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INSOMNIA SYMPTOMS AND DAYTIME DYSFUNCTION

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ABSTRACT

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Repeated episodes of acute sleep disturbance significantly increase vulnerability to the development of chronic insomnia (Buysse et al., 2008) - a perseverative and complex sleep disorder associated with onset of psychiatric illness (Sivertson et al., 2014). Through cognitive modelling, the processes which serve to maintain chronic insomnia are well understood. In contrast, daytime dysfunction in non-clinical populations is poorly understood despite sound reasoning for dysregulation to broader (non-sleep specific) attention, decision making and arousal systems. Insomnia symptoms encompass sub-clinical levels of poor sleep quality and concern about daytime function and can involve a greater underlying sleep debt and more severe daytime cognitive dysfunction than is often substantiated in chronic presentations (Ellis, Gehrman, Espie, Riemann & Perlis, 2012), providing incentive for profiling daytime performance in sufferers. Healthy young adults with psychosocially disturbed sleep are an important population where insomnia symptoms are elevated (Benitez & Gunstad, 2012). Study One investigated the extent to which insomnia symptoms are associated with impairment to attentional mechanisms and executive control using sensitive neurocognitive tasks. Mild attentional impairment on the Attention Network Task was associated with aspects of poor sleep quality, whereas executive control appeared unaffected. Study Two extended the investigation of attentional control to the phenomenon of thought intrusions because difficulty inhibiting negative thoughts is an established feature of insomnia disorder which is, as yet, unexamined in poor sleepers. Using a behavioural measure of thought intrusions insomnia symptoms were found to be associated with greater distractibility and spontaneously activated negative thoughts. Study Three was designed to profile physiological arousal response to situational stress associated with insomnia symptoms because inter-dependent cognitive and physiological processes are posited to initiate and maintain the complaint. Situational stress was created using the 7.5% CO_2 challenge but associations between autonomic activity, state anxiety and insomnia symptoms were not revealed. The final study investigated risky decision making, where, despite good overall performance on a modified gambling task, those with greater insomnia symptoms were more likely to gamble on trials with explicitly unfavourable odds. Collectively, results provide evidence for dysregulated attention, increased distractibility, negative intrusive thought and unfavourable decision making. Daytime dysfunction associated with insomnia symptoms could establish a vulnerability for sleep disorder and could precede clinically important affective disturbance found in clinical insomnia.

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DECLARATION OF AUTHORSHIP

I Louise Baker declare that this thesis and the work presented in it are my own and has been generated by me as the result of my own original research.

Insomnia Symptoms and Daytime Dysfunction

I confirm that:

1. This work was done wholly or mainly while in candidature for a research degree at this University;
2. Where any part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated;
3. Where I have consulted the published work of others, this is always clearly attributed;
4. Where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;
5. I have acknowledged all main sources of help;
6. Where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;
7. None of this work has been published before submission.

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Date: 01.02.2015

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Chapter One

Complaint of Insomnia Symptoms and Daytime Impairment

Acute insomnia: recent reconceptualisation

Acute insomnia is increasingly recognised as an important condition for treatment, particularly when it is recurrent and associated with daytime impairment and distress (Morin et al., 2009). For some, acute insomnia will develop into a chronic, complex and debilitating syndrome which is extremely challenging to treat effectively (Ellis, Gehrman, Espie, Riemann & Perlis, 2012). Despite this, the pathogenesis and aetiology of acute insomnia are unknown and our understanding of the factors involved in the development of chronic insomnia is poor. In 2005 the National Institute of Health highlighted the urgent need to profile the developmental stages of insomnia in the 'State of the Science' statement. Since then, modest but important progress has been made in targeted research and clinical recognition of acute insomnia. The work described in this thesis specifically addresses the need to better profile daytime cognitive impairment in those with (predominantly acute) 'insomnia symptoms', a term which encompasses sub-clinical levels of poor sleep quality and concern about daytime function. An improved understanding of associations between insomnia symptoms and daytime dysfunction should not only help to inform new treatment programmes for the complaint, but could also increase awareness of mechanisms involved in the vulnerability to further sleep problems, mental and physical ill-health (Benca, 2001).

The Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association, 2013) does not provide specific diagnostic criteria for acute insomnia, however, inferred diagnosis can be made from Insomnia Disorder, the only difference being the duration of complaint (Ellis et al., 2012; Morin, 2012). In Insomnia Disorder individuals report trouble falling asleep, maintaining sleep, early morning awakenings and/or feeling unrefreshed following sleep. This is accompanied by an appraisal that insomnia impacts negatively upon quality of life, causing significant distress within social, occupational or other personally important domains. The sub-clinical phases of insomnia are considered to be 'acute' (3-14 days), 'transient' (2-4 weeks) and 'subchronic' (1-3 months). There is debate around whether the number of nights per week of unsatisfactory sleep should be a criterion for treatment-worthiness in acute insomnia due to

the considerable night-to-night variability and periods of remission seen in recurrent cases. Nevertheless, consistent with DSM-5 defined chronic insomnia, three nights of unsatisfactory sleep per week are considered clinically important where sleep onset latency (SOL) and wake after sleep onset exceed 30 minutes (Ellis et al., 2012). Currently, there are no quantitative criteria for severity of insomnia, and clinicians and researchers rely upon well-established retrospective self-report measures in order to capture this aspect of the condition, e.g. the Insomnia Severity Index (ISI; Bastien, Valliéres & Morin, 2001).

There are conceptual differences between the DSM-5, the International Classification of Diseases 10th edition (World Health Organisation, 1992) and the International Classification of Sleep Disorders 2 (ICSD; American Academy of Sleep Medicine, 2005) classification systems for insomnia in relation to whether an identifiable trigger should be a requirement for diagnosis. Some experts provide counter-arguments referring to cases where patients are not aware of the causal factor in sleep disturbance (Morin, 2012; Bastien, Vallières & Morin, 2004). Indeed, acute insomnia could be the end result of cumulative stressful events over time (Ellis et al., 2012) and/or there may be delay between insomnia which is 'acceptable' to the individual and eventual help-seeking. The ICSD 2 is most restrictive in criteria for a diagnosis of acute insomnia, referring to the condition as 'adjustment insomnia'. Adjustment insomnia lasts up to three months, must be directly related to an identifiable stressor and must not involve behavioural responses or learned associations considered to be characteristic of chronic insomnia. In contrast to DSM-5 criteria, daytime dysfunction is directly related to the stressor rather than the sleep disturbance. This last criterion may be overly-restrictive given that the average person believes sleep to be important and it is not uncommon for healthy individuals to worry about their sleep routine and patterns (Espie, 2010).

DSM-5 changes for the classification of Insomnia Disorder reflect a conceptual shift from DSM-V logic which inherently suggested causal and directional influences of sleep disturbance and physical or psychological comorbidities. The term Insomnia Disorder is now applied without differentiation between primary and secondary insomnia, reflecting the relationships between insomnia, medical and psychiatric disorder as bidirectional and interactive (Reynolds & O'Hara, 2013). These changes are important for facilitating earlier detection of and intervention for significant sleep disturbance. Furthermore, additional guidelines provided by Ellis et al. (2012) provide a useful operational framework for standardising the current insomnia definitions. However, as emphasised by Morin (2012), these are yet to be validated formally and there are no established cut-offs for differing

criteria (e.g. duration, frequency, severity); furthermore sensitivity and specificity indices have not been explored. For this reason, it is important to investigate 'insomnia symptoms' in non-clinical populations to determine how aspects of poor sleep are represented and associated with daytime function, e.g using the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman & Kupfer, 1989) where a score greater than five identifies a 'poor sleeper'). In order to harmonise criteria across research studies it has been recommended that the term 'insomnia symptoms' is used to label any aspect of an insomnia complaint (i.e. difficulty initiating or maintaining sleep, early morning-awakenings), of non-specific duration and severity, which may be accompanied by daytime impairment (Lichstein, Durrence, Taylor, Bush & Riedel, 2003; Edinger et al., 2004). By implication, findings from research into insomnia symptoms are likely to be useful for understanding the transition from disturbed sleep to acute insomnia.

For most, nocturnal 'insomnia symptoms' (which could be regarded as 'poor sleep') are a natural response to stress and subside with cessation of the emotional impact of a stressor (Spielman, Caruso & Glovinsky, 1987). In such cases, intervention is largely regarded as unnecessary, (although sleep hygiene education and stress management have been shown to be proactive preventative measures even at this stage, e.g. Brown, Buboltz & Soper, 2002). However, in populations exposed to repeated episodes of poor sleep, or in those more vulnerable to the development of Insomnia Disorder, sleep may not return to normal. In such cases, additional concern about the daytime consequences of sleep loss indicates the potential for development of persistent insomnia (Spielman, Caruso & Glovinsky, 1987). It is therefore, important to investigate both poor sleep and insomnia symptoms in populations with regularly disrupted sleep in order to understand how daytime function is perceived and impaired. Indeed, dissatisfaction with daytime function is the main reason for help-seeking for insomnia, over and above complaints related to nocturnal sleep disturbances (Morin, LeBlanc, Daley, Gregoire & Merette, 2006).

Prevalence and comorbidities

Insomnia Disorder often begins in young adulthood (American Psychiatric Association, 2000), also a sensitive period for first onset of mood and anxiety disorders (Beesdo, Knappe, & Pine, 2009). It has a higher prevalence in females, where the risk is between one and a half and two times that in males (Léger, Guilleminault, Dreyfus, Delahaye & Paillard, 2000; Klink, Quan, Kaltenborn & Lebowitz, 1992). Whilst the most frequent trajectory for sleep disturbance measured over a three year period is persistent insomnia for both syndromal (meeting diagnostic criteria for Insomnia Disorder) and sub-

syndromal insomnia (symptoms of Insomnia Disorder), for those with sub-syndromal insomnia a period of remission is more likely than the development of chronic insomnia (Morin et al., 2009).

An estimated 20% to 35% of the general population experience 'insomnia symptoms', and 10% of cases reach clinical significance (Ohayon, 1997; Léger, Guilleminault, Dreyfus, Delahaye & Paillard, 2000; Ford & Kamerow, 1989; Weissman, Greenwald, Nino-Murcia & Dement, 1997; Ohayon, 2002). There is a reluctance to engage in help-seeking behaviour for early insomnia perhaps due to the belief that disturbed sleep is "benign, trivial, or a problem one should be able to cope with alone" (Stinson, Tang, & Harvey, 2006). However, when recognised as clinically important, insomnia is highly persistent, with 46% of sufferers reporting problems three years later (Morin et al., 2009). Health related quality of life decreases with severity of the sleep complaint, independent of anxiety and depression comorbidities (Ancoli-Israel & Roth, 1999; Léger, Scheuermaier, Phillip, Paillard & Guilleminault, 2001).

Insomnia is comorbid with an anxiety disorder in 32.5% of cases, and the likelihood of an insomnia diagnosis is increased four-fold in this group when compared to the healthy population (Roth et al., 2006). Ohayon and Roth (2003) in a large-scale cross-sectional study (*N*=14,915) reported insomnia as highly comorbid with panic disorder (61%) and generalised anxiety disorder (GAD) (44%), the latter finding being replicated in a large epidemiological study (Stewert et al., 2006). Insomnia is also highly comorbid with depression, and predicts subsequent depressive episodes when it persists for greater than two weeks (Buysse et al., 2008; Ohayon & Roth, 2003). In the Nord-Trøndelag Health Studies (HUNT) involving longitudinal assessment of and clinical data collection from 24, 715 individuals, insomnia at time one was associated with incident onset of anxiety, depression and help-seeking for mental disorder 11 years later, after adjusting for demographic variables and physical health conditions (Sivertson et al., 2014).

In a community based sample of 1041 adolescents, Johnson, Roth and Breslau (2006) provided persuasive evidence for insomnia having independent, and distinct directional associations with anxiety versus depression. In those with comorbid complaints, anxiety preceded insomnia in 73% of cases, whereas insomnia preceded 69% of cases of depression. Prior anxiety disorder was found to increase risk of insomnia by three and a half times compared to those without anxiety (adjusting for depression) whereas depression was not associated with risk for onset of insomnia. Prior insomnia was not associated with risk for onset of anxiety disorder, but increased risk of depression by more than three and a half

times that of those without insomnia. The suggestion, therefore, is that in young people anxiety can lead to Insomnia Disorder and Insomnia Disorder may then lead to depression. This profile of directional associations is consistent with previous epidemiological studies (e.g. Ohayon & Roth, 2003; Breslau, Roth, Rosenthal & Andreski, 1996), but it is also possible that with repeated and persistent insomnia, associations between depression, anxiety and insomnia become bi-directional (Johnson et al., 2006).

Importantly, when comorbid with anxiety or depression, insomnia is more often associated with distress and can facilitate remission of the other disorder when treated (Harvey, Tang, Browning, 2005, Franzen, 2008). Eaton, Badawi and Melton (1995) reported that 47% of incidents of depression at a one year follow up could have been prevented had sleep disorder been treated at baseline. Changes to the conceptualisation of insomnia in the DSM-5 reflect the importance of recognising insomnia as both an isolated distressing complaint and as a complaint which occurs with a wide range of psychiatric and physical disorders. This emphasises the importance of treatment for the complaint of insomnia, yet presents a challenge in how best to empirically investigate the effects of insomnia upon daytime function in cross-sectional research.

The consensus for research approaches to insomnia (across DSM-5, recommendations from the NIH and published guidelines from the American Academy of Sleep Medicine Working Group (Edinger et al., 2004)) is that insomnia should be treated as a general complaint without exclusions for other disorders which may be involved in its maintenance. However, in non-clinical populations it is likely that secondary analyses examining the uniqueness of association between poor sleep, insomnia symptoms and daytime dysfunction are likely to be valuable in addition to primary analyses. Given the proposed directional path between anxiety and insomnia (Johnson et al., 2006) it is particularly interesting to tease apart the contribution of anxiety and insomnia to observable deficits in non-clinical populations. This approach might help to explain how individuals are affected by poor-sleep and ultimately how poor-sleep exacerbates personal distress and increases the risk of mental ill health (e.g. depression).

Cognitive impairment is well established in both anxiety and depression (Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari & Lönnqvist, 2008), but little is known about whether and how poor sleep and insomnia symptoms impact cognition, independently of these complaints in otherwise healthy populations. Preliminary evidence suggests the importance of examining cognitive function associated with poor-sleep. Nebes, Buysse, Halligan, Houck, and Monk (2009) using the PSQI in conjunction with a battery of

attention and executive performance tasks, found that in community based healthy older-adults, individuals classified as poor sleepers showed worsened cognitive function. The association between poor sleep quality and executive functions was maintained, after controlling for depressive symptoms. Benitez and Gunstad (2012) replicated these observations in a sample of young adults, where diminished attention and executive functions were independently associated with PSQI defined poor sleep. Specifically, subjective sleep quality, sleep duration, and sleep medication use predicted cognitive performance when controlling for emotional reactivity and demoralisation (constructs common to both anxiety and depression). These studies also highlight the concurrent validity of the PSQI for examining cognitive dysfunction in poor-sleeping healthy adults. As emphasised in Benitez and Gunstad (2012), if healthy individuals endorse poor sleep on the PSQI this may be an important indicator of the need for further psychological assessment or treatment.

When investigating insomnia symptoms (i.e. not reaching diagnostic criteria for insomnia) in healthy populations, self-report measures, such as the PSQI and the ISI, are endorsed as 'best-practise' methods of assessment (Buysse, Ancoli-Israel, Edinger, Lichstein & Morin, 2006). Given that insomnia symptoms are heterogenous, a multifaceted approach is optimal (Morin, 2003), e.g. combined usage of a questionnaire designed to measure more objective aspects of sleep quality (e.g. duration, disturbance, latency) and a questionnaire assessing both nocturnal and daytime cognitive aspects of insomnia (e.g. distress, perceived impaired functioning). Self-assessment is not only more practical and efficient for large samples but is also considered to be most sensitive to the nature of a complaint which is diagnosed on the basis of subjective symptomatology. Indeed, objective measurement of sleep parameters have been considered inappropriate in the assessment of insomnia (Kushida, Littner & Morganthaler, et al, 2005).

Characteristics of poor sleep and insomnia symptoms in young adults

Due to the divergent range of measures used to assess sleep, and the limited number of published studies in young adulthood (18-25 college and early career age), it is difficult to make reliable estimates of the prevalence of poor sleep and insomnia symptoms in this population. Indeed, estimates of insomnia have ranged from 8.7% to 69% (Bramoweth & Taylor, 2012; Brown, Soper & Buboltz, 2001; Sing & Wong, 2011; Taylor et al., 2011). Reporting insomnia symptoms, one study found that 15% of US college students experience poor sleep quality, 12-13% with problems with SOL, early morning awakenings or disrupted sleep at least three times a week. In Taylor et al. (2011) 9.4% of US students

reported problems initiating or maintaining sleep for three or more nights per week for at least six months. Although these studies used DSM-5 criteria associated with acute and chronic insomnia, they did not assess perceived daytime impairment. In Bramoweth and Taylor (2012) 8.7% of students reported symptom of chronic insomnia, accompanied by daytime impairment, for at least six months.

In Lund et al. (2010) 60% of 1,125 university students were poor sleepers as defined by a PSQI score greater than five. Tension and stress accounted for the most variance in PSQI scores and academic stress and emotional upheaval were overwhelmingly reported as the cause for sleep problems. Twenty percent of students reported stress interfering with sleep at least once a week, e.g. "stress about college" "racing thoughts" "worry about the future." This accounted for 24% of variance in PSQI scores. Academic stress was identified as the most common reason for poor-sleep, accounting for 39% of answers, followed by emotional causes (25%). Environmental factors such as lights/noise, illness and pain, and co-sleeping accounted for 17%, 8% and 4% of responses respectively. The authors reported 'chronically restricted sleep' in this population where 38% had PSQI scores over seven, the average student slept for 7.02 hours (25% less than 6.5 hours) and only 29.4% reported eight or more hours sleep as recommended (Ferrara & De Genaro, 2014). The average weekday rise time was 8.02am (10 am weekend days), and the average bedtime was 1.44am. The primary contributors to PSQI global scores were reduced total sleep time and an inability to fall asleep within 30 minutes.

Of note, in several studies of poor sleep in young adults the average total sleep time does not seem markedly lower than recommended (e.g. Lund et al., 2010; Medeiros, Mendes, Lima & Araujo, 2003). This could reflect difficulty in discerning sleep time from 'time in bed,' the importance of other sleep variables in this population (e.g. disturbance, SOL, efficiency, daytime distress), and/or that modest increases in sleep duration over time (e.g. 30 mins) may have a profound effect upon daytime function (Lack, 2010). Furthermore, measures that do not discriminate between weekday and weekend hours of sleep (e.g. PSQI) may output an average total sleep time which disguises the large discrepancy between these two time points which contributes to poor sleep in this population (e.g. Trockel, Barnes & Egget, 2000).

