# UNIVERSITY OF SOUTHAMPTON

Faculty of Social and Human Sciences

School of Psychology

## Associations between Severity of Insomnia, Attentional Control and Negative Thought Intrusions in Healthy Volunteers

by

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## General Abstract

Recent research has found an association between insomnia and both negative affect and weakened attention control, yet despite this association and the high prevalence of sleep difficulties to be found in the general population, very little research has explored the association between these factors as a result of acute sleep loss in a healthy population. The literature review therefore explores the existing evidence base for relationships between poor sleep and both emotional and cognitive functioning, by drawing upon research pertinent to insomnia and anxiety disorder. The review concludes and supports an association between attentional control and reduced emotional regulation in poor sleep, with a particular significance of increased hyperarousal, reduced inhibition of negative schema and thought intrusions as a result of sleep loss. However, further studies are needed to clarify these relationships, particularly in healthy individuals experiencing transient, acute or chronic sleep disturbance. An improved understanding of these relationships is imperative for developing better recognition, diagnosis and treatment of sleep disorders in the general population. Consistent with this suggestion, the empirical paper describes a study which examined whether specific components of acute sleep loss were associated with attentional deficits observed in anxiety; specifically a reduced ability to focus attention, and an increase in difficulties inhibiting negative thoughts. The study used a cross sectional research design involving 112 undergraduates, who all completed self-report measures of insomnia severity, trait anxiety, attentional control and cognitive failures. Participants also completed a 3-stage worry task, during which thoughts were sampled, including: an initial 5-minute breathing interval, a 5-minute worry period and a 5-minute post-worry breathing interval. Results found that increased insomnia severity, independent of anxiety, was significantly associated with increased deficits in attentional control and increased negative thought intrusions during a five-minute breathing focus period in a healthy population. Considering this impact of sleep loss, the findings recommend that a better awareness of sleep difficulties is established in both health professionals and in the general population, to enable earlier recognition and diagnosis, with the aim of preventing the development of more complex forms of insomnia and improving available treatment protocols.

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## Declaration of Authorship

I, the undersigned, confirm the work I have presented as my thesis is entirely my own work. Reference to, quotation from, and discussion of the work of any other person has been correctly acknowledged within the work in accordance with University guidelines for production of a thesis.

**Signed:** ……..
**Date:**……

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# Literature Review Paper

## Poor Sleep, Anxiety and Cognitive Processing: A Review of the Literature

*Running head: Insomnia, Anxiety and Cognitive Processing*

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**Abstract**

There is a high prevalence of sleep difficulties in the general population, which consistently impact on an individual's functioning, in particular, emotional and cognitive processing (Mental Health Foundation, 2011). Recent research has found an association between insomnia and both negative affect and weakened attentional control, yet despite this association and the high prevalence of sleep difficulties within the general population, very little research has explored the associations between these factors, as a result of acute sleep loss. The literature review therefore explores the existing evidence base for relationships between poor sleep and both emotional and cognitive functioning, by drawing upon research pertinent to insomnia and anxiety disorder. The review explores the impact of insomnia, especially on cognitive dysfunction and emotion processing difficulties, highlighting evidence which supports these impacts. Furthermore, in order to understand the processes involved in the development and maintenance of insomnia the review examines relevant models, including the behavioural, psychobiological, hyperarousal and cognitive models and the evidence supporting them. These models reveal significantly similar processes between insomnia and anxiety (and more specifically worry), in particular attentional dysfunction and emotional dysregulation, which are explored in terms of supporting literature. The review concludes and supports an association between attentional control and reduced emotional regulation in poor sleep, with a particular significance of increased hyperarousal, reduced inhibition of negative schema and thought intrusions as a result of sleep loss. However, further studies are needed to clarify these relationships, particularly in healthy individuals experiencing transient, acute or chronic sleep disturbance. An improved understanding of these relationships is imperative for developing better and earlier recognition, diagnosis and treatment of sleep disorders in the general population.

# Literature Review

## Overview of Sleep

The general population exhibit a high prevalence of sleep difficulties, as evidenced by the first large scale Great British Sleep Survey by the Mental Health Foundation (2011). This survey revealed that only 38% of respondents were considered "good sleepers" and 36% of the total sample suffered from difficulties getting to sleep, or getting back to sleep after waking in the night. These findings support other estimates which suggest that one third of the general population experience difficulties maintaining and/or initiating sleep at least once a week (LeBlanc et al., 2009). In addition to high prevalence rates sleep difficulties, and more specifically sleep disorders (for example insomnia), can be persistent and therefore complex disorders to treat. For example, a longitudinal study by Morin et al. (2009) found that 74% of individuals diagnosed with insomnia disorder suffered with the complaint one year later and 46% suffered three years later. High prevalence rates and the frequent long term nature of sleep difficulties and sleep disorders (particularly insomnia) are consequently a considerable significant public health concern, resulting in significant economic costs. These costs can include greater work absenteeism and significantly higher health service utilisation, even after controlling for other socio-demographic variables and comorbid conditions (Novak, Mucsi, Shapiro, Rethelyi, & Kopp, 2004). Research has estimated that the overall cost of insomnia, specifically, in the UK to be equivalent to two billion pounds per year (Leger, Levy, & Paillard, 1999), with 76% of the annual expenditure related to insomnia, attributable to loss of productivity (Daley, Morin, LeBlanc, Gregoire, & Savard, 2009).

Poor sleep, in general, can have substantial consequences on an individual's physical and mental wellbeing given the important role that it plays in the restoration of processes including tissue growth and maintenance of energy (Maquet, 2001). Sufficient sleep enables neural recovery and regeneration, further promoting both memory consolidation and learning, daytime cognitive functioning (Kopasz et al., 2010) as well as emotion processing (Payne & Kensinger, 2010).

Due to the restorative function of sleep it is often described and perceived as a cognitive and emotional resource beneficial for managing stress (Drake, Richardson, Roehers, Scofield, & Roth (2004). Research suggests that it is the cognitive energy gained during sleep which increases positive affect through effective emotional regulation and reduces negative affect during cognitive and emotional conflict (Zohar, Tzischinsky, Epstein, & Lavie, 2005). These findings suggest that a sleep deficit will impact greatly on an individual's emotional wellbeing, cognitive ability and performance.

Despite the high prevalence of sleep difficulties and sleep disorders in the general population, as well as the consistent impacts on daily functioning, research has found it challenging to understand and relate subjective reports of poor sleep with objective evidence (Espie & Kyle, 2008). This difficulty has consequently limited further understanding of the relationships between poor sleep and the effectiveness and efficiency of both cognitive and emotional processes.

**Aim of Review**

The aim of this review is to therefore explore the existing evidence base for the relationships between, and the consequences of, poor sleep in cognitive and emotional processing in healthy individuals. This review will focus and draw primarily upon research pertinent to insomnia and anxiety disorder, of which there is

the strongest relationship and overlap of features, especially when compared to other psychological difficulties, for example, depression. The strength of this relationship and the similarity in features is hoped to aid an understanding of how insomnia may develop over time. Therefore, the relationship between insomnia and other psychological disorders is beyond the scope of this review (see Harvey, 2011 for a review). A greater understanding of daytime dysfunction in disorders characterised by chronic sleep disturbance, notably insomnia will provide enhanced awareness and insight into the impact of sleep loss at each stage and severity of the complaint. Improved understanding will enable more efficient, individually tailored treatment to be developed, in order to target the most prominent and distressing aspects of insomnia as it presents and develops. Current treatment options for insomnia are limited and poorly researched, with particular concerns regarding the current over-dependency and use of pharmacological interventions in the treatment of insomnia. This over-reliance on medication is despite evidence for Cognitive Behaviour Therapy (CBT) being more effective, cost efficient (in the long term) and having longer lasting effects.

To date, there has been limited research in the areas outlined above, despite the high prevalence rates of insomnia in the population. Thus far, research has predominantly examined the components involved in the *maintenance* of chronic insomnia and the associated consequences thereof, but has not examined processes involved in the *development* of sleep difficulties nor processes that further compromise cognition and emotion processing in poor sleepers. Therefore, this review will begin by introducing the concept of insomnia, specifically looking at diagnostic criteria and prevalence rates and will then explore the impact of insomnia, especially on cognitive dysfunction and emotional difficulties. Furthermore, in order

to understand the processes involved in the development and maintenance of insomnia, this review will examine relevant behavioural, psychobiological, hyperarousal and cognitive models. In addition, mechanisms of sleep deprivation will be highlighted and reviewed with particular reference to those mechanisms that are common to insomnia and anxiety.

To identify pertinent articles, online searches of the databases PsycInfo, Web of Science, Medline and Embase were conducted using different combinations of the search terms; "sleep," "deprive*," "insomnia," "anxi*," "attention," "emotion*," regulat*," "emotion* processing," "emotion dysreg*," "cognitive ability" and "cognitive control". Additional articles were identified by manual searches of the reference lists sourced from downloaded articles. Studies using samples of children (<15 years old) were excluded, as were studies exploring insomnia with any other comorbidities other than emotional difficulties (for example physical health complaints). Studies which focused on sleep disorders other than insomnia were also excluded.

## Overview of Insomnia

### Diagnosis

Insomnia is a complex and multi-dimensional disorder, in which sleep may be difficult to both initiate and maintain and can result in a lack of feeling refreshed when awake. Therefore, in general terms, insomnia is defined as dissatisfaction with quality or quantity of sleep (Holbrook, Crowther, Latter, Cheng, & King, 2000). This disorder is also often accompanied by decreased daytime functioning, due to fatigue, mood disturbance, concentration impairment and difficulties in social and occupational areas of functioning (American Psychiatric Association, 2000). Despite an accumulation of knowledge and information relating to insomnia over the

last decade, the complexity and multidimensional nature of the disorder has resulted in a lack of agreement on a universal definition, which has limited our understanding, diagnosis and research into insomnia. The three main classification systems; DSM-IV (American Psychiatric Association, 2000), ICD-10 (World Health Organisation, 1992) and the International Classification of Sleep Disorders (ICSD; American Academy of Sleep Medicine, 2001) have each proposed varying definitions of insomnia, although all agree on three core diagnostic components. First, being dissatisfied by the quality or quantity of sleep, either attributable to difficulties initiating or maintaining sleep, or resulting from a deficiency in refreshing sleep. Secondly, for the sleep difficulty to have occurred for at least three times per week for at least one month, or to have developed despite having adequate opportunities and circumstances for sleep. Finally, for the sleep difficulty to be impacting on daily living and functioning. The DSM-IV and ICD-10 also specify that there must be no associated medical, organic or neurological causative factors for the sleep complaint.

More specific definitions of insomnia in the literature sometimes consider and refer to the duration of the sleep complaint as either "transient," "acute" or "chronic" in nature. Sleep difficulties that last for less than one week, typically between one and three days and frequently caused by environmental or life stressors are referred to as transient insomnia. Acute or short term insomnia are definitions used to describe sleep difficulties that last for longer than one week and less than one month and sleep difficulties that last longer than one month are referred to as chronic insomnia (Silber, 2005).

**Prevalence**

Insomnia is the most common sleep complaint and the most commonly reported mental health complaint in the UK (Singleton, Bumpstead, O'Brien, Lee, & Meltzer, 2000). Studies suggest that between 30% and 37% of individuals in the general population experience one or more symptoms of insomnia (LeBlanc et al., 2009; Morphy, Dunn, Lewis, Boardman, & Croft, 2007; Roth, 2007), whilst between 4% and 10% of the general population meet criteria for a diagnosis of insomnia disorder (LeBlanc et al., 2009; Roth & Roehrs, 2003). However, estimates of insomnia prevalence vary considerably, with some research suggesting that epidemiological figures are likely to be understated with only 1 in 20 individuals actually presenting to healthcare professionals with insomnia related symptoms (Earl-Slater & Walley, 2001). A lack of awareness, stigma about poor sleep (Dement & Mitler, 1993) and/or an individual's preference to self-treat using alternative methods, for example relaxation or substance use (Chilcott & Shapiro, 1996) are suggested as factors in, and explanations for, individuals under-reporting and under-recognising insomnia disorder. Prevalence rates can also vary depending on the particular definition of insomnia being used. For example, a review by Ohayon (2002) revealed that the prevalence of insomnia is commonly reported according to four main categories; insomnia symptoms, insomnia symptoms associated with daytime consequences, dissatisfaction with sleep quality or quantity and insomnia disorder diagnosis. This review highlighted that, rather problematically, studies do not often specify a category of diagnosis for their sample which can, in turn, result in comparison difficulties between studies and may explain often reported discrepancies in the literature.

Considering the complexities involved in comparing epidemiological insomnia research it is understandably important for studies to make a clear distinction between the four categories, in terms of the definition of insomnia they are using. In addition, there is a notable lack of consistency in usage of the severity and chronicity categorisation terms of transient, acute and chronic insomnia. These inconsistencies result in difficulties when comparing studies, for example, an individual with a complaint of transient insomnia (lasting less than a week) is unlikely to experience the same impairment as an individual with chronic insomnia, which has lasted for years. Improvements in these areas (for example establishing a universal agreement on the categorisation of insomnia) would therefore ensure consistency across the research field.

## Development and Maintenance of Insomnia

In order to understand the developmental course of insomnia and the various variables involved in the maintenance of the disorder, the following contemporary theoretical models were consulted and reviewed:

### Behavioural Model of Insomnia

The most widely acknowledged and influential model used in understanding the development and maintenance of insomnia is Spielman and Glovinsky's "3 - P model" (predisposing characteristics, precipitating events and perpetuating attitudes and behaviours) (Spielman & Glovinsky, 1991). This model is often referred to in the literature as the behavioural model (Perlis, Giles, Medelson, Bootzin, & Wyatt, 1997; Perlis, Shaw, Cano, & Espie, 2011) due to the emphasis placed on the role of maladaptive behavioural coping strategies in the development of short-term and acute insomnia. This model views these behavioural strategies as fundamental in the transition from acute insomnia to more chronic forms of insomnia. Although

described in the literature as a behavioural model, this model does also consider wider factors, including biological and social contributors to insomnia (see below for examples). However, these are seen as vulnerability factors for insomnia rather than causal as per the behavioural coping strategies.

