 2007 software. These categories consist of words and word stems (e.g., light*) pertaining to
32 word subcategories (see Table 5 & 6) tapping into 5 psychological constructs (affective, cognitive, social, perceptual, and biological processes) and 7 personal concern categories (work, achievement, home, leisure activities, money, religion, and death).

Additional categories can also be obtained: 4 general descriptor categories (total word count, words per sentence, percentage of words captured by the dictionary, and percent of words longer than six letters), 22 standard linguistic dimensions (e.g., percentage of words in the text that are pronouns, articles, auxiliary verbs, etc.), 3 paralinguistic dimensions (assents, fillers, non-fluencies), and 12 punctuation categories (periods, commas, etc). These additional categories were omitted from data extraction with the exception of total word count and percentage of words captured by the dictionary. The latter 2 categories were used as covariates in our analysis to control for negative dream entry length and words unrecognised by the software.

A total of 87 negative dream reports were provided, however, in order to not conflate means on study variables such as the Beck Depression Inventory – II, only one report per participant was selected for analysis from those participants (n= 25) who had submitted more than one negative dream during the course of the diary. The negative dream reports were selected based on stepped criteria: (i) nightmares over bad dreams - reports where the negative dream had woken up the participant were selected above those which had not awoken participants; (ii) for either multiple nightmares or multiple bad dreams in the absence of nightmares, the dream with the highest cumulative severity score (intensity, vividness and distress) was selected; and (iii) in the event that multiple negative dream reports had the same cumulative score, the report with
the highest score on distress was selected. This method ensured that only one report was selected from each participant. A total of 47 negative dreams (17 bad dreams and 30 nightmares) were selected for the analyses.

4.2.6. Data analysis procedure

Means and standard deviations were calculated for both SH group and no SH group participants. The LIWC automatically scores categories as zero if no words in the participants report correspond to this category. Additional descriptive statistics for groups following self-harm recency (no SH, history of SH, and current SH) were calculated for exploratory analyses (see Appendix I).

To test the hypothesis that participants with a history of self-harm would use more death words compared to participants with no history of self-harm, ANCOVA was performed. Participant grouping (SH group vs. No SH group) was entered as the between participant factors. Depressive symptoms, word count and percentage of word captured by the dictionary were entered as covariates. The ‘death’ word category was entered as the dependent variable.

To assess if that participants with a history of self-harm would differ compared to participants with no history of self-harm in their use of words pertaining to the 5 psychological constructs (affective, cognitive, social, perceptual, and biological processes) and remaining 6 personal concerns (work, achievement, home, leisure activities, money, and religion), MANCOVA was performed. Participant grouping (SH group vs. No SH group) was entered as the between participant factors. Depressive symptoms, word count and percentage of word captured by the dictionary were entered as
covariates. The 5 psychological constructs and 6 personal word categories were entered as the dependent variables.

Further exploratory analyses investigating self-harm recency were performed and are reported in Appendix I. Method for these analyses are detailed below.

Our first exploratory analysis of self-harm recency (no SH, history of SH, and current SH) assessed group differences on the 5 psychological constructs (affective, cognitive, social, perceptual, and biological processes) and 7 personal concerns (work, achievement, home, leisure activities, money, religion and death) was performed using MANCOVA. Participant grouping (no SH/SH history/current SH) was entered as the between participant factors. Depressive symptoms, word count and percentage of word captured by the dictionary were entered as covariates. The 5 psychological constructs and 7 personal word categories were entered as the dependent variables.

A further exploration of psychological constructs’ subcategories was performed where group differences (no SH/SH history/current SH) had been found for the one of the 5 psychological constructs. This was performed using MANCOVA where participant group was entered as the between group factor. Depressive symptoms, word count and percentage of word captured by the dictionary were entered as covariates. The subcategories of interest were entered as the dependent variables.
4.3. Results

4.3.1. Descriptive statistics

Descriptive statistics for the no SH and SH history groups are reported in Tables 5 and 6. There were no significant differences between the no SH group ($M=22.21, SD=4.23$), SH history group ($M=21.33, SD=3.71$) on age, $t(45)=-.72, p>.05$. Moreover, there were no significant differences between groups on word count ($t(45)=.33, p>.05$) or percentage of words captured by the dictionary ($t(45)=-.08, p>.05$). However, SH history participants reported significantly higher levels of depressive symptoms than the no SH group, ($t(45)=2.46, p<.05$).
<table>
<thead>
<tr>
<th></th>
<th>No SH (n= 14)</th>
<th>SH history (n= 33)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Depressive symptoms</strong></td>
<td>11.57 (6.33)</td>
<td>18.94 (10.40)</td>
</tr>
<tr>
<td><strong>Word count</strong></td>
<td>98.43 (80.87)</td>
<td>108.15 (98.39)</td>
</tr>
<tr>
<td><strong>% captured by dictionary</strong></td>
<td>92.13 (6.97)</td>
<td>92.01 (3.79)</td>
</tr>
<tr>
<td><strong>Social processes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Family</em></td>
<td>1.61 (2.28)</td>
<td>.75 (1.27)</td>
</tr>
<tr>
<td><em>Friends</em></td>
<td>.58 (1.45)</td>
<td>.57 (1.13)</td>
</tr>
<tr>
<td><em>Humans</em></td>
<td>.92 (1.38)</td>
<td>1.07 (1.34)</td>
</tr>
<tr>
<td><strong>Affective processes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Positive emotion</em></td>
<td>.61 (.91)</td>
<td>1.56 (2.56)</td>
</tr>
<tr>
<td><em>Negative emotion</em></td>
<td>4.41 (3.91)</td>
<td>3.74 (2.75)</td>
</tr>
<tr>
<td><em>Anxiety</em></td>
<td>.76 (1.40)</td>
<td>1.22 (1.59)</td>
</tr>
<tr>
<td><em>Anger</em></td>
<td>1.34 (1.53)</td>
<td>1.12 (1.49)</td>
</tr>
<tr>
<td><em>Sadness</em></td>
<td>1.72 (3.60)</td>
<td>.97 (1.22)</td>
</tr>
<tr>
<td><strong>Cognitive processes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Insight</em></td>
<td>3.53 (3.64)</td>
<td>2.21 (2.29)</td>
</tr>
<tr>
<td><em>Causation</em></td>
<td>1.59 (1.71)</td>
<td>1.12 (1.34)</td>
</tr>
<tr>
<td><em>Discrepancy</em></td>
<td>.98 (1.46)</td>
<td>1.19 (1.28)</td>
</tr>
<tr>
<td><em>Tentative</em></td>
<td>1.11 (.95)</td>
<td>3.24 (4.79)</td>
</tr>
<tr>
<td><em>Certainty</em></td>
<td>.55 (.89)</td>
<td>1.38 (2.03)</td>
</tr>
<tr>
<td><em>Inhibition</em></td>
<td>.64 (1.22)</td>
<td>.73 (1.74)</td>
</tr>
<tr>
<td><em>Inclusive</em></td>
<td>6.06 (3.44)</td>
<td>7.78 (3.77)</td>
</tr>
<tr>
<td><em>Exclusive</em></td>
<td>2.55 (1.80)</td>
<td>2.83 (2.44)</td>
</tr>
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Table 6 - Linguistic frequency per self-harm categories (cont.)

<table>
<thead>
<tr>
<th></th>
<th>No SH (n= 14 )</th>
<th>SH history (n= 33 )</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Perceptual processes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>See</td>
<td>2.01 (1.91)</td>
<td>4.47 (3.72)</td>
<td></td>
</tr>
<tr>
<td>Hear</td>
<td>.62 (.84)</td>
<td>2.26 (2.48)</td>
<td></td>
</tr>
<tr>
<td>Feel</td>
<td>.31 (1.04)</td>
<td>.55 (.99)</td>
<td></td>
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<tr>
<td><strong>Biological processes</strong></td>
<td></td>
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<tr>
<td>Body</td>
<td>.79 (1.43)</td>
<td>.98 (1.56)</td>
<td></td>
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<tr>
<td>Health</td>
<td>.76 (1.55)</td>
<td>.43 (1.04)</td>
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<tr>
<td>Sexual</td>
<td>.17 (.65)</td>
<td>.11 (.39)</td>
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<tr>
<td>Ingestion</td>
<td>.58 (1.49)</td>
<td>.29 (.80)</td>
<td></td>
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<tr>
<td>Relativity</td>
<td>15.23 (6.20)</td>
<td>15.69 (4.98)</td>
<td></td>
</tr>
<tr>
<td>Motion</td>
<td>2.20(1.89)</td>
<td>2.86 (2.30)</td>
<td></td>
</tr>
<tr>
<td>Space</td>
<td>9.5 (5.91)</td>
<td>8.61 (3.41)</td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>3.35 (2.66)</td>
<td>4.31 (2.75)</td>
<td></td>
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<tr>
<td><strong>LIWC Personal Concerns</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Work</td>
<td>1.50 (2.77)</td>
<td>1.61 (3.12)</td>
<td></td>
</tr>
<tr>
<td>Achievement</td>
<td>1.47 (1.72)</td>
<td>.74 (1.01)</td>
<td></td>
</tr>
<tr>
<td>Leisure</td>
<td>2.46 (2.24)</td>
<td>1.96 (2.63)</td>
<td></td>
</tr>
<tr>
<td>Home</td>
<td>1.09 (2.42)</td>
<td>1.45 (2.20)</td>
<td></td>
</tr>
<tr>
<td>Money</td>
<td>.62 (1.33)</td>
<td>.06 (.36)</td>
<td></td>
</tr>
<tr>
<td>Religion</td>
<td>.00</td>
<td>.03 (.17)</td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>.66 (1.67)</td>
<td>.61 (2.10)</td>
<td></td>
</tr>
</tbody>
</table>

4.3.2. Do participants with a history of self-harm dream of death?

An ANCOVA examined the effect of self-harm grouping (no SH vs. SH history) on death category words while controlling for the effects of depressive symptoms, word count and percentage of words captured by the LIWC dictionary.

Levene’s test of equality of error variance indicated homogeneity assumptions had been met, $F(1, 45)= 0.01, p>.05$. The ANCOVA did not reveal any significant difference between SH groups on death words after controlling for depressive symptoms, word count and percentage of words captured by the dictionary, $F(1, 42) = .003, p>.05$, partial $\eta^2 = .00)$. Results
indicate that participants with a history of self-harm and those without do not express differing amount of death related words in their negative dream reports.

4.3.2. Do the negative dreams of participants with a history of self-harm differ from those without on psychological constructs and personal concerns?

A MANCOVA examined the effect of self-harm grouping (no SH vs. SH history) on the 5 psychological constructs (affective, cognitive, social, perceptual, and biological processes) and remaining 6 personal concerns (work, achievement, home, leisure activities, money, and religion) while controlling for the effects of depressive symptoms, word count and percentage of words captured by the dictionary.

In the first instance, Box’s M test could not be computed. This was due to religion words having a zero value for the No SH group. Religion was removed from our dependent variables. MANCOVA was performed with religion removed. A significant Box’s M test (Box M = 12.17, p<.05) indicated assumptions of homogeneity of covariance had been violated. As such Pillai's trace was used as the multivariate test criterion. Pillai’s trace is the most conservative estimate recommended when homogeneity assumptions are not met (Tabachnick & Fidell, 2001). The multivariate effect was non-significant, $F(11, 32) = 1.58, p>.05$, $\lambda_{Pillai} = .35$, partial $\eta^2 = .35$. Similarly, the test of between participant effects showed no significant differences between SH group and no SH group on the 5 psychological constructs; social processes ($F(1, 42) = .05, p>.05$, partial $\eta^2 = .001$), affective processes ($F(1, 42) = .13, p>.05$, partial $\eta^2 = .003$), cognitive processes ($F(1, 42) = .65, p>.05$, partial $\eta^2$
= .015), perceptual processes \((F(1, 42) = 3.60, p=.065, \text{partial } \eta^2 = .079)\), and biological processes \((F(1, 42) = .54, p>.05, \text{partial } \eta^2 = .013)\). Moreover, there were no significant differences between SH group and no SH group participants on personal concerns relating to work \((F(1, 42) = .04, p>.05, \text{partial } \eta^2 = .001)\), achievements \((F(1, 42) = 3.19, p=.081, \text{partial } \eta^2 = .071)\), leisure activities \((F(1, 42) = .85, p>.05, \text{partial } \eta^2 = .020)\), home \((F(1, 42) = .37, p>.05, \text{partial } \eta^2 = .009)\). However, negative dreams reports of no SH group participants were higher in money words than SH group participants, \((F(1, 42) = 6.44, p<.05, \text{partial } \eta^2 = .133)\).

Religion was run through ANCOVA. This revealed no significant differences between groups, \((F(1, 42) = 1.41, p>.05, \text{partial } \eta^2 = .032)\).
4.4. Discussion

4.4.1. Do participants with a history of self-harm dream of death?

It was predicted that negative dream reports from participants with a history of self-harm would contain significantly more linguistic content related to death than participants without a history of self-harm. However, this hypothesis was not supported in the present study upon controlling for the effects of depressive symptoms, length of dream reports or percentage of words captured by the default LIWC dictionary. Thus, contrary to previous findings, (Langs, 1966; Ralphing, 1970; Firth et al., 1986; Evans, 1990) participants with a history of self-harm did not report elevated levels of death related words irrespective of depressive symptoms compared to their control counterparts. Furthermore, our exploratory analysis reported in the Appendix I indicated that participants currently engaging in self-harm (<1 month) did not report any death related words within their negative dream reports.

Explanations for this discrepancy may lie within the different methodological approaches between the literature and the present study. Previous studies utilised thematic text analysis, and whilst perhaps rigorous in its implementation, the use of subjective methods and its reliance on inter-rater reliability remains open to bias. However, the present study, with its use of computerised text analysis software provides an objective measurement of frequency of words utilised by participants. Though it is true that LIWC analysis does not take into account contextual factors as thematic analysis would, Tausczik & Pennybaker (2010) argue that word frequency within text reflects attentional focus. Thus, death related words should be reported more often in participants whose focus revolves around death. A replication of this
study specifically measuring suicidal intent and its relation to negative dream content would provide further insight.

In addition, the retrospective nature of data gathering methods in the literature, with a delay ranging from 2-21 days (Ralphing, 1970) and even up to 6 months (Firth et al., 1986) is problematic, potentially affecting the detail of their reported memories. Participants were asked to recall the worst dream they had experienced prior to their suicide attempt, which could have led participants to subjectively reinterpret of their dreams in the context of the salient suicide attempt, and introduced a stronger death-themed bias within their reports. This is in stark contrast to our longitudinal methodology where dream reports are obtained on the day of dream occurring, ensuring low levels of such contextual influence on interpretation and maximising level of details.

Alternatively, the present study investigated negative dream content by comparing participants with a history of self-harm regardless of suicidal intent (and those currently engaging in self-harm – see Appendix I), to participants without any prior self-harm episode. The literature meanwhile focused on participants who had attempted suicide, implying clear levels of intent. It is therefore possible that intent could be a deciding factor in relation to the level of death related content within nightmare reports. Thus the discrepancy between our finding and those of the literature could be accounted for by the dilution of suicidal intent across our self-harm participants due to the inclusion of all self-harm episodes regardless of intent or motivation to die.
4.4.2. Exploratory analysis

The results of the exploratory analysis yielded interesting between group differences. The no SH group provided reports containing more references to money than the SH group. Additionally, a marginally non-significant difference in perceptual processes was detected indicating SH group participants used more perceptual words compared to the so SH group. A further exploratory analysis of self-harm recency (Appendix I) indicated that currently self-harming participants use more words related to the ‘feel’ subcategory of perception and more words related to the ‘body’ subcategory compared to participants without self-harm and those with a self-harm over one months ago.

Dreams are thought to reflect recombined memories for fear extinction (Nielsen & Levin, 2007). More precisely, the current affective load experienced by an individual is said to dictate the need for the formation of new fear extinction memories. These memories are created during the dream process which dissociate and recombine attributes of fear memories. The memories are then recreated into a new potentially fear extinguishing context. However, nightmares occur due to a failure within this process whereby the recombined memory is consistent to waking state fear memories, akin to phobias or social anxiety. The increased frequency of words relating to perception specifically feeling, and body, in individuals with current self-harm may reflect the incorporation of distressing memories related to self-harm into negative dream content. Thus, this content type may be more prevalent due to the increased affective load associated with sensations and body parts in participants currently engaging in self-harm compared to those who have not
recently self-harmed (history of SH >1 month) or those who have never self-
harmed.

Though exploratory, our results suggest group differences independent of
the effects of depressive symptoms, word count, and words captured by the
default LIWC dictionary. Importantly, our exploratory analysis showed that
the linguistic frequency of perception, specifically feeling, and body categories
differed between current and self-harm history individuals. As such, changes in
an individual’s dream content relating to the aforementioned categories may
serve as a marker of increased vulnerability for self-harm.

4.4.3. Limitations and further research

Due to the exploratory nature of our results, the findings regarding
differences in perceptual processes detailed above should be interpreted with
cautions. Replication with a priori hypothesis investigating differences in
perceptual processes, ‘feel’ and ‘body’ words needs to be performed. In
addition, a larger sample of participants currently engaging in self-harm, which
could not be recruited specifically on this criterion due to ethics restrictions, is
advised to validate these findings. Moreover, while death categories words did
not appear to be more prevalent in participants with self-harm compared to
those without, the discrepancy between our findings and the literature may be
linked to the literature’s focus of behaviours with clear suicidal intent.
Replications which control for suicidal intent at the time of the dream report
may help elucidate if suicidal intent is reflected in negative dreams via themes
of death.
The negative dream reports analysed in this study were collected in conjunction with the data presented in Chapter 3. As such, it is possible to link specific negative dreams with specific occurrences of self-injurious thoughts and behaviours (SITBs). A comparison of negative dreams content which elicit SITBs against those which do not would provide a clearer temporal link between content and SITBs. This could possibly allow for the identification of specific triggering content. However, such an analysis could not be performed at this time due to the low number of negative dreams directly linked to post-sleep SITB occurrence (n=9). Replication should consider such an analysis should statistical power allow for these comparisons.

While the LIWC averts common pitfalls of qualitative analysis, it lacks subtlety and omits contextual descriptions in reports and as such pertinent themes (e.g. absence of death words while participant describe jumping off a roof) may go undetected. Therefore, blinded thematic analysis of negative dream reports could be performed. However, it is recommended that dreams be obtained in the longitudinal fashion of the present study to minimise re-interpretation and memory biases.

4.4.4. Key points from Chapter 4

- Prior research of nightmare content of suicidal individuals has several methodological issues – retrospective acquisition of content and subjective methods of analysis.
- The longitudinal method of acquisition for nightmares in self-harming individuals and the use of LIWC software to analyse content has not previously been reported in the literature.
- Contrary to findings in the literature, participants with a history of self-harm do not report more words pertaining to death.
- Exploratory analysis indicates individuals who have recently self-harmed report more words relating to perceptions (see, feel) and the body (arm, leg).

4.4.5. Implications for the next chapters

- Findings of from the diary study (previous and present chapter) indicate differences between individuals with and without a history of self-harm, e.g. lack of morning SITBs reported in participants without self-harm and differences in content.
- The next chapter will focus of modeling the psychological mechanism linking nightmares to self-harm informed by the literature and empirical findings reported in Chapters 2 and 3.
Chapter 5: Investigating the Mechanism Linking Nightmares to Increased Self-Harm Risk Using Structural Equation Modeling

5.1. Introduction

The literature clearly indicates a robust association between nightmares and suicidal behaviour (Bernert & Joiner, 2007). Several articles have theorised the potential mechanism linking nightmares and increased suicidality, utilising knowledge from the dreaming and sleep literature. Notably, Cukrowicz et al., (2006) built upon the idea of emotional exhaustion put forward by Krakow et al., (2000) and suggested that the content of nightmares could increase stress levels and negative cognition experienced during waking hours. This assertion is in line with the dream literature which has demonstrated that the relationship between pre-sleep and post-sleep affect is moderated by dream affect (Agargun & Cartwright, 2003). That is, negative affect pre-sleep can be reduced or increased depending on the level of negative affectivity experienced during dreams. Furthermore, it reflects recent theoretical propositions made by Nielsen and Levin (2007, 2009) which frame nightmares as a dysfunction of normal regulatory processes of dreaming. Cukrowicz et al. (2006) further posited that those who had experienced nightmares would suffer from increased levels of negative affect in the morning due to the reduction of overnight emotional regulation. This increased negative affectivity would make individuals who had experienced nightmares more sensitive to stressors and suicidal cues during the day due to the reduction in coping capacities stemming from mood dysregulation. In turn, these cues would lead people to be at increased risk for suicidal ideation. While theoretically sound, detailed description of the constructs and methods
of measurement as well as empirical support for such a mechanism yet to be established.