Most recently, Taylor, Bramoweth, Grieser, Tatum and Roane (2013) using prospective sleep diaries and retrospective self-report measures for assessment of insomnia, reported the following prevalence of sleep profiles in a sample of 1, 1039 college students assessed against DSM-5 criteria: 57.1% were good sleepers, 9.5% had chronic insomnia,

6.5% reported an insomnia complaint but did not meet severity, frequency *and* duration criteria, and 26.9% met severity, frequency and duration criteria but did not report insomnia. The average duration of complaint was 3.34 years. This profile of insomnia in young adults parallels that observed in the general population (e.g. Ohayon & Roth, 2001; Lichstein, Durrence, Riedel, Taylor & Bush 2004), and highlights that persistent sleep trouble is a feature of this group. Chronic insomnia was also associated with significantly elevated levels of anxiety, depression and stress, and lower quality of life ratings. Collectively, these studies highlight that insomnia is as important a complaint in young adults as it is the general population and should not be trivialised due to lifestyle factors.

Taylor et al. (2013) note a large proportion of students who did not link their daytime symptoms with insomnia. This finding is consistent with a previous study where students did not rate their subjective sleepiness and cognitive dysfunction as highly as expected, despite convincing objective evidence for sleep related daytime impairment (Curcio, Ferrara & De Gennaro, 2006). Given the environmental and social factors disrupting sleep at this time, it is possible that individuals are unaware of the link between their sleep and daytime performance, attributing daytime impairment to other factors or considering poor sleep as 'par for the course' of college life and something that will resolve in time. A lack of effective health education in school and college about the importance of good sleep patterns may feed into the low awareness that severe sleep disturbance could result in mental health problems worthy of intervention (Gallasch & Gradisar, 2007). On the other hand, given that only 14 minutes differentiated those with insomnia from those without in Taylor et al. (2013), it is possible that attributing poor-sleep to college life rather than an inability to initiate sleep may protect individuals against an escalating psychological problem with sleep (Taylor et al., 2013).

One study investigated coping strategies of 845 first year college students reporting PSQI defined sleep problems (57% insufficient sleep, 14.4% insomnia symptoms defined as difficulty initiating and maintaining sleep, 8.9% sleepiness/fatigue, 8.5% erratic sleep schedule, 5.5% poor sleep quality). Interestingly, participants with insomnia symptoms and poor sleep quality endorsed 'I have no means of coping' and 'sleep-promoting activities' most highly, whereas those with insufficient sleep and sleepiness endorsed napping as the most effective coping strategy. Furthermore, 'ignore the problem' was highly endorsed across all groups. Increased exposure to sleep interfering events compounded by ineffective coping (e.g. the tendency to 'internalise' emotional conflict through inhibition, denial, and repression of difficulties during the day; Kales, Caldwell, Soldatos, Bixler &

Kales, 1983) puts this population at risk for persistent sleep trouble. Extended rise times, going to bed early, napping, problem-solving in bed, worrying and ruminating can all further interfere with sleep by sensitising individuals to their wakefulness (Harvey, 2002). Behaviours such as watching TV and eating are incompatible with sleep and may become associated with arousal and frustration around sleeplessness (Stimulus Control Theory; Bootzin & Nicassio, 1978). Sleep disturbance in a student population, therefore, is likely to go beyond a simple stress-response, is detrimental, involves a cognitive component and can elicit maladaptive coping skills.

It has been demonstrated that poor sleep and insomnia symptoms in young adults are important indicators of vulnerability to Insomnia Disorder. Students may be particularly at risk due to the high-levels of habitual sleep disturbance (e.g. restricted sleep opportunity and cumulative sleep loss) and sleep problems attributed to perceived 'stress', the major reason for development of insomnia (Roth & Drake, 2004). Longitudinal studies comparing degrees of life change in insomniacs and controls showed that over a five year period, increased numbers of stressful events, environmental changes, and illness are important in precipitating insomnia (Healey, Kales & Monroe, 1981). However, in this population stress may be predisposing, precipitating or perpetuating in insomnia (an in-depth review of developmental models of insomnia is found between p.12 and p.18). Lund, Reider, Whiting and Prichard (2010) explore one potential developmental profile of insomnia. Stress-related events associated with college lifestyle, e.g. erratic schedules and high-pressure exam periods, initially disrupt sleep. This may load onto an already hyperaroused state resulting from maturational changes in the neuroendocrine system, e.g. HPA axis alterations which originate from adolescence where increased perisleep onset cortisol secretions are promoted (Forbes et al., 2006). Finally, with significant changes in lifestyle and a new independence, young adults may lack sufficient coping skills to manage levels of stress and may internalise thoughts and feelings, e.g. rumination and worry (Jose & Ratcliffe, 2004).

Young adulthood involves novelty and change in lifestyle; leaving home, independence, academic pressures at university, occupational challenges in the workplace, new social situations, and exposure to alcohol and drugs (Taylor, et al., 2013). These life changes can be dramatic and individuals cope differently with the challenges they are faced with; those who are vulnerable to poor sleep may find this period particularly disruptive (Kales & Kales, 1984). In light of these changes it is not surprising that young people with poor sleep often self-medicate in order to relieve psychological distress associated with insomnia (Stasio, Curry, Sutton-Skinner & Glassman, 2008). Increased alcohol consumption

and over the counter hypnotics are common methods for dealing with disrupted sleep-wake schedules (Lund et al., 2010). Haario, Rahkonen, Laaksonen, Lahelma and Lalluka (2013) reported bidirectional relationships between alcohol consumption and insomnia such that insomnia at baseline predicted heavy drinking and physical inactivity five years later (adjusting for age, gender, drink at baseline, marital status, occupational class, sleep duration and mental health). Heavy drinking and binge drinking at time one predicted subsequent insomnia symptoms.

An important aspect of sleep in young adults is the influence of a desynchronised sleep-wake cycle. The circadian pacemaker which regulates the sleep-wake cycle can be disrupted by work, study and social schedules which interfere with normal exposure to light and dark and social contact (Medeiros, et al., 2003). This can lead to a delayed sleep onset which in turn can lead to a shorter sleep duration if rise time is early (Lack, 2010). Prolonged sleep at weekends is considered largely due to a reduction in sleep length during the week, whereas the delay of bedtime is due to a tendency of the human circadian system to maintain a delayed phase (Valdez, Ramirez & Garcia, 1996). It is therefore, common practise to measure the influence of 'morningness-eveningness' in sleep disturbed populations (Horne & Östberg, 1976). A tendency towards 'eveningness' is predicative of poor sleep (e.g. accounting for 2% of variance in PSQI score; Lund et al., 2010) and correlates reliably with measures of poor sleep quality (Yang, Wu, Hsieh, Liu, & Lu, 2010).

There is sound evidence for the importance of investigating insomnia symptoms *and* poor sleep in young adult (student) populations. Previous work has focussed primarily upon insomnia and its relationship to mental health in young people, where mental health complaints have provided an index of daytime impairment. Very few studies, however, have measured both daytime and nighttime aspects of poor sleep and insomnia symptoms, and further investigated associations with objective daytime performance.

Daytime consequences of insomnia

Daytime impairment in insomnia is reported across all important domains of functioning, e.g. cognition (attention, concentration, memory), mood (dysphoria and irritability), daytime sleepiness, reduced motivation, energy and initiative, accident and error proneness and somatic complaints (Fortier-Brochu, Beaulieu-Bonneau, Ivers & Morin, 2012). Social relationships, occupational function and academic performance are affected (American Psychiatric Association, 2000; American Academy of Sleep Medicine, 2005) and quality of life is significantly lower than for healthy controls (Léger et al., 2001). For some individuals the most debilitating aspect may be emotional dysregulation, whereas for others

it may be the effect upon cognition (Jansson & Linton, 2007). Despite the higher prevalence of Insomnia Disorder in females, they are no more likely than males to experience daytime cognitive impairment associated with the sleep complaint (Léger, Partinen & Hirschkowitz, Chokroverty, Touchette & Hedner, 2010).

Research shows that college students who have insomnia symptoms have longer reaction times and poorer accuracy on tests of vigilance, lower grades, higher levels of daytime sleepiness, fatigue, worry, mental health complaints and a higher risk for traffic accidents (Lindsay, Hanks, Hurley, & Dane, 1999; Means, Lichstein, Epperson, & Johnson, 2000; Taub & Berger, 1978; Taylor et al., 2011; Trockel, Barnes, & Egget, 2000).

In the workplace, accidents are between 2.5 and 4.5 times more likely to be caused by those suffering from insomnia (Balter & Uhlenhuth, 1992; National Sleep Foundation, 1991). Cross-sectional studies estimate that the overall economic cost of insomnia in the UK is equivalent to two billion pounds per year, with 76% of insomnia annual expenditure attributable to loss of productivity at 27.5 days (Daley, Morin, LeBlanc, Gregoire & Savard, 2009). A large proportion of 'direct' costs involve use of alcohol as self-management of insomnia (58%) and time off for consultations (33%). Driving is also impaired in those with insufficient sleep, which increases the risk of falling asleep momentarily at the wheel and of road traffic accidents (Pack, Pack, Rodgman, Cucchiara, Dinges & Schwab, 1995; Knipling & Wang, 1994; Carskadon, 1990). Psychomotor deficits during driving are comparable to the deficits seen at or above the legal alcohol limit (Dawson & Reid, 1997). Daytime sleepiness is largely responsible for the low-level lapses in attention which cause work errors and accidents, and is considered to reflect underlying sleep debt or insufficient sleep duration (Lim & Dinges, 2010).

Several reviews have confirmed that academic achievement is dependent upon sleep patterns and associated daytime impairment (Samkoff & Jacques, 1991; Fallone, Owens & Deane, 2002; Wolfson & Carskadon, 2003). Later rise times, delayed sleep onset, sleep irregularity and reduced sleep length have predicted poorer exam performance and grade outcome (Trockel et al., 2010; Medeiros et al., 2003). There is a paucity of research, however, investigating the mechanisms which underlie these complaints, especially in young adults. Whilst it has been established that memory and learning are severely impaired (see Curcio, Ferraro & Gennaro, 2006) for review of rapid eye movement (REM) sleep and non-rapid eye movement sleep (NREM) functions in procedural and declarative memory) research also suggests that attention may be fundamentally and adversely affected in insomnia symptoms, i.e. 'readiness to engage' in the processing of relevant stimuli and the

ability to 'maintain focus' upon stimuli is weakened (Jackson, et al., 2013). This will inevitably affect learning of new material.

It is less clear whether executive performance is impaired in insomnia. Converging positron emission tomography (PET) and electroencephalogram (EEG) evidence implicates the prefrontal cortex as particularly sensitive to sleep deprivation supporting the rationale for a selective deficit in this area (review of the relevant literature can be found in a later chapter) (e.g. Finelli, Baumann, Borbély, & Achermann, 2000). Furthermore, tasks which require switching of attention and multiple active cognitive sets most reliably reveal impairment in those with insomnia and are most consistent with the nature of impairment reported by sufferers (Shekleton et al., 2014). There is a current debate whether the executive control deficits observed using certain neuropsychological tasks are true or whether non-executive dysfunction is driving the 'overall' measure of executive performance, i.e. the intrinsic properties of the tasks prevent dissociation between levels of processing (Jackson et al., 2013). Another perspective is that executive performance is associated with more subjective aspects of sleep (e.g. perceived sleep quality) rather than with more objective indices of sleep deprivation (e.g. duration, latency) (Benitez & Gunstad, 2012). By implication, this would suggest that non-executive cognitive impairment (e.g. attentional preparedness and vigilance) is most strongly associated with poor sleep but those with sleep-related anxieties/concerns may also show executive performance impairment more characteristic of anxiety disorders.

Models relevant to acute insomnia

To date, a model specifically relevant to our understanding of daytime dysfunction associated with acute insomnia has not been developed. Indeed, there has been little attempt to validate the daytime complaints of poor sleepers and to identify key processes which serve to escalate sleeplessness into a syndrome. This is surprising given the difficult task of revealing daytime dysfunction in clinical samples where compensatory strategies, comorbid mood and anxiety disorder, and hyperarousal disguise underlying deficits associated with sleep (Schmidt et al., 2010). Very little research has been conducted in populations where naturally disturbed sleep is a regular occurrence and is not associated with clinical confounds, e.g. student populations, new mothers, military personnel.

Cognitive models of the maintenance of chronic insomnia have proved successful in furthering our understanding of key disorder processes and we rely on such models in order to identify important variables in the investigation of daytime consequences of acute

insomnia symptoms. Due to the current lack of research in this area, Ellis et al. (2012) reports

"there is no reason to suppose that during the acute form of the disorder the individual is or is not focused more on the occurrence of the insomnia versus the precipitant. Similarly, there is no reason to assume differences between the acute and chronic phases in terms of frequency (p.8)."

However there are several key differences between these forms of sleep complaint. Firstly, acute insomnia is most often a direct consequence of life stress (internal or external) (Ellis et al., 2012), whereas chronic insomnia is maintained by maladaptive coping strategies, sleep-focused information processing and other dysfunctional cognitive responses to persistent sleep trouble, unrelated to the initiating event (Harvey, 2002). Secondly, acute insomnia may be more stably underpinned by severe sleep debt, potentially greater in severity than in chronic insomnia (Ellis et al., 2012) where individuals often overestimate their sleep disturbance when compared to objective measures (Dorsey & Bootzin, 1997). Daytime sleepiness related to acute insomnia can be more profound than in chronic insomnia, and insomnia with short sleep duration is the most severe insomnia phenotype strongly connected with adverse health outcomes (Vgontzas & Fernandez-Mendoza, 2013). Caution must be taken when developing hypotheses in relation to acute insomnia because the phenomena of dysfunctional thinking, heightened sleep-related worry, cortical hyperarousal, sleep misperception, automaticity and attentional bias to sleep stimuli which are found in chronic insomnia (Espie, 2002) cannot necessarily be assumed to be features of acute insomnia. Nevertheless, there are several important human models which directly hypothesise about the course of acute insomnia, and inferences can also be drawn from popular models of chronic insomnia.

Current models of insomnia have much in common when considering the processes important to disorder development and maintenance. The main differences are whether insomnia is underpinned by cognitive, physiological or emotional hyperarousal and whether the focus is the individual's general level of arousal or their arousal response to certain conditions (Lundh & Broman, 2000). In the following sections we discuss models relating to both insomnia development and maintenance in order to understand how daytime dysfunction may be characterised in acute insomnia (and insomnia symptoms). An important consideration across all models is that is it very difficult to determine whether a variable is predisposing for insomnia, involved in the disorder development, or is fundamental to disorder maintenance. Indeed, variables may be more or less influential at

any stage of a sleep complaint and the association between variables may be bi-directional (Ellis et al., 2012).

The '3P' model of the natural history of insomnia.

Spielman and colleagues' 1986/1987 model of the natural history of chronic insomnia remains highly relevant today and has been supported by high efficacy of chronic insomnia therapies derived from it (Ellis et al., 2012). In the 'Three P' model (see *Figure 1.1*), *predisposing* factors which are primarily biopsychosocial (e.g. higher trait anxiety, a less robust sleep-wake cycle) determine the threshold for which an individual will experience acute insomnia when a life-stressor/threat, known as a *precipitating* factor, occurs (e.g. exam stress, divorce, illness). The accumulated sleep debt which characterises acute insomnia cannot be easily maintained due to the homeostatic drive for sleep. Therefore, most individuals recover normal sleep within a few days to weeks. However, in some cases short-term insomnia will develop where the sleep complaint starts to involve not only residual distress from the initiating event but also additional distress involving maladaptive coping strategies and sleep-focussed concern (eventually these *perpetuating* factors alone maintain chronic insomnia).

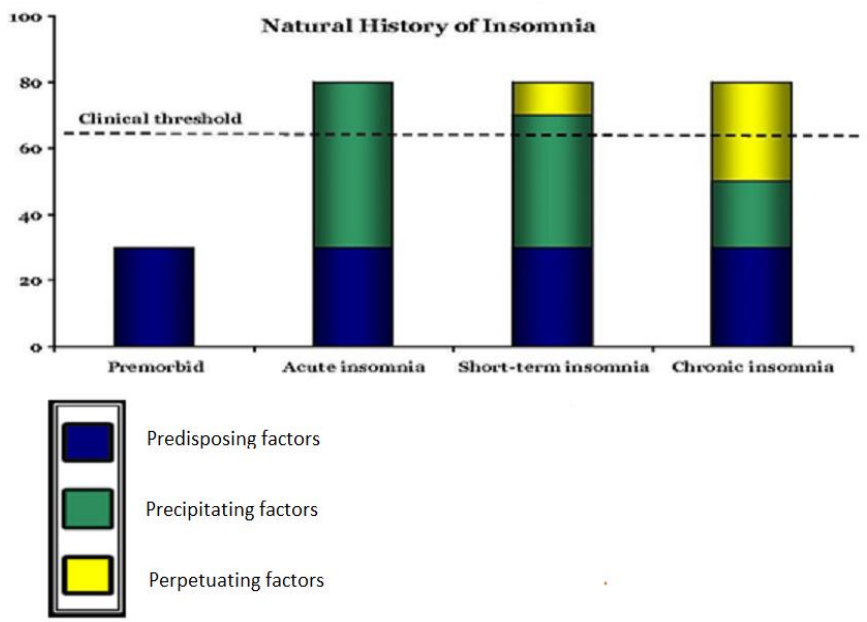

Figure 1.1 The Natural History of Insomnia (adapted from Spielman & Glovinsky, 1991).

Because acute insomnia has received very limited research attention, current knowledge of predisposing and precipitating factors in this condition is limited. However,

as previously described (p.7), Lund et al. (2010) apply this model to provide a highly plausible model for insomnia development in young adults.

'Sleep reactivity' and susceptibility to insomnia.

An encouraging programme of research initiated by Drake, Richardson, Roehrs, Scofield and Roth (2003) investigates individual responsivity to stress and the impact this has upon sleep. Conceptualised most recently in a psycho-bio-behavioural model of vulnerability to insomnia (Harvey, Gehrman & Espie, 2014; *Figure 1.2*), this theory explains how some individuals (i.e. those with neuroticism trait and/or the related 5HTTLPR genetic polymorphism) are predisposed to respond to stress with increased sympathetic nervous system activation. This arousal then leads to increased stress reactivity which causes vulnerability to insomnia.