The 3 - P model suggests that predisposing factors influencing an individual's vulnerability to developing insomnia may include: biological traits that reduce sleep drive (for example; genetic predisposition), social factors (including care giving responsibility or parental demands) and psychological traits (such as a tendency for worry as well as anxiety and emotional dysregulation) (Erman, 2007). These factors can result in individual's becoming less effective at managing the effects of transient and acute sleep loss caused by precipitating factors (for example, life stressors, such as unemployment and divorce). Perpetuating factors develop once insomnia is established, whereby individuals may adopt a variety of maladaptive behavioural strategies (for example, extended periods in bed or napping) and cognitive changes (for example, cognitive hyperarousal) to compensate for their sleep loss. These factors serve to maintain the sleep complaint over time and further increase worry and frustration about sleeping (Bootzin & Epstein, 2011). The 3 – P model of insomnia was the first model developed to understand the role of multiple determinants in the development and maintenance of insomnia and provides a useful and concise framework for conceptualising the development of insomnia through illustrating how transient insomnia can evolve into persistent insomnia. However, it is predominantly a behavioural model, so therefore does not explore in any detail the cognitive processes that may also be involved in the development of insomnia. In addition, the majority of insomnia research only utilises the latter part of the model, which focuses on the maintenance of insomnia. This narrower focus is

unsurprising considering the complexities involved in understanding the independent and distinctive contributions of sleep loss, worry and anxiety to the development of insomnia.

**Psychobiological and Hyperarousal Models of Insomnia**

Physiological models of insomnia place an emphasis on the role of arousal in the development and maintenance of insomnia. In particular, the psychobiological inhibition model of insomnia (Espie, 2002) suggests that stressful life events precipitate both physiological and psychological arousal, which are reported to interfere with homeostatic and circadian regulation of sleep known to inhibit wakefulness (Espie, Broomfield, MacMahon, Macphee, & Taylor 2006). This model proposes that insomnia may resolve if the stress is limited or diminished, or may be perpetuated if cognitive processes such as attentional biases (thought to be implicated in insomnia), are directed towards the insomnia complaint. This model also suggests that in the early stages of insomnia, difficulty with sleep initiation or sleep maintenance can occur due to alterations in the functioning of neurobiological mechanisms of automaticity (involuntary and automatic nature of sleep initiation and maintenance) and plasticity (ability of sleep processes to adjust and/or accommodate situational factors that disrupt normal sleep-wake functioning). The psychobiological model proposes that these alterations occur due to cognitive processes, jointly referred to as the attention-intention pathway (Espie et al., 2006), which are thought to worsen transient-acute insomnia into a more chronic, self-perpetuating insomnia. This attention-intention pathway involves an individual's attention being drawn to any initial sleep difficulties, which can result in increased attentional bias for arousal and threat-related cues. More specifically, individuals may selectively attend to sleep-related stimuli and interpret them as confirmation of

their inability to sleep. As a consequence, this process could result in individuals striving to increase their sleep efforts, subsequently preventing the inhibition of wakefulness.

The model places a significant emphasis on the role of cognitive features in the development and maintenance of insomnia, which is consistent with previous findings of associations between intrusive cognitions, heightened levels of attentional bias and an effortful preoccupation with sleep and insomnia development (Jones, Macphee, Broomfield, Jones, & Espie, 2005). However, the model also expands on this cognitive emphasis by specifically addressing the role of selective attention and the inability to de-arouse or disengage from the active wake processes that interfere with the initiation of sleep processes (Bastien, 2011). Initial evidence for these processes has been established by Bastien, St-Jean, Morin, Turcotte, and Carrier (2008) who found that individuals with insomnia were unable to disengage from wake processes or inhibit further information processing, resulting in difficulties in the initiation and maintenance of sleep. The psychobiological inhibition model is the first to propose that sleep continuity disturbance occurs in association with a failure to inhibit wakefulness, as opposed to conditioned hyperarousal (Riemann et al., 2009).

The hyperarousal perspective of insomnia (Perlis et al., 1997; Riemann et al., 2009) has been increasingly researched over the last decade and is seen as an integrative approach encompassing both psychological and physiological factors in the development and maintenance of insomnia. A commonly cited hyperarousal model in the literature is the neurocognitive model (Perlis et al., 1997) which expands on the concept and role of cognitive and somatic arousal in insomnia, discussed in the models above. This top-down model hypothesises that cortical

arousal may act as an additional perpetuating factor in insomnia, alongside cognitive and somatic domains of arousal. This model proposes that cortical arousal is a conditioned response occurring during the period of sleep onset, which is itself elicited from visual stimuli (for example the bedroom) and/or temporal cues (for example bedtime). It is proposed that this cortical arousal acts as a biological precipitant to cognitive arousal (in the context of chronic insomnia) and contributes to sleep continuity disturbance through enhanced sensory and information processing and long term memory formation. Support for the theories proposed by this model has been shown and observed by heightened electroencephalography (EEG) beta activity (indicative of cortical arousal) in individuals with both sleep onset insomnia (Freedman, 1986) and primary insomnia (Perlis, Smith, Orff, Andrews, & Giles, 2001).

Riemann et al. (2009) provide an extension to the neurocognitive model presented above, by proposing a bottom-up neural explanation of hyperarousal to explain the development of insomnia. Riemann suggest that a dysfunctional "key switch" in the hypothalamus is responsible for closing off the arousal system during sleep. It is this consequent imbalance between sleep-activating and arousal-promoting areas of the brain which they suggest may be fundamental in the development of primary insomnia. This imbalance may account for sleepiness and an inability to initiate sleep in individuals with insomnia due to the coexisting hyperarousal. Consequences thereof can include cognitive and emotional difficulties, especially anxiety and daytime impairment. Despite this model providing a useful additional perspective on the development of insomnia, predictions regarding a neural vulnerability and consequent imbalance between arousing and sleep-inducing brain activity have yet to be tested in a sample of

individuals with insomnia. However, preliminary support has been provided by exposing rats to a psychological stressor in order to generate sleep disturbances, similar to those observed in stress-induced insomnia in humans (Cano, Mochizuki, & Saper, 2008). This study found simultaneous activation of sleep-promoting and arousal related areas in the brain. They hypothesised that the sleep promoting areas of the brain are activated as a result of circadian and homeostatic pressure, but cannot shut down the arousal system which is simultaneously activated by the limbic system. This process results in unique circumstances whereby sleep circuitry reveals neuronal activity similar to that of sleep, but neuronal activity in the cortex and limbic system resemble that of a fully waking state.

**Cognitive Model of Insomnia**

In addition to the models above, integrated cognitive-behavioural approaches to understanding insomnia provide a helpful framework for developing an awareness of the interactions between cognitive, affective and behavioural components of sleep disorders and related mental health problems (Jansson & Linton, 2007). Harvey (2002) proposed a cognitive model of the maintenance of insomnia, emphasising the role of intrusive negative thoughts relating to consequences of sleep loss and concerns about daytime functioning. These worries trigger arousal, distress and associated anxiety, with a consequent increase in attentional focus on the sleep difficulty (Cisler & Koster, 2010). With anxiety directing attentional resources towards threat (Dagleish & Watts, 1990), the cognitive model suggests that individuals with insomnia will experience attentional narrowing towards both internal (for example physiological sensations) and external (for example looking at the clock to see how long it is taking to fall asleep) sleep related cues. These cues may be taken as evidence for insufficient sleep and impaired coping and functioning

during the day, which can in turn, exacerbate discrete thoughts such as; "I'm never going to get to sleep" and perpetuate broader worry and negative rumination (Harvey, 2002). Hypervigilance and monitoring for such cues is further increased in the pre-sleep period by anticipatory anxiety about the likelihood and consequences of poor sleep (Clark, 1999).

Harvey (2002) suggests that biased processing of sleep-related material and elevated anxiety can distort an individual's perception of the sleep deficit such that they feel, believe and report that the sleep problem might be worse than it objectively is. These distorted beliefs, along with counterproductive behaviours (discussed earlier as perpetuating factors), are ultimately responsible for reinforcing the original negative thoughts regarding sleep and therefore play a role in the maintenance of insomnia. A consequence of this process is that escalated anxiety and distorted perceptions of sleep can eventually culminate in a real deficit of sleep and daytime functioning due to increasingly catastrophic worry, physiological arousal and high levels of distress (Espie, 2007). In support of this cognitive model, several studies have found a relationship between both intrusive thoughts and worry and disrupted sleep. In particular, individuals with insomnia were found to generate more catastrophic thoughts about the consequences of not sleeping, as well as giving higher likelihood ratings that they will have greater difficulty sleeping than good sleepers (Harvey & Greenall, 2003). In addition, significant associations have been found between non-sleep related ruminations and poor subjective sleep quality, delayed sleep latency and sleep disturbances, even after controlling for mood in individuals with insomnia (Thomsen, Mehlsen, Christensen, & Zacheriae, 2003).

**Summary of Models**

Until recently, insomnia has been primarily understood in the literature as a psychological disorder, triggered by psychosocial stress (for example life stressors) and maintained by maladaptive behaviours (for example self medicating with drugs and/or alcohol) (Spielman & Glovinsky, 1991). However, Riemann et al. (2009) suggest that, given recent research establishing the additional importance of cognitive hyperarousal and underlying neural mechanisms, it would be more accurate to describe insomnia as a 'psychobiological' disorder, which also captures the involvement of social, psychological factors, as well as explicitly acknowledging and emphasising the role of biological factors in insomnia. Although there is significant overlap between these definitions (psychological and psychobiological) and theoretical models, each variant has explored and placed a different emphasis on the variables that promote insomnia (for example, social and biological). This does not imply that the spectrum of ideas and factors considered in the various models and definitions are mutually exclusive, nor are they contradictory of one another. Instead, they are highly interdependent and interrelated and should therefore be consulted together to aid a more comprehensive understanding of the development and maintenance of insomnia. The models described above provide valuable frameworks for understanding the sleep disorder, its functional consequences, and also broader problems in emotional dysregulation and cognitive dysfunction that may increase risk for comorbid mood disorder and anxiety.

## Impact of Insomnia

Implications of insomnia include a reduced quality of life (when compared to healthy controls) impacting on physical ability, decreased psychological and emotional wellbeing and a reduction in social interactions (Leger, Scheuermaier,

Philip, Paillard, & Guilleminault, 2001). The consequences of insomnia can vary substantially between individuals, with some finding impairments in cognition the most challenging, whilst for others the emotional impact of poor sleep may be the most difficult (Jansson & Linton, 2007).

**Insomnia and Cognitive Dysfunction**

The cognitive based models above (Espie, 2002, Harvey, 2002) have made predictions about the important role of cognition in insomnia. In particular, they specify the association between sleep and the following factors; arousal, attentional deficits for everyday stimuli, attentional biases to sleep related stimuli and an increase in negative intrusive thoughts. These associations have been supported in sleep deprivation studies, revealing increased functional impairment in areas including cognitive control, attention, working memory, vigilance and risk taking in healthy individuals (Alhola & Pola-Kantola, 2007). However, despite the suggestion that poor sleep contributes to cognitive impairment, relatively few studies have investigated these deficits in individuals with insomnia and those studies that have done so, have often produced inconsistent findings. Some studies, for example, have found little or no difference between individuals with insomnia and healthy controls in tasks of alertness, attention, visual and verbal memory, verbal fluency, and psychomotor speed (Fulda & Schulz, 2001). However, individuals with insomnia have been found to perform worse than healthy controls when performance is measured by accuracy rather than speed (Shekleton, Rogers, & Rajartnam, 2010). Estimates suggest that significant differences in cognitive functioning between individuals with insomnia and healthy controls are only found in approximately one-fifth of studies (Riedel & Lichstein, 2001). Such findings have been interpreted as evidence that insomnia is not associated with deficits in cognition (Riedel &

Lichstein, 2001), whilst others argue that the limited evidence is due to methodological weaknesses within these studies (Espie & Kyle, 2008).

Recently, McKnight-Eily et al. (2011) found that insufficient sleep was associated with risky decision making associated with ten of eleven primary health risk behaviours (including alcohol use, risky sexual behaviours, cigarette use, marijuana use and suicidal ideation). This study cited the maladaptive effect of chronic sleep loss on cognitive control and decision making as central to the range of behavioural problems evident in adolescents with poor sleep. Other studies have found associations between alcohol misuse (Chilcott & Shapiro, 1996) and recreational drug use (Hasler, Smith, Cousins, & Bootzin 2012) with insomnia. In addition, the cognitive functional impairments noted above, often place individuals at increased risk of accidents within the work environment (Roth, 2007). Accidents are reportedly between 2.5 and 4.5 times more likely to be caused by individuals with insomnia than those with good sleep (Balter & Uhlenhuth, 1992; National Sleep Foundation, 1991).

**Attentional Dysfunction**

A meta-analysis of twenty four studies investigating the effects of insomnia on cognitive performance (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012) revealed that individuals with insomnia performed worse than healthy controls (although not significantly so) on tasks measuring the following attentional components; reaction time, information processing and selective attention. However, there were no differences observed on tasks of divided attention, alertness and sustained attention or vigilance between individuals with insomnia and healthy controls. Although significant differences were not found in specific tests of attention, significant differences were found between the two groups on measures of

memory, including working memory and episodic memory, where individuals with insomnia performed significantly worse than healthy controls. Fortier-Brochu et al. (2012) found substantial variation in the number of studies investigating each cognitive function, suggesting low statistical power as an explanation for this variation and for the limited differences found between individuals with insomnia and healthy controls, especially in tasks concerning attention. This meta-analysis has extended and improved previous findings, in which no significant differences in cognitive functioning have been found between healthy individuals and those with insomnia. For example, a review of 13 studies by Riedel & Lichstein (2000) found that 76% of cognitive tasks (including assessment of attention, vigilance and executive functioning) revealed no significant differences between individuals with insomnia and healthy controls. Riedel and Lichstein (2000) proposed that the limited differences found between the two groups may be as a consequence of small sample sizes within the individual studies of the meta-analysis failing to detect small to moderate differences between the groups. This suggestion does find some support from the fact that the study with the largest sample size (and therefore more statistical power) (Hauri, 1997) revealed the largest effect (size) of insomnia on daytime dysfunction.