The above mechanism, whilst focused on suicidal ideation, appears transposable to self-harm regardless of suicidal intent. Indeed findings reported in Chapter 2 have demonstrated that the link between nightmares and suicidal ideation extends to self-harm regardless of intent or motivation to die. Moreover, findings from our diary study (see Chapter 3) highlight that nightmares increase the risk for post-sleep self-injurious thoughts and behaviours (SITBs), while self-harm thoughts or behaviour prior to the sleep period do not increase risk of nightmares. Thus, the predictive directional relationship of nightmares on SITBs and the partially mediating role of post-sleep negative affect between nightmares and post-sleep SITBs supports initial assertions in the literature made by Cukrowicz et al. (2006) and Bernert & Joiner (2007).

The present study aims to further examine the regulatory mechanism proposed in Cukrowicz et al. (2006) in relation to self-harm risk regardless of suicidal intent by implementing a large scale psychometric survey encompassing variables of interest to create a structural equation model. Currently, details relating to this mechanism within the literature are vague and ill defined; precise definitions of increase sensitivity to stressors and suicidal or in this case self-harm cues are not elaborated upon (see section 1.2.6. pp. 31).

In addition to investigating levels of nightmares, self-harm behaviour and negative affect, which are clearly defined constructs, a latent variable will
be created with respect to ‘self-harm cues’. Latent variables are unobserved variables measured via proxies, creating a hypothetical construct representing observed behaviours or cognitions. As they are not directly measured, they benefit from a lack of measurement error. Moreover, multiple latent indicators in SEM avoid collinearity problems and inflated variance explained common to multiple regression techniques (Bollen, 1989). ‘Self-harm cues’ have not been elaborated upon by Cukrowiz et al. (2006) making direct measurement unfeasible. Hence a latent variable will be used to measure these ‘self-harm cues’.

The latent indicator will include experiential avoidance; a class of behaviours with the function to avoid or escape unwanted internal or external experiences (Chapman et al., 2006), due to its association with self-harm and emotion regulation (Chapman et al., 2006). Moreover, defeat from the Cry of Pain model (Williams, 1997) and hopelessness, defined as a cognitive style of negative attributions regarding ones future and helplessness enacting improvement (Klonsky, Kotov, Bakst, Rabinowitz, & Bromet, 2012), will serve as additional latent variable indicators for ‘self-harm cues’. Defeat and hopelessness have been found to be strong predictors of suicidal spectrum behaviours including self-harm (O’Connor, 2003; Rasmussen et al., 2010). Also, significantly higher levels of defeat while controlling for depressive symptoms have been found in participants suffering from elevated nightmare levels (see Chapter 2), further supporting the inclusion of defeat in the present study. Fitting with theory it follows that higher levels of negative affectivity would create a bias increasing perception of events as being defeating and of prospects being hopeless, and increase the need to avoid the phenomenon in
those with elevated experiential avoidant tendencies. Hence, the inclusion of these factors into a latent variable such as ‘self-harm cues’ is deemed appropriate.

Studies have also linked hyperarousal, a state of increased physiological and psychological arousal which reduces tolerance to stressors (Joiner et al., 1999), to increased pain sensitivity and anxiety, exaggerated startled responses, and increased suicidal behaviour (Busch & Fawcett, 2004). While hyperarousal is often linked to insomnia, the disrupted sleep pattern of nightmare sufferers has been suggested to resemble that of chronic insomnia sufferers and stem from hyperarousal (Riemann et al., 2010). More recently, McCall & Black (2013) suggest hyperarousal to mediate the relationship between nightmares and suicidality. McCall and Black do not explicitly test this mediation but suggest it is based on previous findings in the literature (Han, Kim, & Shim, 2012). As such hyperarousal appears to be an apt construct to measure relating well to the vaguely described ‘increased sensitivity to stressors’ mentioned by Cukrowicz et al. (2006).

5.1.1. Aims

This study aims to create a structural equation model relating to the regulatory mechanism proposed by Cukrowicz et al. (2006) with the inclusion of more precisely defined constructs as mentioned above. The resulting model would provide a framework from which to test the assumptions of the mechanism linking nightmares to increased likelihood of self-harm. Previous findings (see chapter 3) indicated that links between nightmares and self-harm thoughts and behaviours were not computable in participants who reported no
history of self-harm. This suggested the link between nightmares and self-harm is constrained to those with who have lifetime history of self-harm engagement. As such, the present study will focus on modeling the underlying mechanism linking nightmares to self-harm in participant reporting self-harm issues with the aim of providing a predictive model of current self-harm (<1 month).

In line with the aforementioned literature, a successful structural equations model should incorporate the 4 following assumptions:

(i) Nightmares significantly predict increased negative affect.

(ii) High levels of negative affect will significantly predict elevated levels of hyperarousal (sensitivity to stressors).

(iii) Hyperarousal will significantly predict elevated suicidal cues (defeat, experiential avoidance, hopelessness).

(iv) High levels of self-harm cues will significantly predict current self-harm (<1 month).

The model, meeting goodness of fit criterion and the four assumptions listed above shall be retained in view of assessing the strength of the indirect effect of nightmares on current self-harm.
5.2. Methodology

5.2.1. Design and procedure

A cross-sectional questionnaire study design was implemented. Data was again collected by means of a web survey similar to that of our first survey (see Chapter 2). The design of the questionnaire and the order of the scales followed recommendations for internet based surveys by Dillman, Smyth & Christian (2008). The order of the measures followed the general order advertised to promote engagement, with relevant questions to the wider population (nightmares) were listed first to encourage continued participation. The most sensitive measure (self-harm) remained at the end of the questionnaire. The survey following a multiple page design where by each scale would be fitted onto a single page. The response format for each scale was selected to reduce completion time.

Participants complete the survey in one sitting without time limit although they were instructed not to over think their answers too much and to provide their initial responses. Participants were first given instructions and an electronic consent form followed by simple demographic questions. Each questionnaire was presented on a single page. Prior to the self-harm measure, an instruction page was displayed to remind participants that their answers would be anonymous and that their honest answers would be of the outmost importance to this study. Having completed and submitted their responses, participants were redirected to the Personality Social Psychology and Health research group website where they could obtain further information on our group’s research activities. Participants who completed the online survey (and provided a contact email address) were entered into a £100 lottery.
Prior to data collection, the survey was piloted on a small sample derived from postgraduate students from the School of Psychology at the University of Nottingham. This allowed for verification of layout and text proof reading as well as the checking the functionality of the survey’s “exit from questionnaire” link permitting participants to withdraw from the study at any time. Piloting the survey revealed completion time to be approximately 15 minutes.

5.2.2 Participants

A total of 1151 participants attempted the questionnaire. Participants were recruited through convenience sampling. The questionnaire was advertised to participants as “Nightmares, mood and wellbeing”. Participants could access the questionnaire by following a web link circulated in the recruitment e-mail and on social networking sites. The questionnaire was advertised online (Facebook) and by email throughout the University of Nottingham schools and departments over the course of 10 months.

Participants were excluded from the analysis due to improper completion (i.e. they did not attempt all question sets) or omitted demographic information (age, gender, occupation, current use of medication or psychiatric diagnosis). Participants who had attempted all questions set but who had small amounts of missing data values (no more than 1 per scale) where included. Participants were excluded from the study if replying positively to current use of medication; as many pharmaceuticals affecting dopamine, serotonin, GABA, norepinephrine or acetylcholine have been linked to the induction of nightmares as a side effect (Pagel & Helfter, 2003). Similarly, participants replying positively to having psychiatric diagnoses were also excluded as they
could confound findings in relation to nightmares and self-harm behaviour due
to co-morbidity and potential for medication use which could alter normal
sleep. Means were calculated and listwise deletion was used to remove
participants with missing mean scores. Three hundred and eight participants
withdrew or did not adequately complete the measures within the survey and
were thus excluded (71 of which did not provide demographic information).
Following our exclusion criteria relating to current psychiatric diagnosis and
medication use, a further 281 participants were excluded. The remaining 562
participants (100 males) aged 18-58 ($M= 20.74$ years old, $SD= 4.71$)
adequately completed all measures. This sample was comprised of 204
participant with a history of self-harm (>1month) and of 48 participants with
current self-harm (<1month). The remaining 308 participants reported no self-
harm issues. As previously mentioned, the present study aims to build a
predictive model of current self-harm (<1 month) from participants reporting
self-harm issues (any self-harm over the lifetime). Thus, our analysis focuses
on the 252 participants (35 males) reporting either current or a history of self-
harm. These participants were aged 18-45 years old ($M= 20.45$ years old, $SD= 3.60$).

5.2.3. Measurements

The survey was composed of seven psychometric scales. Additional
demographic questions were included at the start of the survey. Scales are
detailed below in the order of their appearance:

The *Disturbing Dream and Nightmare Severity Index* (DDNSI; Krakow,
2006) assesses frequency and severity of participants’ disturbing dreams and
nightmares of the past seven days. The five item scale measures frequency of nights of nightmare, the number of nightmares, the frequency of nightmare related awakening, participants’ perceived severity of the problem and, the intensity of the nightmare experienced. Scores on this scale range from 0-37 with higher scores indicating greater severity of nightmare complaint. It was selected to measure participant’s nightmare levels due to its brevity and ability to predict clinically significant nightmares (Krakow, 2006) and will be used as the exogenous variable in our model. The Cronbach’s alpha of the DDNSI for this sample was $\alpha = .80$.

The **Negative Affect subscale** (Watson, Clark & Tellegen, 1988) from the *Positive Affect and Negative Affect Schedule* (PANAS) measures state or trait predisposition to positive and negative affect depending on instructions associated with time (last week or currently) given to participants. For the purpose of this study only the negative affect (NA) subscales is used in the analysis with time instructions relating to affect experienced in the last week to reflect the time instructions of other measure within the survey (e.g. DDNSI, IES-R). However, both the positive and negative affect subscales were presented to participants in a set randomised order. The 10 item NA subscale contained negative adjective rated on 5 point Likert like scale ranging from 1 to 5. Higher scores represent participants’ increased identification with the item (‘very slightly or not at all’ to ‘extremely’). The subscale is brief and easily self-administered, thus was chosen to explore if levels of negative affect in our participants. Our previous findings (see Chapter 2 and 3) obtained using the NA subscale and the literature (Cukrowicz et al., 2006; Cartwright, 2010)
support the use of the subscale in the present study. The Cronbach’s alpha of the NA subscale was $\alpha = .83$.

The **Defeat scale** (Gilbert & Allan, 1998) assesses feelings of defeat and loss of status. This 16 item scale is scored on a 5 point Likert like scale ranging from 0 to 4 (‘never’ to ‘always/all the time’). Higher ratings on an item reflect participants increased feeling of defeat. Items (2, 4, and 9) are reverse scored items where statements are indicative of success rather than defeat. This scale has been validated with students (Gilbert & Allan, 1998) and has been used with those experiencing first time and repeat self-harm (Rasmussen et al., 2010). The defeat scale has been selected to form part of the latent variable referred to by Cukrowicz et al (2006) as self-harm “cues”. Findings from our previous study (see Chapter 2) which demonstrated defeat’s link to nightmares and defeat’s key role in the Cry of Pain model (Williams, 1997) tested on self-harming participants by Rasmussen et al. (2010), provide support for the inclusion of defeat within our model. The Cronbach’s alpha of the defeat scale for this sample was $\alpha = .90$.

The **Acceptance and Action Questionnaire** (AAQ; S. C. Hayes et al., 2004) is a 9 item scale measuring experiential avoidance. Scored on a 1 to 7 Likert like Scale (‘never true’ to ‘always true’), higher total scores indicate greater levels of experiential avoidance. For scoring purposes items 1, 4, 5, and 6 are reversed. The AAQ has been validated on multiple large samples of students, civil servants and psychiatric patients. It was selected to measure participant’s level of experiential avoidance, closely implicated with self-harm (Chapman et al., 2006). It was selected to form part of the self-harm “cues” latent variable due to the strong theoretical links between experiential
avoidance, negative affect and hyperarousal (Chapman et al., 2006; S. C. Hayes et al., 2004). The Cronbach’s alpha of the AAQ was $\alpha = .70$. See Appendix K for a copy of the AAQ.

The Impact of Event Scale revised (IES-R; Weiss, 2007) hyperarousal subscale (items 4, 10, 15, 18, 19 & 21) was used to reflect the ‘increased sensitivity to stress’ construct mentioned by Cukrowicz et al. (2006). The IES-R and its hyperarousal subscale have been shown (Weiss, 2007) to be an appropriate instrument to measure subjective responses to hyperarousal (anger, irritability, hypervigilance, difficulty concentrating, and heightened startle and physiological arousal). The instructions on the scale were altered in order to be general rather than trauma specific and ask participants to relate to answer the items in reference to the last 7 days. Items are scored on a 5 point Likert like scale ranging from 0 to 4 (‘not at all’ to ‘extremely’). While there is no specific cut-off score for the IES-R, higher scores indicate greater symptoms count. The IES-R has shown good reliability and validity (Weiss, 2007). The Cronbach’s alpha of the hyperarousal subscale was $\alpha = .74$. See Appendix J for a copy of the IES-R Hyperarousal subscale.

Beck’s Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974) is comprised of 20 dichotomous items (true or false) assessing hopelessness, negative expectations for the future. Participants’ agreement with the items is scored 1, with items 1, 3, 5, 6, 8, 10, 13, 15, and 19 being reverse scored. Greater total scores reflect higher levels of hopelessness. The BHS and the construct of hopelessness was selected to form part of the self-harm “cues” latent variable, along with defeat and experiential avoidance, due to its strong empirically validated link to self-harm (Slee, Spinhoven,
Garnefski, & Arensman, 2008). The Cronbach’s alpha of the hopelessness scale was $\alpha = .91$. See Appendix L for a copy of the BHS.

The *Deliberate Self-Harm Inventory* (DSHI; Gratz, 2001) is a 17 item scale assessing self-harm behaviours corroborated by clinical observations (Gratz, 2001). The scale assesses self-harm behaviour on a yes/no dichotomy. The DSHI has been validated on multiple populations with good test-retest reliability (Gratz, 2001). Instructions to participants were deliberately altered to omit mention of intent, reflecting the definition of self-harm used in this thesis to encompass all self-harm acts regardless of intent or motivation to die (Hawton et al., 2011). One additional item was included asking participant to indicate the last time of their last self-injurious act if applicable. This allowed for the labelling of participants as currently self-harming (<1 month) or history of self-harm (>1 month) which was used as this study’s dependent variable. The Cronbach’s alpha of the DSHI for this sample was $\alpha = .65$.

5.2.5. *Data analysis procedure*

SEM and path analysis relies on parametric assumptions of normality. However, such assumptions have been found to be ignored in large swath of the literature utilising such techniques (Micceri, 1989). This is mainly due to the propensity of non-normal multivariate data and small sample sizes often used in SEM (see West, Finch, & Curran (1995) for a review of the impact of non-normality in SEM). Moreover, SEM has difficulties computing models which include dichotomous variables due to the large resulting standard error which greatly inflate chi-square values. However, several methods reviewed by Kupek (2005) have been proposed to enable researchers to overcome these
difficulties and compute models with non-normal variables. Firstly, it is proposed by Browne (1984) that asymptotic distribution free (ADF) estimators be used to account for problematic kurtosis in multivariate data sets. However, several restrictive assumptions complicate the use of ADF, such as, the need for very large data sets, a relatively low number of variables included in a model and, the assumption of an underlying continuous scale in categorical variables. Bollen & Stine (1993) alternatively advocated for using robust maximum likelihood (ML) estimation or the use of bootstrapping. The latter also being recommended by West et al. (1995). However, Green, Akey, Fleming, Hershberger, & Marquis (1997) note that binary variables greatly influence chi-square statistic used as an indicator of model fit. So, although models with categorical variables of 3 or more categories would benefit, models with binary variables would continue to suffer from chi-square inflation. Jöreskog & Sörbom (1996) recommend the use of tetrachoric, polychoric or polyserial correlations between non-normal variable pairs assuming the variable to have an underlying continuous scale. The correlation is then to be used in the model. Lastly, Muthen (1993) advocates employing probit or logit estimators for categorical variables within the model in the first instance to ascertain model fit. If model fit during the initial analysis is deemed acceptable, SEM is then re-run (with ADF or robust ML depending on model) to obtain model coefficients. This is necessary as probit regression model do not provide significance values for pathways within models. Linear probability model residuals violate the homoscedasticity and normality of errors assumptions of Ordinary Least Square regression resulting in invalid standard errors and hypothesis tests.
With the issues of non-normal distribution and our use of dichotomous variables (current self-harm coded as 1 and history of self-harm coded as 0) in mind, the assumptions of multivariate normality and linearity were evaluated for the present data using PASW Statistic 18. Kolmogorov–Smirnov statistic, box plots and scatter-graphs of the standardised residuals were used to assess normality. The data was found to be non-normally distributed with large positive skewed for all variables (see Table 7).

Structural equation models were carried out using IBM SPSS AMOS 21. AMOS 21 (Arbuckle, 2012) provides the user with the ability to fit models specified to have an endogenous categorical binary variable (see AMOS user’s guide example 33) using Bayesian methods only. Estimation options for our analysis were set to Maximum likelihood, mean and intercept estimation were also computed. Bayesian SEM estimation employed MCMC (Makrov Chain Monte Carlo) algorithm. This process automatically rescales our binary variable and allowing AMOS to run a probit model.

Bayesian SEM does not provide the user with the familiar chi-square index of model fit nor does it offer modification indices, valuable for model refinement. Instead, posterior predictive P (PPP) values (Meng, 1994) are provided to indicate model fit. PPP values and their interpretations differ from R. A. Fisher’s (1925) traditional p-value and established threshold. Model fit is deemed good the closer the PPP is to .5 (Meng, 1994) with values closer to 1 and 0 (.95 and .05 thresholds) indicating poorer fit. Moreover, Bayesian estimation should be interpreted differently form ML estimates. The confidence intervals are used to indicate 95% probability that the estimates fall between the upper and lower values of the interval. When the intervals does
not include zero, the effect is assumes to be present and the null hypothesis can be rejected.

Thus, the present analysis was performed following the technique recommended by Muthen (1993) assessing models for goodness of fit using PPP values in the first instance and obtaining Bayesian coefficient estimates. Models were then re-run using bootstrapped ML estimation to verify Bayesian estimates by comparing with ML coefficients and, obtain modification indices for model refinement. Following model modification indices and in keeping with theoretical underpinning, the model regression pathways were refined. The process was repeated until a model with a suitable goodness of fit was obtained. Upon identification of a fitting model, direct of the variables and the indirect effects of nightmares of current self-harm was computed.
5.3. Results

5.3.1. Descriptive Statistics

Means and standard deviation for our sample and z-score for skew and kurtosis are reported in Table 7. Zero order correlations of our continuous variables are provided for the entire sample in Table 8.