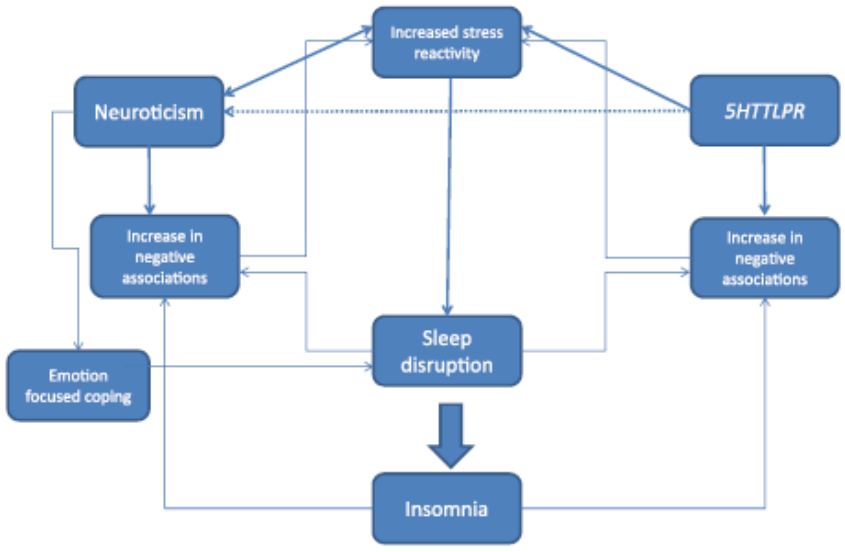

Figure 1.2 Psycho-bio-behavioural model of vulnerability to insomnia (Harvey, Gehrman & Espie, 2014).

These hypotheses are consistent with the 'sleep interfering processes' proposed in Lundh and Broman (2000) where individuals with insomnia are considered to have a basic elevated arousal level which can be life-long, genetic or induced (e.g. those with a chronic tendency to worry and ruminate, consistent with high trait anxiety). The authors further propose that individuals with a proneness to insomnia may have increased arousability *in response* to stimuli. For example, Coren (1988) reported that trait-like arousability was predictive of several key indices of sleep disturbance using the Arousability Predisposition Scale containing items relating to arousability (e.g. "I startle easily') and items relating to slow habituation to stimuli (e.g. "I find that my heart keeps beating fast for a while after I

have been 'stirred up' "). The frequency and intensity of arousing events can be particularly problematic for those who have a lower threshold for arousability.

Central to both the psycho-bio-behavioural model and Lundh and Broman's sleep-interfering processes is the interaction between cognitive *and* physiological arousal where the two phenomena are considered manifestations of the same underlying problem. That is, the level of arousal elicited by a situation/stressor is the consequence of both an individual's basic physiology and their psychological response. The cognitive consequences of stress reactivity can be measured as rumination or worry, and its physiological correlate can be assessed using autonomic measures which reflect the activity of the HPA axis. Whereas the phenomenon of 'hyperarousal' (both autonomic and cognitive) in chronic insomnia has been extensively investigated (Riemann, 2010), research has not investigated whether increased autonomic responding is a feature of acute insomnia. If parallel processes operate during the day and night to maintain sleeplessness then increased autonomic reactivity (both at baseline and in response to stimuli) may be an aspect of daytime impairment in this complaint.

Work by Ellis et al. (2014), provides detail on *how* stress reactivity results in sleep disruption. They propose that acute insomnia can be triggered by stress related to any event that causes a reduction in quality of life or distress at a current situation. A significant life event, an accumulation of daily hassles or a chronic, persistent stressor causes increased stress reactivity resulting in insomnia. This is due to the perception of having inadequate resources to cope or due to a loss of actual resources necessary for effective stress management. Stress reactivity, therefore, is likely to be an important aspect of insomnia symptoms: increasing the likelihood of repeated insomnia episodes which in turn increase arousal.

Harvey's cognitive model of insomnia maintenance.

Harvey's cognitive model of the maintenance of insomnia (2002; see *Figure 1.3*) has been embraced both in research and clinical practice, and continues to inspire novel and testable predictions, particularly those relating to daytime dysfunction. Ellis et al. (2012) acknowledge that although this model informs us primarily about processes involved in chronic insomnia, it is important because the factors involved in the maintenance of insomnia could also be predisposing factors for the onset of acute insomnia episodes. The strength of this model is its ability to account for why some individuals do not spontaneously recover from insomnia. Harvey acknowledges the work of Clark (1999) in developing this model, because several of the processes identified and validated as

preventing self-correction of anxiety are also important in insomnia, which is consistent with the high comorbidity between these conditions.

This model takes excessive, negatively toned cognitive activity as responsible for the maintenance of insomnia. Whilst this model states that such activity is primarily characterised by sleep-related thoughts (both during the nighttime *and* daytime) in chronic insomnia, it is conceivable that for individuals with acute insomnia this cognitive activity involves more general negative intrusive thought. These thoughts in turn cause autonomic arousal and dysregulation to emotion processing, and this effectively primes a state of anxiety. Once this is established, it becomes easy for dysfunctional cognitive and behavioural processes to operate. Indeed, attentional narrowing and selective attention to threat is a feature of anxiety (Dalgleish & Watts, 1990) and individuals begin to preferentially process and assimilate information in line with their current fears/concerns. Individuals then sub-consciously accumulate evidence (in the form of night-time and day-time experiences) to confirm a feared 'inability to sleep' and the detrimental effects upon daytime function. In acute insomnia, it is conceivable that individuals experience more profound negative information processing biases, which cause them to interpret even ambiguous, neutral information in a threat-related manner, regardless of whether the information concerns sleep or not.

Harvey stipulates that the accumulation of sleep-related information believed to confirm concerns of total sleeplessness causes a distorted perception of the sleep-problem, where individuals overestimate their deficit in sleep and daytime performance. In relation to acute insomnia, however, an extensive discrepancy between subjective and objective measures of sleep is unlikely. Individuals with acute insomnia, by definition, remit within a month which suggests that dysfunctional cognitive processes may not be such a salient feature of this complaint, although this has not been empirically investigated to date.

Importantly, the model also considers the role of precipitating/perpetuating factors which may be relevant to acute insomnia experience. In particular, dysfunctional beliefs about sleep are emphasised, e.g. that one must have eight hours of sleep in order to function well the next day or if one night sleep is disturbed that it is essential to recover the lost sleep at the next opportunity. These beliefs mean that individuals are less likely to accept alternative explanations for their daytime experiences, e.g. interpreting yawning as a sign of sleep debt rather than the natural slump in circadian rhythm. These sleep-focussed cognitive biases add conviction to the concern that individuals are unable to sleep. Individuals may also have existing tendencies to worry, catastrophize, or may experience

general health hypochondriasis, in which lost sleep is a significant event and worry is considered by sufferers as beneficial, e.g. problem solving. Individuals may demonstrate safety behaviours, or maladaptive coping strategies (e.g. avoiding social events, missing work) which prevent them from managing their symptoms, learning skills to cope, and ultimately recognising their ability to perform in spite of sleeplessness. Avoidance behaviours in particular may cause further symptoms because low mood may follow the cessation of previously pleasurable and goal-fulfilling activities.

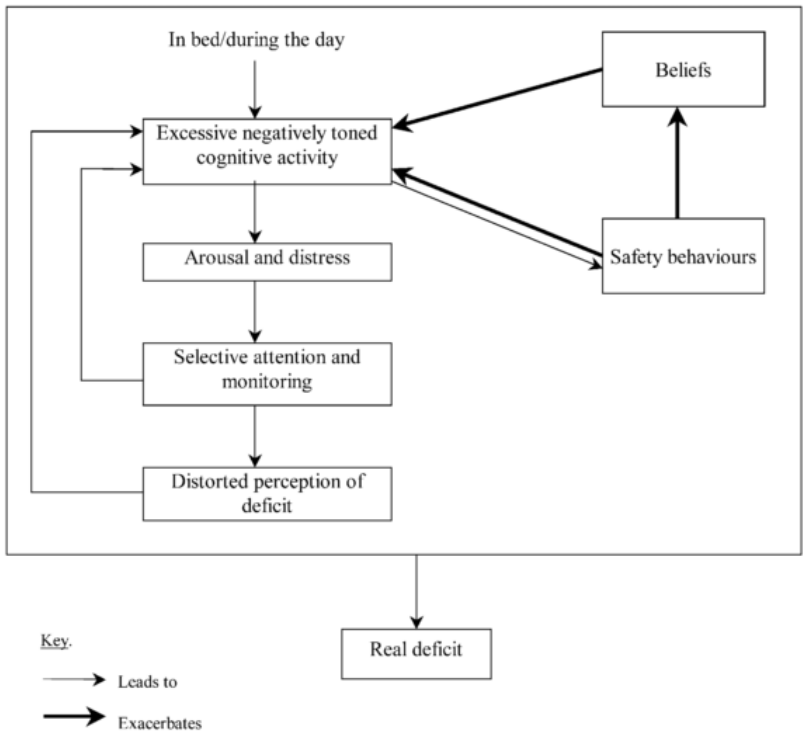

*Figure 1.3***.** Cognitive model of the maintenance of insomnia (Harvey, 2002).

Having reviewed several important models of insomnia it is clear that there is limited knowledge about how daytime dysfunction is characterised in insomnia symptoms and we are largely limited to models of chronic insomnia to inform predictions. The psycho-bio-behavioural model is an explanatory model of how acute insomnia develops but does not predict how daytime impairment is characterised. Despite this, this model highlights worry and over-arousal as predisposing factors for insomnia which may also be daytime impairments characteristic of those with insomnia symptoms. Although models of chronic insomnia are primarily concerned with cognitive-behavioural responses to sleep loss and emotional aspects of the disorder, it is clear that attentional dysregulation and autonomic arousal are core processes facilitating these responses. As such, these aspects of insomnia may be most relevant in early stages of disorder, escalating sleeplessness and

setting the foundations for more psychologically distressing features of the disorder. Very little work has been conducted into cognitive/physiological processes involved in acute insomnia and we hypothesise more general and fundamental dysregulation than has been highlighted in models of chronic insomnia (where sleep-related bias is emphasised). Indeed, the psycho-bio-behavioural model suggests that insomnia associated with the stress response leads to more generalised functional impairment.

Treatment approaches to poor sleep and insomnia

The importance of understanding the similarities and differences in daytime dysfunction between acute insomnia versus chronic insomnia is highlighted by a notable lack of early, preventative treatment for those experiencing acute symptoms. Very recently a pilot randomised control trial of the effects of Cognitive Behavioural Therapy for Insomnia (CBT-I) on sleep and daytime functioning was conducted in college students with DSM-5 defined Insomnia Disorder (Taylor et al., 2014). It was reported that sleep efficiency (defined as percentage of time in bed spent asleep), sleep latency, night time awakenings, sleep quality, (PSQI) insomnia severity (Insomnia Severity Index), dysfunctional beliefs about sleep, daytime fatigue and global sleep quality were all significantly improved following treatment (and durable at three month follow up). Total sleep time and time spent in bed after awakening showed no improvement between pre and post treatment. Change in nocturnal measures was supported using actigraphy. Daytime impairment measures included measures of anxiety, fatigue, sleepiness, mood and quality of life. Differences were not reported between the waitlist control group and the CBT-I group on daytime measures (except fatigue) which the authors suggest is due to limited statistical power (medium effect sizes found for all measures). In this study, CBT-I comprised stimulus-control, sleep hygiene, relaxation training and cognitive restructuring. Importantly, CBT-I was demonstrated as an effective treatment in college students with Insomnia Disorder and that treatment responses are similar to that in the general population.

With regard to individuals with insomnia symptoms, it is not routine practise to provide early intervention. This may reflect a lack of understanding of how daytime function is subjectively perceived and impaired in this complaint. The importance of providing education programmes and interventions in this population has been recognised. This approach is likely to have longer-term effectiveness than pharmacologic treatments which are often prescribed during acute insomnia episodes (Morin, Culbert & Schwartz, 1994). However, recent campaigns in college populations have focused primarily upon nocturnal aspects of sleep, neglecting management of daytime impairment (e.g. Orzech,

Salafsky & Hamilton, 2011). It is imperative to understand whether more general, daytime attentional dysregulation, executive control impairment (e.g. risky decision making) and autonomic reactivity are characteristic of those with insomnia symptoms. This way, treatment approaches for acute insomnia may be adapted to provide early intervention techniques, e.g. promoting attentional control and effective coping skills for the (physiological and cognitive) consequences of stressful life events.

Sleep deprivation studies: dysregulation within neural circuitry

As previously discussed, acute insomnia is likely to be characterised by significant underlying sleep loss and, therefore, it is appropriate to look to sleep deprivation studies for an understanding of the effects upon cognition. Attention is likely to be particularly vulnerable to disruption given anxiety associated with the stress-response, its key role in maintaining chronic insomnia, and in light of convergent neuroimaging studies. The amygdala and prefrontal cortex (PFC) have been implicated as brain regions particularly affected following sleep deprivation (Yoo, Gujar, Hu, Joelsz & Walker, 2007). These regions have been well-established within the anxiety literature, playing a specific role in executive functions such as attentional control and in the regulation of emotion (Bishop, 2009).

Yoo et al. (2007) revealed that in young healthy adults, sleep deprivation for 35 hours was associated with significantly greater amygdala activation in response to negative aversive stimuli (versus neutral stimuli) when compared to controls. Specifically, sleep deprivation increased amygdala activation by 60%, and increased expressed volume three-fold in response to aversive pictures. The control group showed significantly stronger connectivity between the PFC and amygdala than the sleep deprived group, which is suggestive of greater stability in affective processing given that depressive symptoms result from dysfunction in this area (Davidson, 2002). Interestingly, sleep deprived participants showed greater amygdala connectivity with autonomic activating centres of the brain stem. Together, these findings suggest that sleep deprivation is associated with a failure of top-down control mechanisms to moderate the sub-cortical, heightened response to threat. Identification of this neural network has led to many studies investigating how cognitive processes are affected in sleep deprivation (several of these are discussed in this thesis).

Partial sleep deprivation studies: useful for investigating insomnia symptoms?

Although far more studies have looked at neurocognitive performance associated with total sleep deprivation (≤ 45 hours), studies of partial sleep deprivation could more closely approximate the nature of acute insomnia (Goel et al., 2009). Killgore (2010)

confirmed amygdala-prefrontal dysregulation following chronic partial sleep deprivation, suggesting that the same network is affected by sleep restriction but to a lesser extent. Goel, Rao, Durmer and Dinges (2009) reported the growing consensus that four or more days of partial sleep deprivation (<7h per night) causes cumulative negative effects upon neurobehavioural function (Van Dongen, Maislin, Mullington & Dinges, 2003; Drake, Roehrs, Burduvali, Bonahoom, Rosekind & Roth, 2001; Dinges et al., 1997; Belenky et al., 2003) and repeated nights of between three and six hours sleep results in increased daytime sleep propensity (Drake et al., 2001; Carskadon & Dement, 1981).

In a well- controlled dose-response study cognitive performance (psychomotor vigilance, working memory, cognitive 'throughput') following total sleep deprivation was compared to that following chronic partial sleep deprivation where participants were allowed four or six hours per night for two weeks (a control group slept for eight hours) (Van Dongen et al., 2003). Experimentally induced partial sleep deprivation may closely reflect the type of sleep restriction experienced by students where the opportunity to obtain optimal sleep over extended periods is limited by social, environmental and work-related pressures. Participants were tested every two hours between 07.30 and 23.30. Two weeks of four hours sleep resulted in cognitive performance equivalent to two nights of total sleep deprivation and two weeks of six hours sleep was comparable to one night of total sleep deprivation. Control participants did not show observable cognitive deficits. Interestingly, despite linear, cumulative cognitive deficits in partial sleep deprivation, subjective ratings of sleepiness did not increase to the same extent, suggesting a discrepancy between objective measures of performance and subjective feelings of sleepiness. Given that total sleep deprivation results in the quickest and most profound cognitive impairment, when compared to the same amount of sleep lost over days (Van Dongen et al., 2003; Drake et al., 2001), it has been argued that individuals habituate to partial sleep deprivation (Drake et al., 2001). However another argument is that daytime cognitive impairments are a consequence of the accumulated time spent awake over and above usual wakefulness period (Van Dongen et al., 2003). In a sample of 48 adults (21-38 years) Van Dongen et al. (2003) estimated that 15.84 hours of wakefulness (and associated sleep period equal to 8.16 hours) is the outer boundary for preventing cognitive impairment.

Interestingly, a meta-analysis reported by Pilcher and Huffcutt (1996) concluded that partial sleep deprivation resulted in greater cognitive dysfunction than did total sleep deprivation. Following partial sleep deprivation, performance fell two standard deviations below control performance, whereas total sleep deprivation fell one standard deviation

below: however, the range of tasks included in this analysis could mean that more sensitive measures were employed in the partial sleep deprivation studies (Goel et al., 2009). Goel et al. (2009) provided a detailed review of the partial sleep deprivation literature and concluded that receiving under seven hours sleep over a prolonged period results in significant detrimental effects to cognitive performance.

When investigating daytime function following cumulatively restricted sleep it is important to maintain an awareness of how changes to normal sleep architecture may impact cognition and emotion. Across several nights of partial sleep deprivation/sleep restriction, REM sleep (which is rich during late-night sleep) is significantly reduced, whereas slow wave sleep (SWS) is largely preserved across restricted nights (Brunner, Dijk, Tobler & Borbély, 1990). In Brunner et al. (1990) participants were permitted to sleep at their habitual sleep time but were restricted to only four hours of sleep for two nights, followed by two nights of sleep recovery. This resulted in a REM sleep debt of 131%, which induced a potent compensatory response involving increased REM sleep pressure (reduced latency to enter REM sleep) and recovery (increased time in REM sleep) across the whole night and within the first part of the night. This well-documented rebound effect persists until sufficient REM sleep has been recovered and highlights REM sleep as essential for optimal brain function.

REM sleep is thought to be critical for next-day learning of emotional information, for affective memory consolidation and for the fundamental regulation and perception of emotion (see van der Helm & Walker, 2010 for a review of relevant literature). It may be cumulatively restricted in student populations but for those with insomnia symptoms this could be particularly debilitating due to psychological processes which interfere with the REM rebound effect responsible for resetting the emotional brain (van der Helm & Walker, 2010). The emotional aspects of insomnia (e.g. impulsivity, negative automatic thoughts, symptoms of anxiety and depression) may, therefore, be most strongly related to REM sleep. However, the timing of sleep restriction is influential in determining the impact upon sleep architecture. Individuals with sleep restricted to four hours during the latter part of the night receive significantly more REM sleep than those with sleep restricted to the first part of the night, consistent with a circadian disposition for REM sleep to occur at this time of day (Tilley & Wilkenson, 1984; Czeisler & Guilleminault, 1980). Later night restricted sleep involves greater REM sleep and stage 4 sleep and a reduction in stage 2 sleep when compared to early night restricted sleep (Tilley & Wilkenson, 1984). Therefore, although

REM sleep deprivation for any period results in daytime cognitive and emotional impairment, the timing of sleep restriction may affect the severity of these impairments.

It should be noted that sleep deprivation and insomnia are quantitatively and qualitatively different conditions. Studies previously referred to in this section guide the development of research hypothesis in this thesis but results cannot be generalised to the population under investigation who predominately experience acute insomnia symptoms. Given the wealth of studies which have investigated cognitive impairment associated with sleep deprivation it is valuable to contrast this literature with findings in Insomnia Disorder. However, the following limitations should be carefully considered throughout the thesis.