A recent study by Edinger, Means, Carney, and Krystal (2008) used a comprehensive study procedure (including polysomnography, multiple sleep latency tests, a battery of performance tasks and subjective measures) and found that individuals with insomnia demonstrated greater performance deficits on tasks of reaction time and switching attention than good sleepers. More specifically, results indicated that individuals with insomnia had longer reaction times and greater behavioural and attentional instability during testing, than a control group of healthy

individuals. This study by Edinger et al. (2008) addresses the many limitations noted in previous studies (for example, small sample sizes and a limited range of measures) and therefore provides good evidence for attentional deficits existing in individuals with insomnia. In addition, Spiegelhalder, Espie, Nissen, and Riemann (2008) found that individuals with insomnia have an attentional bias towards sleep-related word cues (measured by a modified stroop test) when compared to professionals working in the field of sleep, although not when compared with healthy controls. These findings provide support for existing evidence of increased sleep-related monitoring and negative thought intrusions, especially during the pre-sleep period (Harvey, 2000). Emotionality is commonly negatively toned in insomnia, especially due to the close relationship with anxiety and worry. However, despite this common theme of negativity, research in this area (especially understanding the processes of sleep involved in emotional dysregulation and emotion processing) has been particularly limited.

**Insomnia and Comorbidity with Emotional Difficulties**

Individuals presenting with insomnia are estimated to be 5 times more likely to experience a psychological disorder than individuals without insomnia, with increasing severity of insomnia correlating with greater psychological difficulties (Sarsour, Morin, Folwy, Anupama, & Walsh 2010). Poor sleep, especially restorative sleep, may trigger psychological disorders by limiting an individual's ability to manage increased levels of distress and hyperarousal, which may exacerbate negative affect and blunt the experience of positive emotion (Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2010). More specifically, a large cross sectional study across four countries (N=14,915) by Ohayon and Roth (2003), revealed that insomnia was particularly comorbid with both panic disorder (61%)

and Generalised Anxiety Disorder (GAD) (44%) (see related findings in UK samples by Stewart et al., 2006). A review by Marcks & Weisberg (2009) provides further evidence of this comorbidity, by finding between 50% and 70% of individuals with GAD had insomnia. In addition, they found that sleep disturbances, in turn, appeared to exacerbate and trigger GAD related symptoms, suggesting a bidirectional relationship. The above statistics provide evidence of comorbid mental health difficulties in insomnia (and vice versa), however, the level of comorbidity remains unclear with estimates varying between 26% and 100% in those presenting at a sleep clinic and between 3.6% and 79.8% in community samples of individuals with insomnia (see Harvey, 2001 for a review).

Within these studies the bidirectional relationship between insomnia and anxiety appears to be the most significant, with anxiety having the highest comorbidity with insomnia (Lee & Douglas, 2010; Ohayon & Reynolds, 2009). This relationship is perhaps due to a number of shared common characteristics, for example; hyperarousal, heightened vigilance and attention to threat, persistent cognitions (for example worry and rumination), increased daytime impairment and increased night-time waking (Uhde, Corteses, & Vedeniapin, 2009).

Historically, insomnia was viewed as secondary to, or as a symptom of, psychological disorders such as anxiety and depression. However, it is now accepted that insomnia may be a risk for, or even causal in, the development of psychological disorders (Harvey, 2001; Lichestein, 2000), as well as existing as a primary chronic disorder without a clinical level of psychological distress (Roth, 2007). Harvey (2001) suggests that caution should be exercised by both clinicians and researchers when attributing insomnia as a consequence of psychological disorders, due to the possibility that insomnia may have developed first and may

therefore be the primary disorder (particularly if it causes the individual the most distress, or is responsible for the maintenance of both disorders). Considering this bidirectional relationship, research by Ohayon and Roth (2003) found that insomnia more commonly precedes mood disorders (especially prior to relapse in 56% of cases) rather than being a consequence thereof, whilst in relation to anxiety disorders they found that insomnia mostly appeared at the same time or after the anxiety disorder. Their findings suggest that insomnia is most probably a risk factor for the development of depression, whereas anxiety is likely to be a risk factor for the development of insomnia.

Despite the large amount of research that has been conducted into the impact of insomnia, research in this field has faced some criticism. In particular, Leger et al. (2001) argue that it is difficult to assert causative factors to the effects of insomnia, when many of the relationships can be bidirectional. They suggest that accuracy would be improved if research described an association between disorders, rather than specify a cause or a consequence, unless longitudinal evidence identifies the direction of causation. Further criticisms focus on methodological weaknesses, in particular, failure to describe samples in terms of diagnostic criteria, for example transient, acute or chronic insomnia (Ohayon, 2002). More generally, a review by Espie and Kyle (2008) highlighted the prevalence within insomnia research of small, poorly designed samples, using inadequate measures which have proven powerless in detecting the objective deficits and nature of the insomnia complaint.

In order to address these weaknesses and to effectively understand cognitive and emotional dysfunction throughout the developmental course of insomnia, it is essential for research to address questions/hypotheses that are generated from contemporary theoretical models.

**Sleep deprivation and emotional processing**

To date research into the effects of sleep on emotional processing and emotional dysregulation has focused on acute sleep deprivation studies. Findings from these studies cannot be used to estimate the effects of early-onset insomnia due to the use of artificial experimental conditions (when compared to circumstances in which insomnia would naturally develop), however, these studies still provide an important insight into the early effects of an acute sleep complaint. In particular, Zohar, Tzischinsky, Epstein, and Lavie (2005) investigated the effects of sleep disturbance on mood in new medical resident staff over a 5-7 day period. Measures of affective events were taken by asking residents to establish the frequency of goal disruptive events, for example "During the last 15 minutes: has someone or something disrupted your scheduled activity?" and goal enhancing events, for example "During the last 15 minutes: have you had an opportunity to perform a medically challenging task?" Results found that sleep deficits amplify both negative emotions and fatigue following goal disruptive events and reduce the effects of positive emotion following goal enhancing events. In addition, sleep deficits were found to result in an overall elevated baseline for experiencing positive emotions.

A study by Walker and Stickgold (2006) investigated the impact of sleep deprivation on memory encoding of both emotional and non-emotional material. In this study healthy participants were assigned to either a sleep deprived group, where they were deprived of sleep for 36 hours, or allocated to a control group, where they were allowed to sleep normally, prior to a memory encoding session of emotionally rated words (negative, positive and neutral). Participants were then tested following two nights of restorative sleep, in order to facilitate testing of retention in a sleep rested state for both groups. Overall, participants in the sleep deprived condition

demonstrated a 40% deficit in memory encoding, relative to participants who had slept normally prior to learning. A significant difference was observed between individuals in the sleep deprived condition and the control group, with the control group exhibiting the highest retention for positive emotional memories (59%), which is consistent with the suggestion that positive emotion facilitates memory encoding (Phelps, 2004). In the sleep deprived group, the highest retention was seen for negative emotional memories (81%) whereas a significant encoding impairment was evident for neutral, and, most significantly, for positive memories. These outcomes indicate that sleep deprivation impairs the ability to commit new experiences, especially positive ones, to memory. In addition, these findings support theories suggesting that there is a reduction in working memory capacity and top-down attentional control during sleep deprivation, resulting in hypervigilance and greater awareness of negative stimuli. This could help explain a continued ability to encode emotionally negative memories, rather than positive or neutral ones.

## Anxiety and Insomnia

### Common Neurocognitive Processes

Neurocognitive models of anxiety share much in common with the models of insomnia outlined above, particularly with respect to the role of top-down (cortical) and subcortical (thalamic) mechanisms implicated in cognitive control and emotional activation and regulation (Bishop, 2007). Specifically, functional brain imaging research suggests that anxiety, or more specifically worry, involves activation of automatic bottom-up cognitive processes, (for example impairment in cognitive control) initiated due to over-activity in the amygdala and hippocampus regions of the brain (Bishop, Duncan, Brett, & Lawrence, 2004). In addition, reduced activity in prefrontal control structures restricts appropriate top-down regulation of amygdala

activity, and further results in impairment of broader cognitive control and a failure to appropriately regulate the negative schemata that characterise anxiety and worry (Clark & Beck, 2010). Recent research by Yoo, Gujar, Hu, Jolesz, and Walker (2007) confirms the role of similar mechanisms in disturbed sleep. They assigned 26 participants to either a 35 hour sleep deprivation group or a control group (in which participants slept normally at home). During functional magnetic resonance imaging (fMRI), participants from both groups were presented with 100 images from a standardised picture set, ranging on an experimentally controlled gradient from emotionally neutral to increasingly aversive. Although both groups displayed significant activation within the amygdala region in response to increasingly negative stimuli; individuals in the sleep deprived condition demonstrated 60% greater amygdala activation and a three-fold increase in the amount of amygdala volume stimulated, compared to healthy controls in response to increasingly negative emotional stimuli. In addition, the sleep deprivation group demonstrated a significant reduction in functional connectivity between the amygdala and the medial prefrontal cortex when compared to the control group of non-sleep deprived individuals, suggesting an impairment of top-down cognitive, pre-frontal control (Sotres-Bayon, Bush, & LeDoux, 2004). These preliminary results suggest that both sleep deprivation and anxiety feature impaired top-down control processes and a consequent increase in cognitive activity. This evidence supports earlier findings from Walker and Stickgold (2006, discussed above) that sleep deprivation prevents individuals from effectively controlling their responses to threatening stimuli. Consequently, sleep-disturbed individuals may experience anxiety due to impaired cognitive control and interpretation of the threatening stimuli in a negative manner.

### Common Cognitive Mechanisms

As noted earlier, recent cognitive models of insomnia emphasise the role of a range of biases in information processing (for example, selective attention to threat, poor cognitive/attention control) in the development and maintenance of disturbed sleep (Harvey, 2002, Espie 2002). Insomnia and anxiety share a number of features, including; increased vigilance and attention to threat stimuli, hyperarousal and enduring cognitions (for example worry and rumination), as well as similar patterns of dysregulation in neural activity, resulting in parallel attentional deficits and inaccuracies in emotional processing (Dahl & Bjorvatn, 2009). Despite these similarities and the wealth of evidence indicating the strong associations between anxiety and insomnia, research has not specifically addressed aspects of cognition that mediate the association between sleep difficulties and anxiety. In addition, there has been limited research exploring the independent contributions of the above similarities in the development of insomnia, with the distinct possibility that sleep difficulties are more strongly associated with certain symptoms of anxiety than others.

## Evidence of Common Attention Dysfunction in Sleep and Anxiety

Concluding the evidence presented above, it appears that impaired attentional processes have been identified as a primary cognitive factor underlying the development and maintenance of both anxiety (Eysenck, Derakshan, Santos, & Calvo, 2007) and insomnia (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006). For example, individuals with insomnia selectively attend and monitor their environment for sleep related cues (Harvey, 2000), whilst individuals with anxiety have a hypervigilance and selective attention for any possible threat (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007). Due to

attention playing a significant role in both anxiety and insomnia it is important to consult theories of attentional control to establish a better understanding of these disorders and their most common and significant features; worry and impaired top-down control. The attentional control theory (Eysenck, Derakshan, Santos, & Calvo, 2007) proposes that anxiety disrupts the balance between a goal-directed top-down attentional system and a stimulus driven bottom-up system, resulting in less inhibition and control of negative stimuli and an increase in attention towards more emotionally salient material (see also Sadeh & Bredemeier, 2011). An increase in anxiety can therefore escalate the allocation of attention to threat related stimuli (either internally, for example, worry thoughts or externally, for example, environmental cues), resulting in reduced attentional focus on tasks that do not involve threatening stimuli.

The theory of attentional control has been well researched within the anxiety literature, particularly in relation to individuals with high anxiety who demonstrate a greater attentional bias towards threatening stimuli, compared to non-anxious individuals (Cisler & Koster, 2010). A meta-analysis by Bar-Haim et al. (2007) also noted a significant threat-related bias in individuals with anxiety, but not in non-anxious individuals. This meta-analysis indicated consistency of this bias across a variety of experimental conditions and in different types of anxious populations (for example, individuals with GAD or Obsessive Compulsive Disorder), although only a low to medium effect size was found ($d$=0.45). Attentional biases in anxiety have been observed using several different experimental settings and tasks, including, the visual search task (Ohman, Flykt, & Estevesm, 2001), the dot probe task (Macleod, Mathews, & Tata, 1986) and the modified stroop task (Stroop, 1935) (which is the most commonly used task in this area) all of which support the robustness of the

evidence that attentional bias is a central feature of anxiety (see Cisler and Koster, 2010 for a review).