Table 7 - Descriptive statistics for full sample and split by self-harm recency

<table>
<thead>
<tr>
<th></th>
<th>Full sample (n=252)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Age</td>
<td>20.45 (3.60)</td>
</tr>
<tr>
<td>Nightmares</td>
<td>7.19 (4.55)</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>24.57 (7.21)</td>
</tr>
<tr>
<td>Hyperarousal (sensitivity to stress)</td>
<td>1.24 (.77)</td>
</tr>
<tr>
<td>Defeat (cues)</td>
<td>23.55 (10.29)</td>
</tr>
<tr>
<td>Experiential Avoidance (cues)</td>
<td>33.35 (6.51)</td>
</tr>
<tr>
<td>Hopelessness (cues)</td>
<td>6.65 (5.25)</td>
</tr>
</tbody>
</table>

All correlations were significant and effect size ranged from small positive to large positive associations. The correlations revealed that, in keeping with past literature, nightmares were significantly positively correlated to increased negative affect, hyperarousal and, in keeping with our findings reported in Chapter 2, associated to elevated levels of defeat. Nightmares were also significantly positively correlated to experiential avoidance and hopelessness. Hyperarousal scores showed a particularly strong significant positive association to negative affect. A very strong positive correlation was also observed between defeat and hopelessness.
Table 8 - Zero order correlations for continuous variables

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Nightmares</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>2. Negative Affect</td>
<td>.257***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Hyperarousal</td>
<td>.373***</td>
<td>.620***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Defeat</td>
<td>.287***</td>
<td>.660***</td>
<td>.550***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Experiential Avoidance</td>
<td>.194***</td>
<td>.527***</td>
<td>.459***</td>
<td>.647***</td>
<td></td>
</tr>
<tr>
<td>6. Hopelessness</td>
<td>.248***</td>
<td>.503***</td>
<td>.445***</td>
<td>.732***</td>
<td>.605***</td>
</tr>
</tbody>
</table>

*** = p<.001

5.3.2. Structural equation model – Model 1

Following the description of the mechanism by Cukrowicz et al. (2006), an initial model was created. Model no.1 follows the basic linear relationship between variables described in the literature. Namely, that nightmares regress onto negative affect; negative affect regresses onto hyperarousal (‘sensitivity to stress’) which in turn regresses onto a latent variable composed of defeat, experiential avoidance and hopelessness (note that hopelessness is used a weighting again which other latent indicators are estimated). This latent variable, ‘self-harm cues’ then regresses onto our endogenous variable current self-harm (model is predicting outcome current self-harm). This model is depicted in Figure 5 with standardised ML model estimates.
Model convergence set at $< 1.002$, occurred after approximately $n= 5200$ samples. Bayesian estimation indicated posterior predictive p-value $= .00$. This indicates poor fit for Model 1. Model 1 was re-run using robust ML estimation (bootstrap 200 re-sample) treating the dichotomous variable as continuous, thus providing modification indices for model refinement. This method similarly demonstrated poor model fit $[\chi^2 (df=14, n=252) = 107.18, p<.001; \text{CFI} = .868; \text{RMSEA}= .163 \text{ (90\% C.I. } =.135 - .518)]$. However, pathway coefficient estimation shown in Table 9 indicated all paths to be significant at $p<.001$ level. Bayesian estimates indicate unstandardized total effect. Note the lack of estimates for residual 2 as this is fixed at 1 when variable is specified to be dichotomous.
## Table 9 - Model 1 Bayesian and ML estimates (unstandardised)

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Bayesian Estimate</th>
<th>S.E.</th>
<th>S.D.</th>
<th>95% Lower bound</th>
<th>95% Upper bound</th>
<th>ML Estimate</th>
<th>S.E.</th>
<th>C.R.</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Regression weights</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Negative Affect</em> &lt;--- <em>Nightmares</em></td>
<td>.260</td>
<td>.003</td>
<td>.062</td>
<td>.135</td>
<td>.375</td>
<td>.259</td>
<td>.061</td>
<td>4.221</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>Hyperarousal</em> &lt;--- <em>Negative Affect</em></td>
<td>.633</td>
<td>.002</td>
<td>.05</td>
<td>.534</td>
<td>.732</td>
<td>.633</td>
<td>.051</td>
<td>12.507</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>Self-harm cues</em> &lt;--- <em>Hyperarousal</em></td>
<td>.568</td>
<td>.002</td>
<td>.06</td>
<td>.450</td>
<td>.692</td>
<td>.571</td>
<td>.059</td>
<td>9.694</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>Hopelessness</em> &lt;--- <em>Self-harm cues</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>Experiential Avoidance</em> &lt;--- <em>Self-harm cues</em></td>
<td>.756</td>
<td>.003</td>
<td>.063</td>
<td>.642</td>
<td>.886</td>
<td>.754</td>
<td>.062</td>
<td>12.242</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>Defeat</em> &lt;--- <em>Self-harm cues</em></td>
<td>.893</td>
<td>.002</td>
<td>.062</td>
<td>.779</td>
<td>.022</td>
<td>.891</td>
<td>.061</td>
<td>14.702</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>Current Self-harm</em> &lt;--- <em>Self-harm cues</em></td>
<td>.725</td>
<td>.008</td>
<td>.147</td>
<td>.453</td>
<td>.036</td>
<td>.200</td>
<td>.032</td>
<td>6.307</td>
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<tr>
<td><em>Nightmares</em></td>
<td>.705</td>
<td>.002</td>
<td>.060</td>
<td>.595</td>
<td>.832</td>
<td>.687</td>
<td>.061</td>
<td>11.203</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>e1</em></td>
<td>.666</td>
<td>.002</td>
<td>.061</td>
<td>.560</td>
<td>.802</td>
<td>.649</td>
<td>.058</td>
<td>11.203</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>e2</em></td>
<td>.458</td>
<td>.001</td>
<td>.041</td>
<td>.387</td>
<td>.545</td>
<td>.447</td>
<td>.04</td>
<td>11.203</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>resd1</em></td>
<td>.423</td>
<td>.003</td>
<td>.064</td>
<td>.311</td>
<td>.571</td>
<td>.410</td>
<td>.058</td>
<td>7.08</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><em>e3</em></td>
<td>.127</td>
<td>.001</td>
<td>.026</td>
<td>.077</td>
<td>.180</td>
<td>.122</td>
<td>.025</td>
<td>4.899</td>
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<tr>
<td><em>e4</em></td>
<td>.323</td>
<td>.002</td>
<td>.037</td>
<td>.257</td>
<td>.398</td>
<td>.316</td>
<td>.034</td>
<td>9.4</td>
<td>&lt;.001</td>
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<tr>
<td><em>e5</em></td>
<td>.360</td>
<td>.001</td>
<td>.045</td>
<td>.276</td>
<td>.451</td>
<td>.349</td>
<td>.043</td>
<td>8.115</td>
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<tr>
<td><em>resd2</em></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.128</td>
<td>.012</td>
<td>10.918</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>
5.3.3. Structural equation model – Model 2

A second model was created following modification indices obtained in Model 1. This model is depicted in Figure 6 with standardised ML model estimates. This model diverged from the description made by Cukrowicz et al. (2006) as displayed in Model 1 by the inclusion of a direct predictive pathway from nightmares to hyperarousal which is supported by the literature on hyperarousal (Riemann et al., 2010; Simor et al., 2012). Moreover, as suggested by the modification indices, hyperarousal is now predicted by both nightmares and negative affect and, in turn predicts self-harm cues. The remainder of the model was not altered from Model 1.

![Figure 6 - Model 2 with standardised ML estimates](image-url)
Model convergence set at < 1.002, occurred after approximately n= 6200 samples. Bayesian estimation indicated posterior predictive p-value = .28. This indicates adequate model fit. However, further refinement is needed. Model 2 was re-run using robust ML estimation (bootstrap 200 re-sample) treating the dichotomous variable as continuous to obtain model modification indices. Robust ML estimation similarly demonstrated model fit which did not diverge significantly from the data [$\chi^2$ (df=12, n=252) = 19.99, $p$=.067; CFI= .989; RMSEA= .052 (90% C.I. =.000 - .090)]. As with the previous model, pathway coefficient estimation shown in Table 10 indicated all paths to be significant at $p<.001$ level. Bayesian estimates indicate unstandardized total effect. Residual 2 estimates are missing as this is fixed at variance of 1 when variable is specified to be dichotomous. While this model improves upon Model 1 and shows moderate fit to the data, further refinement is needed.
<table>
<thead>
<tr>
<th>Pathway</th>
<th>Bayesian Estimate</th>
<th>S.E.</th>
<th>S.D.</th>
<th>95% Lower bound</th>
<th>95% Upper bound</th>
<th>ML Estimate</th>
<th>S.E.</th>
<th>C.R.</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Regression weights</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative Affect &lt;--- Nightmares</td>
<td>.26</td>
<td>.001</td>
<td>.062</td>
<td>.141</td>
<td>.381</td>
<td>.259</td>
<td>.061</td>
<td>4.221</td>
<td>&lt;.001</td>
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<tr>
<td>Hyperarousal &lt;--- Nightmares</td>
<td>.235</td>
<td>.002</td>
<td>.053</td>
<td>.133</td>
<td>.337</td>
<td>.235</td>
<td>.051</td>
<td>4.639</td>
<td>&lt;.001</td>
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<tr>
<td>Hyperarousal &lt;--- Negative Affect</td>
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<td>.001</td>
<td>.051</td>
<td>.474</td>
<td>.675</td>
<td>.573</td>
<td>.05</td>
<td>11.399</td>
<td>&lt;.001</td>
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<tr>
<td>Self-harm cues &lt;--- Negative Affect</td>
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<td>.001</td>
<td>.064</td>
<td>.386</td>
<td>.636</td>
<td>.51</td>
<td>.063</td>
<td>8.048</td>
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<tr>
<td>Self-harm cues &lt;--- Hyperarousal</td>
<td>.247</td>
<td>.001</td>
<td>.059</td>
<td>.136</td>
<td>.364</td>
<td>.249</td>
<td>.058</td>
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<td>Hopelessness &lt;--- Self-harm cues</td>
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<tr>
<td>Experiential Avoidance &lt;--- Self-harm cues</td>
<td>.765</td>
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<td>.064</td>
<td>.646</td>
<td>.901</td>
<td>.763</td>
<td>.063</td>
<td>12.098</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Defeat &lt;--- Self-harm cues</td>
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<td>.001</td>
<td>.061</td>
<td>.812</td>
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<td>.925</td>
<td>.061</td>
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<td>.584</td>
<td>.838</td>
<td>.687</td>
<td>.061</td>
<td>11.203</td>
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<tr>
<td>e1</td>
<td>.663</td>
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<td>.06</td>
<td>.56</td>
<td>.794</td>
<td>.649</td>
<td>.058</td>
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<td>e2</td>
<td>.423</td>
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<td>.022</td>
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<td>.033</td>
<td>9.704</td>
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<td>.478</td>
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<td>.042</td>
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<td>resd2</td>
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<td>-</td>
<td>-</td>
<td>.129</td>
<td>.012</td>
<td>10.961</td>
<td>&lt;.001</td>
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</tbody>
</table>
5.3.4. Structural equation model – Model 3

A 3rd and final model was created refining Model 2 following theoretical and empirical literature. This model is depicted in Figure 7 with standardised ML model estimates. The structure of Model 2 was retained. However, additional covariance constraints were added between the negative affect error term (e1) and the error terms of the latent variable indicators. That is, the error terms for defeat (e3), experiential avoidance (e4) and hopelessness (e5). Rationale for doing so stems from the strong theoretical associations between negative emotional states and the constructs forming the latent indicator (Chapman et al., 2006; Williams, 1997). The error term for hyperarousal (e2) was co-varied to the residual of current self-harm (resd2). The rationale for this additional constraint stems from findings of Busch & Fawcett (2004) linking hyperarousal to suicidal spectrum behaviours and cognitions.
Model convergence set at < 1.002, occurred after approximately n= 8200 samples. Bayesian estimation indicated posterior predictive p-value = .52. This indicates good model fit close to the .5 prescribed by Meng (1994) as an indicator of a model fit. Model 3 was re-run using robust ML estimation (bootstrap 200 re-sample) treating the dichotomous variable as continuous to obtain model estimates and pathway significance levels. Robust ML estimation similarly demonstrated good model fit which did not diverge significantly from the data [$\chi^2$ (df=8, n=252) = 6.78, $p$=.56; CFI= 1.000; RMSEA= .000 (90% C.I. =.000 - .066)]. Pathway coefficient estimations are shown in Table 11 with Bayesian estimates indicative of unstandardised total effects. Residual 2 estimates are missing as this is fixed at variance of 1 when variable is specified to be dichotomous. Regression coefficients were significant at $p$<.001 level.
However, covariance estimates were only significant for hyperarousal error (e2) to current self-harm residuals (resid2)\(^8\). Model 3 will be retained as it is fitting to the dataset and meets the four assumptions made in the literature, namely that:

(i) Nightmares display a positive predictive relationship towards negative affect.

(ii) Negative affect shows a positive predictive relationship towards hyperarousal (sensitivity to stressors).

(iii) Elevate levels of hyperarousal significantly predict elevated self-harm cue levels (defeat, experiential avoidance, hopelessness).

(iv) Self-harm cues significantly predict current self-harm (<1 month).

\(^8\) A tentative variant of Model 3 omitting non-significant covariance estimates (e1 to e3, e1 to e4 and, e1 to e5) while retaining the structure of model 2 and the significant covariance coefficient (e2 to residual 2) was re-analysed. This model showed moderate fit with posterior predictive p-value being .36, an improvement on model 2, yet less fitting than Model 3. Coefficient estimates in this model did not differ from those presented in Model 3.
Table 11 - Model 3 Bayesian and ML estimates (unstandardised)

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Bayesian Estimate</th>
<th>S.E.</th>
<th>S.D.</th>
<th>95% Lower bound</th>
<th>95% Upper bound</th>
<th>ML Estimate</th>
<th>S.E.</th>
<th>C.R.</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Regression weights</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative Affect &lt;--- Nightmares</td>
<td>.249</td>
<td>.002</td>
<td>.062</td>
<td>.131</td>
<td>.373</td>
<td>.258</td>
<td>.06</td>
<td>4.283</td>
<td>&lt;.001</td>
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<td>Hyperarousal &lt;--- Nightmares</td>
<td>.242</td>
<td>.002</td>
<td>.052</td>
<td>.142</td>
<td>.342</td>
<td>.243</td>
<td>.05</td>
<td>4.85</td>
<td>&lt;.001</td>
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<td>.051</td>
<td>.467</td>
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<td>.569</td>
<td>.05</td>
<td>11.32</td>
<td>&lt;.001</td>
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<td><strong>Self-harm cues &lt;--- Negative Affect</strong></td>
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<td>.004</td>
<td>.155</td>
<td>.455</td>
<td>1.054</td>
<td>.818</td>
<td>.233</td>
<td>3.517</td>
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<td>.069</td>
<td>.122</td>
<td>.39</td>
<td>.253</td>
<td>.068</td>
<td>3.71</td>
<td>&lt;.001</td>
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<td>.094</td>
<td>.455</td>
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<td>Defeat &lt;--- Self-harm cues</td>
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<td>.088</td>
<td>.618</td>
<td>.962</td>
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<td>.005</td>
<td>.124</td>
<td>.311</td>
<td>.797</td>
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<tr>
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<td>-.064</td>
<td>.002</td>
<td>.074</td>
<td>-.192</td>
<td>.088</td>
<td>-.092</td>
<td>.099</td>
<td>-.931</td>
<td>.352</td>
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<td>.002</td>
<td>.077</td>
<td>-.2</td>
<td>.103</td>
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<td>.085</td>
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<td>.593</td>
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<td>.099</td>
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<td>.003</td>
<td>.061</td>
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<td>Nightmares</td>
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<td>.061</td>
<td>.596</td>
<td>.831</td>
<td>.687</td>
<td>.061</td>
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<td>.058</td>
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<td>.001</td>
<td>.039</td>
<td>.358</td>
<td>.509</td>
<td>.412</td>
<td>.037</td>
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<td>.05</td>
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<td>-</td>
<td>.13</td>
<td>.012</td>
<td>10.957</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>
As Model 3 was retained, the direct and indirect effects were estimated from the Bayesian structural equation model. Direct and indirect effects reported in Table 12 are standardised to ease interpretation.

<table>
<thead>
<tr>
<th></th>
<th>Nightmares</th>
<th>Negative Affect</th>
<th>Hyperarousal</th>
<th>Self-harm cues</th>
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</thead>
<tbody>
<tr>
<td><strong>Standardized Direct Effects</strong></td>
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<td></td>
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<tr>
<td>Negative Affect</td>
<td>.257</td>
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<td>0</td>
<td>0</td>
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<tr>
<td>Hyperarousal</td>
<td>.236</td>
<td>.555</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Self-harm cues</td>
<td>0</td>
<td>.648</td>
<td>.205</td>
<td>0</td>
</tr>
<tr>
<td>Defeat</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1.022</td>
</tr>
<tr>
<td>Experiential Avoidance</td>
<td>0</td>
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<td>0</td>
<td>.775</td>
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<tr>
<td>Hopelessness</td>
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<td>0</td>
<td>0</td>
<td>1.054</td>
</tr>
<tr>
<td>Current Self-harm</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>.373</td>
</tr>
</tbody>
</table>

|                      |            |                 |              |               |
| **Standardized Indirect Effects** |          |                 |              |               |
| Negative Affect      | 0          | 0               | 0            | 0             |
| Hyperarousal         | .143       | 0               | 0            | 0             |
| Self-harm cues       | .244       | .114            | 0            | 0             |
| Defeat               | .250       | .778            | .21          | 0             |
| Experiential Avoidance | .189     | .59             | .159         | 0             |
| Hopelessness         | .258       | .803            | .216         | 0             |
| Current Self-harm    | .091       | .284            | .077         | 0             |

As can be seen from the indirect effect of nightmares on current self-harm, an increase of 1 standard deviation in nightmare score will indirectly increase the relative risk of a participant being identified by the model as currently self-harming by 9.1%. Meanwhile the direct effect of the self-harm cue latent variable on current self-harm indicated that a 1 standard deviation increase in self-harm cues will increase probability of participants being classified as currently engaging in self-harm by 37.3%.
5.4. Discussion

5.4.1. Modeling the mechanism linking nightmares to increased self-harm risk

The models presented within this chapter have sought to model the mechanism linking nightmares to increased risk of self-harm behaviour by using structural equation modelling technique. Model 3 showed the best fit to the data and was retained. The direct and indirect effects tabulated in Table 5.6 in conjunction with significant pathways co-efficient show that increases in nightmare by the magnitude of one standard deviation with increased likelihood of participants currently self-harming by 9.1%. This supports findings from Chapter 3 which indicated nightmares to increase the risk of self-harm thoughts and behaviours in participants reporting having self-harmed at least once over their lifetime. Moreover, it follows findings of increased risk of suicide attempts by those experiencing nightmares depicted in Sjöström and colleagues (2007, 2009).

While broadly following the description of Cukrowicz et al. (2006), the model differs in several key ways. Firstly the "self-harm cues" latent variable comprised of experiential avoidance, defeat and hopelessness; which is significantly predictive of current self-harm and is itself significantly predicted by negative affect and hyperarousal, is a stark contrast to the ill-defined “suicidal cues” mentioned by Cukrowicz and colleagues (2006). This latent variable has been assembled from constructs supported by theory and empirical evidence from the literature (Brown et al., 2005; Glanz, Haas, & Sweeney, 1995; Rasmussen et al., 2010; Williams, 1997, 2001). Moreover, findings from Chapter 2 of this thesis lend credence to the inclusion of defeat. Similarly, Cukrowicz and colleague describe negative affect as increasing sensitivity to
stressors yet do not define this construct clearly. The selection of hyperarousal, due to the theoretical construct’s definition and literature linking it to sleep disorders (Busch & Fawcett, 2004; McCall & Black, 2013), has been validated by the significant interaction it displayed with the other variables in the model. The present model goes beyond the existing literature by clearly defining the constructs to be measured in the mechanism linking nightmares to self-harm risk. Moreover, the present study establishes a precedent, to good effect, by selecting psychometric tools to empirically measure the underlying mechanism proposed by Cukrowicz et al. (2006).

5.4.2. The role of negative affect

Negative affect appears to be pivotal to the model. As predicted, negative affect was associated with hyperarousal which partially mediates the relationship between negative affect and self-harm cues. Moreover, our modification indices suggested a direct effect of negative affect on self-harm cues. The regression coefficient suggested by our modification indices (direct path of negative affect on self-harm cues) was shown to be significant in the retained model. Cukrowicz and colleague’s description of the mechanism indicated mediation by hyperarousal, but did not explicitly claim a direct effect from negative affect on self-harm cues. It is possible that this direct effect was suggested by the modification indices due to the latent variable indicators (defeat, hopelessness and experiential avoidance) which form ‘self-harm cues’ and their close links to negative affect. This is highlighted by the medium positive correlations between negative affect and the latent indicators shown in table 5.2. However, this does not detract from the findings here due to the strong theoretical and empirical (Brown et al., 2005; Chapman et al., 2006;
Glanz et al., 1995; Rasmussen et al., 2010; Williams, 1997, 2001) support which informed the rationale for including these particular latent indicators. Moreover, focusing on the experiential avoidance theoretical standpoint, self-harm cues should include negative affect. Affect regulation being a key function of self-harm (Chapman et al, 2006); it follows that a high level of negative affect be related to increased need for avoidance, which can be obtained via self-harm engagement.