The typical symptoms of insomnia differ from those following sleep deprivation. Current sleepiness, an index of underlying sleep debt (Carskadon & Dement, 1982), is more reliably associated with sleep deprivation in comparison to insomnia where it is often disguised or even absent in more severe cases where individuals report daytime 'fatigue' and tiredness (Chambers & Keller, 1993; Stepanski, Zorick, Sicklesteel, Young & Roth, 1986). The extent to which sleepiness is a feature of acute insomnia is unknown. The discrepancy between objectively measured sleep debt and self-reported sleep debt associated with chronic insomnia may be a feature specific to this population, (Harvey, 2002) going beyond errors of estimation found in the general population, or in poor sleeping healthy individuals. Individuals with insomnia experience simultaneous arousal and fatigue such that on the gold-standard test of sleepiness, the Multiple Sleep Latency Test (MSLT), they do not show evidence of sleep propensity despite adequate opportunity to sleep (e.g. Seidel & Dement, 1982; Sugarman, Stern & Walsch, 1985). Therefore, the discrepancy between subjective and objective measures of sleep in insomnia prevents comparison with highly controlled sleep deprivation studies. Furthermore, insomnia is most commonly initiated by psychosocial stressors (which involve an element of emotional disturbance) which is vastly different to induced sleep deprivation which has a 'beginning' and 'end' point.

There is evidence to support differential effects of sleep deprivation and insomnia symptoms upon daytime cognitive function. For example, where insomniacs show increased arousal response to new stimuli and a slower habituation of the orienting responses (as measured by electrodermal activity), in sleep deprivation a markedly different pattern of responding is found with a slower shift of attention to novel stimuli, a decreased orienting response amplitude and a significantly faster habituation of the orienting response (Waters et al., 1993; McCarthy & Waters, 1997). Finally, sleep deprivation studies involve two sources of inter-individual variability, basal sleep-need and vulnerability to cognitive

dysfunction following sleep-loss which may profile differently in insomnia (Van Dongen, Baynard, Maislin & Dinges, 2004).

The relationship between sleep and anxiety: implications for investigating cognitive dysfunction in a poor sleeping population

In light of evidence that acute insomnia is a short-term, maladaptive response to internal/external stress and that sleep deprivation leads to amygdala-prefrontal disconnect associated with weakened attentional control (a feature of anxiety) it makes sense to consider the interplay between acute insomnia and anxiety which may affect daytime performance. As previously discussed, the relationship between anxiety and sleep disturbance is key in early development of insomnia in young adults, increasing vulnerability to future development of mood disorder (Johnson et al., 2006). The complex relationship between anxiety and insomnia can be explained by two competing models: the risk-factor model and the common-cause model (Klein, Wonderlich and Shea, 1993). According to the 'risk-factor model', one disorder predisposes an individual to the development of the other disorder. As such, chronic insomnia may be a trait marker for individuals at risk of anxiety disorders and/or repeated episodes of acute insomnia may confer significant risk for anxiety (Dahl & Bjorvtan, 2009). This model received support from a review investigating epidemiological findings of comorbidity (Dahl & Bjorvtan, 2009) and may be particularly relevant when considering development of sleep disorder in young adults (Johnson et al., 2006). The 'common cause' model proposes that insomnia and anxiety disorders have shared aetiology arising from a 'common core liability' but contends that these conditions are not necessarily causally related. Whilst this model can also successfully account for the high comorbidity between disorders, we do not yet know what core liability/liabilities may underlie these two disorders. This thesis assumes equal status of both models which together support the application of a popular model of cognitive control in anxiety to the investigation of attentional dysregulation in insomnia.

Attentional control: A useful construct for insomnia research?

As previously mentioned, attentional processes in chronic insomnia are key to the perpetuation of the disorder and to its maintenance. We are well-informed about the sleep-related attentional bias in this clinical group which serves to sensitise individuals to the experience of nocturnal wakefulness and to ambiguous daytime experiences that can be interpreted within the context of sleep-loss (e.g. Espie, 2007; Harvey, 2002). However, we know less about attentional processes during acute insomnia episodes which often subside and reoccur for susceptible individuals, without clinical levels of sleep-focussed worry.

Based on neuroimaging evidence of significant amygdala-prefrontal dysregulation following sleep deprivation (Yoo et al., 2007; Chuah et al., 2010) and concurring behavioural evidence using well-established neurocognitive tests of attention in sleep deprivation and in acute insomnia, there is a sound basis for expecting similar mechanisms of action in anxiety and in insomnia.

The concept of 'attentional control,' typically applied to understanding anxiety, is also likely to be relevant to the study of insomnia symptoms. Trait individual differences in the ability to focus and shift attention, resist distraction and flexibly control thought (Rothbart, Ellis & Posner, 2004; Derryberry & Reed, 2002) are likely to be important in determining the association between insomnia and daytime cognitive function. Furthermore, emotional dysregulation associated with insomnia is well documented and weak attentional control is associated with increased negative emotionality (Moriya & Tanno, 2008; Muris, van der Pennen, Sigmond & Mayer, 2008). Attentional Control Theory (Eysenck, Derkashan, Santos & Calvo, 2007; ACT) is well established within anxiety research as measuring a robust phenomenon (Cisler & Koster, 2010), with an aggregate effect size of d=.45 in a meta-analysis (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenberg & van IJzendoorn 2007). Furthermore, attentional control as a self-report construct has reliably predicted performance on several standard neurocognitive measures of attention as reported in a review by Cisler and Koster (2010), (e.g.. Dot probe task; MacLeod, Mathews & Tata, 1986; Visual search task; Öhman, Flykt & Esteves, 2001; Stroop task; Stroop, 1935).

Individuals with anxiety demonstrate a readiness to attend to threat information (Bar-Haim et al., 2007), which the model suggests is due to the dynamic interaction between a top-down volitional attentional system and a 'bottom-up' stimulus driven system. The top-down system is important for maintaining attention on current task goals whereas the bottom-up system instantly allocates attention towards cues that hold potential threat-salience (Pashler, Johnston & Ruthruff, 2001). In high trait-anxious individuals this attentional system is disrupted such that the influence of the bottom-up system is increased whilst top-down mechanisms are concurrently weakened (Lavie, 2005). This imbalance leads to on overall distractibility by task-irrelevant stimuli and a deficit in available attentional resources for maintaining optimal performance. This is particularly evident in situations presenting high state anxiety or situational stress (Eysenck, 1992). Specifically, 'worry' has been identified as an underlying cause of attentional resource depletion due to its chronic, excessive and uncontrollable form (Eysenck et al., 2007). Excessive and persistent worry is an established feature of GAD, however, it is also a very important

feature of insomnia (Sunnhed & Jansoon-Frojmark, 2014) and, therefore, we can expect a similar effect of worry on attentional control between disorders.

Anxiety monopolises attentional resources which is especially detrimental during challenging experimental paradigms involving counter-intuitive task heuristics. Individuals perform at a lower level to controls due to a lack of available attentional resources, preventing efficient modulation of an initial bias towards distractor stimuli (Derakshan, Ansari, Shoker, Hansard & Eysenck, 2009). Specifically, executive control of attention is most heavily burdened in these circumstances, such that 'inhibition' and 'shifting' functions are weakened. Inhibition down-regulates the instinctual response to allocate attentional resources towards perceived threat and resists distraction from task-irrelevant stimuli (Friedman & Miyake, 2004) whilst shifting facilitates the cognitive switch between competing cognitive sets so that individuals can meet the demands of multiple task requirements (Eysenck et al., 2007). Combined, these functions are critical to coordinate thoughts in relation to internal goals (Pashler et al., 2001).

Attentional Control Theory provides predictions about the way in which attention is affected under conditions of situational stress, worry and state anxiety. Although acute insomnia is likely to involve direct pathways to impaired performance deficits due to sleep debt, ACT explains how executive control deficits may also be a feature of the condition. The effect of state anxiety is that processing efficiency, "the relationship between effectiveness of performance and effort or resources spent in task performance, as measured by reaction time," (Eysenck et al., 2007, p.337) is impaired on cognitively demanding tasks over and above any effect on performance effectiveness, "quality of task performance indexed by standard behavioural measures (p.337)" and indexed by error rate. A conceptual awareness of ACT is helpful because standardised measures have been developed in order to assess 'attentional control,' and these have been well validated (e.g. ACS; Derryberry & Reed, 2002; Cognitive Failures Questionnaire, CFQ; Broadbent, Cooper, Fitzgerald & Parkes, 1982). Currently, there are no recommended measures of daytime cognitive function associated with insomnia. This deficit has been highlighted and researchers encouraged to use and report appropriate candidate measures (Buysse et al., 2006).

In this review, the role of anxiety in insomnia symptoms has been considered from an aetiological perspective and within important theoretical models of insomnia. It is clear that anxiety is an important aspect of an acute insomnia complaint and is important to the developmental trajectory of chronic insomnia. Attentional Control Theory is helpful in

considering how insomnia symptoms may affect cognitive performance and throughout this thesis there is an intention to explore attentional control as a mechanism which could be susceptible to dysregulation. Furthermore, given the close associations between anxiety and acute insomnia symptoms it was appropriate to be guided by experimental paradigms well validated in anxiety research in order to investigate cognitive performance in acute insomnia. Similarly, the ACS and CFQ were used to measure self-reported cognitive decline (executive control) associated with insomnia symptoms because of their validated psychometric properties in anxiety research. By taking this approach, the work undertaken in this thesis was novel and measures of cognitive performance were considered sensitive to the nature of insomnia symptoms in a healthy population.

Future directions and thesis aims

A review of the research important to understanding insomnia symptoms highlighted several areas of daytime function which warrant further empirical investigation, particularly because of the implications for young adults. Young adults are a comparatively neglected population in sleep research (Lund, Reider, Whiting & Prichard, 2010) yet are often likely to experience poor sleep due to increasing social, emotional and academic pressures, and are therefore at increased risk of developing a sleep-complaint that is not otherwise accounted for by developmental factors or physical health (as seen in child and older adult populations respectively). This thesis concentrated upon cognitive and physiological aspects of insomnia symptoms in this population because these are most strongly related to increased sleep onset latency and total sleep time (Broman & Hetta, 1994; Lamarche & Ogilvie, 1997). In addition, the work conducted in this thesis provided important evidence for the prevalence of poor sleep and insomnia symptoms in healthy young adults which is a greatly under-reported demographic.

Careful consideration was given to the selection of measures used to assess sleep in this programme of work because both subjective (e.g. questionnaires, sleep-diaries) and objective tools (e.g. actigraphy, polysomnography) are commonly used to meet specific research aims. The following criteria were used to determine the most suitable method of sleep assessment for the purpose of exploratory, cross-sectional, correlational study design: comprehensive assessment of perceived sleep and insomnia symptoms, short completion time, encouraged accuracy of reporting, practical for participants and research team. It was decided that self-report measures completed at one time point would be used to capture sleep profiles, selected on the basis of sound psychometric properties (see Chapter Two for details).

The PSQI is the most widely-used standardised measure of sleep quality which is reliable, valid, discriminates between good and poor sleepers, provides an index which is easy for participants to use and experimenters to interpret, and is brief and clinically useful in determining a variety of sleep disturbances which affect sleep quality (Carpenter & Andrykowski, 1998; Buysse, Reynolds, Monk, Berman & Kupfer, 1988). The sound psychometric properties of the PSQI were originally reported in a middle aged sample of 52 healthy subjects and 96 individuals with mixed sleep problems (including initiating and maintaining sleep, excessive somnolence and sleep problems related to depression). Since then, however, the inventory has been applied extensively within psychiatric and health related research and clinical practise, in healthy populations, in young people, adults and older adults (see Carpenter & Andrykowsky, 1998 for a review). Several studies have confirmed the appropriateness of the PSQI for measuring sleep parameters in college students specifically, reporting sound psychometric properties (Taylor et al., 2013; Lund et al., 2010).

The PSQI measures sleep quality over the past month, bridging the gap between post-sleep inventories (assessing only previous night of sleep) and survey type assessment (e.g. over the past year). Post-sleep inventories have the advantage of accurate measurement of night-to-night variation in sleep parameters but are limited in that they do not provide information about frequency or duration of problems. Survey-type questionnaires can provide a general idea of habitual sleep behaviour but do not capture sleep presentation at a given time point. The PSQI produces seven components which are considered to represent key areas routinely assessed by sleep clinicians; quality, latency, duration, efficiency, disturbance, daytime dysfunction and medication use. Items require various responses including usual bed time, usual wake time, time before sleep onset and hours slept and forced choice Likert choices. These component scores are added to produce a global score which identifies good sleepers and poor sleepers.

The ISI provides a different dimension to the assessment of sleep in young adults, developed specifically to align with DSM 5 criteria for insomnia assessed over the past two weeks (Morin et al., 1993). Specifically, this measure considers daytime cognition as a central and determining feature of an insomnia complaint. This measure includes three items relating to nocturnal aspects of sleep (sleep onset latency, wake after sleep onset and early morning awakenings) and four further questions assessing daytime aspects of insomnia; satisfaction with sleep, noticeability of symptoms, interference with daytime function, personal distress. The original validation study involved 223 patients, 145 17-82

year olds with insomnia complaints and 78 older patients who participated in a treatment study for insomnia. Sound psychometric properties were reported including moderate to strong correlation coefficients between individual items and corresponding variables on sleep diaries (Morin et al., 1993). The ISI provides scoring guidelines for identifying cases of 'no clinically significant insomnia,' 'subthreshold insomnia,' 'clinical insomnia' (moderate severity) and 'clinical insomnia' (severe). The ISI has been validated within adolescents, young adults and healthy college students (e.g. Chung, Kan & Yeung, 2011; Backhaus, Junghanns, Broocks, Riemann & Hohagen, 2002; Nadroff, Nazem & Fiske, 2011), however, there is little consistency in cut-off criteria for insomnia in these groups and mean values are referenced for comparison between studies.

Study One (Chapter Two) investigated the efficiency and effectiveness of attention during well-established neurocognitive tasks. Specifically, we were interested in determining which aspects of attention are associated with insomnia symptoms, i.e. whether deficits in alerting and orienting are most characteristic of insomnia symptoms or whether executive control deficits are also present. These components of attention can be differentiated using the Attention Network Test (ANT; Posner & Rothbart, 2007; Fan, McCandliss, Sommer, Raz & Posner, 2002) and the Switching Attention Task (SAT; Edinger, Means, Carney & Krystal, 2008) which, so far, have been used limitedly within insomnia research and are novel to the investigation of acute insomnia. Response inhibition was also investigated, which requires executive control of attention using the Stop Signal paradigm (SSP; Lappin & Erikson, 1966; Logan & Cowan, 1984). Through selection of sound experimental measures, this chapter aims to profile how poor-sleep in young adults is associated with attention and executive control. This research should inform us as to how concentration, memory and ultimately learning may be affected in this population.

Study Two (Chapter Three) investigated associations between insomnia symptoms (measured by poor sleep quality and insomnia severity), distractibility and thought intrusions. The thought intrusions task (Hirsch, Hayes & Mathews, 2009) is a novel objective measure of spontaneous, intrusive thought which has previously been used within anxiety and worry research but is also highly appropriate for poor-sleeping populations. The phenomenon of intrusive thought, and the related efficiency of attentional control associated with insomnia symptoms were of primary interest. This aspect of daytime function in young adults was considered important because of the risk for further mental health problems such as chronic worry and anxiety. Distractibility by negative daytime thoughts relating to current concerns may indicate dysregulation within broad attentional

(and related emotional) networks. This is in contrast to the sleep-focused intrusive thought often reported in chronic insomnia at night-time.

Study Three (Chapter Four) investigated autonomic arousal associated with insomnia symptoms which typically accompany cognitive arousal (investigated in Study Two, Chapter Three). We were interested in whether insomnia symptoms were associated with increased autonomic arousal at baseline and, importantly, following an acute stress manipulation. The 7.5% CO_2 model of anxiety (Bailey, Kendrick, Diaper, Potokar & Nutt, 2006; Bailey, Papadopolous & Nutt, 2009) was used to induce acute stress symptoms and autonomic activity was measured by blood pressure, heart rate and self-report state anxiety.

In the final study we investigated risky decision-making, an area which has received a lot of research attention in recent years in relation to sleep-deprivation. Using a novel modified Risky Choice Task (RCT; Fairchild et al., 2009), we were interested in associations between insomnia symptoms and the likelihood for young-adults to make high-risk decisions. This was considered to be a particularly important area of investigation given potentially severe consequences of irrational decisions in this population, e.g. relating to finance, health, sexual health, driving, drugs and alcohol. Many previous studies into decision-making and sleep loss have suffered criticisms related to the inability of paradigms to determine *how* this aspect of cognition is affected by sleep loss. The RCT has a major strength of being able to capture the profile of decision making according to quantifiable 'risk' and value contingencies.

Across all studies, the analytical approach determined the strength of associations between objective performance and poor-sleep quality and insomnia symptoms. Although this approach is typical for the investigation of insomnia symptoms, as a secondary analysis the uniqueness of these associations were assessed beyond the effects of anxiety and self-report deficits in cognitive control. This is because elevated trait anxiety can predispose for disturbed sleep and in a healthy population may account for more variance in performance than insomnia. The primary measures were self-reported sleep quality, and insomnia symptoms which were correlated with objective performance measures. Measures of attentional control and cognitive error proneness were also taken to explore the role of cognitive control in insomnia-related daytime performance.

In summary, the importance of this thesis has been highlighted by the recent call for increased understanding, improved conceptualisation and early intervention for acute insomnia (Ellis et al., 2012). Acute insomnia is known to predict more psychologically complex and persistent problems with sleep in the form of chronic insomnia; itself a

significant risk factor for psychiatric illness. A review of the relevant literature has informed predictions about daytime dysfunction in those with insomnia symptoms. There is a strong case for anticipating dysfunction within broader cognitive systems (particularly relating to attention) in the acute stages of sleeplessness which may in part be associated with sleep-interfering stress reactivity. Through improved profiling of cognitive dysfunction associated with insomnia symptoms we will be better informed of the mechanisms likely to be involved in the transition to clinically important insomnia. Furthermore, where there is currently no recommended early intervention for acute insomnia, we may begin to consider the value of attention-based treatments for increasing resilience to the daytime impact of insomnia symptoms.

Chapter Two

Insomnia Symptoms and the Cognitive Control of Attention

Impaired daytime function is the most important reason why people with insomnia seek help (Morin et al., 2006). Individuals commonly report symptoms of fatigue and irritability, impaired concentration and performance, in social and occupational domains (Moul et al., 2002). Insomnia strongly predicts absenteeism from the workplace (Léger, Massuel & Metlaine, 2006; Ozminkowski, Wang & Walsh, 2007) difficulty carrying out workplace duties and increased workplace errors (Léger, Guilleminault, Bader, Levy, & Paillard, 2002). Young adults in full-time education report that concentration, learning, and retention of information is a particular 'struggle' requiring extra mental effort. (Kyle, Espie & Morgan, 2010). The resulting fatigue has a knock-on effect reducing the pleasure received from social and leisure activities and may serve to further escalate psychological distress (Kyle et al., 2010).