It is hypothesised that the worry aspect of anxiety may deplete top-down attentional resources which are required for sustaining focus on current goal demands and tasks (Eysenck et al., 2007). This may be due to the chronic, excessive and uncontrollable nature of worry, which is a defining feature of GAD (APA, 1994). Eysenck and Calvo (1992) propose that worry causes a reduction in working memory capacity due to the automatic processing of task-irrelevant stimuli (Baddeley, 2002), making it more difficult for individuals to successfully maintain attention on the task at hand. In particular, it is proposed that the central executive component of working memory is affected in impaired attentional control and therefore anxiety. This component is responsible for both inhibition, which regulates an individual's instinctual response to allocate attentional resources to threat and withstand distraction from task-irrelevant stimuli (Friedman & Miyake, 2004) and shifting, which enables individuals to cognitively shift to meet the demands of multiple tasks (Eysenck, et al., 2007). Together these functions are critical for effective working memory and for coordination of thoughts in relation to internal goals (Pashler, Johnston, & Ruthroff, 2001). A recent study by Hayes, Hirsch, and Mathews (2008) assigned 32 healthy participants to either a low worry group or a high worry group (level of worry was determined by Penn State Worry Questionnaire (PSWQ), Meyer, Miller, Metzger, & Borkovec, 1990). All participants were asked to complete a random key press task whilst thinking about either a personally relevant worry topic or a positive personally relevant thought topic. Participants were asked to rate valence of thoughts as positive, negative or neutral and also rate their mood on visual analogue scales after each thought

condition. This research demonstrated that high worriers had less residual working memory capacity during worry than when thinking about a positive topic, compared to low worriers. These findings remained significant when the authors controlled for mood, confirming that the reduction in working memory capacity in high worriers was related to the worry component of anxiety, rather than mood state. These findings are supportive of the attentional control theory which proposes that worry causes a deficit in top-down attentional resources, in this case specifically working memory capacity. Consequently, individuals have less attentional resources available to redirect their thoughts away from worry (due to impaired inhibition) and to be effective at task and/or daily activities they are performing (due to impaired cognitive shifting). Although the study by Meyer et al. is the first study to provide evidence for depleted working memory in high worriers, it is a small study and therefore further research is warranted in this area, in particular, replication in a clinical GAD sample would provide further support for these initial findings.

Considering the evidence that sleep deprivation dysregulates both neural and cognitive processes in anxiety, it is important to consider the prospect that weakened attentional control can contribute to the development of both insomnia and anxiety.

## Future Research

This review has explored the relationships between insomnia and the effectiveness of cognitive and emotional processes, particularly considering both attentional and emotional control alongside emotional dysregulation, by consulting and drawing upon behavioural, cognitive, neurocognitive and psychobiological models within the literature. These models and the literature reviewed reveal significantly similar processes between insomnia and anxiety and, more specifically, worry. In particular, the research cited has found that impaired attentional control

results in reduced emotional regulation and, more specifically, reduced inhibition of negative schema and thought intrusions. Research also highlights that heightened cognitive arousal is common to both anxiety and sleep disorders. Despite these findings, the impact and role of specific cognitive factors within insomnia remains unclear. In particular, although there is growing research into sleep related worries, very little research has looked specifically at the role of non-sleep specific worry in the development and maintenance of insomnia. Therefore future studies should utilise cognitive measures of emotional processing that are widely used in the anxiety literature, to further clarify biases in emotional processing in individuals with transient, acute and chronic sleep disturbance. This is important, considering chronic insomnia is predicted to develop from repeated exposure to bouts of sleepiness as seen in transient and acute sleep disturbance (Harvey, 2000).

In addition, it is of importance for future research to explore poor sleep and associated emotional and cognitive dysfunction in a healthy sample, experiencing naturally occurring sleep disturbance. Sleep deprivation studies have demonstrated interesting and beneficial findings, for example deficits in working memory, attentional control and cognitive control as well as finding that sleep deficits amplify negative emotions and reduce the effect of positive emotions. However, despite these findings clear limitations arise due to the artificial nature of the sleep disturbance that prevents generalisation to sleep-disturbed populations. To this end it would be helpful to recruit cohorts of young individuals who experience sleep disturbances due to chosen lifestyle factors, rather than samples that are more likely to be confounded by emotional distress (for example worry and negative thoughts) or physical illness (that often affects sleep in later life), (see Stewart et al. 2006 for related discussion).

**Clinical Implications**

This review highlights the need for improved recognition, diagnosis and treatment of sleep difficulties and, more specifically, comorbid sleep difficulties, especially considering the evidence that they are under-recognised and under-treated in the general population (Benca, 2001). However, due to the considerable overlap between sleep disturbance and anxiety disorder, it may be increasingly challenging for clinicians to differentiate between the complaints in order to devise a treatment plan targeting each individual disorder.

The complexities of insomnia make establishing effective, cost effective treatment protocols particularly challenging and problematic. In particular, there is increasing concern surrounding the popularity of prescription drugs such as benzodiazepines and Z-drugs (for example Zaleplon and Zopiclone) for the management of insomnia, with reports suggesting that individuals with comorbid psychological difficulties were 80% more likely to receive a prescription for a benzodiazepine medication than individuals without such comorbidities (Rasu, Shenolikar, Nahata, & Balkrishnan, 2005). Concerns have also been raised regarding the possible side effects of prescription medication, including high dependency effects, depressed mood and cognitive impairment which are in turn likely to exacerbate the effects of sleep deprivation on daytime-function, with very modest improvements to sleep duration and sleep onset latency (Stewart et al., 2006).

There is a large amount of evidence supporting the use of non-pharmacological treatments in insomnia, in particular Cognitive Behavioural Therapy (CBT). A recent meta-analysis by Riemann and Perlis (2009) found that psychological and behavioural treatments administered weekly over a period of four

to eight weeks produced the most robust improvements in sleep continuity for up to two years, concluding that these methods should be the first line of treatment in insomnia. More specifically, and related to the findings in this review, is the discovery that psychological comorbidities do not appear to undermine the outcome of behavioural and cognitive approaches when treating insomnia, with treatment benefits thereof remaining sustained over time (Morin et al., 2006). These findings are supportive of previous research recommending treatment of insomnia independently of the comorbid difficulty (Neubauer, 2009). Despite these findings, several limitations of the content and structure of CBT have been highlighted in the literature, for example neglecting to address underlying maintenance factors of insomnia as well as an individual's perception of sleep (Harvey & Tang, 2012). Considering the findings in this study and due to the complexity of comorbidity, it would seem appropriate and relevant for CBT to be tailored to the individual's needs and, where possible, for both the insomnia and the comorbid psychological difficulty to be addressed independently. To address the complexity of establishing which disorder to treat first, Harvey (2009) proposed a "transdiagnostic sleep intervention" in which poor sleep is addressed through use of CBT, with the option of adding extra components to target the specific psychological difficulty at a later stage. To this end we need to continue to develop our understanding of the cognitive, behavioural and emotional mechanisms that interact to promote sleep-disturbance and emotional disorder across the lifespan. The present paper has identified a common deficit in attention control (and inability to inhibit negative thought intrusions) as a candidate mechanism that might be targeted in therapy to better resolve comorbid sleep problems and anxiety.

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# Empirical Paper

## Associations between Severity of Insomnia, Attentional Control and Negative Thought Intrusions in Healthy Volunteers

***Running head: Insomnia, Anxiety and Cognitive Processing***

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**Abstract**

There is considerable evidence to support an association between poor sleep and both negative affect and weakened attentional control, yet few studies have explored the link between these factors as a result of acute sleep loss. This present study examined whether specific components of acute sleep loss were associated with attentional deficits observed in anxiety; specifically a reduced ability to focus attention, and an increase in difficulties inhibiting negative thoughts. 112 undergraduates completed self-report measures of insomnia severity (including components of severity, impact and satisfaction), trait anxiety, attentional control and cognitive failures. Participants also completed a 3-stage worry task, during which thoughts were sampled, this process included: an initial 5-minute breathing interval, a 5-minute worry period, and a 5-minute post-worry breathing interval. Results found that insomnia severity was associated with higher levels of anxiety, whilst higher scores on both of these scales were associated with increased cognitive failures and decreased attentional control. In the pre-worry period higher insomnia scores, particularly on the severity component, were associated with decreased ability to focus attention upon breathing and an increase of negative thought intrusions, even when controlling for anxiety. In the post-worry period no significant associations were found between insomnia and negative thought intrusions, however, trait anxiety was modestly related to attentional focus and negative thought intrusions. Furthermore, no correlations were found between either anxiety or insomnia and neutral or positive thought intrusions at either time point. In summary, results indicate that in healthy volunteers, increased insomnia severity (independent of anxiety), was associated with increased deficits in attentional control and increased negative thought intrusions during a five-minute breathing focus. These findings support suggestions that acute periods of poor sleep may dysregulate key emotional processing networks implicated in attentional control, as well as increase preferential processing of negative information, highlighting the need for improved recognition, diagnosis and treatment of sleep difficulties in the general population.

# Introduction

## Definition and Diagnostic Criteria

Insomnia is often described as multi-dimensional, comprising several elements including; subjective sleep complaints, associated negative daytime symptoms and severe distress or impairment in social, occupational or other vital areas of functioning (American Psychiatric Association, 2000). Due to the complexity of insomnia, no universal definition has been agreed, which has made understanding, diagnosing and researching the condition of insomnia particularly difficult. The DSM-IV (American Psychiatric Association, 2000), ICD-10 (World Health Organisation, 1992) and the International Classification of Sleep Disorders (ICSD; American Academy of Sleep Medicine, 2001) have each proposed varying definitions of insomnia. However, all agree on three core diagnostic components: (a) acquiring an unsatisfactory quality of sleep, either due to difficulty initiating or maintaining sleep, or due to early morning waking; (b) occurring for a period of time, for example, for at least one month, or occurring despite having adequate opportunities and circumstances for sleep; and (c) causing an impact on daily living and functioning. Both the DSM-IV and ICD-10 state that there must be no other causative factors for the poor sleep, for example organic, neurological or medical conditions.

Added to the complexity of achieving a distinct definition, is that insomnia is a subjective complaint of either insufficient sleep or poor sleep and does not refer to the actual amount of time spent asleep (Soldatos, 1994). Given this complexity, the Insomnia Severity Index (ISI) is often used as measure in research and clinical settings as it addresses the subjective symptoms and consequences of insomnia, as well as the degree of concern or distress caused by those difficulties. Its content,

which specifically addresses impact, severity and satisfaction, (Bastein, Vallieres, & Morin, 2001) also corresponds to the main diagnostic criteria of insomnia (APA, 2001). The ISI is therefore likely to be clinically very relevant as it provides pertinent information for diagnosis and treating planning, which can be specifically tailored to the individual's perception and understanding of their sleep difficulties.

Historically insomnia was assumed to be secondary to a diagnosis of a psychological disorder, for example, depression or anxiety. However, more recently, due to the chronicity and complexity of insomnia, classification manuals (for example the DSM-IV and ICD-10) have proposed that there is a bi-directional relationship between insomnia and co-morbid disorders, meaning that insomnia can now be considered a primary diagnosis in its own right and may consequently be a risk factor for the development of secondary difficulties, such as anxiety and depression.

**Prevalence**

Insomnia is the most common sleep complaint in the general population in the United Kingdom (UK) (Singleton, Bumpstead, O'Brien, Lee, & Meltzer, 2001). A survey of 6,708 individuals conducted in the UK by the Mental Health Foundation (2011) aimed to investigate individual's sleep patterns. Results indicated that 36% of individuals were classified as having insomnia symptoms, similar to other estimates which suggest that between 30% and 37% of the general population experience symptoms of insomnia (LeBlanc et al., 2009; Morphy, Dunn, Lewis, Boardman & Croft, 2007; Roth, 2007). Research has also indicated that approximately 4% to 10% of the general population are thought to meet criteria for a diagnosis of chronic insomnia (LeBlanc et al., 2009; Roth & Roehrs, 2003). However, it is important to note that estimates of insomnia prevalence vary

extensively depending on the definition used (for example, insomnia disorder or symptoms) and the population group used (Harvey, 2001).

Insomnia is viewed as a persistent and complex condition which often develops over time. Morin et al. (2009) completed a three year longitudinal study of individuals with insomnia and found that overall 74% of the sample reported insomnia lasting for at least one year, with a significantly higher rate in the group with a diagnosis of insomnia (84%) than the group with symptoms of insomnia (69%). In addition to this finding, 46% of the sample reported insomnia persisting for the three year duration of the study, again with significantly higher rates in the group with a diagnosis of insomnia (66%) than the group with symptoms on insomnia (37%). The severity of insomnia at baseline therefore appeared to be an indication of its persistence.

**Implications**

Due to the long term nature of insomnia and its high prevalence, it is a considerable and significant public health concern. Implications of insomnia include reduced quality of life and increased functional impairment, especially in cognitive processing where, for example, reaction time, vigilance and attention are all reported to be compromised (Roth & Roehrs, 2003). The most common areas of reported impairment are in an individual's emotional and mental wellbeing (Wilson et al., 2010), with a particularly strong association between both anxiety and depression and insomnia (Riemann et al., 2009). These implications can often have secondary consequences, for example Daley et al. (2009) found that individuals with insomnia had significantly higher implementation rates of self medication with drugs and/or alcohol and also greater work absenteeism. In addition, Leger and Bayon (2010)

reported that individuals with insomnia were at increased risk of physical illness and a greater risk of traffic and work accidents.

These implications consequently have an impact on a society's economy and in particular its health services (Leger, Levy, & Paillard, 1999; Walsh & Englehardt, 1999). It is therefore of importance to establish and understand the mechanisms which underlie the development and persistence of poor sleep, which would help to differentiate between individuals that recover quickly and without treatment from acute episodes of insomnia, compared to individuals who are vulnerable to developing a more enduring complaint, for example chronic insomnia. An enhanced understanding of these processes would enable improvements in preventive measures and in the psychological treatment of insomnia.

Cognitive Behavioural Therapy (CBT) is currently considered to be the treatment of choice for persistent insomnia (Espie, 1999; Jacobs, Pace-Schott, Stickgold, & Otto, 2004). However, a meta-analysis conducted by Morin, Culbert and Schwartz (1994) revealed that between 19% and 26% of individuals failed to show any improvement after CBT treatment and, despite significant improvement in the rest of the sample, the improvement was not enough to move individuals into a "good sleepers" category. Despite more recent research demonstrating the effectiveness of CBT for insomnia, it appears to be less effective at treating insomnia than it is for treating other psychological disorders, such as Generalised Anxiety Disorder (GAD) and depression, suggesting scope for improvement in this treatment approach of insomnia (Harvey & Tang, 2003). There have also been several criticisms raised about the content and structure of CBT for insomnia, arguing that it does not address some of the underlying maintenance factors, nor individuals' perceptions of sleep (Harvey & Tang, 2012). It is therefore imperative

to obtain a greater understanding of psychological difficulties and mechanisms that are involved in individuals experiencing poor sleep, in order to improve CBT interventions for insomnia.