5.4.3. Hyperarousal, a mediator

The model indicated a direct effect of nightmares on hyperarousal, supporting findings from the sleep literature (Riemann et al., 2010; Simor, Horváth, Gombos, Takács, & Bódizs, 2012). Cukrowicz and colleagues (2006) description of the role of ‘sensitivity to stress’, as measured by hyperarousal levels in the present models, was one of mediation between negative affect and cues. However, there clearly appears to be a significant mediation between nightmares and self-harm cues via hyperarousal in addition to the mediational role previously suggested. The addition of this pathway greatly increased model fit to the data as seen from the jump in PPP value from Model 1 to Model 2. McCall & Black (2013) suggested hyperarousal would mediate the effect of nightmares on suicidality.

Further testing of this mediating or potentially moderating effect is required, and should inform subsequent analysis and model building.

5.4.4. Limitations and further research

It is important to note that structural equation modeling techniques are limited by the data with which the models are created; hence, the importance of
the underlying assumption of data normality. The positive skews and our use of a dichotomous variable clearly violated these assumptions. However, the use of Bayesian estimation methods to assess model fit in combination with bootstrapped maximum likelihood estimations facilitated the computation of models and data prompted refinement. While these techniques are not perfect, their combined use has allowed for model fit to be assessed while taking into account inflated error values, which would have likely led to the rejection of our models due to inflated chi-square values. As previously stated, the models are dependent on the data from which they are constructed and assume the data to be representative of the population. Thus, caution should be used when extrapolating or inferring to other populations based on the present model. In particular it must be stressed that the model does not claim to test causal effects. All modeling was performed based on the best available theoretical and empirical findings from the literature and supported by findings from Chapter 2 and Chapter 3 of this thesis. Potential for model misspecification remains as with all new models. Thus, independent replication with larger data sets and in a variety of populations is advised. For now, the model provides an adequate fit to the data and on the whole respects theoretical and empirical findings of the literature.

The literature has been largely homogenous in its use of psychometric tools to measure nightmares, suicidality, and the variables assumed to provide a mechanistic link between the two. Issues of social desirability and concealment or underreporting are common in studies of suicidality (Nock & Banaji, 2007). Behavioural or physiological paradigms can provide an alternative methodology addressing concerns of concealment and
underreporting. The psychometric assessments used throughout this thesis have thus far allowed us to establish a model of the mechanism linking nightmares to self-harm. The empirical testing of model assumptions remains. Chapters 6 will empirically assess the predictions of Model 3 using behavioural and physiological methods.

5.4.5. Key points from Chapter 5

- The present study aimed to model the psychological mechanism linking nightmares to increased risk of self-harm engagement.
- The model was informed by the literature and previous findings reported in chapters 2 and 3.
- A model (Figure 7, pp. 133) fitting the empirical findings of this thesis and those of the literature could be computed with a good fit to the data.
- This model provides the first reported estimate of the relative risk of nightmares on self-harm engagement using Bayesian SEM, with 1SD increase in nightmares increasing the probability of current self-harm (<1 month) by 9.1%.

5.4.6. Implications for the next chapters

- The model provides a framework from which to test predictions.
- Chapter 6 sets out to test the predictions made by the model via an experimental design using behavioural and psycho-physiological methods.
Chapter 6: Testing the Mechanism Linking Nightmares and Self-Harm Using Behavioural and Psycho-physiological Paradigms

6.1. Introduction

Model 3 reported in Chapter 5 (Figure 7, pp.133) built upon previous findings reported in this thesis. For instance, the primary prediction of the model is that individuals suffering from elevated nightmares levels are at increased risk of engaging in self-harm. This prediction received empirical support via the psychometric assessment detailed in Chapter 2, namely that those suffering from clinically significant levels of nightmares (scores of 11 and above on the Disturbing Dream and Nightmare Severity Index - DDNSI) are 2.08 times (1.36 to 3.18, 95% C.I.) more likely to have a history of self-harm. Furthermore, the indirect effect derived from posterior predictive modeling reported in Chapter 5 indicated that, increases in nightmare level by 1 standard deviation (4.55 points on DDNSI) was tantamount to an increase of 9.1% in risk of engaging in self-harm (≤1 month) in participants with a lifetime history of self-harm further supported the model’s primary prediction. Additionally, the unidirectional predictive relationship of nightmares on self-injurious thoughts and behaviours (SITBs), as well as the mediational role of negative affect on the relationship between nightmares and SITBs reported in Chapter 3 informed our retained model.

Moreover, the four descriptive predictions (Cukrowicz et al., 2006; Bernert & Joiner, 2007) regarding the link between nightmares and suicidality, and the theoretical descriptions of nightmares as affect dysregulators which increase negative affective load upon waking (Nielsen & Levin, 2007, 2009) were incorporated into Model 3. That is:
(i) Nightmares displayed a positive predictive relationship towards negative affect.

(ii) Negative affect showed a positive predictive relationship towards hyperarousal (sensitivity to stressors).

(iii) Elevate levels of hyperarousal significantly predicted elevated self-harm cues levels (defeat, experiential avoidance, hopelessness).

(iv) Self-harm cues significantly predicted current self-harm (<1 month).

Our model as therefore established a tangible framework for further testing which had been absent from the literature. Three testable predictions can be derived from Model 3. Firstly, that individuals suffering from elevated nightmare levels would display elevated levels of negative affect. Secondly, that elevated hyperarousal levels would be observed in those suffering from elevated nightmare levels and that as a function of hyperarousal, those suffering from high nightmare levels should have reduced tolerance to stressors. Thirdly, nightmare sufferers should be more susceptible to self-harm cues.

The model fitted self-reported psychometric data. This thesis and the literature (Krakow et al., 2000; Tanskanen et al., 2001; Bernert et al., 2005; Cukrowicz et al., 2006; Sjöström et al., 2007, 2009; Krakow et al., 2011; Nadorff et al., 2011; Susánszky, Hajnal, & Kopp, 2011; Ribeiro et al., 2012) have so far remained homogeneous in their reliance on psychometric measure for correlational studies. Social desirability, concealment, and under reporting are common place in research involving suicidal spectrum behaviours to avoid unwanted intervention (Nock & Banaji, 2007). Therefore, the use of a variety
of measurement methods resistant to concealment and social desirability effect as such as psycho-physiology and behavioural paradigms would be beneficial to test the predictions of our model.

Psycho-physiological testing offers an alternative and complimentary avenue of research to behavioural paradigms when testing our model. For instance, the model’s predictions relating to hyperarousal and negative affect proposed through the literature (Krakow et al., 2000; Agargun & Cartwright, 2003; Busch & Fawcett, 2004; Cukrowicz et al., 2006; Bernert & Joiner, 2007; Cartwright, 2010; Riemann et al., 2010; Simor et al., 2012) and supported by Chapter 5 are amenable to testing by psycho-physiological means. It is predicted that elevated nightmare levels should lead to elevated levels of negative affect and hyperarousal. Hyperarousal being characterised as an individual’s exaggerated responses to stimuli; reduced pain tolerance and increased agitation (Joiner et al., 1999), it follows that the vague description of increased sensitivity to stressors put forth by Cukrowicz et al. (2006) be conceptualised as hyperarousal.

Participants high in hyperarousal should exhibit elevated physiological responses to stimuli. This has been shown to be the case in hyperaroused veterans with post-traumatic stress disorder exhibiting exaggerated skin conductance responses (Wahbeh & Oken, 2013). The International Affective Picture System (IAPS – Lang, Bradley, & Cuthbert, 1999) is a widely used database of affective pictures with standardised affective and arousal values. Exposure to these stimuli would elicit certain physiological responses. A simple exposure paradigm (Bradley, Codispoti, Cuthbert, & Lang, 2001) while recording galvanic skin response and heart rate during, two well used
indicators of sympathetic nervous system activation (Dawson, Schell, & Filion, 2007), can therefore be implemented to test this prediction of the model.

The model also predicts that nightmare sufferers should exhibit increased sensitivity to stress. The literature (Holdwick & Wingenfeld, 1999; Lejuez, Kahler, & Brown, 2003; Tombaugh, 2006) has shown that the Paced Auditory Serial Addition Tasks (PASAT) is consistently reported as stressful by participants. Moreover, Daughters, Lejuez, Kahler, Strong, & Brown (2005) have successfully used the Paced Visual Serial Addition Task-Computerised (PVSAT-C), a computer based visual variant of the PASAT, as a stress induction task. By experimentally inducing stress using this paradigm, measurements of performance or the duration individuals persevere in the task can be obtained. Thus, providing a behavioural measure of participants’ sensitivity to stress, testing the model’s prediction.

To assess the third prediction of the model; that individuals suffering from nightmares should be more sensitive to self-harm cues, is problematic. To the author’s knowledge, no behavioural task combines all of the latent variable indicators used in the model (defeat, experiential avoidance and hopelessness). While a defeat task has been devised (Johnson, Tarrier, & Gooding, 2008), the task measures self-reported induced defeat but does not have a behavioural outcome measure of defeat in itself. Moreover, experimental paradigms measuring hopelessness and avoidance (Overnier & Seligman, 1967; Seligman & Maier, 1967) have been used in the animal literature; however, create an ethical dilemma due to their extensive use of adverse stimuli.
The Go No-go Association Task (GNAT; Nosek & Banaji, 2001) is a discrimination task between distracter items and target items of interest (words related to self-injury). The GNAT typically measures the strength of association between target items and pre-set attributes (good or bad). By examining the contrast in accuracy (d-prime) between go and no-go trials (no-go minus go trials) a measure of automatic preferences and attitude towards self-injury is obtained. Reaction time measurement for responses can be obtained and contrasted between go and no-go trials as an alternative measure of attitude towards target items.

Principles of signal detection theory (D. M. Green & Swets, 1966; Stanislaw & Todorov, 1999) indicate that sensitivity to a particular cue corresponds to one’s ability to correctly identify targets from noise. Therefore, by omitting the contrast between pre-set attributes and relying on measures of accuracy (d-prime), the GNAT provides an applicable measure of sensitivity to ‘self-harm cues’.

6.1.1. Aims & hypotheses

The present studies aims to test Model 3 using behavioural and psychophysiological paradigms. Specifically, the study will investigate if psychophysiological responses to negative emotional stimuli, sensitivity to stress, and sensitivity to self-harm cues differ between participants with clinically significant (DDNSI scores ≥11) levels of nightmare (high nightmare group) compared to participants with subclinical levels of nightmares (low nightmares group).

In line with our model the hypothesis for this study are as follows:
(i) High nightmare group participants will display significantly elevated physiological responses on negative valence stimuli compared to low nightmare group participants.  
(ii) Participants in the high nightmare group will quit the PVSAT-C stress task significantly faster than low nightmare group participants.
(iii) High nightmare group participants will have significantly higher d-prime values on the GNAT (correct identification of target words) than low nightmare group participants.

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\[9\] An exploration of psycho-physiological differences within participants was also performed and is reported in Appendix N. That is, difference on positive negative and neutral stimuli within participants.
6.2. Methodology

6.2.1. Design and procedure

A cross-sectional case control study design (high vs. low nightmare groups) was implemented. Participants completed a short screening questionnaire allowing them to participate in a lab based experiment composed of a psycho-physiological exposure paradigm (Bradley, Codispoti, Cuthbert & Lang, 2001), the GNAT (Nosek & Banaki, 2001), and the PVSAT-C (Lejuez, Kahler & Brown, 2003).

This study was approved by the University of Nottingham School of Psychology ethics committee. Written consent was obtained from all participants prior to participation in both the screening questionnaire and experiment. Written debriefing information and contact details for the researcher and relevant support groups dealing with issues explored in the present study were provided to all participants.

The screening questionnaire consisted of demographic questions (age, sex, use of medication and psychiatric diagnosis) followed by psychometric measures of nightmares (Krakow, 2006), depressive symptoms (Beck, Steer and Brown, 1996), and self-harm (Gratz, 2001). The questionnaire was e-mailed to university departmental mailing lists and advertised on the experiment participation scheme website of the University of Nottingham, School of Psychology. Participants were required to complete the questionnaire in one sitting and could take as long as they wanted to answer questions. Participants were first given instructions and an electronic consent form. Measurements followed in the order given above. Having completed the
questionnaire, participants were asked if they wished to receive information pertaining to the experimental phase of the study. Upon submission of their responses, participants were immediately presented with debriefing information which included positive mood induction and contact numbers for support services. Piloting of the questionnaire revealed the average completion time to be approximately 10mins.

Participants scoring 11 and above on the DDNSI were categorised as belonging to the high nightmare group, while those with of 10 and less were assigned low nightmare group. To ensure a sufficient number of participants exhibiting high levels of nightmares would be enrolled, invitations to take part in the experimental section of the study were sent to high nightmare participants first. Enrolment of the high nightmare group took precedence over low nightmare groups in order to ensure group balance on gender. Information on the experiment was e-mailed to all participants who had completed the screening and registered an interest. A formal invitation accompanied this e-mail and included potential times slots to take part in the experiment. All testing times offered were scheduled a minimum of 24hrs (maximum of 7 days from questionnaire completion) after sending of the e-mail to give participants time to consider their participation.

On arrival to the session, participants were briefed and required to provide written consent. Participants were asked to complete a nightmare recall questionnaire and indicate their highest mathematics qualification to date (a covariate for analysis of PVSAT-C data). They were then instructed to wash and dry their hands using non-abrasive soap to remove dirt which could impede the application and functioning of physiological sensors (Dawson et al., 2007).
Participants were sat in a comfortable recliner chair. Electrodes and PPG were placed on the participant’s non-dominant hand following recommendations of Dawson et al. (2007). The room lights were dimmed to reduce stimulation from the surrounding environment. Participants completed the physiological paradigm in silence with the researcher hidden behind a partition, monitoring incoming physiological data.

Having completed the psycho-physiological section of the study, GNAT and PVSAT-C followed. Tasks were administered in this order as the psychological stress induced by the PVSAT-C could confound results, particularly with psycho-physiological measurements, or makes participants reluctant to continue with testing. On completion of the tasks, participants were provided with full written debriefing material which includes web resources and counselling services contact details. A short positive mood induction intervention consisting of short passage from McGreevy’s (2006) book of howlers was provided at the end of the session. Such an intervention has been shown to be effective in boosting mood by Göritz (2007) and applied presently to negate any potential adverse effects from the study.

6.2.2 Participants

A total of 382 participants attempted the screening questionnaire. Participants for the online screening were recruited through convenience sampling. Participants were excluded from the study due to improper completion of the screening questionnaire (i.e. they did not attempt all question sets) or omitted demographic information (Age, Gender, Occupation, current use of medication or psychiatric diagnosis). Participants were also excluded
from the study if they indicated current use of medication or existing psychiatric diagnosis. This criterion aimed to control for the artificial conflation of nightmare occurrences induced by common psychoactive medications (Pagel & Helfter, 2003). Nielsen et al. (2006) have shown nightmare rates are stable in early adulthood though frequency reduces in both males and females as a function of ageing. Therefore, entry to the experiment was restricted to participants between 18 and 35 years of age. This was done so that sample homogeneity in age would allow for the nightmare measure obtained during screening to representative of participants’ average nightmare experience. Two-hundred and ninety seven participants were omitted as they withdrew or did not adequately complete the screening (n= 212) leaving no contact details or omitting question set and demographic; expressed no interested to be contacted further (n= 45); did not respond to invitations to participate in the experiment (n=36) or, did not meet the remaining inclusion criteria (n=4) (were above 35 years old, on medication for medical/mental health issue). The sample consisted of 85 participants (12 males) aged 18-31 (M= 20.95 years old, SD= 3.25). All participants were University students. Participants were naïve to the hypothesis of this study. Research credits and a £5 cash incentive were granted in exchange for participation.

6.2.3. Materials & apparatus

54 pictures were selected from the International Affective Pictures Systems (IAPS; Lang et al., 1999) Pictures were chosen according to their standardised affective valences and arousal levels. Pictures were categorised as affectively neutral, positive and negative. Each category contained 18 pictures
(see Appendix M for stimuli list). Negative and positive categories were matched on standardised arousal values.

Digital versions of the selected pictures were displayed using a desktop computer running windows XP on a NEC 19 WV LCD monitor (19inch/48.3cm, 1440x900 pixels) situated approximately .5m from the participant. All pictures were displayed in full screen and 32 bit colour using PsychoPy v1.3.75 (Peirce, 2007).

Stimuli control and physiological data acquisition was achieved via a combination of PsychoPy and a MP 150 BIOPAC. Signals were acquired using a BIOPAC MP150 GSR100C - GSR EDA Galvanic Skin Response Amplifier and PPG100C - Pulse Plethysmogram Amplifier modules. The GSR amplifier was calibrated to detect activity in the range of 0-80μS. Data acquisition and reduction was performed on AcqKnowledge 3.7.3 software. Physiological signals were sampled at 1000Hz for the duration of the paradigm on all data acquisition channels. Heart rate activity was measured using a photo-electric plethysmograph (PPG) placed on the volar surface of the distal phalange of the middle finger. To reduce interference between electrode sets, Galvanic Skin Response (GSR) Ag/AgCl electrode cup filled with BIOPAC isotonic paste were placed on the volar surface of the medial phalanges of the index and ring finger as recommended in Dawson et al. (2007) to measure skin conductance response (SCR). All behavioural paradigms were performed on the same computer and display monitor as the psycho-physiological paradigm using PsychoPy v1.3.75.
6.2.4. Psychometric Measures and Behavioural paradigms

The Disturbing Dream and Nightmare Severity Index (DDNSI; Krakow, 2006) was used as a measure of nightmare frequency and severity. Scores on this scale range from 0-37 with scores ≥11 being indicative of clinical levels of disturbing dreams and nightmares (Krakow, Melendrez, et al., 2002). This measure was used to identify participants for the nightmare group using the cut-off recommended by Krakow and colleagues.

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) was used to assess the presence and severity of depressive symptoms over the last two weeks. The BDI-II has previously shown good reliability (Cronbach’s alphas = .91) and validity (A. T. Beck, Steer, Ball, et al., 1996). It is used in the present study to control for the impact of depressive symptoms in our analyses.

The Deliberate Self-Harm Inventory (DSHI; Gratz, 2001) was used to assess participants’ self-harm history over the lifetime. Seventeen items dichotomously assessed self-harm behaviours (No/Yes). Participants responding positively to any of the 17 items were categorized as having a history of self-harm (SH group) while those reporting no self-harm behaviours were categorised as having no history of self-harm (no SH group). As with previous studies, the instructions to participants were modified to encompass all acts of self-harm by removing any mention of suicidal intent. This was done to obtain a measure of self-harm history regardless of motivation or intent to die. Self-harm history was used as a covariate to control for the impact of past self-harm behaviour in our analyses.
A Nightmare recall questionnaire was used to assess the occurrence of nightmares the night prior to the study. Nightmare recall was dichotomised (Yes/No). Participants answering “yes” to recall additionally asked to write their recollection in as much detail as possible. They were also asked to rate the nightmare on a 5 point Likert Scale on criteria of intensity, vividness and distress\(^{10}\). Nightmare recall was used as a covariate to control for the effect of nightmares on our analyses. That is, to ensure that the occurrence of a nightmare on the day of the study did not impact on the conflate results for low nightmare or high nightmare group participants.

The Psycho-physiological IAPS display paradigm (Bradley, Codispoti, Cuthbert and Lang, 2001) was used to obtain physiological data relating to hyperarousal and biases towards negative affect. Participants were exposed to a randomised set of 54 affectively valenced pictures (18 neutral, 18 positive and 18 negative) from the International Affective Pictures System (IAPS; Lang, Bradley & Cuthbert, 1997) while connected to a MP 150 BIOPAC. Electrodes and PPG were attached following instructions detailed previously to record GSR and heart rate. Each trial consisted of a 2 second white fixation cross displayed in the centre of a black background. The image was then displayed full screen for 6 seconds. A 10 second inter-stimuli interval consisting of a black screen followed. Skin conductance response (SCR) and heart rate (HR) were obtained. See Table 13 for a summary of physiological outcome measures computations.