It is well known that sleep deprivation negatively affects cognitive performance (Drummond, Paulus & Tapert, 2006), although the way in which attentional mechanisms are impaired is unclear and disputed (Orff, Drummond, Nowakowski & Perlis, 2007). Specifically, there is no consensus on whether sleep deprivation predominantly reduces the ability to maintain and shift attention or whether executive control is also independently and directly impaired (Cain, Silva, Chang, Ronda & Duffy, 2012). This is an important but unexplored issue for the complaint of insomnia symptoms[1], which is underpinned primarily by sleep debt (Ellis et al., 2012). Previous research has investigated executive control deficits in sleep deprivation and in clinical insomnia samples. These studies are informative for the investigation of insomnia symptoms but also reflect distinct conditions which are associated with different predictions for cognitive performance. An increased understanding of the mechanisms which bring about cognitive dysfunction following natural, acute sleep loss may help inform early interventions for acute insomnia and ultimately preventative treatment for chronic insomnia.

One of the most influential models of attention was originally proposed by Posner and Raichle (1994) (as cited in Jugovac & Cavallero, 2012). According to the model, there

[1] 'Insomnia symptoms' measured using the PSQI which focuses primarily on nocturnal aspects of poor sleep (with a 'daytime dysfunction' subscale). In subsequent chapters both the PSQI and the Insomnia Severity Index were used to capture important daytime aspects of the complaint as well as indices of sleep.

are three attentional networks; 'alerting', 'orienting' and 'executive control.' Achieving and maintaining a state of arousal and attentional preparedness is known as 'alerting.' Orienting is the selection of information from sensory input which involves the ability to prioritise allocation of attention to novel or salient stimuli, at the expense of processing competing stimuli (Mirsky, Anthony, Duncan, Aheam, & Kellam, 1991). Finally, executive control is required for resolving conflict between competing actions and comes under the broader umbrella term of 'executive function,' the ability to initiate, monitor and stop actions, and achieve goals (Phillips, 1997). The three components of attention, alerting, orienting and executive control, interact to achieve functional efficiency (Fan et al., 2009).

It is a robust finding that total sleep deprivation, chronic partial sleep deprivation and wakefulness beyond 16 hours diminishes alertness, reflected in longer response latencies on simple attention and vigilance tasks (see Basner & Dinges, 2011). Greater moment-to-moment variability of attention, or 'lapses' are believed to be caused by the interaction of the homeostatic drive for sleep, the circadian drive for wakefulness, and compensatory effort to perform (Doran, Van Dongen & Dinges, 2001). This creates an unstable state that changes within seconds where an individual is neither fully awake nor asleep (Doran et al., 2001). Performance variability in sleep deprivation is measured using tasks that require sustained attention for periods exceeding 10 minutes, e.g. the Psychomotor Vigilance Test (PVT; Dinges & Powell, 1985), a reaction time task measuring response time to on-screen stimuli presented at random inter-stimulus intervals (10s-20s). The effects of sleep loss upon covert orienting (where participants momentarily shift attention without moving the eyes) and re-orienting have been reported following partial sleep deprivation (Cavallero et al, 2002; Versace, Cavallero, De Min Tona, Mozzato & Stegagno, 2006), although findings are inconsistent across studies (e.g. Casagrande et al., 2006).

Impaired alerting and orienting, however, are not reliable features of cognitive dysfunction in Insomnia Disorder (Orff et al, 2007; Bonnet & Arand, 1995; Mendelson et al, 1984; Altena, Van Der Werf, Strijers & Van Someren, 2008). In a recent review, Shekleton et al. (2014) reported no difference in performance between participants with Insomnia Disorder and healthy controls on the PVT, even when cognitive load was increased by increasing the complexity of the task. Similarly, orienting ability has been reported as comparable to healthy participants on tasks requiring attentional vigilance and shifting (Edinger, Glenn, Bastien & Marsh, 2000; Edinger et al., 2008; Edinger et al., 1997; Mendelson, Garnett & Linnoila, 1984; Broman, Lundh, Aleman & Hetta, 1992; Backhaus et

al., 2006). Several studies, however, reported decreased accuracy and increased response times on the PVT when distractor stimuli were included (recruiting executive control) (Sugerman, Stern & Walsh, 1985; Hauri, 1997; Varkevisser & Kerkhof, 2005; Altena et al., 2008). Indeed, increased error rate (despite no difference in reaction time) has also been observed (e.g. Schneider, Fulda & Schulz 2004; Lamoureux, Bastien & Morin, 2000) and may be an important, under-reported index of performance.

According to some researchers, executive control is most profoundly affected by sleep loss because prefrontal functionality is selectively impaired (Harrison, Horne, & Rothwell, 2000). Accordingly, the ability to flexibly and adaptively shift the focus of attention in line with task goals is reduced (e.g. Harrison & Horne, 1999; Harrison et al., 2000; Harrison & Horne, 1998; Horne, 1988). This hypothesis is supported by converging EEG and PET evidence showing sensitivity of the prefrontal cortex to sleep deprivation (Finelli et al., 2000; Thomas et al., 2000) yet currently there is no consensus on whether this hypothesis is true.

Two studies previously reported that the ability to stop a prepotent response was impaired on the Go/No Go task following 24 hours and 64 hours of sleep deprivation (Chuah et al., 2006; Drummond et al., 2006). However, the same ability tested by the Stroop Task was not impaired following 36 hours of sustained wakefulness (Sagaspe et al., 2006). If attention is so fundamentally affected by sleep-loss, then decreased performance and variability in performance would be consistently observed across a wide range of tasks which is not reliably the case (see Tucker et al., 2010 for a review). This may be in part due to experimental tasks providing 'impure' measurement of executive performance where executive function cannot be dissociated from basic attentional processes (Phillips, 1997). However, in Sagaspe et al. (2006), non-executive and executive processes of the Stroop task were discriminated. Increased response time and error rate were reported in the absence of impairment to response inhibition during proactive interference. Cain et al. (2012) further optimised the detection of executive control deficits on this task through the addition of congruent word trials, reducing the tendency for individuals to suppress word reading. They also concluded that 40-hours of constant-routine wakefulness did not impair executive control.

Despite extensive research into the effects of sleep deprivation upon executive function, this aspect of cognition is the most under-researched aspect of performance in insomnia (Shekleton, Rogers & Rajaratnam, 2010) and deficits in this area have been most evident when compared to healthy controls. (Edinger et al., 2000; Edinger et al., 2008;

Edinger et al., 1997). In these studies, a battery of cognitive performance measures was used to profile deficits in multiple facets of attention. The task required participants to maintain concentration and attend to the position and orientation of presented stimuli. Later stages of the task required inhibition of a pre-potent response in line with a previously displayed textual command, itself at odds with the position/orientation of the presented stimulus 50% of the time. As such, this task requires concentration, attention, response inhibition and rapid decision making which may approximate the nature of cognitive dysfunction in insomnia (Edinger et al., 2008). Across studies, participants with Insomnia Disorder did not reliably show impairment to performance on a Simple Reaction Time task or Continuous Performance Task, but did so on the Switching Attention Task (SAT) which reflected weakened executive control. Most recently, Shekleton and colleagues (2014) reported that compared to controls, participants with Insomnia Disorder did not show impairment to sustained attention (on the PVT) but showed significantly impaired working memory (N-Back Task) and cognitive switching (SAT). Furthermore, participants did not report being more sleepy than controls, suggesting that the ability to maintain attention was relatively unaffected.

The lack of consensus on the association between poor sleep and executive control warrants an investigation using sensitive cognitive tasks capable of revealing performance deficits in those who experience naturally poor sleep. On the one hand, sleep deprivation research provides most convincing evidence for alerting and orienting impairment and provides mixed results in relation to executive performance. On the other hand, Insomnia Disorder appears to be characterised by executive performance deficits, evident when cognitive resources are heavily burdened. It is possible that alerting and orienting are preserved in Insomnia Disorder because the underlying sleep debt is not sufficient to cause fundamental cognitive dysfunction. In those with 'insomnia symptoms,' however, alerting and orienting may be impaired as a result of sleep loss, and impairment may extend to executive control.

The Attention Network Test has the advantage of being able to discriminate between the three components of attention. This task has been extensively used in the investigation of attention control (Posner & Rothbart, 2007), is simple and timely (20 minutes), and the executive control component has particularly strong test-retest reliability α=.77(Fan et al., 2003). The ANT requires participants to classify the direction of a middle arrow, surrounded by flanker arrows. The flanker arrows may point in the same direction as the middle arrow (congruent condition), or may be opposing the middle arrow (incongruent

condition). The difference between reaction time (RT) on these conditions is considered to reflect executive control efficiency. Visual cues either signal that the stimulus is to appear soon, that it will appear in a particular location, or both, providing information on both alerting (improved performance following a non-spatial warning cue) and orienting (additional benefit when cue correctly indicated target location). To the best of the author's knowledge, two studies (published after the conception of the current study) (Martella, Casagrande & Lupiáñez, 2011; Jugovac & Cavallero, 2012) have applied the ANT to sleep disturbance using 24 hour wakefulness designs. Both studies revealed impairment to all three attentional networks in the sleep deprived group, confirming the appropriateness of the ANT for investigating cognitive control in sleep-disturbed populations (by way of cue effects and conflict effects).

The Switching Attention Test (SAT) involves a series of increasingly complex attentional tasks, i.e. ranging from a key press in response to target location, to target direction, and finally to categorisation following cognitive conflict e.g. an incongruent trial may instruct a participant to categorise the 'side' of the screen a stimulus (arrow) appears when the arrow points in the opposite direction. According to Edinger and colleagues (1997; 2000; 2008) this final switching component (requiring concentration, attentional shifting, response inhibition and rapid decision making) most closely approximates the type of impairment reported in Insomnia Disorder. This task has established reliability and validity for detecting subtle cognitive impairment, e.g. resulting from chronic low-level neurotoxin exposure (Arcia & Otto, 1992; Baker, Letz & Fidler, 1985; Mahoney, Moore, Baker & Letz, 1988).

The Stop Signal Paradigm (SSP) is a measure of inhibitory control, or the ability to stop a prepotent response to a stimulus. This task is particularly appropriate for assessment of executive function because it effectively distinguishes between inhibitory motor control and other cognitive functions including base reaction time (Sagaspe, Philip & Schwartz, 2007). This task involves making simple key-press responses to either a square or circle stimulus on the screen, unless participants hear a bleep which cues them to withhold their response. The task utilizes a staircase algorithm that tracks each participant's performance to ensure that the beep occurs at a 'critical' point for each individual, thus presenting a significant cognitive challenge. The SSP is considered a more sensitive and cognitively purer measure of response inhibition than traditional Go/NoGo Tasks (Aron, Robbins & Poldrack, 2004) previously used. In the first study to use the SSP in relation to insomnia, Sagaspe and colleagues (2007) did not find response inhibition deficits compared to

controls. However, Covassin, de Zambotti, Sarlo, De Min Tona, Sarasso and Stegagno (2011) did find impaired response inhibition (reaction time) on the SSP, accompanied by cardiovascular hyperarousal.

Common to all three tasks is 'cognitive conflict', a property that has been found to activate frontal areas of the brain, namely the anterior cingulate cortex (ACC) and prefrontal cortex (PFC), (Bush, Luu & Posner, 2000; MacDonald, Cohen, Stenger & Carter, 2000) which are themselves well established as critical to efficient executive control (Bishop, 2007). Specifically, the flanker task of the ANT has been found to activate the ACC (Fan et al., 2003), and damage to the PFC has been found to negatively affect response inhibition (Aron et al., 2004) which is required in the SSP.

This study investigated how the three dissociable components of attention,-; namely alerting, orienting and executive control (Fan, McCandliss, Fossella, Flombaum & Posner, 2005) relate to poor sleep quality using the ANT. The study also examined how attention is affected during increasingly complex attentional tasks which discriminate between simple classification performance and switching of attention performance requiring executive control using the SAT. Finally, the study investigated response inhibition requiring executive control of attention using the SSP. The primary hypothesis was that executive performance deficits are associated with insomnia symptoms (assuming direct effects of sleep deprivation upon prefrontal function). Insomnia symptoms were also predicted to negatively associate with simple response reaction time, alerting and orienting of attention. Given established associations between current sleepiness, anxiety and cognitive performance, a secondary aim was to examine associations between insomnia symptoms and performance beyond the contribution of these variables.

Method

Participants. Undergraduate participants were recruited from the Psychology department of the University of Southampton by means of online and poster advertisement. Course credits were exchanged for participation. Seventy two participants took part, nine (12%) were male, and the mean age was 20.17 years (*SD*= 2.94). All participants provided written informed consent and all study procedures were reviewed and approved by the Ethics Committee at the School of Psychology, University of Southampton, UK.

Design. A cross-sectional correlational research design was used, comprising self-report measures and computerised tasks for investigating cognitive control mechanisms.

Self-report measures of sleep quality, anxiety and current sleepiness were correlated with performance measures from the ANT, SSRT and SAT.

Self-report measures.

Sleep Quality. The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman & Kupfer, 1989) measures sleep quality[2] over the past month. Nineteen items relate to normal sleep habits covering seven areas of sleep; subjective sleep quality (PSQI quality), e.g. 'How would you rate your sleep quality overall?,' sleep latency (PSQI latency), e.g. 'how long has it usually taken you to fall asleep at night?,' sleep duration (PSQI duration), e.g. 'how many hours of actual sleep did you get at night?,' habitual sleep efficiency (PSQI efficiency) calculated from time in bed vs. time asleep ratio, sleep disturbances (PSQI disturbance), e.g. 'how often have you had trouble sleeping because of waking up in the middle of the night/early morning?', use of sleep medication (PSQI meds.), e.g. 'how often have you taken medicine to help you sleep?' and day time dysfunction (PSQI day dysfunction), e.g. 'how often have you had trouble staying awake while driving, eating meals, or engaging in social activity'.

Responses are made on a 0-3 subscale (3 is the negative pole) to reflect the majority of days and nights during the month (total score [GPSQI] range 0-21). The PSQI has been shown to have strong internal validity (a = 0.83) and temporal stability (Pearson r = .85 for an average of 28.2 days) (Buysse et al., 1989). It's effectiveness as a screening tool for significant sleep disturbance (Backhaus et al., 2006) and its diagnostic sensitivity (89.6%) and specificity (86.5%) in groups of good and poor sleepers have been confirmed (Buysse et al, 1989). Internal validity of the measure in the current sample was good (a=.77).

*Trait Anxiety***.** The State-Trait Anxiety Inventory- Trait (STAI-T; (Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983) has 20 items which participant rate on a 4 point scale (1 = almost never, 4 =almost always) where higher scores reflect a greater general disposition for anxiety, a trait that is situationally and temporally stable (Tovilovic, Novovic, Mihic & Jovanovic, 2009). Example items include 'I am content' and 'I am a steady person.' The STAI-T is the most widely used measure of trait anxiety in clinical and non-clinical populations (Spielberger, 1989) with high test-retest reliability and internal reliability a = .86 (Barnes, Harp & Jung, 2002) and high discriminant and convergent validity with other

[2] 'Sleep quality' refers to the overarching construct measured by the PSQI. 'PSQI quality' refers to a subscale comprised of specific items assessing perceived standard of sleep.

measures of anxiety and related constructs (Spielberger et al., 1983). Chronbach's alpha was .82 in the current study.

Current Sleepiness. The Stanford Sleepiness Scale *(*SSS; Hoddes et al., 1973) measures current level of sleepiness at the time of survey administration. The SSS is well established at assessing sleepiness both inside and outside of laboratory settings, corresponding to sleep disturbance, previous time awake and performance (Hoddes et al., 1973; Gillberg, Kecklund, & Akerstedt, 1994). The SSS uses a seven point scale where 1 is "Feeling active, vital, alert or wide awake" and 7 is "No longer fighting sleep, sleep onset soon; having dream-like thoughts."

Attentional Control. The Attentional Control Scale (ACS; Derryberry & Reed, 2002) consists of 20 items measuring the ability to control attention in relation to positive or negative goals. Items such as "It's very hard for me to concentrate on a difficult task when there are noises around" reflect attentional focusing and "It is easy for me to alternate between two different tasks" reflected attentional shifting. Items are rated on a four-point Likert scale (1= almost never, 4= always) with possible scores ranging from 20 to 80. 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 (*a*= .88) and extensive literature exists to support attentional control deficits associated with sleep disturbance in clinical and non-clinical samples (e.g. MacMahon, Broomfield, Marchetti & Espie, 2006; Ree, Pollitt & Harvey, 2006). Chronbach's alpha was .72 in the current study.

Cognitive Error Proneness. The Cognitive Failures Questionnaire (Broadbent et al., 1982) contains 25 items inquiring about minor mistakes and slip-ups over the last six months across three areas; perception, memory and motor function. Items include "Do you fail to notice something is there?" "Do you forget which way to turn on a familiar road?" and "Do you bump into people?" Responses are made on the 5-point Likert-like scale (0= never, 4 =always) with a total score range from 0 -100. The scale has very high internal consistency (*a*= 0.96) and has previously been shown to correlate well with subjective sleepiness in undergraduates (Wallace, Vodanovich & Restino, 2003). Chronbach's alpha was .84 in the current study.[3]

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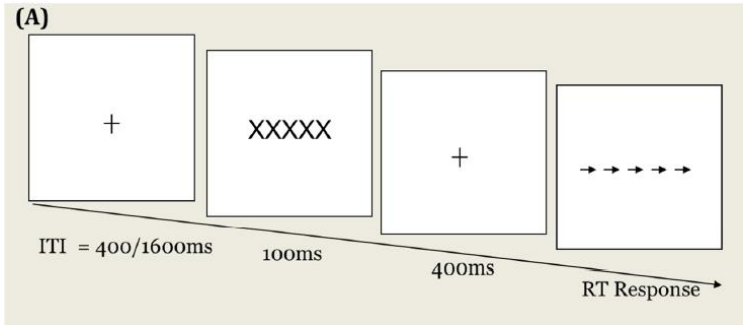
[3] 'Morningness- eveningness' trait was also measured using the Morningness-Eveningness Questionnaire (MEQ; Horne & Östberg, 1976) because young adults are susceptible to circadian rhythm disruption which could cause cognitive impairment (see Chapter One, p. 19 for discussion). However, the average participant was an 'intermediate type' (*M* =46.47, *SD*= 10.16) and correlations were not observed between MEQ scores and any self-report or objective performance measures.

Objective Performance Measures.