**Psychological Mechanisms in Insomnia**

To understand the mechanisms involved in the persistence of insomnia, several cognitive models have been proposed (Espie, 2002; Harvey, 2002; Lundh & Broman, 2000; Morin, 1993). These models have emphasised the important role of cognitive, behavioural and psychological features in the maintenance of insomnia (Jansson & Linton, 2007). The holistic and methodical nature of these cognitive models have contributed to an understanding of the mechanisms that play a role in the persistence of insomnia and their interaction with one another (Harvey & Greenall, 2003). However, despite facilitating an increased understanding of insomnia, research has primarily focused on the factors that contribute to the maintenance of insomnia for example, hypervigilance, selective attention towards threat and sleep related worry (Harvey, 2002), perhaps overlooking the opportunity and importance of exploring the independent explanatory power of other factors in the early development of insomnia.

An important area to consider is the individual's subjective experience of sleep, in particular addressing the components of severity, satisfaction and impact (introduced earlier and discussed in more detail in the method section). Harvey et al. (2008) found that in a population of healthy sleepers, the impact of severity of poor sleep (for example "whether you got enough sleep") was the most important factor for judging sleep quality, which was broadly similar to those described by individuals with insomnia. It is therefore important to understand the association of each of these independent components in relation to attention and emotion

processing, in particular worry, as research has indicated that it is primarily an individuals' interpretation of their current sleep and expectations of desired sleep which can significantly impact their levels of cognitive and emotional arousal. A study by Edinger et al. (2000), for example, found that individuals who subjectively reported insomnia, despite objectively having normal sleep displayed greater levels of depression, anxiety and dysfunctional beliefs about sleep, compared to individuals who did not complain about their sleep, despite having objectively poor sleep. Research in this area may help to differentiate between those people that recover quickly and without treatment from an acute episode of insomnia from those who are vulnerable to developing a more enduring complaint.

**Psychological Difficulties and their Relationship with Insomnia**

The cognitive models cited above have all emphasised the role of anxiety and its effect on poor sleep. However, despite the implied significance of a relationship existing between anxiety and insomnia, this is poorly understood within the literature. Historically, this may have been hindered by flawed assumptions of insomnia being a secondary symptom to psychological difficulties. It is therefore important to explore and establish a deeper understanding of the mechanisms through which insomnia leads to an increased risk of psychological difficulties and vice versa. This improved understanding will aid in enhancing existing therapies such as CBT, as well as improve an understanding of the role of intervention in the prevention of both anxiety and insomnia.

Ohayon and Roth (2003) found that insomnia more commonly precedes mood disorders rather than being a consequence. In relation to anxiety disorders they found that insomnia mostly appeared at the same time or shortly after the anxiety disorder, suggesting that insomnia is most probably a risk factor in the

development of depression, whereas anxiety is most probably a risk factor in the development of insomnia. Jansson and Linton (2006) found that in a healthy population of individuals, depression was closely related to sleep initiation difficulties, whilst anxiety was strongly related to sleep maintenance difficulties. Their findings indicated that high anxiety increased the risk of developing insomnia by more than three times, suggesting that anxiety is the most influential factor in accounting for new cases of insomnia. The most commonly shared component of both anxiety and insomnia is worry, which due to its overlap across the disorders may have an explanatory value in the development of insomnia. Sibrava and Borkovec (2006) defined worry as a primary cognitive characteristic of anxiety which typically concerning uncertain outcomes, possibly negative, regarding future events.

**Models of Insomnia**

Spielman, Curasto, and Glovinsky (1987) were the first to develop a model to understand the development and maintenance of insomnia. Most commonly referred to as the "3-P Model" (predisposing characteristics, precipitating events and perpetuating attitudes and behaviours) it proposes that social factors, for example, parental demands as well as psychiatric disorders including anxiety, may be part of the predisposing factors in the development of insomnia. These predisposing factors serve to exacerbate precipitating factors such as life stressors or physical illness. When these factors are activated, it affects an individual's ability to regulate their thoughts, emotions and behaviours due to depleted cognitive resources, such as energy and attention (Zohar, Tzischinsky, Epstein, & Lavie, 2005). As a consequence of developing insomnia individuals may make various changes in order to compensate for their sleep difficulties, for example sleeping in the afternoon and

waking up later, which may maintain the current sleep difficulties. Individuals may also become more hypervigilant and aware of their sleep difficulties, causing them, in turn, to become aroused and anxious about sleep in general, consequently perpetuating the difficulties of poor sleep and insomnia.

According to the 3-P Model, insomnia is likely to develop with sleep deprivation and it is the individual's interpretation and response to this experience which determines the development path of insomnia. This bottom-up model has been broadly supported by more recent models such as the hyperarousal model of insomnia (Riemann et al., 2010) which suggests that insomnia can occur when a genetically determined dysfunction in sleep wake cycles interacts with adverse circumstances, for example precipitating life stressors. This interaction may consequently lead to sleep disruption as well as to cognitive and emotional disturbances including worry.

Despite bottom-up models being influential, top-down models also help facilitate an equally comprehensive understanding of the contribution of autonomic, cognitive, cortical and emotional vulnerability in the causation of insomnia (Baglioni, Spiegelhalder, Lombardo & Riemann, 2010). Perlis, Giles, Mendleson, Bootzin and Wyatt (1997) proposed a top-down model which suggested that increased cortical hyperarousal, (for example dysfunctional beliefs and intrusive thoughts) can result in insomnia and consequent autonomic arousal. It is important to highlight that top-down and bottom-up models are not mutually exclusive, nor contradictory of one another; in contrast, they are highly interdependent and interrelated and should be consulted together to provide valuable insight into the development and maintenance of insomnia.

**Anxiety and Insomnia**

Recent research into sleep deprivation has revealed comparable patterns of neural dysregulation between insomnia disorder and anxiety. Anxiety disorder involves an imbalance between two modes of information processing; first, bottom-up processing which involves increased activity in the amygdala region, resulting in activation of automatic processes likely to activate negative schema and impair cognitive control and inhibit negative material. Secondly, top-down goal directed processing, involving activation of prefrontal areas (anterior cingulate cortex and lateral prefrontal cortex) which involves activation of cognitive control and can contribute to relief and deactivation of negative schema (Clark & Beck, 2010).

Similar processes have been observed in a sleep deprivation study by Yoo, Gujar, Hu, Jolesz, and Walker (2007). They found that individuals who had experienced approximately 35 hours of sleep deprivation demonstrated 60% greater amygdala activation and a three-fold increase in the amount of amygdala volume activated, compared to healthy controls in response to increasingly negative emotional stimuli. In addition the sleep deprivation group demonstrated a reduction in functional connectivity between the amygdala and the medial prefrontal cortex when compared to the control group of non-sleep deprived individuals, suggesting an impairment of top-down cognitive, pre-frontal control (Sotres-Bayon, Bush, & LeDoux, 2004). Although this was a small and novel study, these findings suggest that both anxiety and sleep deprivation feature inhibited top-down cognitive control processes and consequently an increase in cognitive activity.

In order to further examine the associations between anxiety, insomnia, the experience of worry and weakened top-down control it is important to bring together the two research areas of insomnia and anxiety. Attentional control is a common

feature of the above factors and an element of weakened top-down control. The attentional control theory (Eysenck, Derakshan, Santos, & Calvo, 2007) supports the work discussed previously by Clark and Beck (2010) by proposing that anxiety disrupts the balance between a goal-directed top-down attentional system and a stimulus driven bottom-up system, resulting in less inhibition and control of negative stimuli and an increase in attention towards more emotionally salient material (Bishop, 2007; Sadeh & Bredemeier, 2011). An increase in anxiety can therefore increase the allocation of attention to threat related stimuli (either internal, for example worry thoughts, or external, for example, environmental cues), resulting in a reduced attentional focus on tasks that do not involve threatening stimuli (Koster, Crombez, Verschuere, Van Damme & Wierseman, 2006). More specifically, it is hypothesised that the worry aspect of anxiety may deplete these top-down attentional resources which are required for sustaining focus on current goal demands (Eysenck et al., 2007). This may be due to the chronic, excessive and uncontrollable nature of worry, which is a defining feature of Generalised Anxiety Disorder (GAD) (APA, 1994). Eysenck and Calvo (1992) proposed that worry causes a reduction in working memory capacity, consequently making it more difficult for individuals to successfully maintain attention on the task at hand. A study by Hayes, Hirsch and Mathews (2008) supported this proposal, by finding that individuals with high levels of worry had less residual working memory capacity when focused on a personal worry topic, than when focused on a positive topic, compared to individuals with low levels of worry. However, this is the first study to investigate the direct effects of worry on working memory capacity, it is therefore important for more research to be carried out in this area.

The suggestion that deficits in attentional control underlie vulnerability to anxiety continues to be supported and strengthened by the literature (Clark & Beck, 2010; Eysenck et al., 2007). Further research in this area by Bishop (2007) found that weakened cognitive control of pre-frontal mechanisms (in which individuals are easily distracted by stimuli) is primarily related to trait, but not state anxiety, indicating that it reflects a deficit that is associated with individuals who have an anxious predisposition rather than in individuals who are reacting to an immediate emotional stressor. Considering the evidence that sleep deprivation dysregulates both neural processes and cognitive processes in anxiety it is important to consider the prospect that weakened attentional control can contribute to the development of both insomnia and anxiety. This phenomenon has been well researched within the anxiety literature, in particular in relation to individuals with high anxiety demonstrating a greater attentional bias towards threatening stimuli, compared to non-anxious individuals (Cisler & Koster, 2010). A meta-analysis by Bar-Haim et al. (2007) supported these findings by discovering a significant threat-related bias in individuals with anxiety, but not in non-anxious individuals. The study indicated that this finding is consistent across a variety of experimental conditions and in different types of anxious populations (for example, individuals with GAD or Obsessive Compulsive Disorder), however, only a low to medium effect size was found ($d$=0.45).

Whilst there has been a significant amount of research exploring the association between attentional control and anxiety, there has been less research looking at the association between attentional control and insomnia. Although these studies do exist across a variety of different experimental conditions (see Spiegelhalder, Espie, Nissen and Riemann, 2008 for a review) they have shown

inconsistent results, mostly due to small and poorly characterised samples and inappropriate selection and use of measures (Espie & Kyle, 2008). There has also been limited research using analogue studies with individuals suffering from naturally occurring and regular sleep disruption, furthermore, research has not looked systematically at the independent roles of insomnia and anxiety on an individual's ability to perform tasks.

Relatively limited research has focused upon transient and acute episodes of poor sleep which are commonly experienced in the general population. Therefore we remain relatively uninformed about attentional control and emotional processing deficits in this population of individuals. A study cited earlier, by Zohar, Tzischinsky, Epstein and Lavie (2005) provided support that a sleep deficit can amplify the effects of disruptive and negative events on an individual's emotional state and reduce the positive effect on emotion after positive events. These are commonly reported effects of sleep loss, alongside individual's accounts of feeling fatigued, stressed, irritable and apprehensive (Hamilton, Nelson, Stevens, & Kitzman, 2007).

In addition to attentional control, worry has also been emphasised as having a strong association with insomnia, especially in the maintenance thereof (Harvey, 2002). However, there has been limited research into understanding the role of worry in the development of insomnia. Whilst sleep-focused worry has been investigated in sleep deprived populations, especially during the pre-sleep period (Harvey & Payne, 2002; Tang & Harvey, 2004), very limited research has explored the more general effects of worry in individuals with poor sleep. It could be hypothesised that early stages of sleep deprivation involves a more general influence of worry, which is un-related to subjective sleep perception (Watts, Coyle, & East,

1994). It is also possible that thought intrusions and general worry may be experienced during the daytime which would be consistent with the theory that insomnia is a 24 hour disorder (Wilson et al., 2010).

**Aims**

Considering the highlighted significance of both cognitive and psychological mechanisms in the development and maintenance of sleep disturbances, the present study sought to examine whether early sleep difficulties were related to more general (non-sleep specific) difficulties with attention and emotion processing. A particular focus was given to the extent to which poor sleep increases spontaneous negative thought intrusions in an established thought-sampling worry task. A secondary aim was to examine which sleep components from the ISI (severity, impact and satisfaction) were associated with difficulties on measures of attention and emotion processing, in particular negative thought intrusions. An undergraduate sample was selected, as this population typically experience a great degree of disturbed sleep due to their lifestyle, thus providing a useful and appropriate sample within which to explore links between sleep, worry and cognition in the early stages of a sleep disorder.

**Hypotheses**

1. Insomnia severity total (ISI) will be positively correlated with (a) lower levels of self-report attention control and increased cognitive failures, and self-report worry and (b) negative thought intrusions before and after the instructed worry task. Associations between sleep and outcome measures will remain when controlling for predicted correlations between sleep quality and anxiety, and anxiety and dependent measures.

2. The severity and impact component of the ISI will have the greatest associations with the dependent measures in this study, in line with previous research (Edinger et al., 2000).

# Method

## Ethical Approval

All study procedures were reviewed and approved by the University of Southampton Ethics Committee, UK and Research Governance Office (see Appendix A).

## Participants

Participants were primarily undergraduate students recruited from the University of Southampton, all of whom had responded to an advertisement posted within the university building and on the school of psychology intranet. All participants received course credits for their participation in the study. The sample comprised of participants between 18-43 years of age ($n$= 112, *Mean age*= 20.8, $SD$= 4.50). The sample consisted of 85% of females ($n$= 95, *Mean age* = 20.6, $SD$ = 4.77) and 15% of males ($n$= 17, *Mean age* = 20.4, *SD = 2.48).*

## Design

The study used a cross sectional research design, comprising self report measures in conjunction with a breathing focus task (Ruscio & Borkovec, 2004). Self report measures of insomnia severity, anxiety, attentional control and cognitive failures (detailed below) were investigated in relation to number of successful breathing focuses and number of thought intrusions (participant-classified by valence).