The Go No-go Association Task (GNAT; Nosek & Banaji, 2001) is a discrimination task between distracter items and target items of interest for the

\(^{10}\) Qualitative data and nightmare ratings are not included in this thesis.
study. Participants must correctly identify target and selected attributes from a random sequence of aforementioned words. The target and selected attribute (e.g. positive words) are flashed on screen along with distractor words (e.g. negative words). The sought after attributes and target words are clearly indicated to participants via instructions prior to a block. They are also displayed in the top left and top right corners of the screen during the trials themselves to provide continuous reminders to participants. Each word is displayed for 800ms. Participants must respond in that time. Participants undertake a total of 160 randomised trials. Trials occurred in 4 blocks of 40 (target words n=10, target attribute, n=10 and distractor n=20). The target attribute of the 4 trial blocks are block randomised ensuring participants exposure to 2 positive and 2 negative trial blocks. Scores are calculated following Signal Detection Theory principles of Hits and False Alarms to obtain a measure of accuracy (d-prime). D-prime for positive blocks and negative blocks can then be compared to obtain a measure of attitude, ‘d-prime difference’ (Nosek & Banaji, 2001). While the GNAT typically assesses the strength of association (d-prime difference) between target words (self-harm regardless of intent: cutting, overdose, hanging, suicide, self-harm) and pre-set positive (excellent, happy, good, pleasant, wonderful) or negative (bad, unpleasant, nasty, terrible, horrible) attributes, the present study focused on sensitivity to cues. As such, target word identification accuracy (d-prime) was obtained as the outcome measure rather than attitude towards target words (d-prime difference). The dependent variable of the GNAT (d-prime) was computed by subtracting the Z score for false alarms (false positive) from the Z score for hits (true positive), following instruction from Green & Swets (1966).
Higher d-prime values were indicative of greater sensitivity to target words (self-harm). The Paced Visual Serial Addition Task - Computerised (PVSAT-C; Lejuez, Kahler & Brown, 2003) is a validated visual variant of the PASAT. The PVSAT-C was used as a stress induction task. Numbers (1 to 9) are flashed at the centre of the screen for a specified duration. Participants must add the number currently on the screen with the number preceding it and give a verbal response. New numbers are indicated by a flashing red box surround the digit so that participants are aware of the change should the digit appearing be the same at the one which preceded it (e.g., 2 followed by 2). It is specifically designed to incorporate 3 difficulties (i.e. 3 varying latencies for numerical display). The first block of the task has 3 second latency between numbers and last for 3 minutes, allowing the participant to settle into the task. The second block has a latency of 2 seconds between numbers and last for five minutes. A two minute mandatory rest period is given to participants prior to beginning final block. The third block has a latency of 1 second and can last for a maximum of ten minutes; however participants are instructed during the rest period that due to the difficulty they are allowed to end this block at any time by pressing a designated key on the terminals’ keyboard. Duration of the third block provides a time measure for resistance to psychological stress. As this is likely to be moderated by mathematical ability, an objective measure of ability (highest maths qualification: 0=none, 1=GCSE, 2=AS-Level, 3=A-Level, 4=1stDegree) will be recorded for use as a covariate.

Nosek & Banaji warn of ceiling effects in d-prime and recommend the use of a correction when calculating d-prime should accuracy be too high (d-prime – [0.35/ n of trials]).
6.2.6. Physiological data reduction

Reactions in skin conductance and heart rate were extracted as shown in Figure 8. SCR baselines were recorded for 2000ms prior to stimuli onset. A 2000ms delay was used prior to sampling SCR response data over a 6000ms duration. The delay represents the latency of sweat gland activation by the sympathetic nervous system estimated to be approximately 1.1 seconds after onset (Benedek & Kaernbach, 2010a, 2010b). Responses occurring between 1 and 5 seconds are deemed and at amplitudes of 0.01 to 0.05 mS are deemed to have been elicited by the stimulus (Dawson et al., 2007). Heart Rate (HR) data were extracted with baseline HR being sampled for 1000ms prior to stimuli onset. HR response was also sampled across the 6000ms duration of stimuli onset. Due to the relative immediacy in HR variability detected by the PPG (approx. 1/2 beat lag) compared to SCR, no measurement delay was required.
A wide variety of outcome measure can be computed from physiological data. To keep physiological analysis succinct, SCR mean and HR mean were selected as outcome measures. In addition, SCR MRA (mean response amplitude) was calculated by subtracting SCR baseline mean from SCR response maximum. Table 13 summarises the physiological measures used, the method for their calculation and their indexical reference.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Unit</th>
<th>Calculation</th>
<th>Index of arousal when exposed to stimuli compared to baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCR MRA</td>
<td>µS</td>
<td>RSPmax - BLmean</td>
<td>Average arousal (skin conductance) during stimuli exposure</td>
</tr>
<tr>
<td>SCR mean</td>
<td>µS</td>
<td>average response means</td>
<td>Average arousal (skin conductance) during stimuli exposure</td>
</tr>
<tr>
<td>HR mean</td>
<td>bpm</td>
<td>average response means</td>
<td>Average arousal (heart rate) during stimuli exposure</td>
</tr>
</tbody>
</table>

Physiological scores for negative stimuli were averaged. Log transformations were carried out to normalise positively skewed SCR mean data using the following formula (Log[1+SCR]) recommended by (Ben-Shakhar & Dolev, 1996; Lykken & Venables, 1971). Log transformation could not sufficiently reduce the skew in SCR MRA data due to the large within-participant variations. As such SCR MRA data was standardized within each participant using the following formula (raw SCR MRA/ standard deviation) prior to calculating group averages. HR mean is an absolute measure and as such incurred less error, thus skews were acceptable and HR mean data did not require transformation or standardizing.

6.2.7. Data analysis procedure

Means were calculated and pairwise deletion was used to remove participants with missing mean scores from task specific analysis. This missing
data method was selected due to number of tasks employed in the study and the potential for psycho-physiological non-responders, that is, individuals with outlier level baseline responses. As such, loss of individual participants’ data in one task did not reduce power of the analysis in subsequent tasks.

MANCOVA was performed to test whether high nightmare group participants would display significantly elevated physiological responses on negative valence compared to low nightmare group participants. The grouping variable (high nightmare vs. low nightmare) was entered as the between participant factor. Depressive symptoms, hyperarousal, self-harm history and nightmare awakening were entered as covariates. Negative stimuli SCR mean, SCR MRA and HR mean were entered as the outcome variables.

ANCOVA was performed to test the hypothesis that high nightmare group participants would quit the PVSAT-C task significantly faster compared to low nightmare group participants. Participant grouping (high nightmare vs. low nightmare) was entered as the between participant factors. Depressive symptoms, self-harm history, nightmare recall, and highest mathematical qualification were entered as covariates. PVSAT-C duration was entered as the dependent variable.

Finally, in order to test if high nightmare group participants would display significantly higher sensitivity to self-harm cues (target words) than low nightmare group participants, ANCOVA was performed. Participant grouping (high nightmare vs. low nightmare) was entered as the between participant factors. Depressive symptoms, self-harm history, and nightmare
recall were entered as covariates. GNAT d-prime was entered as the dependent variables.
6.3. Results

6.3.1. Descriptive Statistics

Means and standard deviation for both high and low nightmare groups are displayed in Table 14. Z-scores for SCR MRA (μS) are provided in addition to raw data. Log SCR means (μS) are given in the table directly as are raw HR mean (beats per minute - bpm). As per the design of this study, the high nightmare groups (M= 14.93, SD= 3.51) did show a significantly greater level of nightmares that low nightmare participants (M= 5.76, SD= 2.67), t(75) = 13.89, p<.001. Frequencies for categorical variables are also reported in Table 14. Physiological measures were recorded from all participants during the psycho-physiological section of this study. However, due to computer errors, one participant was excluded from analyses of physiological measures. Final N on physiological dependent measurements was n= 84 for the whole sample. The usable N for the low nightmare group was n= 41.

Table 14 - Descriptive statistics for high and low nightmare groups

<table>
<thead>
<tr>
<th></th>
<th>Low nightmare (n=42)</th>
<th>High nightmare (n=43)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD) / z-score</td>
<td>Mean (SD) / z-score</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>9.81 (7.48)</td>
<td>17.86 (9.75)</td>
</tr>
<tr>
<td>SCR mean (log)</td>
<td>.40 (.18)</td>
<td>.40 (.20)</td>
</tr>
<tr>
<td>SCR MRA</td>
<td>.07 (.09) / .14 (1.02)</td>
<td>.04 (.09) / -.13 (.97)</td>
</tr>
<tr>
<td>HR mean</td>
<td>71.09 (13.68)</td>
<td>78.89 (25.51)</td>
</tr>
<tr>
<td>GNAT d-prime</td>
<td>3.27 (.80)</td>
<td>3.13 (.73)</td>
</tr>
<tr>
<td>PVSAT-C duration</td>
<td>229.54 (229.36)</td>
<td>157.09 (184.33)</td>
</tr>
<tr>
<td>*Self-harm history</td>
<td>20</td>
<td>34</td>
</tr>
<tr>
<td>*Nightmare recall</td>
<td>3</td>
<td>21</td>
</tr>
<tr>
<td>*Math Qualification</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None (0)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>GCSE (1)</td>
<td>22</td>
<td>24</td>
</tr>
<tr>
<td>AS-Level (2)</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>A-level (3)</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>1st degree (4)</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

*Binomial and *multinomial variables are expressed as count data.
6.3.2. Do nightmare groups differ on physiological responses to negative stimuli?

A MANCOVA examined the effect of nightmare grouping on SCR mean, SCR MRA and HR mean responses to negative stimuli while controlling for the effects of depressive symptoms and self-harm history and nightmare recall.

A non-significant Box’s M test (Box M = 12.17, p>.05) indicated assumptions of homogeneity of covariance had been met. Pillai’s trace was used as the multivariate test criterion as it is the most conservative estimate (Tabachnick & Fidell, 2001). The multivariate effect was non-significant, \( F(3, 76) = 1.31, p>.05, \lambda_{\text{Pillai}} = .05, \text{partial } \eta^2 = .05 \). Similarly, the test of between participant effects showed no significant differences between high and low nightmare groups on SCR mean (\( F(1, 78) = .05, p>.05, \text{partial } \eta^2 = .001 \)), SCR MRA (\( F(1, 78) = 2.09, p>.05, \text{partial } \eta^2 = .026 \)), or HR mean (\( F(1, 78) = 1.70, p>.05, \text{partial } \eta^2 = .021 \)).

6.3.3. Do nightmare groups differ in PVSAT-C endurance?

An ANCOVA examined the effect of nightmare grouping on PVSAT-C duration while controlling for the effects of depressive symptoms, self-harm, nightmare recall, and highest mathematics qualification.

Levene’s test of equality of error variance indicated homogeneity assumptions had been violated, \( F(1, 83)= 10.72, p<.05 \). However, the variance ratio\(^{12}\) between groups did not exceed the recommended value of 2 (Field, 2013). The ANCOVA revealed a significant difference between nightmare

\(^{12}\) Variance ratio calculated as \((\text{group1 SD}^2) / (\text{group2 SD}^2)\). That is, \( 229.54^2 / 184.33^2 = 1.55 \).
groups on PVSAT-C duration after controlling for depressive symptoms, self-harm history and nightmare recall, $F(1, 79) = 5.54, p=.02$, partial $\eta^2 = .066$). Results indicate that the high nightmare group participants quit the task faster than low nightmare group participants.

6.3.4. Do nightmare groups differ in self-harm cues identification accuracy?

ANCOVA was performed to examine the effect of nightmare grouping on GNAT d-prime while controlling for the effects of depressive symptoms and self-harm history, and nightmare recall.

Levene’s test indicated assumptions of homogeneity of error variance had been met, $F(1, 83) = 1.07, p>.05$. The ANCOVA revealed there were no significant differences between nightmare groups on accuracy of target word identification (d-prime), $F(1, 80) = .42, p>.05$, partial $\eta^2 = .008$. 
6.4. Discussion

The present study aimed to test three assumptions put forth by the literature and supported Model 3 (Figure 7, pp.133) in order to validate their claims by other means than psychometric assessments which have permeated the literature (Krakow et al., 2000; Tanskanen et al., 2001; Bernert et al., 2005; Cukrowicz et al., 2006; Sjöström et al., 2007, 2009; Krakow et al., 2011; Nadorff et al., 2011; Susánszky, Hajnal, & Kopp, 2011; Ribeiro et al., 2012). That is, do elevated nightmare levels lead to: (i) increased levels of negative affect and hyperarousal; (ii) reduced tolerance to stressors; and (iii) increased susceptibility to ‘self-harm cues’. These assumptions were tested using behavioural and psycho-physiological paradigms.

6.4.1. Do high nightmare participants display elevated physiological responses to negatively valenced stimuli?

When investigating the effects of nightmares on psycho-physiological responses to negatively valenced stimuli, our analysis revealed that the high nightmare group did not significantly differ from the low nightmare group on SCR means, SCR MRA, or HR mean when controlling for depressive symptoms, self-harm history, and nightmare recall. As such, the null hypothesis could not be rejected.

All psycho-physiological measures yielded insufficiently large differences between participants. Moreover, our exploratory analysis reported in Appendix M indicates that psycho-physiological responses to positive, neutral and negative stimuli within participants were also non-significant. These findings do not support the prediction of the model and are contradictory
to prior findings using psychometric assessments of negative affect (Chapter 2).

Bradley and Lang (2005) indicate that the most reliable positively and negatively valenced stimuli to elicit strong physiological responses appear to be erotic or pornographic and graphic mutilation or threatening weapons, respectively. However, the stimuli used fall in the relatively mild physiological arousal range available within the IAPS database. These mild stimuli may not have been sufficient arousing to elicit responses of the desired magnitude or sufficiently strong to elicit responses differentiable between nightmare groups. Limitations on stimuli selection were imposed to comply with ethical norms and regulations of the University and to reduce potential negative outcomes for participants. However, other studies have used high arousal negatively valenced images with sensitive populations. For instance Glenn, Blumenthal, Klonsky, & Hajcak (2011) have shown clear heightened startle response when exposed to negative stimuli of greater arousal values than those of the present study in individuals who self-harm compared to those with no history of self-harm. As such, replication with higher intensity stimuli is advised, although appropriate ethical consideration of participants and adherence to the institution’s ethics policy should be enforced.

6.4.2. Are high nightmare participants less resistant to stress?

Our findings indicated that participants in the high nightmare group quit the high stress section on the PVSAT-C significantly faster than participants in the low nightmare group. This supports our research hypothesis and indicates that individuals suffering from elevated nightmare levels are less resistant to
stressors beyond the depressive symptoms, their self-harm history and recall of nightmare prior to testing.

This behavioural finding support the inclusion of hyperarousal (arousal which reduces tolerance to stressors [Joiner et al., 1999]) in our model. This result also provides the first empirical support for nightmare levels’ impact on stress tolerance. Moreover, the observed effect is medium, following effect size guidelines set out by Cohen (1988)\textsuperscript{13}. We therefore suggest that individuals experiencing elevated levels of nightmare may benefit from stress resilience training if nightmare reducing treatments are unavailable.

Wells & Matthews (1994) suggest that negative emotional states lead to impaired cognitive performance compared to neutral or positive emotional states. The PVSAT-C has been shown to induce these negative moods and has consistently been reported as being stressful (Holdwick and Wingenfeld, 1999; Lejuez et al., 2003). Therefore, participants in the high nightmare group may have had higher baseline levels of negative affect which the PVSAT-C embellished. Thus, reducing their cognitive performance and increasing stress leading the high nightmare group to quit the task faster than participants in the low nightmare group. This suggestion follows findings from Chapter 2, whereby high nightmare group participants exhibited significantly higher levels of negative affect than their low nightmare group counterparts.

However, the lack of significant difference in psycho-physiological responses to negatively valenced stimuli between nightmare groups, discussed previously, creates ambiguity. On one hand, group difference in responses to

\textsuperscript{13} Partial eta square effect size guidelines (Cohen, 1988): 0.01 = small, 0.06 = medium, 0.13 = large
negative affective stimuli should be visible from the psycho-physiological paradigm if the stress and negative affect induced by the PVSAT-C had built upon baseline negative affect as suggested. Although as discussed, the relatively low arousal value of the stimuli presented during the psycho-physiological paradigm may have been insufficient to elicit physiological responses of the desired magnitude. Results should therefore be interpreted with a level of caution and replication while controlling for baseline levels of negative affect is recommended.

6.4.3. Are participants with high nightmares more sensitive to self-harm cues?

Following Model 3, we hypothesised that high nightmare group participants would be significantly more accurate at identifying self-harm target words due to their increased sensitivity to self-harm cues. However, our analysis revealed no significant difference in accuracy between high and low nightmare groups indicating no observable difference in sensitivity to self-harm cues.

Nightmare groups in the present study significantly differed on nightmare levels an average of 9.17 points on the DDNSI. According to the observed effect of our Bayesian estimates reported in Chapter 5, this 9.17 point difference translates to an increase of approximately 18% in likelihood of current self-harm for participants with an existing history of self-harm engagement\textsuperscript{14}. Our sample included participants without a self-harm history, recruited as recruitment could not ethically be made based on the criterion of self-harm engagement. The inclusion of participants without a history of self-

\textsuperscript{14} Based on estimates of 1SD (4.55 points on DDNSI) increasing risk of current self-harm (<1month) by 9.1% in a population with a history of self-harm.
harm may therefore have diluted potential effects. Replication with a larger sample which includes more participants with a history of self-harm is advised to verify the present findings.

6.4.4. Limitations and further research

Although supportive of the hypothesis that high levels of nightmares reduce stress tolerance, this study failed to show differences between nightmare groups on psycho-physiological responses to negative stimuli, and sensitivity to self-harm cues.

As previously discussed, the lack of psycho-physiological difference between groups may be due to the relatively mild standardised arousal values of the negative stimuli presented to participants. If ethically permissible, replication with stimuli of greater arousal values is advised.

While the PVSAT-C yielded results supporting our hypothesis, further investigation should aim to control for baseline affect to verify these findings. Moreover, further studies should consider mediation analysis to further validate the pathways described by Model 3 linking nightmares to negative affect and hyperarousal (sensitivity to stressors). Moreover, the sample size (n=84 for psycho-physiological paradigm, n=85 for GNAT and PVSAT-C) for this study was based on power calculation assuming large effect sizes. Thus, greater statistical power is recommended in replications, particularly when controlling for numerous covariates which reduce degrees of freedom.

The GNAT paradigm itself offers additional methodological issues which require consideration should this study be replicated. The present study utilised a 800ms stimuli display. Nosek & Banaji (2001) explain that all display
latencies have benefits and inconveniences. For instance, while greater stimuli display latency can increase participant response accuracy, this impacts on d-prime; a ratio of true positive to false positive which requires a level of inaccuracy to be calculated. Conversely, too short a display latency would create a large amount of missing data as participants would be unable to respond prior to the end of the trial. The settings for the present GNAT were a compromise to ensure some degree of inaccuracy (false positives), while allowing for sufficient stimuli display latency to obtain adequate numbers of true positive identifications. As our results indicated no group difference, increasing the difficulty of the task by reducing display latency may allow for group differences to become apparent. Replications of these findings should aim to vary stimuli display latency to ensure greater variability in d-prime.

In sum, it is possible that small group differences could not be detected due to low statistical power, or, that stimuli display latency on the GNAT created a ceiling effect making correct identification of self-harm cues too easy. Similarly, lower power may have hindered the detection of group differences on psycho-physiological measures, or this lack of difference could be due to the mild arousal levels of the stimuli used.

6.4.5. Key points from chapter 6

- This study aimed to test the main prediction of Model 3 reported in Chapter 5 (Figure 7, pp.133).
- Nightmares of clinical severity reduce tolerance to stress, supporting the path between nightmares and hyperarousal indicated in the model.
- No difference due to nightmare grouping was detected on ‘self-harm cue’ sensitivity (d-prime).
No psycho-physiological differences based on nightmare grouping were detected. We suggest this may be due to the mild arousal value of stimuli used, too weak to elicit a substantial response.

This is the first study investigating the links between nightmares and self-harm to implement an experimental design, and to use behavioural and psycho-physiological paradigms.

6.4.6. Implications for next the chapter

The results from this chapter provide partial support for Model 3 using behavioural measures. These results and those of previous chapter will be discussed in Chapter 7 in the context of the literature and a mechanism linking nightmares to self-harm.
Chapter 7: Summary and conclusions

This thesis has investigated a potential psychological mechanism linking nightmares to self-harm regardless of intent or motivation to die. This has been done through a variety of psychometric, behavioural and psycho-physiological methods. This final chapter summarises the aims and findings of the studies reported in this thesis. A conclusion based on the sum of the evidence gathered is presently discussed.