Attention Network Task; alerting, orienting and executive control. Each trial began with a fixation cross presented centrally on-screen for 400-600ms (varying randomly between trials), see *Figure 2.1*. This was followed by a visual cue (a horizontal series of four black asterisks) presented for 100ms which alerted the participant that a target stimulus would soon be presented, to which they should respond. The cue appeared randomly in one of three conditions; above/below the original location of the fixation cross (a single cue which always correctly cued the location of the target), in the centre (replacing the location of the cross), simultaneously above and below the fixation cross (double cue) or no cue. The target arrow and flanker arrows (congruent/incongruent) then appeared simultaneously 400ms after disappearance of cue. The target arrow was presented in a centrally nested position within a row of five horizontal black lines on a white background with arrowheads pointing either left or right. The arrows flanking the target arrow either pointed in the same direction (congruent presentation) or the opposite direction (incongruent presentation) as the target. Apart from no-cue trials, where stimuli automatically disappeared after 1700ms, all arrows immediately disappeared following a response.



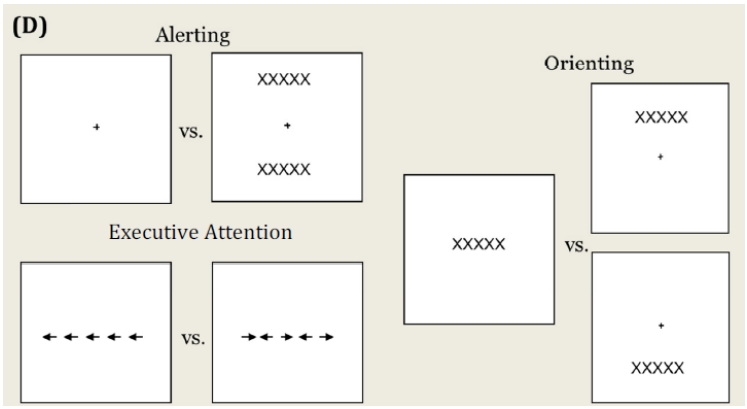


Figure 2.1. Attention Network Task trial types and cues. (A) Temporal sequence of presentation, (B) types of cue, (C) target stimuli, (D) comparisons by which attentional network function calculated.

Stop Signal Paradigm: response inhibition. A white square or circle on a black background in the centre of the screen served as the primary task stimuli (see *Figure 2.2*). Each trial began with a white fixation cross which was then replaced by a primary task stimulus (square or circle) after 250 ms. This remained on-screen until a response was made or until 1,250ms had elapsed. The default inter-stimulus interval was 2,000 ms independent of reaction time. On stop-signal trials, an auditory stop-signal of 750Hz 75ms unexpectedly occurs after a variable delay (Stop Signal Delay; SSD), but is initially set at 250ms, and is adjusted continuously based on previous responses using an algorithm developed by Logan, Schachar and Tannock (1997). This means that when inhibition is successful, the stop-signal delay increases by 50ms to present a greater challenge but when it is unsuccessful it decreases by 50ms to facilitate the next appropriate response (Verbruggen et al., 2008). A 10 second interval occurred between blocks, during which participants were given feedback on their performance in the previous block, i.e. the number of correct responses on no-signal trials, number of missed responses on no-signal trials, mean reaction time (reaction time) on no-signal trials and percentage of correctly suppressed numbers.

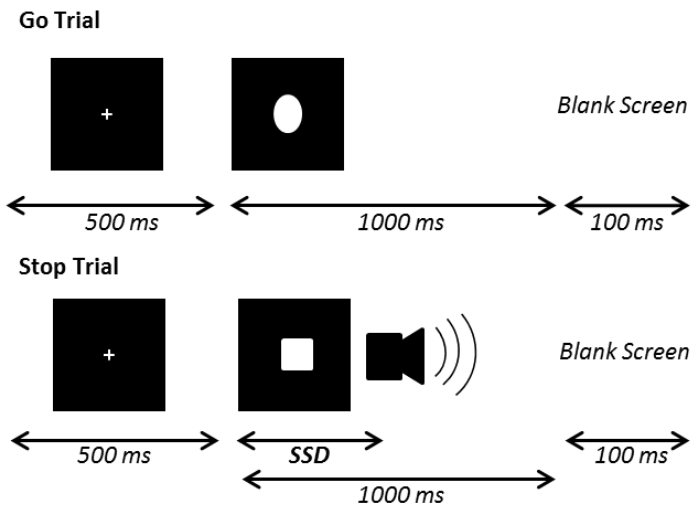


Figure 2.2 Stop Signal Paradigm schematic.

Switching of Attention Test: cognitive conflict. The SAT forms part of the Neurobehavioural Evaluation System (Letz & Baker, 1988) and lasts around six minutes. Participants press keys on the left or right side of the keyboard which correspond to on-screen stimuli. Initially participants complete trials where they categorise which side of the screen a square appears, then which direction a centrally positioned arrow points. In these trials, stimuli remain on screen for 2500ms or until a response is made. In the second part of this task, the word 'SIDE' or the word 'DIRECTION' instructs the participant whether they are to key-press in response to the side of the screen in which the stimulus is presented or to the direction in which the arrow is pointing. According to Edinger et al. (2005) the DIRECTION condition requires greater cognitive effort than the SIDE condition. After 1000ms an arrow is presented on either the left or right side of the screen. Fifty percent of the time the instruction was congruent with the stimulus (e.g. SIDE, arrow presented on the left, pointing left), the remaining trials were incongruent (e.g. DIRECTION, arrow presented on the left, pointing right). Congruent and in-congruent trials were fully randomised. The incongruent trials provoke cognitive conflict which requires executive control, in particular response inhibition and rapid decision making in order to meet the task goal.

Apparatus. Attention Network Task stimuli and SAT stimuli were coded using the Inquisit Millisecond software package (Inquisit 3, 2010) and were presented via Windows XP on the computer monitor. During the ANT motor responses were made using a response box positioned centrally on the desk in front with two keys corresponding to the direction of the target arrow on screen (left or right). Stop Signal Paradigm stimuli were presented and encoded using STOP-IT and ANALYSE-IT software (Verbruggen et al.,

2008). Participant responded by pressing one of two keys on the keyboard ("Z" for square and "/" for circle). All statistical analyses were computed using SPSS 17 statistical software package.

Procedure. Test sessions were conducted in a private testing lab within the Psychology department of the University of Southampton. An information sheet was provided and informed consent taken. Self-report measures were administered with participants instructed to provide instinctual reactions to the scale items. Participants were then seated at a desk 60 cm in front of the computer monitor in the testing booth to perform the ANT followed by the SSP and SAT.

Participants were instructed to remain still with an upright posture for the duration of the tests and to react as quickly but as accurately as possible. Task instructions were explained, demonstrations were given and understanding was checked. Before the task was initiated with lights out the experimenter entered a unique identifying code under which the participant data was saved.

The ANT involved one practice block with 24 trials and two experimental blocks in which the eight stimuli sets were each presented on 12 trials (totalling 96 trials per block). The SPP involved a practice phase of 32 trials and an experimental phase of 64 trials, in which 75% of trials were 'no-signal' trials and 25% of trials were 'stop-signal trials.' The SIDE categorisation task involved six practise trials and 16 experimental trials, the DIRECTION categorisation task involved four practise trials and 16 experimental trials. The switching task involved 8 practise trials and 48 test trials. The ANT and SSP lasted approximately 20 minutes and the SAT lasted six minutes. A full debrief was then given and an information sheet regarding sources of help for those with poor sleep was offered.

Data Analysis. Questionnaires were scored in accordance with original instructions for each scale where higher scores reflected a greater frequency of the related occurrence. One outlier participant was removed from ANT analyses, five from PRS and three from SSRT on the SSP (based on scores that were three times SD beyond sample mean). The 'ANT alerting' variable was calculated by subtracting the mean reaction time of the double-cue conditions from the mean reaction time of the no-cue conditions, the 'ANT orienting' variable was reflected in the difference between reaction time to spatial and centre cues and the 'ANT executive' variable was calculated by subtracting the mean reaction time of all congruent flanking conditions, summed across cue types, from the mean reaction time of all

incongruent flanking conditions, see *Figure 2.1* (Fan et al., 2003; Fan et al., 2009). Parametric assumptions were met for Pearson's correlation coefficient.[4]

Consistent with previous studies, key outcomes on the SAT included mean response latency (MRL) and within-subject standard deviation (SD) of response latencies across stimulus presentations (indices of overall performance and attentional/behavioural instability respectively).[5] Key output variables were: SAT Side (MRL and SD to SIDE trials), SAT Direction (MRL and SD to DIRECTION trials), SAT S Side (switching SIDE subtest score), SAT S Direction (switching DIRECTION subtest score). Switching subtest scores were derived from the second part of the SAT where 50% of the time the correct response to a given trial was in conflict with stimulus presentation (e.g. the instruction to respond to SIDE involved a left pointing arrow on the right side of the screen).

On the SSP the internal tracking procedure causes the SSD to converge on a value where participants successfully inhibit a response 50% of the time. From this value, Stop Signal reaction time (SSP SSRT measured in ms) is computed as mean SSD minus mean 'go-trials' reaction time (see *Figure 2.3*). The SSRT is, therefore, a measure of how long it takes to inhibit a pre-potent response. Errors of commission were also computed (SSP PRS measured in %) to reflect the likelihood when a pre-potent response cannot be stopped.

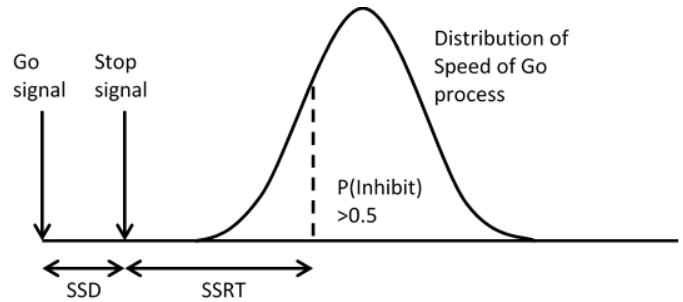


Figure 2.3. Schematic of the underlying horse-race model of 'stopping' and estimation of SSRT.

Results

Table 2.1 shows that 59.9% of the sample was classified as "poor-sleepers" according to the PSQI (Ellis, Mitchell & Hogh, 2007) (see Appendix H for details of

[4] . Bonferroni correction applied to key analyses between sleep measures and performance outcome = adjusted significance level $p<.003$ based on two sleep measures tested against nine dependent measures.
[5] In addition, we also investigated error rate (Error), a variable which was not reported in Edinger et al. (2005) but has been highlighted in previous studies as a potentially important index of performance (see Table A1, Appendix I)

bedtime, sleep onset latency, wake time and total hours of sleep pooled across Studies One to Four). Levels of trait anxiety, attentional control, and current sleepiness fell within the normal range for this population (e.g. Taylor et al., 2005; Derryberry & Reed, 2002; Alapin et al., 2000).

Mean reaction time of alerting and orienting components of the ANT (alerting: 38.30ms, orienting: 31.68ms) were similar to that found in Martella et al. (2011) where individuals underwent 24 hours of prolonged wakefulness (alerting: 39ms, controls: 30ms; orienting: 37ms, controls:21ms). Executive control mean reaction time on the ANT (92.63ms) fell between the values reported for controls (86ms) and sleep deprived individuals (103ms) in Martella et al. (2011). Participants performed similarly to, or better than healthy controls on the SAT (mean SAT: Side MRL=354.55µs; Dir. MRL= 417.32µs; Side S MRL= 355.61µs, Dir. S MRL=420.54µs) compared to participants in Edinger et al. (2008) (although specific descriptive task statistics were not reported). Mean SSRT and PRS (271.87ms; 48.76%) were comparable to that reported in Covassin et al. (2011) (SSRT: 205ms morning, 222ms evening; PRS: 44% morning, 61% evening).

Table 2.1.

Descriptive statistics and Pearson's correlations between self-report measures

			Self-report measures			
Measures			SSS	ACS	CFQ	STAI-T
	M	*SD*				
GPSQI	7.07	3.01	.27*	-.17	.21	.39**
PSQI quality	1.25	.72	.18	-.12	.31**	.36**
PSQI disturbance	1.33	.50	.15	-.06	.31**	.19
PSQI latency	1.68	.90	.16	-.24**	.15	.33**
PSQI duration	.43	.77	.19	.11	-.12	-.02
PSQI efficiency	1.00	1.05	.18	-.07	-.06	.03
PSQI meds.[a]	.29	.72	.06	-.13	.09	.51***
PSQI day dysfunction	1.08	.52	.24*	-.26*	.49***	.43**
SSS	3.27	.98	-	-.36**	.32**	.36***
ACS	48.06	9.70	-	-	-.48***	-.46***
CFQ	46.81	13.93	-	-	-	.47***
STAI-T	39.38	9.91	-	-	-	-

Table 2.2.

Pearson's correlations between self-report measures and performance measures

	Objective performance measures								
Measures	ANT alert.	ANT orient.	ANT exec.	SAT side MRL (SD)	SAT dir. MRL (SD)	SAT S side MRL (SD)	SAT S dir. MRL (SD)	SSP SSRT	SSP PRS
GPSQI	-.13	.16	-.09	-.16 (.10)	.00 (-.08)	-.19 (-.02)	-.02 (-.04)	.19	.14
PSQI quality	-.01	.12	-.02	-.06 (.16)	-.02 (.03)	-.06 (.04)	-.04 (-.13)	.14	.01
PSQI disturbance	-.20	.23	.05	-.13 (-.08)	.07 (.03)	-.15 (-.13)	.10 (.01)	.10	-.03
PSQI latency	-.23	.26*	-.10	-.13 (.01)	.06 (-.04)	-.12 (.04)	.03 (-.13)	.04	.21
PSQI duration	.11	-.12	-.03	-.10 (.08)	-.19 (.22)	-.14 (.06)	-.18 (.07)	.09	-.04
PSQI efficiency	.01	-.05	-.17	-.15 (.09)	.10 (-.17)	-.20 (-.02)	.07 (-.16)	.21	.13
PSQI meds.[a]	.00	.11	.01	.05 (.06)	-.01 (-.02)	.06 (.08)	.00 (.06)	-.00	.05
PSQI day dysfunction	-.28*	.28*	.01	-.15 (.07)	-.05 (.18)	-.17 (-.13)	-.08 (.11)	.23	.13
SSS	.09	-.14	.28*	-.02 (.33**)	-.10 (.29*)	.01 (.08)	-.10 (-.15)	.35**	.10
ACS	.22	-.22	-.19	.01 (-.20)	-.15 (-.17)	-.05 (-.09)	-.06 (.09)	-.10	-.13
CFQ	-.30*	.12	.23	.02 (.12)	.05 (.16)	-.02 (-.08)	.05 (.03)	.20	.26*
STAI-T	-.16	.26*	.12	-.06 (.17)	-.12 (.31*)	-.03 (-.03)	-.12 (.19)	.04	.22

Note. [a] Spearman's Rho analysis performed due to non-normal distribution. ANT (n=71)= Attention Network Task (alerting, orienting, executive control), SAT Side MRL (n=70) = Side subtest of Switching Attention Test; SAT Dir. MRL (n=66)= Direction subtest of SAT; SAT S Side= Switching Side subtest of SAT; SAT S Dir.= Direction Switching subtest of SAT, SSP SSRT (n=69)= reaction time on Stop Signal Paradigm; SSP PRS (n=67)= errors of commission on SSP. *N*: PSQI =72, SSS=72, ACS=71, CFQ=72, STAI-T=69, ANT=71, SSRT=69, PRS=67, SAT Side (S) MRL=70, SAT Dir. (S) MRL 66. *p<.05, **p<.01, *** p<.001

As expected, poor sleep quality (quality, latency, daytime dysfunction, and medication) was moderately and significantly associated with trait anxiety. A moderate association between poor sleep quality and current sleepiness was driven by the association with 'daytime dysfunction' PSQI subscale. There were weak positive associations between 'latency' and 'daytime dysfunction' subscales and poorer attentional control. Moderate associations were observed between 'daytime dysfunction', 'sleep quality', 'sleep disturbance' and cognitive errors proneness.

Attention Network Task

There was evidence of association between poor sleep quality and alerting and orienting ability on the ANT, however the direction of these associations was opposing as shown in Table 2.2. The ability to focus attention towards relevant stimuli (orienting) was moderately and positively associated with greater sleep 'latency', 'daytime dysfunction' and trait anxiety (a trend was observed for 'disturbance' PSQI subscale and attentional control). Partial correlations controlling for trait anxiety revealed that the association between daytime dysfunction and orienting of attention remained significant (r=.25, p=.03) but the association between sleep latency and orienting became non-significant (r=.18, p=.15). The ability to maintain attentional focus over time was moderately and negatively associated with 'daytime dysfunction' and cognitive error-proneness (a trend was observed for 'latency' PSQI subscale and attentional control). However, following adjustment for multiple comparisons, observed associations were non-significant. There was no evidence that impaired executive control was associated with poor sleep quality.

In summary, executive control on the ANT was not associated with poor sleep quality. Daytime symptoms of poor sleep were associated with impaired alerting yet aspects of poor sleep quality were associated with greater orienting ability.

Switching Attention Task

Mean response latency and performance variability on the SAT were unrelated to sleep quality. Current sleepiness was moderately and positively associated with variability in responding to 'side' and 'direction' trials. Trait anxiety was also positively associated with variability in response to 'direction' trials.

Table A1 (Appendix I) shows that error rate on 'side' trials was moderately and negatively associated with poor sleep quality ('total', 'duration', 'efficiency') such that poor sleepers made fewer classification errors. There were no significant associations between error rate on switching trials and sleep variables.

In summary, poor sleep quality was not associated with impaired executive control on the SAT (as measured by switching subtests). Furthermore, those with poorer sleep made fewer errors on the simple response choice part of the task, contrary to expectations.

Stop Signal Paradigm

Poor sleep quality was not associated with executive control as measured by response inhibition. Current sleepiness was positively associated with reaction time to inhibit a prepotent response and the propensity for cognitive errors was moderately, positively associated with errors of commission.

Discussion

This study was designed to examine associations between poor sleep quality over the past month (as measured by the PSQI) and executive control of attention in participants with naturally disturbed sleep. Previous work in this area has been limited to sleep deprivation studies or to clinical samples where confounding variables (such as comorbid mood and anxiety disorder) might mask associations between poor sleep and cognitive dysfunction.

Overall, results from this study do not support an association between poor-sleep over the past month (as measured by the PSQI) and impaired executive control in healthy individuals with poor sleep quality. Mild impairment to alerting was associated with subscales of the PSQI ('latency' and 'daytime dysfunction'), however, impairment did not extend to executive function (switching of attention and response inhibition) nor did associations remain after correction for multiple testing. Individuals with poor-sleep quality showed greater attentional capture by task-relevant stimuli (orienting) despite poorer maintenance of preparatory cognitive activation.

Results are consistent with studies reporting that non-executive functions are foremost affected following sleep loss, and that impairment is generic to cognitive processes subserved by attention (Tucker et al., 2010; Doran, Dongen & Dinges, 2001).
Results are further consistent with state instability theory which proposes that attentional lapses following sleep deprivation impair efficient alerting. However, results challenge the belief that sleep loss selectively impairs executive function because associations between poor sleep quality and executive control were not found.