## Measures

**Anxiety.** The State-Trait Anxiety Index (STAI-T; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) is a widely used measure of anxiety and has been used in over 3,000 studies. This index consists of 20 statements describing anxiety symptoms such as; apprehension, tension, nervousness and worry. Participants rate

these categories for frequency of occurrence. Scores range between 20 and 80, with a higher score indicating greater anxiety. The STAI-T has good internal consistency $\alpha = .89$ and test-retest reliability $\alpha = .88$ (Barnes, Harp, & Jung, 2002). Internal consistency in the present study was good ($\alpha = .92$).

**Insomnia.** The Insomnia Severity Index (ISI; Morin, 1993) is a well-established screening tool for insomnia that, in part, reflects the DSM-IV criteria for a diagnosis of primary insomnia. This questionnaire was selected based on its structure, which reflects sleep onset and maintenance factors, as well as degree of impairment and emotional stress over the past two weeks. Indeed, the ISI is considered to be the most sensitive measure to daytime impairment and affective function associated with insomnia (Bastien, Vallieres, & Morin, 2001). The ISI is a seven item self-report questionnaire, which assesses the nature, severity and impact of insomnia. The seven items are rated on a 5-point Likert Scale (*0 = no problem, 4 = very severe problem*) with total scores ranging from 0 to 28. The total score is interpreted as follows: absence of insomnia (0-7); sub-threshold insomnia (8-14); moderate insomnia (15-21); and severe insomnia (22-28). However, these categories do require additional validation (Smith & Wegener, 2003). Smith and Trinder (2001) proposed that a cut off score of 14 distinguishes insomnia sufferers from healthy controls with sensitivity of 94% and specificity of 94%. More recently, Morin, Belleville, Belanger and Ivers (2011) suggested a cut off score of 10 to be the optimal balance for detecting insomnia symptoms in community samples. A principal component analysis was carried out by Bastien, Vallieres, and Morin (2001) to explore the content validity of the ISI. Their research suggested that three components, which are consistent with the diagnostic criteria of insomnia, captured 72% of the total variance. Component one; labelled “Impact” included items

relating to levels of distress, awareness of impairment and interference with daily functioning. This component accounted for 26% of the total variance. Component two; labelled "Severity" included objective items relating to severity of early morning waking, sleep onset and sleep maintenance difficulties. This component also accounted for 26% of the total variance. The third and final factor; labelled "Satisfaction" included items relating to level of distress, severity of initial insomnia and satisfaction with current sleeping patterns. This component accounted for 20% of the total variance. Components were used from the ISI as proposed by Bastein, Vallieres, and Morin (2001) to ascertain the involvement of differential sleep factors on dependent variables. Internal consistency for all subscales has good reliability $\alpha = .90$ (Morin et al., 2011) and $\alpha = .85$ in this study.

**Cognitive Failures.** The Cognitive Failures Questionnaire (CFQ; Broadbent, Cooper, Fitzgerald, & Parkes, 1982) is a self assessment scale containing 25 items that measure a person's likelihood of committing an error in the completion of everyday tasks involving psychomotor function, memory and attention over the last six months. Participants responded to items such as "Do you bump into people?" "Do you find you forget appointments?" and "Do you daydream when you ought to be listening to something?". Participants were asked to rate how often they make mistakes on a 5-point Likert scale (*0= never, 4 = very often*). The questionnaire is scored by adding up the ratings from the 25 questions, with the highest possible total score being 100. A higher score indicates a higher incidence of cognitive failures. Very high internal consistency of the scale has been found by others ($\alpha = .96$; Wallace, 2004) and good reliability in the current study $\alpha = .74$. This scale has also been found to be stable over long periods of time with a test retest reliability rate of $\alpha = .82$ (Wallace, Kass, & Stanny, 2002), and has also previously shown to correlate

well with subjective sleepiness in undergraduates (Wallace, Vodanovich, & Restino, 2003).

**Attentional Control.** The Attentional Control Scale (ACS) is a self report questionnaire that was developed to measure individual differences in attentional control (Derryberry & Reed, 2001). It consists of 20 items which initially appeared as two subscales comprising; "attentional focusing" and "attentional shifting" (Derryberry & Rotherbert, 1988). However, more recently, the two scales have been combined under the heading of the ACS, whereby the total score is used as a measure of an individual's ability to control attention. Items such as; "My concentration is good even if there is music in the room around me" reflect focus of attention, "It is easy for me to read or write while I'm also talking on the phone" reflects attentional shifting between tasks and "I can become interested in a new topic very quickly when I need to" indicates flexibility of controlling attention (Derryberry & Reed, 2002). Items were rated on a 4-point Likert scale (*1= almost never, 4= always*) with possible scores ranging from 20 to 80; higher scores indicating better attentional control. To the best of the author's knowledge, this scale has not yet been applied within a study of disturbed sleep, however, it has good internal consistency ($\alpha$= .88) and extensive literature exists to support attentional control deficits associated with sleep disturbance in clinical and non-clinical samples (MacMahon, Broomfield, & Espie, 2006; Ree, Pollitt, & Harvey, 2006). Internal consistency in this study indicated good reliability ($\alpha$ = .75). The total score of the ACS also has moderate negative correlations with self-report measures of trait-anxiety (Derryberry & Reed, 2001) whilst Verwoerd et al. (2008) also found that lower ACS scores predicted an increase in diary ratings of intrusive thoughts over

four consecutive days after watching an emotional film fragment, helping to support the predictive validity of the scale.

**Breathing Focus Task.** This task was adapted from a standard behavioural measure of worry originally developed by Borkovec et al. (1983), which was refined by Ruscio and Borkovec (2004) and more recently adapted by Hirsch, Hayes and Mathews (2009) as a worry task that has been used to reveal a greater propensity to worry in high trait anxious individuals (Hayes, Hirsch, Krebs, & Mathews, 2010; Hayes, Hirsch, & Mathews, 2010). The latter version of this task was used in this study and consisted of three phases: a 5 minute pre-worry breathing focus which assessed tendency to worry prior to the active experimental induction, a 5 minute instructed worry period and finally a 5 minute post worry breathing focus which assessed the persistence of the induced worry. During each breathing focus period, participants were instructed to focus their attention on their breathing. During this time, a computer produced 12 beeps at intervals of between 20 to 30 seconds apart (Donaldson, 2004), signalling participants to report if their attention was focused on their breathing by saying "breathing" or if they were experiencing a thought intrusion. If focused on the latter, participants were asked to indicate whether the thought was positive, neutral or negative and asked to provide a brief description of the thought (for example "positive - going on holiday"). The experimenter dictated all verbal communication. After the pre-worry breathing focus period, participants were asked to identify a current worry topic which was discussed briefly with the experimenter to ensure that it was related to a potentially negative future situation (rather than retrospective or depressogenic in content, as specified by the script, see Appendix D). The experimenters and authors were mindful of the potential overlap and co-morbidity of depression and anxiety and although there were no published

guidelines on ensuring a non-depressogenic worry was given by participants, a number of protocols were established to encourage such. First, participants were asked to think of something that was worrying them in 'the moment', thus encouraging anticipatory worry, which is a major feature of anxiety rather than depression. Secondly, participants were asked three questions to further explore and activate the worry. These questions also helped to focus the five minute period upon the potential future consequences of an event that has not yet happened (for example; how catastrophic would it be, how likely is it to happen and how well would you cope?). Participant responses to these questions were used to clarify the relevance and significance of the worry and any uncertainty about the salience of the worry was pursued by the experimenters through further questioning. Participants were then asked to spend 5 minutes worrying about this issue in silence, whilst the experimenter left the room. On the experimenters' return, the second breathing interval was completed. In this final stage, participants were asked to expand on the thought intrusions they reported during the breathing focus periods. The experimenter read aloud each thought intrusion and asked the participants to describe what was going through their minds at the moment they originally had that thought. Participant's descriptions were recorded for later rating by an independent researcher, who then assessed the valence of each thought intrusion.

**Procedure**

Once ethical approval had been obtained from the University (see Appendix A), participants who signed up to the study were asked to collect an information sheet detailing the nature of the study (see Appendix B) and a questionnaire pack two days prior to the testing session. Questionnaires were completed and brought to a single test session where several computerised tasks were performed (not all

related to the core aims of this study and to be reported elsewhere by different authors). Full written informed consent was sought from participants prior to the testing session (see Appendix C). The completed questionnaires included the ISI, STAI-T, CFQ and the ACS as described above. Approximately an hour and a half into the test session the worry task began. The experimenter read aloud standardised instructions (see Appendix D) and checked participant's understanding of the procedure. A practice trial was then conducted where participants focused on their breathing for 45 seconds and provided a brief summary of their thoughts at three time intervals. This trial was followed by the initial breathing task, the worry period and the post-worry breathing task (as described above). Following the experimental session, participants were thanked and given a full debriefing and mood repair stimuli in the form of humorous news stories (see Appendices E and F).

# Results

## Approach to Data Analysis

Data analysis was conducted using SPSS 19. Data was checked for normality using frequency distributions and the Kolmogorov-Smirnov test, all indicating the data was normally distributed. Missing data were dealt with in the correlational analyses using pairwise deletion, i.e. only participants who provided data for both the variables being correlated were included in each analysis.

Morin et al. (2011) recommend caution be exercised when using cut off scores for insomnia screening questionnaires, with their findings suggesting that different cut off scores were needed depending on the research question and population used, for example a lower cut off score may be more preferable to estimate the prevalence of insomnia in the general population than would be necessary if estimating the prevalence in a clinical population However, there is a risk that such an approach could introduce greater subjectivity where cut off scores too low could potentially give false positives whilst those too high may prove to be too stringent. Considering this evidence, and these potential complications, this study chose to reflect the continuum of insomnia severity, without being constrained by categorical cut offs by using correlational and linear regression analyses. These analyses were considered the most fitting for the data in this study, due to the probability of important, sub-threshold and sub-diagnostic levels of sleep disturbance in the student population. This study chose to replicate the components of the ISI, introduced earlier by Bastien et al. (2001) (for example, severity, impact and satisfaction), in order to investigate whether these components of poor sleep were associated with reduced attentional control and biased emotion processing (for example, negative thought intrusions) within a non-clinical population.

**Analysis**

Table 1 shows descriptive statistics for all test measures. The ISI mean score was first interpreted in the context of the original categorical scoring system (Morin, 1993) in order to provide an indication of the average level of sleep disturbance in the sample. The mean score of 8 in this sample is the lowest score used to classify someone as having 'subthreshold insomnia' (Bastien et al., 2001). More recently, in a validation study, Morin (2011) suggested a score of 10 on this scale as the optimum balance between sensitivity and specificity for detecting insomnia in community samples. Taking into consideration the range of scores on this measure and that, according to the aforementioned cut off scores, between 26% and 53% of the sample experienced insomnia, the sample does appear to reflect a continuum of insomnia symptoms (see Appendix G).

Table 1

*Descriptive statistics for self-report measures*

| Measure | N | Min | Max | M | SD |
|---|---|---|---|---|---|
| ISI | 112 | 0 | 21 | 8.22 | 4.57 |
| Impact | 112 | 0 | 11 | 3.12 | 2.30 |
| Severity | 112 | 0 | 10 | 3.08 | 2.08 |
| Satisfaction | 112 | 0 | 11 | 4.79 | 2.42 |
| STAIT-T | 108 | 21 | 72 | 40.14 | 10.40 |
| ACS | 106 | 31 | 99 | 48.46 | 9.78 |
| CFQ | 112 | 8 | 97 | 46.02 | 14.73 |

Mean trait anxiety scores fell within the expected range for a healthy sample (for example 33-44, Spielberger et al. 1983 as did attentional control scores (45.6-58.9: Derryberry & Reed, 2001), however, the mean score on the CFQ was higher

than expected when compared to average scores previously reported in the general population (25-35; Wagle, Barrios, & Ho, 1999).

Descriptive statistics for the worry task are displayed in Table 2.

Table 2

*Descriptive Statistics for Worry Task as per Category of Thought*

| Condition | N | Min | Max | M | SD |
|---|---|---|---|---|---|
| Pre-worry | | | | | |
| Breaths | 111 | 1 | 12 | 8.68 | 1.86 |
| Neutral | 111 | 0 | 5 | 1.15 | 1.20 |
| Positive | 111 | 0 | 5 | 1.26 | 1.26 |
| Negative | 111 | 0 | 6 | .89 | 1.09 |
| Post-worry | | | | | |
| Breaths | 111 | 2 | 12 | 8.41 | 2.17 |
| Neutral | 111 | 0 | 5 | 1.26 | 1.26 |
| Postive | 111 | 0 | 5 | .84 | 1.09 |
| Negative | 111 | 0 | 7 | 1.57 | 1.44 |

Table 3

*Pearson's Correlation Matrix among Self-Report Insomnia Severity, Trait Anxiety and Cognitive Function*

| Measure | | | |
|---|---|---|---|
| | STAI-T | ACS | CFQ |
| ISI | .46** | -.25* | .28** |
| Severity | .26** | -.13 | .20* |
| Impact | .54** | -.23* | .28** |
| Satisfaction | .46** | -.34** | .29** |
| STAI-T | - | -.52** | .60** |

*** Correlation is significant at the .01 level*
** Correlation is significant at the .05 level*

Correlation analysis was performed in order to establish relationships between key variables, as shown in Table 3. As hypothesised, a significant positive correlation was revealed between insomnia and anxiety. Higher insomnia total scores were significantly associated with lower attentional control and increased cognitive failures. A significant positive correlation was also seen between anxiety and increased cognitive failures and a significant negative correlation was found between anxiety and lower attention control. These findings were to be expected given the established burden that anxiety exerts upon the cognitive resources necessary to efficiently perform tasks requiring attentional control.

**Pre-worry period.** As shown in Table 4, and as hypothesised, there was a significant negative correlation between the ISI total score and the number of breathing focuses in the pre-worry period.

Table 4.