7.1. Review of aims and findings of empirical chapters

7.1.1. Chapter 2 Summary

Existing literature demonstrated an association between nightmares and suicidal ideation (Tanskanen et al., 2001; Bernert & Joiner, 2005; Cukrowicz et al., 2006), attempted suicide (Sjostrom et al., 2007), and completed suicide (Sjostrom et al., 2009). By the nature of the cognitions and behaviours being studied, research implied that nightmares were linked to behaviours with suicidal intent. This study aimed to investigate if nightmares were associated to self-harm regardless of intent or motivation to die. Furthermore, negative affect, defeat and entrapment were compared between participants exhibiting clinically significant levels of nightmares and those who reported subclinical levels.

The findings of this study were congruent with the literature and extended established trends of nightmares predicting suicidality by showing a significant association between nightmares and self-harm regardless of intent. This was the case after controlling for the effects of depressive symptoms. Moreover, this study indicated that while nightmares could significantly predict
self-harm history when controlling for depression, insomnia could not. Moreover, our analysis of negative affect performed via ANCOVA supported the premise of the descriptive mechanism proposed by Cukrowicz et al. (2006) and Bernert & Joiner (2007); that negative affect is elevated in participants suffering from nightmares. Similarly, an ANCOVA analysis investigating group differences in defeat and entrapment, prominent variables from the Cry of Pain model of suicide (Williams, 1997, 2001) revealed defeat to be elevated in those experiencing clinical levels of nightmares compared to participants with subclinical nightmare levels. However, the observed effect was small. Entrapment on the other hand, showed no difference between groups.

7.1.2. Chapter 3 Summary

The literature shows that nightmares and suicidal cognitions and behaviours are associated (Tanskanen et al., 2001; Agargun & Cartwright, 2003; Agargun & Beşiroğlu, 2005; Bernert & Joiner, 2007; Goldstein, Bridge, & Brent, 2008; Sjöström et al., 2007, 2009; Nadorff, Nazem, & Fiske, 2011; Susánszky, Hajnal, & Kopp, 2011; Ribeiro et al., 2012). However, the direction of causality has not been established and existing models (McCall & Black, 2013) have assumed nightmares to be predictive of suicidality. As exploring causality was deemed beyond the scope of this thesis due to restrictions in time and resource (ability to perform nightmare reducing intervention – imagery rehearsal therapy; Karkow & Zadra, 2010), this study aimed to explore the direction of the predictive relationship between nightmares and self-injurious thoughts and behaviours (SITBs). In addition, this study explored mood deregulation by measuring pre- and post-sleep negative affect to test if negative affect mediated the relationship between nightmares and SITBs.
Restrictions to our analysis were imposed due the low occurrence of self-injurious acts over the study period. As such, self-injurious acts and self-injurious ideation were combined into the variable SITBs, allowing the computation of our models.

Our GEE analysis indicated that nightmares are potent predictors of SITBs (O.R. 4.01, 95% CI [1.06 to 15.15]) and that this relationship is unidirectional. Moreover, post-sleep negative affect was a significant partial mediator between nightmares and post-sleep SITBs.

7.1.3. Chapter 4 Summary

The literature investigating the link between nightmares and suicidality has focused on the observable occurrences of nightmares; rather than the content of the experience. Moreover, research into dream content and its links to suicidal behaviour has been sparse for over the last 20 years (Langs, 1966; Ralphing, 1970; Firth et al., 1986; Evans, 1990; Maltsberger, 1993) and has focused on exploring the associations between the content of negative dreams and behaviours with suicidal intent. The diary methodology used to obtain data in Chapter 3 provided an opportunity for the longitudinal collection of dream content, allowing for the exploration of associations between content and self-harm regardless of intent or motivation to die. Previous studies (Ralphing, 1970; Firth et al., 1986) had found individuals who had attempted suicide reported dream content more pervasive of death themes. The study presented in Chapter 4 aimed to investigate difference in negative dream content using LIWC software (Pennebaker, 2007) between participants with and without a history of self-harm. Death content was investigated and exploratory analyses
were performed for 5 psychological constructs (affective, cognitive, social, perceptual, and biological processes) and for 6 further personal concern categories to death (work, achievement, home, leisure activities, money, and religion).

The findings of this study were contradictory to the literature (Ralphing, 1970; Firth et al., 1986). There was no significant difference between participants with and without a history of self-harm of linguistic frequency of death words when controlling for the effects of depressive symptoms; length of diary entries and percentage of words recognised by the default dictionary categories. However, there was a marginally non-significant trend (p=.065) towards higher use of perceptual processes words in participants with a history of self-harm. Participants without a history of self-harm reported significantly more money words than participants with a history of self-harm. Exploratory analysis of self-harm recency indicated that participants with current self-harm (<1 month) reported significantly more words relating to the perceptual and biological processes subcategories ‘feel’ and ‘body’ compared to participants without self-harm and those with a history of self-harm (>1 month).

7.1.4. Chapter 5 Summary

Based on the predictive directional relationship between nightmares and SITBs reported in Chapter 3 and the descriptive mechanism proposed by Cukrowicz et al. (2006) and McCall & Black (2013), Chapter 5 aimed to model the psychological mechanism linking nightmares to self-harm behaviour. As our analysis in Chapter 3 revealed that participants without a history of self-harm did not report any SITBs throughout the course of the study, the SEM
model was to be predictive of risk of current self-harm engagement (<1 month) in a population of participants reporting a history of self-harm.

The model aimed to meet 4 requirements to be accepted: nightmares needed to significantly predict increased negative affect; this negative affect had to significantly predict hyperarousal (sensitivity to stressors); and in turn hyperarousal was to significantly predict ‘suicidal cues’ (a latent variable composed of defeat, experiential avoidance, hopelessness). Lastly, ‘self-harm cues’ needed to significantly predict current self-harm (<1 month).

A model was created following the theoretical and empirical literature (Cukrowicz et al., 2006; Bernert & Joiner, 2007; McCall & Black, 2013), and refined based on our data set using modeling modification indices. Model 3 (Figure 7, pp.133) showed good fit with our data and met the 4 aforementioned requirements described. From this model, direct and indirect effects were calculated. The indirect effect of nightmares on current self-harm, indicated that an increase of 1 standard deviation in nightmare score (4.55 points on the Disturbing Dreams and Nightmare Severity Index) would indirectly increase the probability of a participant being identified by the model as currently engaging in self-harm by 9.1%. Additionally, a direct effect of the latent variable ‘self-harm cues’ on current self-harm indicated that a 1 standard deviation increase in ‘self-harm cues’ would increase probability of participants being classified as currently engaging in self-harm by 37.3%.

7.1.5. Chapter 6 Summary

As Chapter 5 provided a testable model, the study presented in Chapter 6 set out to test the assumptions of this model. That is, it aimed to test if
individuals suffering from elevated nightmare levels would display increased responses to stimuli of negative affect; display reduced tolerance to stressors; and display increased sensitivity to ‘self-harm cues’. Moreover, as the literature and the studies presented in this thesis has thus far been reliant and psychometric assessments. This was deemed problematic due to issues of concealment and under-reporting which are prevalent in research of suicidality (Nock & Banaji, 2007). Therefore, the study presented in Chapter 6 aimed to test the predictions of the model using behavioural and psycho-physiological methods.

Our analysis comparing high and low nightmare groups on psycho-physiological responses to negatively valenced stimuli, obtained from an exposure paradigm (Bradley, Codispoti, Cuthbert and Lang, 2001), failed to revealed significant differences. Thus, our analysis failed to demonstrate that participants experiencing clinically significant levels of nightmares responded with greater intensity to negative stimuli. However, an exploratory analysis within participants (all participants included) revealed there to be no differences in responses between positive, negative and neutral stimuli (Appendix N). This may indicate that the stimuli used were too mild to induce responses of a sufficient magnitude for between groups analysis. Similarly, no group differences could be detected when assessing participants’ sensitivity to self-harm cues using d-prime (Stanislaw & Todorov, 1999) obtained from the Go No-go Association Task (GNAT; Nosek & Banaji, 2001). Thus, we could not demonstrate, as assumed by our model, that participants in our clinical nightmare group were more sensitive to ‘self-harm cues’. However, our analysis of sensitivity to stressors revealed that participant in our clinical
nightmare group were significantly faster to quit a stress induction task, the Paced Visual Serial Addition Task – Computerised (PVSAT-C), compared to participants with subclinical nightmare levels. This difference remained upon controlling for depressive symptoms, past self-harm history and mathematical ability. This result provides support for the model’s prediction participants with elevated nightmare levels would display reduced tolerance to stressors.

7.1.6. Theoretical and Methodological Contributions to the Literature.

A number of theoretical and methodological contributions to the literature have been made throughout this thesis, advancing this field of research.

Research on the links between nightmares and suicidal behaviour had to date limited their scope to suicide attempts (Sjostrom et al., 2007) or repeat attempts (Sjostrom et al., 2009). Based on the behaviours studied, the literature had implied (possibly unwittingly) a link between nightmares and suicidal intent. This thesis aimed to explore if the link between nightmare and suicidal behaviour extended to self-harm regardless of intent, due to the continued contention of dichotomizing self-injurious behaviour as suicidal or not (Kapur et al., 2013). To the authors’ knowledge, no studies had yet investigated the links between nightmares and self-harming behaviour regardless of suicidal intent. The study presented in Chapter 2 set out to answer this question and indicated that a link between nightmares and self-harm regardless of suicidal intent was present. This finding provided the foundation for subsequent studies and was replicated in Chapters 3 and 5.
This thesis also aimed to answer a fundamental question, the direction of the relationship between nightmares and self-harm. This had yet to be addressed by the literature due to the cross-section and correlation design of published studies (Bernert et al., 2005; Cukrowicz et al., 2006; Krakow et al., 2011; Nadorff et al., 2011). As such, it was not clear whether nightmares preceded or followed self-injury. Chapter 3 aimed to answer this question using longitudinal prospective diary methodology which had not been applied in this field. The data obtained was therefore less prone to underestimation of nightmares common in retrospective measures (Robert & Zadra, 2008; Zadra & Donderi, 2000). Our findings provided the first empirical support for nightmares’ unidirectional relationship with self-injurious thoughts and behaviours (SITBs). This allowed subsequent studies (Chapter 5) to model the psychological mechanism linking nightmares to self-harm in the knowledge that key assumptions of directionality had been empirically addressed.

Moreover, the inability to model an association between nightmares and post-sleep SITBs in individuals with no history of self-harm provided an important insight. While prior studies had shown links between nightmares and suicidality in student populations as used presently, none had accounted for the effect of self-harm history on this association. Our analysis split our sample by self-harm history to account for its effect. Our inability to compute a model due to the lack of post-SITBs in participants without a history of self-harm revealed that a mechanism modeled should concentrate on participants with a history of self-harm.

In addition to answering questions of directionality, our diary study (Chapter 3) provided empirical support for the partial mediational role of
negative affect on nightmares to post-sleep SITBs. Negative affect had been
described as playing a pivotal role in the association between nightmares and
suicidal behaviours (Cukrowicz et al., 2006; Bernert & Joiner, 2007).
However, to the author’s knowledge, no empirical support for this mediational
role has been reported to date. This result informed the structural equation
model of the psychological mechanism linking nightmares to self-harm
reported in Chapter 5.

Our diary study (Chapter 3) was an opportunity to delve deeper into the
nightmare and suicide literature by exploring nightmare content. The existing
literature had focused on suicide attempters (Evans, 1990; Firth et al., 1986;
Langs, 1966; Maltsberger, 1993; Raphling, 1970). Therefore, this study was
the first investigation of nightmare content in participants with reporting self-
harm regardless of suicidal intent. Moreover, this study is the first to obtain
content data prospectively in order to reduce nightmare reinterpretation and
memory biases (Robert & Zadra, 2008; Zadra & Donderi, 2000). Additionally,
this study is the first exploration of nightmare content in a self-harm population
to utilised linguistic count software, the LIWC 2007 (Pennebaker & Chung,
2007). Contrary to the literature, our findings indicate no difference between
participants with and without a history of self-harm on frequency of death
category words. However, individuals with a history of self-harm describe their
nightmares using more perception words (see, feel) than individuals who have
never self-harmed. While exploratory and needing replication, this study
provided a novel insight into the nightmare to self-harm relationship.

Informed by our prior findings, and the sleep and suicide literatures,
Chapter 5 aimed to model the mechanism linking nightmares to increased risk
of self-harm engagement. This mechanism followed the theorised mechanism put forward by Cukrowicz et al. (2006) and that of McCall & Black (2013). This chapter and the model retained (Model 3) provided the first empirically based mechanism linking nightmares to self-harm. Moreover, due to the Bayesian SEM methodology employed, this model provided an estimate of relative risk of self-harm engagement based on nightmare levels. This is presently the only estimated effect size in the literature indicating the impact of nightmare on risk of self-harm engagement (<1month).

The literature to date has relied on psychometric assessments. Chapter 6 set out to test our newly developed model using behavioural and psycho-physiological methods to remedy this over reliance. As predicted by our model, participants experiencing nightmares of clinical severity indicated significantly more sensitive to stressors. This result is the first empirical support for the effect of nightmare on behavioural measures of stress and provided support for our model and that of McCall & Black (2013).

In sum, this thesis has implemented methodology which has to date been missing from the literature to further knowledge in this field of research. Namely, the use of longitudinal diary methodology to assess directionality of effects, linguistic count software to explore nightmare content, the use of Bayesian SEM to obtain a measure of relative risk of nightmares on self-harm engagement, and the use of behavioural and psycho-physiological methods to test predictions made by our model.
7.1.7. Relationship between thesis findings and existing models of suicidal behaviour

This thesis utilised two prominent models of suicidal behaviour; the Cry of Pain model (CoP; Williams, 1997, 2001) and the Experiential Avoidance Model (EAM; Chapman et al., 2006), to inform the exploration of a psychological mechanism linking nightmares to self-harm. While these models have a different focus (CoP on suicidal behaviour and the EAM on non-suicidal self-injury), they are viewed to be complementary (see page 17 section 1.2.2. (i) for a review of models). Moreover, it is deemed permissible that both be used due to the focus of this thesis. That is, self-harm regardless of suicidal intent which is all encompassing.

Chapter 2

Schneidman (1964) proposed that sleep could act as an interruption of consciousness and provide temporary relief. Following this notion and with the frame work of the CoP (Williams, 1997, 2001), one would expect nightmares to be positively associated to entrapment. That is, if healthy sleep offers relief (escape) from distressing mental state (e.g. feeling defeated) one would expect nightmares to be act as increasing perception of entrapment. However, survey data from Chapter 2 indicated that nightmares could only predict defeat and not entrapment. Moreover, the effect size for nightmares predicting defeat was small.

The association of nightmares to defeat can be explained in several ways. It is possible that nightmares are seen as a failure in and of themselves. That is, the inability of the individual to achieve peaceful sleep may be perceived as a
failure. However, further study beyond the scope of this thesis into attitudes and dysfunctional beliefs towards sleep would be required to explore this notion. Alternatively and following the dreaming literature (Levin & Nielsen, 2007, 2009), the negative affective load associated with a defeating event would increase the need for new fear extinction memories. These memories would therefore be created during dreaming and contain attributes of fear memories which may contain defeat themes. These themes and the evoking the waking of the sleeper from the nightmare would increase perceived defeat. Again, to verify this supposition, further research is needed. Specifically, polysomnographic research where participants’ negative dream themes could be explored in a manner similar to the study performed by Hobson, Pace-Schott, & Stickgold (2003) where participants were woken during REM and asked to recount the content of their dreams.

The elevated levels of negative affect being found in the nightmare group follows the assertions of the existing literature (Bernert & Joiner, 2007) and reflect the EAM’s emotional response to a stressor. And, while the CoP does not directly reference negative emotions, feelings of defeat can be categorised as negatively valenced.

As entrapment was not found to be significantly related to our predictor (nightmares), it was dropped from further investigation. This is to reflect the need for empirical validation of variables which would form an explanatory mechanism linking nightmares to self-harm.
Chapter 3

Findings from the diary study (Chapter 3) showed post-sleep negative affect to mediate the relationship between nightmares and post-sleep SITBs. The EAM (Chapman et al., 2006) frames self-harm as a maladaptive coping strategy which regulates negative affect linked to aversive internal experiences brought on by a stressor. Therefore, in this context, nightmares could be seen as the stressor which elicits negative internal experiences (i.e. the mediating negative affect). This is then regulated by engaging in self-harmful behaviours. Williams (1997, 2001) also suggest that strong negative emotions are present prior to the ‘conservation-withdrawal’ phase which sets in with self-harm of higher suicidal intent. The fact that a model could not be computed on participants without a history of self-harm is also telling. It suggests that participants must have previously self-harmed to display this effect. That is, self-harm must be embedded in their behavioural repertoire. Similarly to the EAM where by self-harm is a conditioned response, SITBs may occur in self-harm participants after a nightmare as a conditioned coping strategy to regulate negative affect.

Chapter 4

The dream report (Chapter 4) and linguistic frequency analysis using LIWC software did not indicate differences between self-harm and non-self-harm groups on categories which would fit easily with key component of either the CoP or the EAM. For instance, there were no group differences on negative affective words (which we might expect in self-harm group according to the EAM). Nor were any differences found on ‘Personal Concerns’ categories
words (e.g. Achievements or Work subcategories) which would likely be related to defeat considering the construct being defined as stress from a loss of status (real or perceived). However, the method of analysis does not allow for evaluation of context in which the words are used. As such pertinent themes relating to either the EAM or CoP may go undetected. Further thematic analysis of dream logs would be required and are recommended.

Chapter 5

Model 3 (figure 9, pp. 188) incorporates negative affect and hyperarousal. Both are closely linked to the EAM. Negative affect reflects the emotional response due to a stressor, and hyperarousal reflects the emotional intensity/difficulties regulating emotions when aroused. Moreover, Model 3 incorporates the latent variable indicators defeat and hopelessness from the CoP, and avoidance from the EAM. All model pathways were found to be significant and the model met the criteria for data fit as such it was retained. This combination of variables is able to predict the probability of current self-harm (<1 month) engagement from a sample of individuals with an existing history self-harm (i.e. where self-harm is already in a behavioural repertoire and may be a conditioned response, reflecting the findings of previous chapters). Thus, the model proposed borrows from both the CoP and EAM. However, it must be noted that prior to this chapter, the literature had been vague on its definitions of ‘cues’. Indicators which make up this latent variable were selected due to prior literature (Klonsky, Kotov, Bakst, Rabinowitz, & Bromet, 2012; O’Connor, 2003; Rasmussen et al., 2010; Chapman et al., 2006; Willimas, 1997, 2001) highlighting their roles as precursors to self-harmful behaviours. As shown by Table 8 (pp. 126), these variables were all significantly associated with
nightmares. However, it is suggested that these latent indicators and their role in a mechanism linking nightmares to self-harm be explored further.

Chapter 6

Chapter 6 provided empirical support for participants with clinical levels of nightmare (DDNSI ≥11) having a lower tolerance to stressors (PVSAT-C task) than participants with subclinical nightmare levels. This reduced tolerance to stressors reflects the EAM’s poor distress tolerance criterion which moderates the link between emotional response to a stressor (e.g. negative affect) and avoidance (Chapman et al., 2006). However, physiological responses to negatively valenced stimuli did not indicate increased physiological responses in clinical nightmare group participants compared to subclinical nightmare participants as would be expected by the EAM. The absence of physiological response differences between neutral and negative stimuli (see appendix N\textsuperscript{15}) with-in participants explains the lack of between group differences. While a modified protocol for a GNAT (Nosek & Banaji, 2001) was implemented to test ‘self-harm cue’ detection, no differences were found between clinical and subclinical nightmare groups. It is suggested that this effect may either be fully mediated by the reduced stress tolerance and thus undetectable, or that this difference does not exist. Further investigation into the components of the latent variable (i.e. defeat, avoidance and hopelessness) should be undertaken with a simpler task. The author suggests a dot probe task (MacLeod, Mathews, & Tata, 1986) utilising words and themes related to defeat, avoidance and hopelessness.