The finding that poor sleep quality is associated with impaired alerting and superior orienting on the ANT in a non-clinical sample is novel and interesting. An increased orienting response is a feature of Insomnia Disorder (Waters et al., 1993) which is not observed following sleep deprivation manipulations (Cavallero et al, 2002). A combination of increased sleepiness and motivation to overcome attentional lapses via extra cognitive effort may have created a situation where individuals were sensitive to goal-relevant stimuli even though their 'baseline' level of preparatory attentional readiness was low. Performance on the most challenging part of the task which required concentration, response inhibition and rapid decision making was unrelated to poor sleep quality. This could be explained by compensatory effort purposefully recruited in order to manage these multiple demands. Indeed, MRL of simple classification during SIDE trials was negatively associated with aspects of poor sleep quality suggesting that poor sleepers were able to compensate for the

effects of sleep loss during this task. Together these findings are consistent with previous research showing that the level of cognitive performance achieved by those with poor sleep is determined by motivation to overcome the effects of sleep loss (Dorrian & Dinges, 2003). Levels of motivation and ability to engage cognitive effort is determined by the severity of sleep loss. However, this compensatory effort cannot fully prevent intrusions of attentional lapses into wakefulness (Durmer & Dinges, 2009), explaining why alerting remained negatively associated with poor sleep quality.

Our findings on the ANT only partly support Martella et al. (2011) and Jugovac and Cavallero (2012) who showed adverse effects on all three ANT components following 24 hours of wakefulness. However, this is understandable given key differences between sleep deprivation and poor sleep (see Chapter One for discussion). A 'normal' sleep routine was a requirement for participation in the previously cited studies whereas in the current study variation in sleep quality, quantity and daytime symptoms was inherent. A direct comparison between studies is therefore not achievable but it is useful to note the differences in profile of cognitive function in poor sleep versus sleep deprivation.

Results on the SAT do not support the findings from Edinger et al. (2008) where it was concluded that switching of attention is impaired in Insomnia Disorder as measured by longer MRL on tasks which provoke cognitive conflict. This difference could in part be due to sample differences. The underlying sleep debt may be more severe in poor sleeping participants (hence impaired alerting) whereas in Insomnia Disorder executive performance may be most strongly related to perceived sleep quality and dysfunctional cognitive processes resulting from persistent psychological distress (Harvey, 2002). Indeed, dysphoric mood and emotional dysregulation may underlie executive performance deficits in Insomnia Disorder (Shekleton et al, 2014). This explanation could account for the absence of alerting and orienting impairment in Edinger et al. (2008).

Another possible reason for the difference in results is that the SAT imposes lower and upper cuts for acceptable response times for analysis (100-600ms) which may disguise valuable variability of response times in the data. Indeed, we observed that current sleepiness was associated with variability in responding during the simple classification trials but was unrelated to MRL variables, findings which are difficult to consolidate.

Consistent with Sagaspe et al. (2007), we did not find that response inhibition impairment on the SSP was associated with poor sleep quality. These findings could be explained by the results from Covassin et al. (2011) where hyperarousal was reported as a reliable marker of response inhibition impairment in those with Insomnia Disorder. As

such, this task may be most sensitive to those experiencing a particularly severe episode of insomnia with elevated physiological arousal. Our sample contained large inter and intra-variability of sleep disturbance which can affect the sensitivity of paradigms for detecting subtle cognitive impairment (Tucker et al., 2007).

Analysis of self-report measures provides evidence that increased poor-sleep covaries with greater anxiety and current sleepiness (e.g. Lund, Reider, Whiting & Prichard, 2010). However, it is surprising that poor-sleep was not associated with poor attentional control and reported cognitive failures. Benitez and Gunstad (2012) reported significant associations between poor-sleep (PSQI) in young adults and poor executive control on a trail-making task. Therefore, it is possible that the items contained in the ACS and CFQ (more cognitively focussed, and more behaviourally focussed respectively) may not be sensitised to the type of cognitive/motor control impairment reported in poor-sleep quality. The lack of associations found between the ACS, CFQ and the ANT, SAT and SSP also indicates that the self-report measures capture different aspects of cognitive control to the objective measures.

Our results challenge a popular theory that sleep loss selectively impairs cognitive functions reliant on the PFC (Harrison & Horne, 2000; Harrison et al., 2000). This study aligns with Tucker et al. (2010) where key functions of executive control (working memory efficiency and resistance to proactive interference) were not 'directly' affected by chronic sleep loss. Here we suggest that response inhibition and cognitive conflict resolution are not selectively impaired in poor sleep. Our results are consistent with the theory of state-instability (Lim & Dinges, 2008) which posits that moment-to-moment attention is fundamentally affected by sleep loss.

Whilst very few studies have attempted to discriminate between executive and non-executive components of neurocognitive tasks, it is likely that by further burdening cognitive resources through increasing task complexity and duration, compensatory strategies will be weakened and such associations may be revealed (Espie & Kyle, 2008). The repeated presentation of stimuli in the ANT, SAT and SSP may promote compensatory strategies which result in faster, more correct responses and obscure natural cognitive decline following sleep loss (Horne, 2013). Indeed, the relatively good level of performance associated with poor sleep may reflect sufficient motivation and available resources to overcome cognitive dysfunction for a short laboratory testing session.

It is also important to consider that laboratory tests of executive control are limited in the extent to which they test the functionality of cognition in day-to-day life (Horne, 2013).

Recently novel decision making tasks involving unpredictable conditions and outcomes have revealed executive impairment associated with sleep loss (Libedinsky et al., 2013). Therefore, it remains a matter of debate whether executive performance deficits reported in poor sleeping populations are a result of sleepiness and associated performance variability or whether sleep loss selectively impairs executive control.

Future replication studies using the ANT, SAT and SSP can provide additional data from poor sleeping populations which may help to clarify these issues. For example, it would be interesting to investigate whether the same profile of cognitive impairment is also found in those with Insomnia Disorder, or whether executive control impairment is a more reliable feature of the latter population . Alongside sensitive self-report measures of cognitive control in poor sleep, it will be beneficial to include an additional sleep measure for assessing daytime cognitive aspects of poor-sleep, (e.g. distress about daytime impairment). Questionnaires such as the Insomnia Severity Index (Bastien et al., 2001) may reveal associations with executive performance if it is true that anxiety and subjective aspects of insomnia (e.g. concern over daytime function) are more reliably associated with executive control impairment (Benitez & Gunstad, 2012). Furthermore, there is evidence that many young people meet DSM-5 criteria without reporting 'insomnia' suggesting the importance of measuring disorder processes in this population (Taylor et al., 2013). These measures could be further complemented by objective measures of sleep (e.g. polysomnography) which capture variability in sleep patterns. Future studies may also benefit from the inclusion of a state measure of anxiety in order to assess current situational and performance related concerns which could affect performance at time of testing.

This novel study investigated executive control in poor-sleeping individuals using tasks that provide dissociation of executive performance from lower-level attentional processes. Associations between subscales of the PSQI and objectively measured alerting are consistent with findings that poor sleep preferentially affects maintenance of attention (Lim & Dinges, 2010). Findings further suggest that executive control is not impaired in poor sleepers.

Chapter Three

Insomnia Symptoms and Daytime Intrusive Thoughts

In Chapter Two it was established that reported poor sleep quality is associated with mild attentional impairment on neurocognitive tasks. The investigation of attentional control in insomnia symptoms was then extended to an important aspect of Insomnia Disorder; namely thought intrusions. Historically, insomnia has been characterised as "the result of an inability to turn off intrusive, affectively-laden thoughts and images at bedtime" (Borkovec et al., 1983, p.9). Whilst this definition captures the important nocturnal aspect of insomnia, the evidence base for parallel daytime processes is increasing, and supports the reconceptualization of insomnia as a '24 hour disorder' (ICSD, 2nd edition, 2005; Diagnostic and Statistical Manual of Mental Disorders, 4th edition., text revision, 2000). Worrisome thought is a central feature of several models of insomnia maintenance (e.g. Espie, 2002; Harvey, 2002; Lundh & Broman, 2000; Morin, 1993) and involves persistent intrusive thought; "spontaneous, unwanted, unbidden, uncontrollable and discrete thoughts that are attributed to an internal origin" (Harvey, Tang & Browning, 2005, p.599).

It is important to determine whether intrusive thought is associated with insomnia symptoms because clarifying the processes involved in the transition from poor sleep to insomnia has been highlighted as a priority in order to improve treatment for the condition (Ellis et al., 2012). Sleep-related worry has been investigated as a process variable within CBT-I which may improve key symptoms (self-reported insomnia severity, total sleep time and wake after sleep onset) in those with a complaint of three to 12 months (Sunnhed & Jansson-Frőjmark, 2014). However, it is unclear whether more general, intrusive negative cognition is also a feature of an initial sleep complaint that should be targeted.

Cognitive hyperactivity in the pre-sleep period is the main cause of sleeplessness within clinical populations, occurring 10 times more frequently than somatic arousal (Lichstein and Rosenthal, 1980). Cognitive activity is worrisome in nature, involving rehearsal, metacognition, problem solving and sensory processing (Harvey, 2005). Nelson and Harvey (2003) found that those with insomnia reported greater verbal thinking, more negatively valenced pre-sleep images compared to good sleepers, and fewer random/non-connected images. These results provide evidence of reduced cognitive and affective activity (i.e. cognitive 'flitting') in good sleepers compared to those with insomnia, who instead engage with worrisome thoughts and negative imagery which can exacerbate physiological and emotional arousal.

In severe cases, thought intrusions relate to fears and concerns about the effects of sleeplessness (Borkovec, Lane & VanOot, 1981). In a novel experiment, Wicklow and Espie (2000) investigated 'live' spontaneous pre-sleep thought processes using a voice-activated audiotape and revealed common intrusions categorised under 'active problem solving', 'present state monitoring' and 'environmental reactivity.'

Experimental studies of cognitive activity in the pre-sleep period suggest a causal relationship between increased cognitive activity and insomnia. In healthy individuals, inducing performance anxiety (threat of giving a speech) before sleep-onset results in longer self-reported sleep onset latency (SOL) (e.g. Tang & Harvey, 2004), longer objective SOL and increased night-time awakenings (Hall et al., 1994). Likewise, tasks that distract insomnia patients from engaging with intrusive thought before bedtime have successfully shortened SOL (e.g. using imagery; Harvey & Payne, 2002). Thus, limiting attentional resources required for worry might quicken sleep onset in individuals with insomnia.

Beyond nocturnal cognitive hyperactivity, aetiological models suggest that poor cognitive/attention control and intrusive thoughts are important daytime features of insomnia. Consistent with evidence that distractability ('mindwandering') is a dominant predictor of negative affect (Killingsworth & Gilbert, 2010), the experience of poor attention control and thought intrusions in poor sleepers might confer risk for comorbid mood and anxiety disorder. However, the effects of insomnia symptoms on daytime thought intrusions have not been examined. Accordingly we investigated the frequency and valence of thought intrusions in a sample of young adults who naturally and markedly vary in their sleep quality.

We measured the frequency and valence of thought intrusions using an established measure of thought intrusions developed by Ruscio and Borkovec (2004) and adapted by Hirsch et al., (2009). This task has been widely used to examine thought intrusions and worry in non-clinical groups (Krebs et al., 2010), individuals with elevated worry, and generalised anxiety disorder (Hayes et al., 2010; Hirsch et al., 2009). The thought intrusions task measures both resting level intrusions, and intrusions that follow a period of active worry on a topic chosen by the participant. This paradigm overcomes limitations associated with self-report questionnaires that ask participants to retrospectively report the frequency with which they worry about a pre-determined set of topics chosen by researchers, and that might be confounded by recall bias.

This study is the first to test whether insomnia symptoms are positively associated with greater daytime thought intrusions, and in particular negative thought intrusions. As a

secondary analysis, the uniqueness of this relationship beyond the effects of anxiety and self-report deficits in cognitive control was assessed (because both variables are known to be elevated in insomnia, and associated with negative thought intrusions).

Method

Participants. Participants were 109 (82% F) university students with a mean age of 20.70 years (*SD*= 4.56) who received course credits for participation. Participants provided written informed consent prior to participation. All study procedures were reviewed and approved by the Ethics Committee at the School of Psychology, University of Southampton, UK.

Design and procedure. A cross-sectional research design examined associations between objectively measured thought intrusions and widely used self-report measures of insomnia severity (Insomnia Severity Index – ISI) and poor sleep quality (PSQI). Secondary self-report measures of attentional control (ACS), trait anxiety (STAI-T) and current sleepiness (SSS) were also included.

Self-report measures.

Insomnia Symptoms. The Insomnia Severity Index (Bastien et al., 2001) measures perceived severity of insomnia over the past two weeks. The ISI has the advantage of being both brief and partly based in DSM-5 criteria and it is accepted as the most sensitive measure to daytime impairment and affective function associated with insomnia (Bastien et al., 2001). It measures degree of satisfaction with sleep, interference with daytime function, noticeability of impairment and distress associated with the complaint. Responses are made on a 0-4 scale with higher scores reflecting a greater problem over the past two weeks. Total scores range from 0 to 28 where 8-14 is considered 'subthreshold- insomnia' and 14-21 is considered 'mild-clinical insomnia.' The scale has previously reported good internal consistency and test-retest reliability (Bastien et al., 2001), with a Cronbach's alpha of .84 in the current study. Convergent validity measured by the correlation coefficient between items on the questionnaire measure and corresponding variables from sleep diaries is sound (Bastien et al., 0.32-0.91). In previous studies with student populations, mean scores of between 7.14 and 11.23 have been reported (Chung, Kan & Yeung, 2011; Wilkerson, Boals & Taylor, 2011).

Three questions relate to problems with; sleep onset, sleep maintenance, and early awakening. The remaining four questions relate to; how satisfied individuals are with their sleep, how noticeable they consider their quality of life to be affected by insomnia, how worried they are about insomnia and how much it interferes with daily functioning.

Previous principal component analyses have suggested a one factor structure accounting for 68.99% of total variance in ISI scores (Sierra, Guillén-Serrano & Santos-Iglesias, 2008), a two factor structure involving 'severity of sleep difficulties' (31.9% variance) and 'impact of sleep difficulties' (30.5%) (Savard, Savard, Simard & Ivers, 2005) and a three factor structure involving 'interference with daily functioning' (26% variance), 'noticeability of impairment' (20% variance) and 'level of distress' (20% variance) (Bastien et al., 2001). A two factor structure was considered to be the most appropriate structure for two reasons. Firstly, items fall coherently and intuitively into either 'night-time problems' or 'daytime impairment.' Secondly, studies reporting two factors have consistently identified the same components whilst three factor structures have multiple item loadings which are difficult to interpret theoretically.

Sleep Quality. The PSQI (Buysse et al., 1989) reported in detail in Chapter Two measured sleep quality over the past month. Chronbach's alpha was .75 in this study.

Trait Anxiety. The STAI-T (Spielberger et al., 1983) reported in Chapter One measured anxiety as a general disposition. A Chronbach's alpha of .93 was found in this study.

Current Sleepiness. The SSS (Hoddes et al., 1973), reported in Chapter Two assessed current levels of sleep propensity.

Attentional Control. The ACS (Derryberry & Reed, 2002) reported in Chapter Two measured the ability to 'focus', or maintain attention on a given task, and the ability to 'shift', or redirect attention to a new stimulus or between multiple competing tasks. Chronbach's alpha was .75 in this sample.

Cognitive Errors. The CFQ (Broadbent et al., 1982) previously reported in Chapter Two, measured the trait-like tendency to make mistakes in perception, memory and motor function. This measure had a Chronbach's alpha of .88 in this study.[6]

The thought intrusions task

Consistent with Hirsch et al. (2009) the thought intrusions task contained three stages; an initial five minute breathing focus, a five-minute worry period and a five-minute post-worry breathing focus. During pre-worry and post-worry breathing focus periods participants were instructed to focus attention on their breathing. If thoughts wandered away from their breath participants were instructed to redirect attention back to their breathing. Within pre- and post-worry periods 12 beeps were presented (between 20-30

[6] MEQ scores (M= 44.36, SD=8.40, intermediate type).

seconds apart). On hearing a beep participants were asked to state one of four response options; if focussing on their breath as instructed, then participants reported "Breathing". However if when probed their attention had wandered from their breath, then they briefly described the content of the thought, and whether it was positive, negative or neutral (e.g. "Looking forward to seeing my friends; positive", "Worried I won't meet my deadline; negative"). The experimenter logged all thought intrusions as they occurred and confirmed the reported valence with the participant at the end of the study.

In between breathing-focus periods participants were asked to identify a current worry. This was briefly discussed with the experimenter to ensure that the worry was characterised by concern about a future event (rather than a retrospective depressive concern). Over 50% of our sample identified a current worry related to workload, deadlines or exam pressure. Other worries included finance, relationships and change of residence. Participants rated (0-100) their worry with respect to i) how likely is this to happen? (Extremely unlikely to extremely likely), ii) how catastrophic would it be? (Not at all catastrophic to Extremely catastrophic), and iii) How well do you think you would cope? (Not at all well to Extremely well). Across the sample VAS ratings confirmed that the self-referential worry topics chosen by participants involved high levels of uncertainty as to likelihood of outcome (M=50.83, SD=21.00), would be moderately catastrophic (M=54.74, SD=25.09), and difficult to cope with (M=43.66, SD=22.49). Participants focused on the worry for five minutes.

Participants completed the ISI, PSQI, ACS, CFQ and STAI two days prior to attending a test session in which they completed the sleepiness measure (SSS) and the thought intrusions task. Following standardised instructions (see Hirsch et al., 2010) participants completed a practice breathing focus trial that lasted 45 seconds and contained three thought sampling beeps, and the thought intrusions task in full. [7]As part of the debrief participants read some amusing news stories to reduce any residual negative effects of the worry induction.

Results

Descriptive statistics and correlations between self-report measures and performance measures are presented in Table 3.1. According to ISI scoring guidelines, 53.2% of our sample experienced insomnia symptoms over the past two weeks. Of these, 79.31% are categorised as having "sub-threshold insomnia" and the remaining 20.69% as

[7] Bonferroni correction for multiple comparison testing involves adjusted significance level $p<.01$ (based on key analyses between two sleep measures and four levels of dependent variable).

having "mild clinical insomnia." According to the PSQI, 72.1% of the sample was classified as "poor-sleepers." Levels of trait anxiety, attentional control, and current sleepiness fell within the normal range (e.g. Taylor et al., 2005; Derryberry & Reed, 2002; Alapin et al., 2000). In order to correct for multiple comparisons, a value of $p<.01$ was used to detect statistically significant associations.