*Pearson's Correlation Matrix among Self-Report Measures, Number of Breathing Focuses, and Number of Thought Intrusions by Valence on the Worry Task*

| | Condition | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Breath | | Neutral | | Positive | | Negative | |
| | Pre | Post | Pre | Post | Pre | Post | Pre | Post |
| Measure | | | | | | | | |
| ISI | -.24* | -.19* | .01 | .02 | .12 | .11 | .25** | .18 |
| Severity | -.30** | -.17 | .11 | .07 | .13 | .10 | .24* | .11 |
| Impact | -.15 | -.16 | -.03 | .02 | .07 | .05 | .20* | .18 |
| Satisfaction | -.18 | -.13 | -.02 | -.04 | .13 | .09 | .17 | .17 |
| STAI-T | -.15 | -.20* | -.09 | -.06 | .13 | -.02 | .21* | .36** |
| ACS | -.01 | .13 | .13 | .23* | -.11 | -.05 | .00 | -.38** |
| CFQ | -.11 | -.13 | -.04 | -.02 | .13 | .02 | .09 | .20* |

** *Correlation is significant at the .01 level*
* *Correlation is significant at the .05 level*

The severity subscale, reflective of more objective sleep deficits, was most strongly associated with the number of breathing focuses in the pre-worry period. Contrary to predictions, trait anxiety was not correlated with the number of breathing focuses during the pre-worry period. However, there was a significant positive correlation between anxiety and negative thought intrusions during the pre-worry period. There was also a significant positive correlation between the ISI total score and the number of negative thought intrusions, as well as a significant positive correlation between the severity and impact components of the ISI and negative thought intrusions in the pre-worry period. There was no evidence for significant correlations between the ISI scores and neutral and positive thought intrusions, as well as no significant correlations between trait anxiety scores and neutral and positive thought intrusions.

A partial Pearson's correlation was performed between the ISI total score and negative thought intrusions whilst controlling for anxiety. As hypothesised, ISI total score remained significantly correlated with negative thought intrusions in this model ($r$ (104) = .19, $p < .05$). Further partial correlations revealed that there was a significant correlation between the severity component of the ISI and negative thought intrusions, whilst controlling for anxiety ($r$ (104) = .21, $p < .05$). However there was no evidence for a significant correlation between the impact component of the ISI and negative thought intrusions ($r$ (104) = .14, $p = .17$).

**Post-worry period.** As shown in Table 4, and as hypothesised, there was a significant negative correlation between both the ISI score and trait anxiety on the number of breathing focuses during the post-worry period. There was no evidence of a significant correlation between the ISI total score and negative thought intrusions in the post-worry period. However an association between the impact

component and negative thought intrusions fell just below significance. A significant positive correlation was found between trait anxiety and negative thought intrusions following the worry period. There was also a significant positive correlation between attentional control and neutral thought intrusions during the post-worry period. However, there was no evidence for significant correlations between any of the other individual independent variables and positive or neutral thought intrusions in this task.

Paired samples t-tests were conducted to look at the difference in performance of the worry task at pre-worry and post-worry intervals. There was no significant difference between the number of breaths at pre-worry ($M$ = 8.68, $SD$ = 1.86) and number of breaths at post-worry ($M$ = 8.41, $SD$ = 2.17) ($t$ (110) = 1.74, $p$ = .85). There was a significant difference between the number of negative thought intrusions at pre-worry ($M$ = .89, $SD$ = 1.09) and number of negative thought intrusions post-worry ($M$ = 1.57, $SD$ = 1.44) ($t$ (110 = 4.96, $p$ = < .001) indicative that the worry manipulation was successful in producing an increase in worry. Individual differences in the degree of change in negative thought intrusions did not co-vary with measures of sleep quality, anxiety or attention control (irrespective of whether baseline pre-negative intrusions were entered as a covariate).

Table 5

*Pearson's Correlations between Change in Negative Thought Intrusions and Self-Report Measures of Insomnia and Anxiety whilst Controlling for Pre-Breath Intrusions.*

| Change | Measure | | | | | |
|---|---|---|---|---|---|---|
| | ISI total | ISI severity | ISI impact | ISI satisfaction | STAI-T | ACS |
| Negative Intrusions | -.04 | -.12 | .01 | .02 | .18 | -.38 |

## Discussion

The present study was the first to utilise the worry task to investigate the effects of both poor sleep and anxiety on attentional control and emotional regulation. It was predicted that there would be a bi-directional positive relationship between insomnia severity and anxiety and in addition that these two variables would be positively associated with self reported cognitive failures and negatively associated with attentional control. It was also predicted that the independent variables of attentional control, self reported cognitive failures, anxiety and insomnia would be correlated with performance indicators on the worry task (for example breathing focus and thought intrusions). In particular, it was hypothesised that the independent variables would be negatively correlated with breathing focus and positively correlated with thought intrusions. In addition, it was predicted that there would be an independent relationship between insomnia severity and the performance indicators beyond shared associations with anxiety.

Consistent with predictions, a significant positive association was found between insomnia (ISI) and anxiety. This result supports previous findings in the literature, reporting similar relationships between insomnia and anxiety in clinical samples (Jansson-Frojmark, & Lindblom, 2008), as well as research which emphasises poor sleep as a risk factor for anxiety and conversely anxiety as a risk factor for poor sleep (Jansson & Linton, 2006). The findings in this study replicate previous associations between increased anxiety and poor self-report attention control (Derryberry & Reed, 2002), but are the first to associate sleep quality with self-report deficits in attentional control and cognitive failures. Furthermore the present study is novel in examining associations between sleep and objective

performance measures of attentional control (breathing focus) and negative thought intrusions in a breathing focus task.

As hypothesised, insomnia severity (ISI) was significantly associated with a decline in focused attention on breathing during the 5 minute pre-worry breathing period. This association was in the absence of a predicted correlation between breathing focus and trait anxiety and self-report attentional control and cognitive failures. Additionally, the severity component of the ISI (ISI-S), which reflects more objective sleep loss (due to late sleep onset and/or sleep maintenance difficulties), was most strongly associated with a reduction in breathing focus (for example, larger effect size). These findings are in line with previous clinical models of insomnia that associate a decrease in sleep quality with a decrease in attention for daily activities, often due to the effects of fatigue, but also due to hypervigilance and increased awareness of threat and worry about poor sleep (Harvey, 2002; Spielman & Glovinsky, 1991).

A unique feature of the present study was to go beyond previous evidence in clinical insomnia and examine sleep-cognition relationships in a predominantly healthy sample experiencing transient and acute episodes of poor sleep (Doghramji, 2006). Healthy young adults (for example, undergraduates) are more likely to experience poor sleep due to lifestyle choice (for example, socialising) rather than worry/negative affect that promotes poor sleep in later life. As such the present sample provides a useful cohort in which to examine associations between sleep, cognition and negative thought intrusions in the absence of notable mood/anxiety disorders (Roth & Roehrs, 2003). Specifically in student populations there can be several causative factors for acute insomnia, including changes to sleep environment, academic pressures, timing of sleep and part time employment responsibilities

(Brown, Buboltz, & Soper, 2002). Whilst research suggests that most individuals will naturally recover from transient sleep deprivation, it is important to consider that chronic insomnia can develop from repeated exposure to acute episodes of insomnia, especially when poor sleep leads to deficits in attentional control and emotional regulation (for example, increased worry) (Ellis, Gehrman, Espie, Riemann, & Perlis, 2012). Thus while individuals differ in their vulnerability to develop transient insomnia (Drake, Scofield, & Roth, 2008), observed associations between poor sleep and objective deficits in both attentional control and worry control suggest a need to monitor and address these problems at early stages of sleep disruption, to help prevent the development of more severe and persistent sleep and mood disorders.

The findings in this study fit well with suggestions that individuals suffering with insomnia experience greater difficulties in attentional control for everyday tasks due to attentional biases towards threatening stimuli, which may take the form of negative thought intrusions (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006). The findings also converge with those from recent neuro-imaging studies that implicate dysfunction in amygdala-prefrontal pathways in sleep-related biases in emotion processing, through specifically increasing amygdala mediated threat detection (triggering bottom-up stimulus driven attention, activating negative schema and impairing attentional control) and reducing prefrontal top-down control of attention and negative schema (Yoo et al. 2007).

In this study it is noteworthy that the significant association between insomnia and negative thought intrusions remained after controlling for anxiety during the 5 minute pre-worry breathing period, with the severity component (ISI-S) being particularly important in this association. This extends evidence that negative thought intrusions and attentional biases are more readily associated with trait

anxiety/worry, and highlight the importance of sleep quality in mental and emotional well-being (Spiegelhalder, Espie, Nissen, & Riemann, 2008). The hypothesis that the impact component of the ISI would be associated with negative thought intrusions was supported, although did not remain significant when anxiety was controlled for. This latter finding is consistent with research which suggests that the perceived/self-reported impact of poor sleep on daytime functioning correlates strongly with anxiety about underperformance (Harvey, 2002) for example, those who worry generally, are also likely to worry about the impact/consequences of poor sleep.

In the post-worry period anxiety was associated with both negative thought intrusions and breathing focus, consistent with the idea that anxiety is characterised by a failure to control/inhibit negative cognitions once activated (as was the case following the explicit "worry" period). In contrast, although insomnia was correlated with breathing focus in the post-worry period, it did not correlate strongly with negative thought intrusions post-worry. Though the observed trend fell just below significance and may require more power to correctly reject the null hypothesis, the effect of sleep on inhibiting activated worries may be a feature of more severe persistent sleep loss rather than transient/acute sleep disturbance examined in the present study.

Finally the observation that poor breathing focus (attentional control) was associated with increased negative (rather than positive and neutral) thought intrusions, fits well with attentional control theory (Eysenck et al., 2007) which suggests that poor attentional control increases the influence of bottom-up processing mechanisms that selectively favour negative information and increase negative affect and negative thought intrusions.

**Clinical Implications**

The findings in this study highlight the importance of, and need for, early intervention in both sleep difficulties and co-morbid psychological difficulties. Recognition of sleep difficulties in the general population needs to be improved, particularly considering evidence that sleep disorders are under-recognised and under-treated, despite the existing high prevalence in the general population (Benca, 2005). In addition, it is reported that 50% or more of individuals with insomnia rarely, if ever, consult their doctor for advice or treatment (Espie, 2011), which suggests that greater effort needs to be made to raise an awareness of sleep difficulties and the treatment options available. Further, awareness needs to be raised in health clinicians, with research suggesting that minimal, if any, time is given to sleep disorders on training courses for General Practitioners (GPs) and Clinical Psychologists (Stores, 1999). With better education and awareness both individuals and clinicians may be better placed to identify both the risk factors for insomnia and predictors of insomnia relapse. Health promotion and a greater awareness of the importance of sleep is crucial for early intervention, especially considering the findings of this study which suggest that even transient insomnia in a healthy population can have significant effects on cognition and emotional processing.

Alongside the importance of improving awareness and recognition of sleep disorders, the findings of this study would suggest that consideration needs to be given to the treatment aspects of sleep disorders and co-morbid psychological difficulties. Traditionally, research encouraged treatment of the psychological disorder, with the expectation that the insomnia would resolve (Buysse, Reynolds, & Kupfer, 1997), however, with insomnia now recognised as a primary disorder in

itself, emphasis has been given to treating insomnia independently of the co-morbid disorder (Newbauer, 2009). CBT is currently recommended as the treatment of choice for insomnia, however several limitations of its current content and structure have been highlighted in the literature, for example neglecting to address underlying maintenance factors as well as an individual's perceptions of sleep (Harvey & Tang, 2012). Considering the findings of this study and due to the complexity of comorbidty, it would seem appropriate and relevant for CBT to be tailored to the individuals needs and where possible, for both the insomnia and co-morbid psychological difficulty to be addressed independently. To address the complexity of establishing which disorder to treat first, Harvey (2009) proposed a "transdiagnostic sleep intervention" in which poor sleep is addressed through the use of CBT, with the option of adding extra components to target the specific psychological difficulty at a later stage. Although this new comprehensive approach would enhance existing treatment options, it warrants further research and evaluation.

**Limitations**

Whilst this study presents some interesting and novel findings, there were several important limitations. First, the affective symptoms of insomnia and attentional control were all assessed by self-report questionnaires, which are inherently associated with methodological weaknesses, for example social desirability bias (Nederhof, 2006), and the risk of inaccurate responses due to cognitive biases and poor memory. However, despite their limitations, there are some advantages of self-report questionnaires, including their inexpensive and time efficient characteristics (Libman, Fichten, Bales, & Amsel, 2000). It has therefore been recommended that a combination of sleep measures should be used in sleep

research, highlighting the importance of using both subjective and objective approaches (Gaina, Sekine, Chen, Hamaishi, & Kagamimari, 2004). In relation to this study, it would have been useful to include an objective measure of sleep quality. Despite not expecting to a see difference between objective and subjective sleep deficits which exist in primary insomniacs (due to the sample comprising healthy individuals), the use of actigraphy in combination with sleep diaries would have supported and validated the subjective data from the self report measures used (Littner et al., 2003). Another limitation concerns the completion of the worry task towards the end of a longer testing session (one hour and a half into testing), at which point it may be expected that participants were more fatigued than they were at the beginning of testing. This may have had an impact on an ability to concentrate on breathing and therefore consequent impacts on the number of thought intrusions experienced. In addition to the above, the use of a university population helped control variables known to affect sleep, for example; age, sex, having children at home, occupation, work schedules, differing living arrangements and social milieus, however the use of a specific population sample does limit the ability to generalize findings to the wider population.

Replications of this preliminary study, applying the recommended improvements would be valuable to further clarify the attentional and emotional processing biases associated with poor sleep. Implementation of this study into clinical and subclinical populations would be particularly useful for exploring the impact of the developing course of insomnia on subjective and objective performance as well as the relationship between insomnia and anxiety. In addition, this exploration over the course of insomnia will be an important step in understanding the development of primary insomnia.