\textsuperscript{15} The lack of differences at with-in participants (negative vs. neutral stimuli) on galvanic skin response appeared to have been caused by faulty GSR electrodes.
Overview

The findings of the present thesis suggest a mechanism linking nightmare to self-harm regardless of intent which reflects aspects of both the Experiential Avoidance Model (Chapman et al., 2006) and the Cry of Pain (Williams, 1997, 2001). However, the EAM appears to be more fitting due to its detailed and dynamic account of the early stages of self-harm compared to the overview of the full suicidal spectrum offered by the CoP. Moreover, as only defeat was found to be significantly associated with nightmares, the absence of entrapment reduces the well-established predictive utility of the CoP (O'Connor, 2003; Rasmussen et al., 2010). The EAM provides a framework which matches well with the mechanism detailed in Model 3. For instance, nightmares echo the initial stressor shown in the EAM model (pp. 16), negative affect reflecting the emotional response, and hyperarousal (reduced tolerance to stressors) reflecting the difficulty in regulating negative emotions when aroused. However, the EAM suggests that hyperarousal be a moderator. This conflicts with descriptions of a mechanism proposed by Cukrowicz et al. (2006) and Bernert & Joiner (2007). Further modeling is required to assess if hyperarousal mediates or moderates\[16\] the nightmare to self-harm relationship.

\[16\] Hyperarousal could potentially be a moderating mediator.
7.2. General conclusions

7.2.1. The role of negative affect

The association between nightmares and negative affect and its role in a mechanism linking nightmares to self-harm has been hypothesised to be of importance due to nightmares’ mood deregulatory effect (Agargun & Cartwright, 2003; Agargun et al., 2007; Bernert & Joiner, 2007; Nielsen & Levin, 2007, 2009). That is, nightmares increase levels of negative affect upon waking due to the recombined memory which comprises the nightmare content to have levels of fear and dysphoria too intense for adequate fear extinction. Thus, the fear experienced during the nightmare remains upon waking and increases levels of negative affectivity.

Chapter 2 demonstrated that levels of negative affect were higher in those suffering from clinically significant levels of nightmares compared to those participants with subclinical levels of nightmares. These findings provided support for Bernert & Joiner’s (2007) hypothesis that negative affect would contribute to increased suicidality. Additionally, although not specifically explored apriori, our GEE analysis (Chapter 3 pp.78-79) indicated that pre-sleep negative affect (entered as a covariate) was a significant predictor of nightmare occurrence. However, this pre-sleep negative affect did not increase the likelihood of self-injurious thoughts and behaviours upon waking, while post-sleep negative affect mediated the relationship between nightmares and self-harm.

This reflects theoretical and empirical research (Agargun & Cartwright, 2003; Nielsen & Levin, 2007, 2009) which show that pre-sleep negative affect
impacts on REM sleep affectivity. Specifically, how negative emotions pre-sleep can increase dysphoric emotions during REM sleep, turning a dream into a nightmare. Thus negative affect appears to be a trigger for nightmares. However, our mediation analysis in Chapter 3 (pp.79-80) highlighted a mediation role for negative affect in a mechanism linking nightmares to self-injurious thoughts and behaviours. This mediational role was further demonstrated in Model 3 (Figure 9), whereby negative affect mediated the relationship between nightmares and hyperarousal, and mediated the relationship between nightmares and the latent variable ‘self-harm cues’.

![Figure 9 - Model 3 (recap.)](image)

Depressive symptoms are known to be prominent in those exhibiting sleeping difficulties (Franzen & Buysse, 2008) and suicidal behaviours (Nock
et al., 2008). As such, our analyses sought to statistically control for depressive symptoms in all analyses reporting in this thesis. Therefore, the increased negative affect displayed in those experiencing high levels of nightmares and the mediational role of negative affect in a mechanism linking nightmares to self-harm cannot be dismissed as a simple manifestation of depressive symptoms.

Additionally, this thesis has sought to explore negative affect’s role in this mechanism by a variety of methods. Cross-sectional self-report methodology which mirrors the prevalent method of measurement found in the literature (Krakow et al., 2000; Tanskanen et al., 2001; Bernert et al., 2005; Cukrowicz et al., 2006; Sjostrom et al., 2007, 2009; Krakow et al., 2011; Nadorff et al., 2011; Susánszky, Hajnal, & Kopp, 2011; Ribeiro et al., 2012) was used in Chapter 2. Longitudinal diary methodology was applied in Chapters 3 and 4. While Chapter 6 tested negative affect differences between high and low nightmare groups using psycho-physiological methodology. Although the latter was not able to show group differences in responses to negatively valenced affective stimuli, possibly due to the mild intensity of the stimuli used limiting the size of the physiological response. This mix of longitudinal (Chapter 3) and cross-sectional (Chapter 2 and 5) designs have shown negative affect to be an integral part of the mechanism linking nightmares to self-harm risk.

In sum, this thesis has contributed to our understanding of the role of negative affect by highlighting its partial mediational role between nightmares and post-sleep SITBs in Chapter 3, and its mediational role between nightmares and hyperarousal (Chapter 5).
When describing a potential mechanism linking nightmares to suicidal ideation to explain their findings, Cukrowicz et al. (2006) posited that the negative emotions elicited by nightmares would lead to increased sensitivity to stressors. Additionally, a review article by McCall & Black (2013) has put forward a descriptive mechanism which links sleep disorders to increased suicidality. This article describes hyperarousal as a mediator between nightmares and suicidal behaviours and cognitions. Hyperarousal (Joiner et al., 1999) was selected as a measurable construct as its characteristics (exaggerated responses to stimuli, reduced pain tolerance, and increased agitation) mirror the description of increased sensitivity to stressors given by Cukrowicz et al. (2006).

Due to the strong theoretical and empirical evidence (Han et al., 2012; McCall & Black, 2013) linking hyperarousal to both sleep disturbances and suicidality, hyperarousal was included in Model 3. Our findings reflected the descriptive model of McCall & Black (2013) as hyperarousal mediated the relationship between nightmares and ‘self-harm cues’. Moreover, SEM modification indices provided during our modeling (Chapter 5) suggested hyperarousal as a mediator between negative affect and ‘self-harm cues’, highlighting the importance of hyperarousal in our dataset.

The increased sensitivity to stressors which characterises hyperarousal was tested in Chapter 6 using a behavioural stress paradigm, the PVSAT-C. Our results indicated that participants with clinically significant levels of nightmares quit this stress inducing task considerably faster than participants
with subclinical nightmare levels. This effect remained upon controlling for depressive symptoms, past self-harm behaviour and mathematical ability. The latter being controlled as the PVSAT-C induces stress through speedy mental arithmetic. Our analysis indicated that the effect observed was of a medium size, according to effect size descriptions of Cohen (1988). A behavioural effect such as this one is not negligible and presents an avenue for intervention. Cost effective stress resilience interventions such as that of Steinhardt & Dolbier (2010) offer an alternative when intervention such as Imagery Rehearsal Therapy (Krakow & Zadra, 2010) is not feasible, or could compliment the latter.

In sum, this thesis has contributed to our understanding of the role of hyperarousal by showing its mediational role between nightmares and ‘self-harm cues’ (Chapter 5), and by showing that individuals suffering from clinical levels of nightmares are hyperaroused and exhibit an increased sensitivity to stressor.

7.2.3. Sensitivity to self-harm cues – undetected or non-existent?

The latent variable ‘self-harm cues’ was composed of prominent variables from the self-harm and suicide literature, that is, experiential avoidance from the Experiential Avoidance Model of self-injury (Chapman et al., 2006), and defeat and hopelessness from the Cry of Pain model of suicide (Williams, 1997, 2001). As the literature has shown nightmares to be associated to both suicidality (Bernert & Joiner, 2007) and emotion dysregulation (Nielsen & Levin, 2007, 2009; Agargun & Cartwright, 2003), while experiential avoidance is associated with self-harm and emotion
regulation (Chapman et al., 2006). Thus, the similarities and potential association between nightmares and experiential avoidance became axiomatic, and the latter was selected to be part of the latent variable ‘self-harm cues’. Likewise, defeat and hopelessness have been found to be strong predictors of suicidal spectrum behaviours including self-harm (O’Connor, 2003; Rasmussen et al., 2010; McMillan et al., 2007). Moreover, our findings in Chapter 2 indicated higher levels of defeat could be found in participants suffering from elevated nightmare levels. While theoretically, hopelessness has been associated with high nightmares (Agargun et al., 1998). Thus, defeat and hopelessness were selected as additional indicators to ‘self-harm cues’.

Our studies failed to show support for ‘self-harm cues’ using a cross-sectional behavioural task, the GNAT. However, Model 3 and the direct and indirect effects obtained from it (Table 12, pp.136) suggest that our lack of detection of direct effects may be due to a full mediation between nightmares and ‘self-harm cues’ by negative affect and sensitivity to stress (hyperarousal). Further research is required to verify this mediation.

In sum, this thesis has contributed to our understanding of ‘self-harm cues’ suggest by Cukrowicz et al. (2006) by firstly providing a measureable latent construct (Chapter 5). Moreover, we have applied behavioural methods in the hopes of measuring ‘self-harm cue’ sensitivity in those experiencing clinical nightmare levels. While not empirically supported, we suggest this may be due to a full mediation based on data from our model (Chapter 5). We suggest further research to explore the role self-harm cues in greater depth, subject to which model refinement should be considered.
7.2.4. *The mechanism linking nightmares to increased risk of self-harm engagement.*

This thesis set out to investigate the mechanism linking nightmare to increased risk of self-harm. Through a variety of methodologies, a model has been created and tested.

Based on these results, we propose that nightmares are a unidirectional risk factor for self-injurious thoughts and behaviours upon waking (Chapter 3). Nightmares increase both, levels of negative affect (Chapters 2, 3 & 5) due to the lack of emotion regulation (Agarrgun & Cartwright, 2003; Nielsen & Levin, 2007, 2009), and sensitivity to daily stressors as individuals are hyperaroused (Chapters 5 & 6). It remains unclear if this increased negative affect and stress sensitivity increase sensitivity to self-harm cues (Chapters 6), although our model suggests this is the case (Chapter 5). The decreased tolerance to stress in combination to the elevated levels of negative affect (and potentially self-harm cue sensitivity – although not corroborated by our findings), leave the individual vulnerable to self-harm engagement.

Nock et al. (2009)’s findings indicate that sleep is an alternative emotion regulating coping strategy when feeling self-injurious needs. Nightmares’ disruption of the emotional regulatory process of dreaming (Nielsen & Levin, 2007, 2009) increased the need for emotion regulation during waking periods. In combination with a decreased tolerance to stressors, the need for emotion regulation may be sufficient for individuals regulate via a maladaptive coping strategy such as self-harm (Chapman et al., 2006; Klonsky, 2009). O’Connor, Rasmussen, & Hawton (2012) have demonstrated individuals who engage in self-harm report significantly more life stress that
self-harm ideators. Nightmares’ impact on stress resilience may form part of these stressors, as demonstrated by our behavioural findings (Chapter 6 – PVSAT-C).

7.2.5. Conclusion and implications for future research

The descriptive models put forth in the literature (Cukrowicz et al., 2006; Bernert & Joiner, 2007; McCall & Black, 2013) which formed the basis of this thesis, have been partially supported. Although the sensitivity of ‘self-harm cues’ requires further study. Our findings have extended a mechanism linking nightmares to suicidal behaviours and cognitions to self-harm regardless of suicidal intent or motivation to die.

While Model 3 (Figure 9) requires refinement and replication, following the current state of evidence, we propose that nightmares lead to post-sleep negative affect and reduced stress resilience. This, in turn, leads to an increasing vulnerability to self-harm in order to regulate emotions; regulation which had previously been interrupted by the occurrence of a nightmare.

Importantly, our findings corroborate and extend independent research and suggestions by theorists (Cukrowicz et al., 2006; Bernert & Joiner, 2007; Klonsky, 2009; Chapman, 2006; McCall & Black, 2013). This thesis and shown the importance of nightmares and their content (perceptual processes and body), to increasing vulnerability to self-harm. Moreover, by investigating the psychological mechanism linking the nightmares to self-injury, this thesis has provided avenues for intervention to complement direct treatments aimed at reducing nightmares such as Imagery Rehearsal Therapy (Krakow & Zadra, 2003).
2010) or Lucid Dreaming (Jaap Lancee, Bout, Spoormaker, & van den Bout, 2010). We offer targets for interventions additional to nightmares themselves; post-sleep negative affect and sensitivity to stress, which can be moderated through stress resilience training.

Moreover, research on the impact of nightmare reduction treatment on the rate of self-harm is needed. A robust, randomised controlled trial, as suggested early in this thesis, would be welcome. Chapter 3 has demonstrated the unidirectional predictive relationship of nightmares on self-harm. However, a controlled trial would as it would help further elucidate the causal direction of this relationship.
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Appendices

Appendix A: Insomnia Severity Index (ISI)

For each question, please CIRCLE the number that best describes your answer. Please rate the CURRENT (i.e. LAST 2 WEEKS) SEVERITY of your insomnia problem(s).

1. Difficulty falling asleep
   Answer: 0= None, 1= Mild, 2= Moderate, 3= Severe, 4= Very Severe

2. Difficulty staying asleep
   Answer: 0= None, 1= Mild, 2= Moderate, 3= Severe, 4= Very Severe

3. Problems waking up too early
   Answer: 0= None, 1= Mild, 2= Moderate, 3= Severe, 4= Very Severe

4. How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern?
   Answer: 0= Very Satisfied, 1= Satisfied, 2= Moderately Satisfied, 3= Dissatisfied, 4= Very Dissatisfied

5. How NOTICEABLE to others do you think your sleep problem is in terms of impairing the quality of your life?
   Answer: 0= Not At All Noticeable, 1= A Little, 2= Somewhat, 3= Much, 4= Very Much Noticeable

6. How WORRIED/DISTRESSED are you about your current sleep problem?
   Answer: 0= Not At All Worried, 1= A Little, 2= Somewhat, 3= Much, 4= Very Much Worried

7. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?
   Answer: 0= Not At All Interfering, 1= A Little, 2= Somewhat, 3= Much, 4= Very Much Interfering

Total scores are calculated as the outcome measure. Total score categories: 0–7 = No clinically significant insomnia, 8–14 = Sub-threshold insomnia, 15–21 = Clinical insomnia (moderate severity), 22–28 = Clinical insomnia (severe).
Appendix B: Disturbing Dreams and Nightmare Severity Index (DDNSI)

1. In the last week how many nights have you had nightmares?

   Answer: 0-7

2. How many nightmares have you experienced in the last week?

   Answer: 0-14 (if over 14, select 14)

3. How often have your nightmares awoken you?

   Answer: 0=Never, 1= Not often, 2= Sometimes, 3= Often, 4= Always

4. How severe is your nightmare problem?

   Answer: 0=No problem, 1= Minimal problem, 2= Mild problem, 3= Moderate problem, 4= Moderately severe problem, 5= Severe problem, 6= Very severe problem

5. How intense are your nightmares?

   Answer: 0=Not intense at all, 1= Minimal intensity, 2= Mild intensity, 3= Moderate intensity, 4= Moderately severe intensity, 5= Severe intensity, 6= Extremely severe intensity

The scale is summed to produce an overall index of nightmare severity (range = 0-37). Scores above 10 are consistent with clinical levels of disturbing dreams and nightmares. Scores above 20 are generally consistent with a more severe nightmare disorder.
Appendix C: Positive Affect and Negative Affect Schedule

Please indicate to what extent you have felt this way during the PAST 2 WEEKS:

1. Interested
2. Distressed
3. Excited
4. Upset
5. Strong
6. Guilty
7. Scared
8. Hostile
9. Enthusiastic
10. Proud
11. Irritable
12. Alert
13. Ashamed
14. Inspired
15. Nervous
16. Determined
17. Attentive
18. Jittery
19. Active
20. Afraid

Item Response Anchors are 1 = Not at all; 2 = A little; 3 = Moderately; 4 = Quite a bit; 5 = Extremely.

PA subscale consists of items: 1, 3, 5, 9, 10, 12, 14, 16, 17& 19.

NA subscale consists of items: 2, 4, 6, 7, 8, 11, 13, 15, 18 & 20.

Total scores are obtained as the outcome measure for each subscale.
**Appendix D: Entrapment Scale**

Participants to indicate on a 5-point scale the degree to which the items represented their thoughts and feelings. The response options for the entrapment scale were `not at all like me` (0), `a little bit like me` (1), `moderately like me` (2), `quite a bit like me` (3) and `extremely like me` (4).

(a) *Internal entrapment*

1. I want to get away from myself
2. I feel powerless to change myself
3. I would like to escape from my thoughts and feelings
4. I feel trapped inside myself
5. I would like to get away from who I am and start again
6. I feel I'm in a deep hole I can't get out of

(b) *External entrapment*

1. I am in a situation I feel trapped in
2. I have a strong desire to escape from things in my life
3. I am in a relationship I can't get out of
4. I often have the feeling that I would just like to run away
5. I feel powerless to change things
6. I feel trapped by my obligations
7. I can see no way out of my current situation
8. I would like to get away from other more powerful people in my life
9. I have a strong desire to get away and stay away from where I am now
10. I feel trapped by other people

Total scores are calculated as the outcome measure.
Appendix E: Defeat Scale

The instructions asked participants to indicate on a 5-point scale the degree to which the items represented their thoughts and feelings. For the defeat scale participants were asked how much they had felt defeated in the previous seven days. (e.g. item 3; I feel defeated by life). Response options were `never' (0), `rarely' (1), `sometimes' (2), `mostly' (3) and `always/all the time' (4).

1. I feel that I have not made it in life
2. I feel that I am a successful person
3. I feel defeated by life
4. I feel that I am basically a winner
5. I feel that I have lost my standing in the world
6. I feel that life has treated me like a punchbag
7. I feel powerless
8. I feel that my confidence has been knocked out of me
9. I feel able to deal with whatever life throws at me
10. I feel that I have sunk to the bottom of the ladder
11. I feel completely knocked out of action
12. I feel that I am one of life's losers
13. I feel that I have given up
14. I feel down and out
15. I feel I have lost important battles in life
16. I feel that there is no fight left in me

Total scores are calculated as the outcome measure. Items 2, 4 & 9 are reverse scored.
Appendix F: Beck’s Depression Inventory – II (BDI-II)

Please read carefully each of the statements below and select one which best describe the way you have been feeling in the LAST TWO WEEKS. Be sure to read all of the statements in each group before making your choice.