## Conclusion

This study provides initial evidence of a relationship between poor sleep, attentional control and negative thought intrusions in a healthy population and is the first to (a) use the worry task in sleep research and (b) reveal in the early stages of poor sleep weakened attentional and propensity to worry, which together confer risk for persistent and severe sleep problems, and mood and anxiety disorders. Thus, present findings recommend that a better awareness of insomnia is established in both health professionals and the general population, to enable earlier recognition and diagnosis, which would hopefully prevent more complex forms of insomnia developing. Furthermore, treatment for insomnia, more specifically CBT, needs to be revised to ensure it encapsulates and addresses the difficulties found in this study.

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# Appendices

Appendix A: Ethics approval

Appendix B: Information sheet and consent form for questionnaires

Appendix C: Information sheet and consent form for testing session

Appendix D: Worry script

Appendix E: Debrief statement

Appendix F: Mood repair stimuli

Appendix G: Mean scores for insomnia severity index

# Appendix A

## Ethics Approval

**From:** ERGO [mailto:DoNotReply@ERGO.soton.ac.uk]
**Sent:** 14 October 2011 20:29
**To:** Baker L.D.
**Subject:** Your Ethics Amendment (Ethics ID:813) has been reviewed and approved
Submission Number 813:
This email is to confirm that the amendment request to your ethics form (The role of sleep in cognitive function and emotion processing (Amendment 1))has been approved by the Ethics Committee.

You can begin your research unless you are still awaiting specific Health and Safety approval (e.g. for a Genetic or Biological Materials Risk Assessment)

Comments
None
Click here to view your submission

---
ERGO : Ethics and Research Governance Online
http://www.ergo.soton.ac.uk
---
DO NOT REPLY TO THIS EMAIL

## Appendix B

### Information Sheet and Consent Form for Questionnaire Completion

*Version 1: Date 10/11/2011*

*Study Title:* ***The role of sleep in cognitive function and emotion processing***

Please read this information carefully before deciding to take part in this research. If you are happy to participate you will be asked to sign below.

We are Louise Kenny (Clinical Psychology Trainee) and Louise Baker (PhD student), Psychology students at the University of Southampton. We are requesting your participation in a study examining the effect of poor sleep on daytime function (e.g. attention, mood).

This part of the experiment will involve the completion of a questionnaire pack which should take approximately half an hour. Two credits are received for participation in this part of the experiment (the experiment is worth 10 credits in total).

Personal information will not be released or viewed by anyone other than researchers involved in the project and all data will be anonymised prior to analysis and storage. No identifying characteristics (e.g. name, age) will be stored with your data or included in the results of the study. Your participation is voluntary and you may withdraw your participation at any time. Published results of this research will maintain your confidentiality. If you choose not to participate there will be no consequences to your grade or to your treatment as a student in the psychology department.

A debriefing statement will be supplied at the end of the experiment. If you have any questions please ask them now, or if you prefer contact my supervisor Dr Matt Garner – m.j.garner@soton.ac.uk (023) 8059 5926.

Sincerely,

Louise Kenny

**Statement of consent**

I_____ have read the above informed consent form.
*(Participants name)*

I understand that I may withdraw my consent and discontinue participation at any time during this experiment without any penalty or loss of benefit to myself. I understand that any data collected as part of this research project will be treated confidentially, and that published results of this research will maintain my confidentiality. In signing this consent letter I am not waiving my legal claims, rights or remedies. A copy of this consent letter will be offered to me.

I give consent to participate in the above study. YES
NO

Cont.

Signature Date:

Name

*(Participants name)*

I understand that if I have questions about my rights in this research or I feel that I have been placed at risk, I can contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ. Phone (023) 8059 5578.

## Appendix C

### Information Sheet and Consent Form for Testing Session

*Version 1: Date 10/11/2011*

*Study Title:* ***The role of sleep in cognitive function and emotion processing***

Please read this information carefully before deciding to take part in this research. If you are happy to participate you will be asked to sign below.

We are Louise Kenny (Clinical Psychology Trainee) and Louise Baker (PhD student), Psychology students at the University of Southampton. We are requesting your participation in a study examining the effect of poor sleep on daytime function (e.g. attention, mood).

This part of the experiment will involve a test session during which you will complete a battery of computerised tasks (these will take about 120 minutes). You will receive 8 credits for your participation.

Personal information will not be released or viewed by anyone other than researchers involved in the project and all data will be anonymised prior to analysis and storage. No identifying characteristics (e.g. name, age) will be stored with your data or included in the results of the study. Your participation is voluntary and you may withdraw your participation at any time. Published results of this research will maintain your confidentiality. If you choose not to participate there will be no consequences to your grade or to your treatment as a student in the psychology department.

A debriefing statement will be supplied at the end of the experiment. If you have any questions please ask them now, or if you prefer contact my supervisor Dr Matt Garner – m.j.garner@soton.ac.uk (023) 8059 5926.

Sincerely,

Louise Kenny

**Statement of consent**

I_____ have read the above informed consent form.
*(Participants name)*

I understand that I may withdraw my consent and discontinue participation at any time during this experiment without any penalty or loss of benefit to myself. I understand that any data collected as part of this research project will be treated confidentially, and that published results of this research will maintain my confidentiality. In signing this consent letter I am not waiving my legal claims, rights or remedies. A copy of this consent letter will be offered to me.

I give consent to participate in the above study. YES
NO

Cont.

Signature Date:

Name

*(Participants name)*

I understand that if I have questions about my rights in this research or I feel that I have been placed at risk, I can contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ. Phone (023) 8059 5578.

## Appendix D

## Worry Script

The next task is completely different. I'll give you a quick overview of how it will run. It has four parts:

- First, I'll ask you to focus on your breathing for a while
- Then I'll ask you to focus on some thoughts
- Then you'll focus on your breathing again for a while
- Finally, I'll ask you about some of your thoughts.

I'll give you more detailed instructions as we go along. Let's start with a practice. I'd like you to focus your attention on your breathing for a short time. Try not to focus on anything except your breathing. It is completely normal for your mind to wander, but if this happens try to refocus your attention back onto your breathing again. Does that make sense? You can shut your eyes if it makes it easier.

Ok, so if you'd like to start focusing on your breathing, I'll let you know when to stop after about 20 seconds. Just concentrate on breathing in and out.

*[Experimenter counts 20 seconds]*

That's great. Was it clear what you needed to do?

When the task begins, I'll ask you to direct your attention to your breathing. Then, at random intervals the computer will beep. If you are concentrating on breathing at the point you hear the beep, say: "breathing". However, if your mind had wandered at that point, I'd like you to:

- Summarise in a few words what was going through your mind at that moment
- Tell me whether you would rate it as positive, neutral or negative

For example, you might say "positive – dinner tonight". I'll ask you to expand upon it later. Before we begin this task we'll practice, so you get a chance to see how it works. The practice is just for 45 seconds.

I'd like you to start concentrating on your breathing, and during the practice you'll hear beeps at random intervals. When you hear a beep signal, if at that moment you were concentrating on your breathing, say "breathing". If your attention has wandered at that point, I'd like you to say a couple of words to describe what was running through your mind, and to tell me whether it was, positive, neutral or negative. There is no need to wait for me to prompt you – just go ahead and make your rating as soon as you hear a beep. Try not to predict when the beep will come, just focus on your breathing.

*[Experimenter sets computer to the 45-second practice and records responses. After the practice, they explain again to the participant what is required if necessary.*

Ok, so now we're going to begin the main task. It will be just like the practice, but it will last 5 minutes, with random intervals. As before, I'd like you to focus on your

breathing and then make a response after each beep. Remember to try not to concentrate on anything except your breathing. If your mind wanders, try to refocus your attention back on your breathing again. OK are you ready?

*[Experimenter sets computer to the 5 minute setting and records responses, prompting participants if necessary, by saying "positive, negative, neutral?]*

What I'd like you to do now is to think of something which you're worrying about at the moment. It could be about work, money or relationships, for example. You should choose something you don't mind telling me a little bit about.

*[Experimenter explores these with the participant, giving empathy. If the worry, is more depressogenic than worrying then she suggests they think of another one.]*

Now I'm going to give you 5 minutes to worry about this issue. During that time, I'll leave the room, and what I'd like you to do is to worry about this as you normally would. Focus on the worry for the whole 5 minute period. If you start thinking about something else, please just refocus back onto thinking about the worry. If you could start now, and I'll come back when the 5 minutes is up.

*[Experimenter leaves the room for 5 minutes and then returns]*

Ok, so now I'll give you another 5 minutes to focus your attention on your breathing again. As before, try not to think about anything except your breathing. It's normal for thoughts to wander, but if this happens try to refocus your attention back on your breathing again. Again, at random intervals the computer will beep. If you're focused on your breathing at that point say "breathing". If you thoughts have wandered at that point, I would like you to first give me a brief description of your thought, and whether it was positive, neutral or negative.

*[Experimenter sets the computer to the 5-miunute setting and records participant responses.}*

Great. Now what I'd like you to do is to try to remember what you were thinking about at each of the beeps during the two periods where you were focusing on your breathing. I'll give you a summary of what you said you were thinking about at each of the beeps in turn. I'd like you to try and expand on these descriptions by telling in more detail what you were thinking. For example, if earlier, you said you were thinking about your dinner, you could expand o whether there were more specific thoughts attached – like what you would be cooking later. We only need information about what your thoughts were at the time of the beep. If you said you were thinking about your breathing, an expanded description isn't needed. If it's OK, I'll record this bit to help me score it. Only your participant number will be recorded.

## Appendix E

## Debrief Statement

***Version 1: Date: 4/11/2010***

***The role of sleep in cognitive function and emotion processing***

Debriefing Statement

The aim of this research was to investigate the relationship between poor sleep and cognitive and emotional dysfunction. The developmental trajectory of primary insomnia according to Spielman & Glovinsky (1984) is considered to involve predisposing (psychological vulnerability), precipitating (life stress) and perpetuating factors (conditioned arousal). Despite the popularity of this model, the type of deficits underlying acute, short term and chronic insomnia remain poorly understood and are likely to change due to cognitive processes such as those outlined in Harvey (2002).

It has been proposed that acute insomnia involves significant sleep deprivation, reflected by general impairment to cognitive and emotional processing. As the sleep complaint persists, however, it is characterised more by sleep-related worry, emotional arousal and distress. This state then further fuels negative pre-sleep cognitive activity responsible for insomnia (Harvey, 2002) and reinforces any objective sleep loss (Roth & Roehrs, 2003).Thus selective attention to sleep related stimuli is expected to be a feature of an escalating sleep complaint.

Many recent studies have been unsuccessful in revealing the daytime deficits associated with an escalating sleep complaint due to insensitive experimental paradigms (e.g. paradigms which fail to prevent individuals from compensating for their dysfunction with extra cognitive effort). Therefore, this study used a battery of performance tasks that are likely to be sensitive to the type of complaint characteristic of insomnia sufferers (e.g. difficulty inhibiting responses and difficulty concentrating) and a range of self-report measures to help predict the traits characteristic of those who develop persistent insomnia.

In addition, evidence from studies using fMRI has shown that suppressed inhibitory action from the medial prefrontal cortex to the amygdala due to poor sleep may result in a deficit in emotional regulation such that individuals will exhibit both selective attention to negative sleep related stimuli as well as have difficulties in regulating their emotions, consequently putting them at a higher risk of developing anxiety compared to normal sleep controls. Your data will help in our understanding of the role of sleep in the regulation of these cognitive mechanisms and their link with anxiety.

Cont.

The following references provide more information about the rationale behind this study:

Harvey, A. G. (2002). "A cognitive model of insomnia." Behaviour Research and Therapy **40**(8): 869-893.

Derryberry, D. and M. A. Reed (2002). "Anxiety-related attentional biases and their regulation by attentional control." Journal of Abnormal Psychology **111**(2): 225-236.

**During this study we have asked you to reflect on certain aspects of your physical and mental health. If at any point during your studies you become concerned about your mental or physical health then please contact your General Practitioner.**

**Finally, if you have more general worries during your time as a student in Southampton then please also be aware that Student Services or your personal tutor are happy to provide support and advice.**

Once again results of this study will not include your name or any other identifying characteristics. The experiment did not use deception. You may have a copy of this summary if you wish and a review of the research findings once the project is completed.

If you have any further questions please contact Matthew Garner at m.j.garner@soton.ac.uk

Thank you for your participation in this research.

Signature _____ Date _____

Name

If you have questions about your rights as a participant in this research, or if you feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ.

Phone: (023) 8059 5578.

**We would appreciate it if you did not discuss any aspect of the study with anyone other than the researchers involved, thank you.**

# Appendix F

## Mood Repair Stimuli

### Pensioner's radio sparks call to police

A bad-tempered German pensioner could be charged with wasting police time after complaining about loud music - from her own radio.
Elsie Weiss, 71, from Mulheim called police late at night to complain she couldn't sleep because of the noise.
But police who turned up to investigate found the music was coming from the pensioner's own radio that she had left on full volume in the back garden earlier in the day.
A police spokesman said they were considering sending her a bill for the time spent on the call and said:
"She had taken the radio outside and left it switched on full volume when she went inside," said a police spokesperson.
A neighbour said: "She always plays her music really loud - for once she gave herself a taste of her own medicine."

### Computer error means £2.3 trillion electricity bill

A man has received a bill from British Gas for £2.3 trillion after a computer mix-up.
Brian Law got an initial bill for £59 last November, but when he forgot to pay it, they sent him a final demand.
The demand for £2,320,333,681,613 was supposed to be for electricity supplied to Mr Law's new home at Fartown, Huddersfield.
The company warned they would take him to court if he didn't pay the bill in full immediately, reports the Yorkshire Post.
But Mr Law said he made numerous efforts to have the matter sorted out, but British Gas failed to return phone calls having left his number with representatives.
He said: "Eventually, I decided the only way I was going to sort it out was to go to court and offer a penny a week."
But after local media intervened, British Gas said there had been mistake with a computer mixing up the reference number for the property.
"We have agreed that I owe £59 and I will set up a direct debit for the future," said Mr Law.
A British Gas spokeswoman said Mr Law was told the bill was a "simple clerical mistake.

## Appendix G

## Mean Scores for Insomnia Severity Index (ISI)