1. Sadness
0 - I do not feel sad
1 - I feel sad much of the time
2 - I am sad all the time
3 - I am so sad or unhappy that I can’t stand it

2. Pessimism
0 - I am not discouraged about my future
1 - I feel more discouraged about my future than I used to be
2 - I do not expect things to work out for me
3 - I feel my future is hopeless and will only get worse

3. Past Failure
0 - I do not feel like a failure
1 - I have failed more than I should have
2 - As I look back, I see a lot of failures
3 - I feel I am a total failure as a person

4. Loss of Pleasure
0 - I get as much pleasure as I ever did from the things I enjoy
1 - I don’t enjoy things as much as I used to
2 - I get very little pleasure from the things I used to enjoy
3 - I can’t get any pleasure from the things I used to enjoy

5. Guilty Feelings
0 - I don’t feel particularly guilty
1 - I feel guilty over many things I have done or should have done
2 - I feel quite guilty most of the time
3 - I feel guilty all of the time

6. Punishment Feelings
0 - I don’t feel I am being punished
1 - I feel I may be punished
2 - I expect to be punished
3 - I feel I am being punished
7. Self-Dislike
0- I feel the same about myself as ever
1- I have lost confidence in myself
2- I am disappointed in myself
3- I dislike myself

8. Self-Criticalness
0- I don't criticise or blame myself more than usual
1- I am more critical of myself than I used to be
2- I criticise myself for all of my faults
3- I blame myself for everything bad that happens

9. Suicidal Thoughts Or Wishes
0- I don't have any thoughts of killing myself
1- I have thoughts of killing myself, but I would not carry them out
2- I would like to kill myself
3- I would kill myself if I had the chance

10. Crying
0- I don't cry anymore than I used to
1- I cry more than I used to
2- I cry over every little thing
3- I feel like crying, but I can't

11. Agitation
0- I am no more restless or wound up than usual
1- I feel more restless or wound up than usual
2- I am so restless or agitated that it's hard to stay still
3- I am so restless or agitated that I have to keep moving or doing something

12. Loss of Interest
0- I have not lost interest in other people or activities
1- I am less interested in other people or things than before
2- I have lost most of my interest in other people or things
3- It's hard to get interested in anything

13. Indecisiveness
0- I make decisions about as well as ever
1- I find it more difficult to make decisions than usual
2- I have much greater difficulty in making decisions than I used to
3- I have trouble making any decisions
14. **Worthlessness**
0- I do not feel I am worthless
1- I don't consider myself as worthwhile and useful as I used to
2- I feel more worthless as compared to other people
3- I feel utterly worthless

15. **Loss of Energy**
0- I have as much energy as ever
1- I have less energy than I used to have
2- I don't have enough energy to do very much
3- I don't have enough energy to do anything

16. **Changes in Sleep Pattern**
0- I have not experienced any change in my sleep pattern
1- I sleep somewhat more than usual
1- I sleep somewhat less than usual
2- I sleep a lot more than usual
2- I sleep a lot less than usual
3- I sleep most of the day
3- I wake up 1-2 hours early and can't get back to sleep

17. **Irritability**
0- I am no more irritable than usual
1- I am more irritable than usual
2- I am much more irritable than usual
3- I am irritable all the time

18. **Changes in Appetite**
0- I have not experienced any change in my appetite
1- My appetite is somewhat less than usual
1- My appetite is somewhat more than usual
2- My appetite is much less than before
2- My appetite is much greater than usual
3- I have no appetite at all
3- I crave food all the time

19. **Concentration Difficulty**
0- I can concentrate as well as ever
1- I can't concentrate as well as usual
2- It's hard to keep my mind on anything for a long time
3- I find I can't concentrate on anything
20. **Tiredness or Fatigue**
0- I am no more tired or fatigued than usual  
1- I get more tired or fatigued more easily than usual  
2- I am too tired or fatigued to do a lot of the things I used to do  
3- I am too tired or fatigued to do most of the things I used to do  

21. **Loss of Interest in Sex**
0- I have not noticed any recent change in my interest in sex  
1- I am less interested in sex than I used to be  
2- I am much less interested in sex now  
3- I have lost interest in sex completely  

Total scores are calculated as the outcome measure.  
Classification Total Score Level of Depression:  
1-10 These ups and downs are considered normal  
11-16 Mild mood disturbances  
17-20 Borderline clinical depression  
21-30 Moderate depression  
31-40 Severe depression  
40+ Extreme depression
Appendix G: Deliberate Self-Harm Inventory (DSHI) [modified]

Please answer "yes" to a question only if you did the behaviour intentionally, or on purpose, to hurt yourself. Do not respond yes if you did something accidentally (e.g., you tripped and banged you head on accident). Also, please be assured that your responses are completely confidential.

Have you ever intentionally (i.e., on purpose):

1. Cut your wrist, arms, or other area(s) of your body
2. Burned yourself with a cigarette?
3. Burned yourself with a lighter or a match?
4. Carved words into your skin? (not tattoo)
5. Carved pictures, designs, or other marks into your skin? (not tattoo)
6. Severely scratched yourself, to the extent that scarring or bleeding occurred?
7. Bit yourself, to the extent that you broke the skin?
8. Rubbed sandpaper on your body?
9. Dripped acid onto your skin?
10. Used bleach, comet, or oven cleaner to scrub your skin?
11. Stuck sharp objects such as needles, pins, staples, etc. into your skin, not including tattoos, ear piercing, needles used for drug use, or body piercing?
12. Rubbed glass into your skin?
13. Broken your own bones?
14. Banged your head against something, to the extent that you caused a bruise to appear?
15. Punched yourself, to the extent that you caused a bruise to appear?
16. Prevented wounds from healing?
17. Done anything else to hurt yourself that was not asked about in this questionnaire? If yes, what did you do to hurt yourself? (eg. self-poisoning with medication)

Participants indicating ‘yes’ to any item was categorised have having a history of self-harm. Follow up questions relating to the last occurrence of behaviours was used to distinguish between current self-harm (<1 month) and history of self-harm (>1month).
# Dream, Nightmare & Mood Diary

**Participant No.:_______**

## Day 1 - Pre-sleep (Complete before sleep)

**Date:** __/__/____  
**Time:** ____ pm

Q1. Please indicate to what extent you CURRENTLY (right now) feel the feelings and emotions described:

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Q2. TODAY, have you at any point:

A) had thoughts of deliberately self-injury?  
   Yes □  No □

B) deliberately injured yourself?  
   Yes □  No □
Day 1 - Post-sleep (Complete when you wake up)
Date: __/__/____
Time: am

Q1. Please indicate to what extent you CURRENTLY (right now) feel the feelings and emotions described:

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<th>Very slightly/ Not at all</th>
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Q2. SINCE WAKING UP, have you at any point:

A) had thoughts of deliberately self-injury? Yes □ No □
B) deliberately injured yourself? Yes □ No □

*If you had an unpleasant or negative dream last night, please complete the supplementary page describing that dream (See page 3).

Thank you for completing Day 1 of your Sleep diary.
S1. Please describe the negative dream or nightmare you remember most from last night in as much detail as possible (Try to include details on: Descriptive elements e.g. time, Characters, Activities, Events, Interaction, Settings, Objects, Success or failures, Fortune or misfortune & Emotions).

If you need more space to describe the dream please write on additional space on page 4.

S2. Did this dream/nightmare wake you up? □ Yes □ No

S3. Is this dream/nightmare recurrent? □ Yes □ No

Q5. Please rate the properties of your dream/nightmare on the following scale:

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<th>Very slight/ Not at all</th>
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Vividness

Intensity

Distress
Appendix I: Chapter 4 exploratory analysis

II. Does the negative dream content differ as a function of self-harm recency?

Exploratory analysis of self-harm recency (no SH, history of SH \( \geq 1 \) month, and current SH\([\leq 1\text{month}]\)) to assess group differences on the 5 psychological constructs (affective, cognitive, social, perceptual, and biological processes) and 7 personal concerns (work, achievement, home, leisure activities, money, religion and death) was performed using MANCOVA. Means and standard deviations for these groups are reported in table 4.2.
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<th>Table 11 - Linguistic frequency per self-harm recency</th>
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As with our prior analysis, Box’s M test could not be computed due to zero values for money, religion, and death impairing covariance matrices. These categories were removed and analysed separately using ANCOVA.

A significant Box’s M test (Box M = 1.40, p < .05) for our MANCOVA indicated assumptions of homogeneity of covariance had been violated. Pillai’s trace was used as the multivariate test criterion recommended when homogeneity assumptions are not met (Tabachnick & Fidell, 2001). The multivariate effect was non-significant, \( F(18, 68) = 1.20, \ p > .05, \lambda_{\text{Pillai}} = .48 \), partial \( \eta^2 = .24 \).

Tests of between participant effects showed no significant differences between the no SH group, SH history and current SH group on social processes \( (F(2, 41) = .10, \ p > .05, \text{partial } \eta^2 = .005) \), affective processes \( (F(2, 41) = .72, \ p > .05, \text{partial } \eta^2 = .025) \), biological processes \( (F(2, 41) = 1.40, \ p > .05, \text{partial } \eta^2 = .005) \), and psychological processes \( (F(2, 41) = 1.00, \ p > .05, \text{partial } \eta^2 = .005) \).
p > .05, partial $\eta^2 = .034$), cognitive processes ($F(2, 41) = .32, p > .05$, partial $\eta^2 = .015$), and biological processes ($F(2, 41) = 1.38, p > .05$, partial $\eta^2 = .063$). However, perceptual processes revealed a significant between group difference ($F(2, 41) = 3.81, p < .05$, partial $\eta^2 = .157$). Post-hoc simple contrasts indicated that the current SH group reported significantly more perceptual processes in their negative dream reports compared to the no SH group ($p < .01$). However, while current SH participants did report more perceptual processes compared to SH history participants, this difference was marginally non-significant ($p = .058$).

There were no significant differences between the no SH, history of SH and current SH groups personal concerns relating to work ($F(2, 41) = .04, p > .05$, partial $\eta^2 = .002$), achievements ($F(2, 41) = 1.72, p > .05$, partial $\eta^2 = .077$), leisure activities ($F(2, 41) = .46, p > .05$, partial $\eta^2 = .022$), and home ($F(1, 42) = .77, p > .05$, partial $\eta^2 = .036$).

ANCOVA revealed no significant difference between the 3 groups for religion, ($F(2, 41) = .70, p > .05$, partial $\eta^2 = .033$) or death ($F(2, 41) = .44, p > .05$, partial $\eta^2 = .021$). However, there was a significant difference between groups in reports of money words, ($F(2, 41) = 3.50, p < .05$, partial $\eta^2 = .146$). Post-hoc simple contrasts revealed that the no SH group reported a significantly higher number of money words than the current SH group ($p < .05$). Similarly, history of SH participants reported fewer money words than no SH participants ($p < .05$). There were no difference between SH history and current SH groups on money word usage ($p > .05$).
I2. Exploring constructs of interest as a function of self-harm recency?

Our previous analysis revealed perceptual processes to differ based on self-harm recency. An exploratory MANCOVA of subcategories of the perceptual processes LIWC dictionary was performed investigating differences between self-harm recency groups (no SH, SH history, current SH) while controlling for depressive symptoms, word count and percentage of words captured by the dictionary.

A significant Box’s M test (Box M = 41.52, p<.05) for our MANCOVA indicated assumptions of homogeneity of covariance had been violated. Pillai's trace was used as the multivariate test criterion. The multivariate effect was non-significant, $F(6, 80) = 2.00, p>.05$, $\lambda_{\text{Pillai}} = .26$, partial $\eta^2 = .13$. Tests of between participant effects showed no significant differences between the no SH group, SH history and current SH group on ‘see’ ($F(2, 41) = 1.84, p>.05$, partial $\eta^2 = .082$) or ‘hear’ words ($F(2, 41) = .25, p>.05$, partial $\eta^2 = .012$). However, there was a significant difference between groups in reports of ‘feel’ words, ($F(2, 41) = 3.94, p<.05$, partial $\eta^2 = .161$). Post-hoc simple contrasts indicated that the current SH group reported significantly more ‘feel’ words compared to the no SH group ($p<.05$). Similarly, current SH participants reported more ‘feel’ words compared to SH history participants ($p<.05$). However there were no differences between no SH and SH history groups on frequency of ‘feel’ words reported ($p>.05$).
Appendix J: Impact of Event-Revised Hyperarousal subscale

Below is a list of difficulties people sometimes have after stressful life events. Please read each item, and then indicate how distressing each difficulty has been for you DURING THE PAST SEVEN DAYS.

How much were you distressed or bothered by these difficulties?

1. I felt irritable and angry.
2. I was jumpy and easily startled.
3. I had trouble falling asleep.
4. I had trouble concentrating.
5. Reminders of it caused me to have physical reactions, such as sweating, trouble breathing, nausea, or a pounding heart.
6. I felt watchful and onguard.

Item Response Anchors are 0 = Not at all; 1 = A little bit; 2 = Moderately; 3 = Quite a bit; 4 = Extremely.

Mean scores are calculated as the outcome measure.
Appendix K: The Acceptance and Action Questionnaire (AAQ)

Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Use the following scale to make your choice.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Never True</td>
<td>Very rarely True</td>
<td>Seldom True</td>
<td>Sometimes True</td>
<td>Frequently True</td>
<td>Almost Always True</td>
<td>Always True</td>
</tr>
</tbody>
</table>

1. I am able to take action on a problem even if I am uncertain what is the right thing to do.

2. I often catch myself daydreaming about things I've done and what I would do differently next time.

3. When I feel depressed or anxious, I am unable to take care of my responsibilities.

4. I rarely worry about getting my anxieties, worries, and feelings under control.

5. I'm not afraid of my feelings.

6. When I evaluate something negatively, I usually recognize that this is just a reaction, not an objective fact.

7. When I compare myself to other people, it seems that most of them are handling their lives better than I do.

8. Anxiety is bad.

9. If I could magically remove all the painful experiences I've had in my life, I would do so.

Ratings on Items 1, 4, 5, and 6 are reversed for scoring purposes.

Total scores are calculated as the outcome measure.
Appendix L: Beck’s Hopelessness Scale (BHS)

This questionnaire consists of a list of twenty statements. Please read the statements carefully one by one.

If the statement describes your attitude for the past week, including today, write ‘T’ or ‘true’ (1). If the statement is false for you, write ‘F’ or ‘false’ (0). Please be sure to read each sentence.

1. I look forward to the future with hope and enthusiasm
2. I might as well give up because there’s nothing I can do to make things better for myself
3. When things are going badly, I am helped by knowing that they can’t stay that way for ever
4. I can’t imagine what my life would be like in ten years
5. I have enough time to accomplish the things I most want to do
6. In the future I expect to succeed in what concerns me most
7. My future seems dark to me
8. I happen to be particularly lucky and I expect to get more of the good things in life than the average person
9. I just don’t get the breaks, and there’s no reason to believe that I will in the future
10. My past experiences have prepared me well for my future
11. All I can see ahead of me is unpleasantness rather than pleasantness
12. I don’t expect to get what I really want
13. When I look ahead to the future I expect I will be happier than I am now
14. Things just won’t work out the way I want them to
15. I have great faith in the future
16. I never get what I want, so it’s foolish to want anything
17. It is very unlikely that I will get any real satisfaction in the future
18. The future seems vague and uncertain to me
19. I can look forward to more good times than bad times
20. There’s no use in really trying to get something I want because I probably won’t get it

Ratings on Items 1, 3, 5, 6, 8, 10, 13, 15, and 19 are reversed for scoring purposes.

Total scores are calculated as the outcome measure.
## Appendix M: Chapter 6 psycho-physiological paradigm stimuli list

Table M1 - IAPS stimuli for Psycho-physiological paradigm

<table>
<thead>
<tr>
<th>Negative valence</th>
<th>Neutral valence</th>
<th>Positive valence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1022.jpg Snake</td>
<td>1450.jpg Gannet</td>
<td>4623.jpg Romance</td>
</tr>
<tr>
<td>1050.jpg Snake</td>
<td>2210.jpg Neutral Face</td>
<td>5700.jpg Mountains</td>
</tr>
<tr>
<td>1051.jpg Snake</td>
<td>2235.jpg Butcher</td>
<td>1440.jpg Seal</td>
</tr>
<tr>
<td>1200.jpg Spider</td>
<td>5001.jpg Sunflower</td>
<td>2071.jpg Baby</td>
</tr>
<tr>
<td>1201.jpg Spider</td>
<td>5531.jpg Mushroom</td>
<td>2216.jpg Children</td>
</tr>
<tr>
<td>1220.jpg Spider</td>
<td>5731.jpg Flowers</td>
<td>8500.jpg Gold</td>
</tr>
<tr>
<td>1205.jpg Spider</td>
<td>5740.jpg Plant</td>
<td>8501.jpg Money</td>
</tr>
<tr>
<td>1280.jpg Rat</td>
<td>6150.jpg Outlet</td>
<td>8502.jpg Money</td>
</tr>
<tr>
<td>1303.jpg Dog</td>
<td>7002.jpg Towel</td>
<td>8510.jpg Sports Car</td>
</tr>
<tr>
<td>2692.jpg Bomb</td>
<td>7009.jpg Mug</td>
<td>1710.jpg Puppies</td>
</tr>
<tr>
<td>6242.jpg Gang</td>
<td>7041.jpg Baskets</td>
<td>1811.jpg Monkeys</td>
</tr>
<tr>
<td>6244.jpg Aimed Gun</td>
<td>7130.jpg Truck</td>
<td>2080.jpg Babies</td>
</tr>
<tr>
<td>6571.jpg Car Theft</td>
<td>7150.jpg Umbrella</td>
<td>4625.jpg Couple</td>
</tr>
<tr>
<td>2120.jpg Angry Face</td>
<td>7170.jpg Light Bulb</td>
<td>5260.jpg Waterfall</td>
</tr>
<tr>
<td>2691.jpg Riot</td>
<td>7185.jpg Abstract Art</td>
<td>5480.jpg Fireworks</td>
</tr>
<tr>
<td>6212.jpg Soldier</td>
<td>7205.jpg Scarves</td>
<td>7200.jpg Brownie</td>
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<tr>
<td>6213.jpg Terrorist</td>
<td>7233.jpg Plate</td>
<td>7330.jpg Ice Cream</td>
</tr>
<tr>
<td>9421.jpg Soldier</td>
<td>7038.jpg Shoes</td>
<td>8170.jpg Sailboat</td>
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Appendix N: Chapter 6 exploratory analysis - Exploring within participant psycho-physiological differences to positive, neutral and negative stimuli

To test if the strength of psycho-physiological responses differed based on stimuli valence three one-way repeated measures ANCOVAs were performed. Depressive symptoms were entered as a covariate. SCR mean for positive, neutral and negative stimuli were entered as the first with-in subject factor. SCR MRA and HR mean for negative, neutral and positive stimuli were used as the second and third with-in subject factor respectively. Means, standard deviations and Z-scores for SCR mean, SCR MRA and HR mean are reported in Table N1.

Table N1 – Psycho-physiological means for control and NM group

<table>
<thead>
<tr>
<th></th>
<th>Total sample (n=85)</th>
<th>Control (n=42)</th>
<th>NM group (n=43)</th>
<th>F-ratio</th>
<th>p</th>
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<tr>
<td>Depressive symptoms</td>
<td>13.88 (9.55)</td>
<td>9.81 (7.48)</td>
<td>17.86 (9.75)</td>
<td>18.19</td>
<td>&lt;.001</td>
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<tr>
<td>SCR mean negative (log)</td>
<td>.40 (.19)</td>
<td>.40 (.18)</td>
<td>.40 (.20)</td>
<td>0.01</td>
<td>.933</td>
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<tr>
<td>SCR mean neutral (log)</td>
<td>.40 (.20)</td>
<td>.40 (.18)</td>
<td>.39 (.21)</td>
<td>0.02</td>
<td>.894</td>
</tr>
<tr>
<td>SCR mean positive (log)</td>
<td>.39 (.20)</td>
<td>.39 (.19)</td>
<td>.39 (.21)</td>
<td>0.00</td>
<td>.975</td>
</tr>
<tr>
<td>SCR MRA negative</td>
<td>.06 (.09) / .07 (.14)</td>
<td>.04 (.09) / .13 (.97)</td>
<td>1.49</td>
<td>.226</td>
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<td>SCR MRA neutral</td>
<td>.02 (.04) / .02 (.06)</td>
<td>.02 (.04) / .06 (.12)</td>
<td>0.29</td>
<td>.594</td>
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<tr>
<td>SCR MRA positive</td>
<td>.03 (.05) / .04 (.07) / .25 (.29)</td>
<td>.02 (.03) / .24 (.52)</td>
<td>5.24</td>
<td>.025</td>
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<td>HR mean negative</td>
<td>75.08 (22.64)</td>
<td>71.09 (13.68)</td>
<td>78.89 (25.51)</td>
<td>2.53</td>
<td>.115</td>
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<tr>
<td>HR mean neutral</td>
<td>75.03 (22.82)</td>
<td>71.39 (19.29)</td>
<td>78.49 (25.48)</td>
<td>2.06</td>
<td>.155</td>
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<tr>
<td>HR mean positive</td>
<td>74.71 (22.99)</td>
<td>70.29 (20.38)</td>
<td>78.92 (24.74)</td>
<td>3.03</td>
<td>.086</td>
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Our first one-way repeated measure ANCOVA controlling for depressive symptoms assessed if SCR means (μS) differed based on stimuli valence. Mauchly’s test indicated assumptions of sphericity had been violated, χ²(2) = 16.01, p<.001. As such Greenhouse-Geisser estimates (ε =.85) were used to
correct for degrees of freedom. The results indicated no effect of stimuli valence on SCR means, $F(1.69, 137.135) = 0.47, p>.05$.

The second one-way repeated measure ANCOVA controlling for depressive symptoms assessed if SCR MRAs (µS) differed based on stimuli valence. Mauchly’s test indicated assumptions of sphericity had been violated, $\chi^2(2) = 26.39, p<.001$. Greenhouse-Geisser estimates ($\varepsilon = .78$) were used to correct for degrees of freedom. The results showed no significant differences in SCR MRA between stimuli of varying valence stimuli valence, $F(1.56, 126.47) = 0.76, p>.05$.

The final one-way repeated measure ANCOVA controlling for depressive symptoms assessed if HR mean (bpm) differed based on stimuli valence. Mauchly’s test indicated assumptions of sphericity had been met, $\chi^2(2) = 5.85, p>.05$. The results showed no significant difference in HR mean based on stimuli valence, $F(1.87, 151.33) = 0.46, p>.05$. 