Associations between sleep quality, trait anxiety, attention control and sleepiness.[8] Poor sleep (GPSQI) and insomnia severity (ISI total) were associated with increased trait anxiety, where daytime impairment subscales of these measures were the strongest contributors. Both GPSQI and ISI total scores were further associated with reduced attention control and increased sleepiness where associations were strongest for daytime impairment (ISI daytime, PSQI day dysfunction) and moderate with sleep quality and sleep disturbance (PSQI). Following the worry period there was a reduction in breathing focus ability [pre-mean = 8.72 (SD = 1.75), post-mean = 8.38 (SD = 2.15), (t (108)=2.61, $p<.05$]. Participants were also more likely to experience negative thought intrusions [pre-mean = 0.90, (SD = 1.1), post-mean = 1.58 (SD = 1.44), t (108) = 4.90, $p<.001$] and less likely to experience positive thought intrusions [pre mean = 1.25 (SD = 1.23), post-mean = 0.83 (SD = 1.08), t (108)=3.49, $p<.01$]. Likewise the worry period increased subjective feelings of worry (pre-worry mean= 20.33, post-worry mean = 42.67, t (106)= 7.79, $p<.001$) and anxiety (pre-anxiety mean= 17.04, post-anxiety mean = 35.39, $t(106)= 8.18$, $p<.001$).

Associations between sleep quality, anxiety, attention control and thought intrusions at baseline. During the pre-worry period the ability to focus attention on breathing was negatively associated with nocturnal symptoms (ISI), sleep disturbance (PSQI) and daytime dysfunction (PSQI) (concurrently, increased thought intrusions across valence were observed). Trait anxiety was not significantly correlated with overall thought intrusions during the pre-worry period.

Frequency of negative thought intrusions was positively associated with insomnia severity (ISI total) and poor sleep quality (GPSQI), and also associated with trait anxiety and current sleepiness. Follow-up partial correlations confirmed that poor sleep was significantly associated with negative thought intrusions beyond the effect of current sleepiness (ISI: $r=.21$, $p=.03$, GPSQI: $r=.23$, $p=.02$) and trait anxiety (ISI: $r=.20$, $p=.04$, PSQI: $r=.23$, $p=.02$). Self-report attentional control was unrelated to the ability to focus on breathing.

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[8] MEQ scores associated with sleep quality ($r=-.19$, $p=.06$), and insomnia symptoms ($r=-.17$, $p=.08$) such that being an evening type predicted poorer sleep. Scores did not correlate with any dependent measures.

Self-report measures were unrelated to positive and neutral thought intrusions in this period.

Associations between sleep quality, anxiety, attention control and thought intrusions following worry. Following the five-minute worry period (see Appendix J for categories of 'worry' and related frequencies), breathing focus ability remained negatively associated with poor sleep quality (disturbance) and daytime dysfunction due to poor sleep, and in addition was further associated with insomnia severity and trait anxiety. Partial correlations confirmed that insomnia severity was not associated with greater thought intrusions, beyond the effect of anxiety (ISI: r=-.11, p=.27).

Contrary to findings in the pre-worry (baseline) period, there were no associations between negative thought intrusions and measures of sleep. Instead, frequency of negative thought intrusions remained moderately associated with trait anxiety, and further associated with reduced self-report attention control and greater daytime sleepiness

Table 3.1

Sample characteristics and associations between sleep, anxiety and thought intrusions

Measures			SSS	ACS	CFQ	STAI-T	Baseline breathing focus	Baseline negative intrusions	Post-worry breathing focus	Post-worry negative intrusions
	M	*SD*								
ISI total	8.21	4.50	.28**	-.28**	.27**	.47***	-.18 (p=.06)	.25**	-.21*	.19 (p=.05)
ISI daytime	5.15	3.00	.28**	-.32**	.28**	.54***	-.14	.20*	-.18	.14
ISI nighttime	3.06	1.98	.19	-.15	.20*	.27**	-.20*	.27**	-.20	.13
GPSQI	6.55	3.18	.31**	-.27**	.28**	.47***	-.16	.26**	-.14	.13
PSQI quality	1.26	.68	.28**	-.24*	.23*	.35***	-.10	.27**	-.02	.05
PSQI disturbance	1.23	.47	.24*	-.26**	.30**	.45***	-.22*	.22*	-.22*	.13
PSQI latency	1.58	.87	.23*	-.13	.11	.19	-.05	.14	-.06	.17
PSQI duration	.29	.63	.05	-.01	.05	.19	-.17	.18	-.15	.14
PSQI efficiency	.87	1.03	.17	-.13	.19	.34**	-.09	.16	-.11	.02
PSQI meds.	.17	.49	.31**	-.02	-.01	.06	-.05	-.04	.06	.04
PSQI day dysfunction	1.15	.71	.31**	-.42***	.36**	.54***	-.28**	.20*	-.20*	.19*
SSS	3.03	.90	-	-.29**	.21*	.33**	.08	.23*	-.10	.14
ACS	47.78	8.43	-	-	-.57**	-.57***	.13	-.12	.20*	-.40**
CFQ	46.11	14.87	-	-	-	-.60**	-.12	.10	-.14	.20*
STAI-T	40.17	10.54	-	-	-	-	-.15	.21*	-.21*	.37**

Note. N: thought intrusions task=109, ISI=109, PSQI=104, SSS=109, ACS=103, CFQ=109, STAI-T=105. *p<.05, **p<.01,*** p<.001

Discussion

The role of thought intrusions in insomnia is poorly understood and limited to broad discussions of cognitive hyperactivity and negative cognitive activity in the pre-sleep period. Furthermore these observations have been primarily in those with clinical insomnia and where confounding variables (such as comorbid mood and anxiety disorder) might mask associations between poor sleep and thought intrusions.

This is the first study to examine on-line, daytime intrusive thought associated with insomnia symptoms using the thought intrusions task. We revealed unique, positive associations between poor sleep (over the past month), insomnia severity (over the past two weeks) and negative thought intrusions at baseline (prior to explicitly activating worry). Night-time and daytime aspects of poor sleep contributed equally to these associations, suggesting that negative thought intrusions are related to both sleep disturbance and its daytime consequences in this population, beyond the current level of reported sleepiness. Consistent with previous findings (Hirsch et al., 2009), the worry period increased feelings of anxiety and worry, increased negative thought intrusions and reduced breathing focus. Trait anxiety and self-report attention control (but not sleep measures) were strongly associated with negative thought intrusions following the period of active worry.

Why might sleep quality be associated with increased negative thought intrusions in the pre (but not post) worry period, whereas anxiety is more strongly associated with negative intrusions after a period of worry? It is likely that the worry period activated concerns that were particularly salient for those with elevated trait anxiety, and which remained primed for re-activation during the post-worry breathing focus period (consistent with classification systems that emphasise *persistent* worry as a core symptom of generalized anxiety disorder e.g. DSM-5, 2013). Conversely disturbed sleep was associated with the spontaneous activation of negative thoughts, consistent with evidence of negative thoughts in the pre-sleep period (Harvey, 2002).

We also revealed significant negative associations between both night-time and daytime aspects of poor sleep and the ability to focus and maintain attention on breathing at baseline (however, these associations were only observed for 'disturbance' and 'daytime dysfunction' components of the PSQI and night-time symptoms of the ISI). Surprisingly, we did not find an association with trait anxiety. Following the worry period, associations between insomnia severity and breathing focus were strengthened, as too were associations with trait anxiety. These results highlight the high level of shared variance between anxiety and poor sleep, and "persistent" worry.

Interestingly, we did not find associations between self-reported attentional control, cognitive errors and breathing focus/negative thought intrusions at baseline, although these were revealed in the post-worry breathing period. The ACS asks individuals to report on their ability to control attention in situations where it may be challenged, e.g. "When I am working hard on something I still get distracted by events around me". Thus associations between self-report ACS and behavioural measures of attention control (e.g. thought intrusions) might occur only during conditions of high cognitive load/distractibility e.g. following a period of active worry. Similarly, the CFQ with a focus on perceptual, memory, and motor mistakes, may not be sensitive to the moment to moment attentional control tested in the thought intrusions task.

The results of this study are important for several reasons. Using a novel experimental task, more frequent thought intrusions, (particularly negative intrusions) were associated with naturally disturbed and variable sleep in young adults who commonly experience alterations to sleep environment, shifts to the timing of sleep onset and acute stress (Roth & Roehers, 2003). Due to environmental and biopsychosocial factors, this population is 'at risk' for development of more persistent insomnia, and increased negative thought intrusions may be a marker of this.

Results are consistent with findings from acute sleep deprivation studies that reveal dysfunction in key emotion neural networks implicated in emotion activation (e.g. amygdala) and regulation (e.g. prefrontal-cortex) following sleep loss (Yoo et al., 2007; Tempesta et al., 2010). They are also consistent with aetiological models of insomnia which consider heightened cognitive arousal as important within insomnia development. Finally, results show that in a 'poor-sleeping' population, daytime intrusive thoughts are not characterised by sleep-related concerns, as is commonly observed in clinical populations, but rather by broader concerns that characterise generalized worry.

Although we have discussed the possibility of negative thought intrusions as a risk factor for an escalating insomnia complaint, the cross-sectional design means that we cannot discriminate between factors which may predispose to poor-sleep, those which may perpetuate poor sleep, and those which arise from an acute poor-sleep episode. Nevertheless, by assessing sleep over the past month in non-treatment seeking individuals, it is likely that we predominantly measured initial acute sleep disturbance.

The thought intrusions task provided a sensitive measure of daytime thought intrusions in a poor sleeping population, before and after a period of worry. Thought samples were spontaneous and unique at the level of the individual, overcoming limitations

of previous analogue studies which used a standardised stressor to evoke state worry (i.e. speech threat) in order to measure associated cognitive activity. This study is novel and requires replication within other poor-sleeping populations, including those with a clinical sleep disorder. Future studies could extend the use of this task to profile thought intrusions across varying levels of poor sleep/insomnia severity in order to test theories relating to the development of insomnia which emphasise the increasing importance of sleep-related worry (Harvey 2002; Espie, 2002). Furthermore, in line with emerging evidence that attention training towards benign appraisals of threatening stimuli can reduce worry and anxiety in high-worriers (Hirsch et al., 2009), the thought intrusions task may be useful within CBT-I as an outcome tool for measuring change to sleep-related attentional bias, which is a reliable feature of insomnia disorder (Marchetti et al., 2006).

Currently, our understanding of factors involved in the transition from poor sleep to insomnia is limited. Our finding that negatively valenced thought intrusions and poor attentional control are associated with poor sleep and insomnia symptoms highlights the potential importance of this mechanism in the development of sleep disorders.

Chapter Four

Insomnia Symptoms and Autonomic Reactivity

Historically, researchers attempted to establish insomnia as a disorder of cognitive hyperarousal or a disorder of physiological hyperarousal. Very few studies considered how both cognitive and physiological aspects of the disorder may operate together to escalate and maintain the complaint (Harvey, 2002). It is now accepted that the psychophysiological nature of insomnia disorder reflects over-arousal within inter-dependent cortical, cognitive, emotional and autonomic systems (Baglioni, Spiegelhalder, Lombardo & Riemann, 2010) and that these process are mutually exacerbating. That is, if someone is cognitively aroused (e.g. worrying) they will become physiologically aroused and that if they are physiologically aroused (e.g. heart pounding) they will seek an explanation and become cognitively aroused (Harvey, 2002). Extensive research conducted in recent years has established cognitive *and* physiological arousal as fundamental to chronic insomnia (Riemann et al., 2010), e.g. excessive, negatively-toned sleep-related cognitive activity, elevated heart rate and skin conductance at nighttime (Waters et al., 1993). However we do not know about the physiological daytime characteristics of insomnia symptoms which are likely to be closely related to the stress system and may feed into cognitive aspects of the complaint.

In Chapter Three we revealed associations between insomnia symptoms in healthy young adults and spontaneous activation of negative intrusive thoughts during a goal-directed activity. Similarly, Chapter Two revealed mild attentional dysregulation associated with daytime impairment in those with poor sleep. In line with these observations of cognitive dysfunction, and consistent with proposed physiological underpinnings of acute insomnia (Harvey et al., 2014) it was considered important to understand the relationship between insomnia symptoms and daytime autonomic function. In particular, physiological response to situational stress was considered important because poor sleeping young adults most often report perceived or actual 'stress' as the reason for current sleep problems (Taylor et al., 2011). Irritability, and emotional reactivity follow sleep loss (Baglioni et al., 2010) may further contribute to heightened arousal (physiological and cognitive), particularly in response to novel stressors, thus maintaining insomnia symptoms. Stress-reactivity is, therefore, likely to be an important feature of disturbed sleep which may have consequences for the way everyday challenges are performed.

There is increasing evidence from human and rodent studies to suggest that reactivity of the stress system may be fundamentally altered following both total sleep deprivation

(one to two days) and following a week of sleep restriction (Meerlo, Koehl, van der Borght & Turek, 2002). As highlighted in Ellis et al. (2012) stress can accumulate over a prolonged period to result in insomnia, often before individuals are consciously aware of the effect of sleep loss on their daytime function (Taylor et al., 2010). In the same way, chronic sleep loss may induce neurobiological changes that are not immediately evident but accumulate over time and increase risk for cardiovascular diseases as well as psychiatric disorder (Meerlo, Sgoifo & Suchecki, 2008). Autonomic reactivity to stress is likely to be an important variable for investigation in acute insomnia symptoms because several studies suggest that it is more strongly related to disrupted and discontinued sleep (seen in young adult populations) than sleep deprivation as measured by duration of continuous wakefulness (Irwin, Thompson, Miller, Gillin & Ziegler, 1999; Tiemeier, Pelzer, Jonck, Moller & Rao, 2002; Ekstedt, Akerstedt & Soderstrom, 2004).

A decrease in sympathetic nervous system input during night-time co-occurs with an increase in parasympathetic nervous system activation to initiate sleep (Burgess, Trinder, Kim & Luke, 1997). In sleep deprivation an *increase* in sympathetic activity has been observed and directly associated with increased heart rate and blood pressure, reaching levels seen during wakefulness (Irwin et al., 1999; Lusardi, Mugellini, Preti, Zoppi, Derosa & Fogari, 1996; Tochikubo, Ikeda, Miyajima & Ishii, 1996). These results suggest an increase in baseline autonomic activity following sleep deprivation. Further stress upon this basal sympathetic activity, therefore, may result in a heightened autonomic response (Meerlo et al., 2008). Whilst controlled studies have investigated sleep deprivation and its effect upon basal activity of stress systems, very few studies have attempted to establish how reactivity to new stimuli/stressors is affected. The difficulty, however, in using deprivation manipulations to investigate autonomic stress reactivity is that it becomes impossible to separate out the stress associated with the sleep deprivation manipulation itself from the natural effect of sleep loss on stress reactivity As such, these studies are confounded and have limited external validity (Meerlo et al., 2008).

It is understood that the relationship between activity of stress systems and sleep loss is complex and bidirectional such that "in everyday life, stress and insufficient sleep often go hand in hand and make up a vicious circle in which stress keeps a person awake and the inability to sleep may increase the feeling of stress" (Meerlo et al., 2008, p.198). This is consistent with many aspects of insomnia where cause and consequence are too intimately linked to be easily disentangled. Within Spielman's 3P model of insomnia development (1987; see Chapter One for detailed description), stress reactivity in the form of

sleeplessness could be a predisposing factor for insomnia and/or a consequence of insomnia. Drummond, Smith, Orff, Chengazi and Perlis (2004), propose that the increased arousal seen in insomnia populations is a symptom of insomnia and/or a compensatory mechanism to perform daytime tasks. Indeed, there is much support for compensatory effort in this population (Dorrian & Dinges, 2003). On the other hand, life stress and a predisposition for arousability have been reported as amongst the strongest predictors of insomnia development in epidemiological studies (LeBlanc et al., 2009; Riemann et al., 2010; LeBlanc et al., 2007). In their recent psycho-bio-behavioural model of vulnerability to insomnia, Harvey, Gehrman and Espie (2013) place stress reactivity at the centre of the model. According to this model, neuroticism and 5HTTLPR increase stress reactivity leading to sleep disruption (5HTTLPR may be related to neuroticism, as might stressful life experiences). Following emotion-focused coping, neuroticism may also increase negative associations with disrupted sleep further escalating arousal. Although this model requires further validation, it emphasises the importance of capacity to effectively manage stress in order to protect good sleep.

Given that arousability in healthy individuals could reflect a vulnerability to insomnia-related hyperarousal and that repeated acute episodes of insomnia may predispose for chronic insomnia through increased sympathetic nervous system activation (Harvey et al., 2013) it is unsurprising that hyperarousal is a defining 24 hour feature of chronic insomnia (Riemann et al., 2010). Those with chronic insomnia show increased and sustained activation of the central nervous system (CNS) as indexed by cortisol output, increased activation in neural emotion networks and increased heart rate (Vgontsaz et al., 1998; Vgontsaz et al., 1998; Nofzinger et al., 2004; Covassin et al., 2011; Bonnet & Arand, 1998). Although it is currently unclear whether and how hyperarousal features in those with acute insomnia episodes, these studies emphasise the psychobiological underpinnings of insomnia in its chronic form, where the interplay between cognitive and physiological arousal is dominant (Harvey, Gehrman & Espie 2013).

In healthy participants, Bonnet and Arand (2003) showed that stress disrupts sleep consistently across situations. They investigated 1) the 'first night effect' where a new environment creates mild stress 2) the effect of 400mg caffeine intake 30 minutes prior to sleep onset causing mild physiological stress, 3) three hour phase advance where participants are required to sleep three hours earlier which is normally prevented by natural circadian rhythm and 4) six hour phase advance. Good sleepers with 'first night' disrupted sleep also had greater sleep disruption across the remaining three situations. Increased heart

rate, increased low-frequency (sympathetic nervous system activation), and decreased high-frequency (decreased parasympathetic nervous system activation) electrocardiogram power were found in 'situational insomniacs' only, defined as those with sleep disruption in situation 2,3 and 4 compared to baseline (night two of lab sleep). Higher Multiple Sleep Latency Test (MSLT) scores confirmed 'situational insomniacs' as less able to readily fall asleep and as being more 'aroused' than those whose sleep was robust across all conditions.

Drake, Jefferson, Roehrs and Roth (2006) reported that healthy individuals for whom insomnia is a feature of stress reactivity had higher scores on the Multiple Sleep Latency Test (MSLT) supporting Bonnet and Arand (2003). This finding was further strengthened by results from polysomnography (PSG) recording where latency before persistent sleep (i.e. first 20 continuous epochs of sleep) was greater on the 'first night' of lab sleep in those with increased insomnia stress reactivity, even when excluding those with previous reported insomnia episodes. These results could not be explained by differences in basal sleep systems because two week diary measures confirmed no difference in baseline sleep between high and low insomnia responsiveness groups. Interestingly, the between group difference in MSLT scores became non-significant after controlling for previous insomnia episodes. As highlighted by the authors, this is consistent with knowledge that a past episode of insomnia is the greatest predictor of a new episode (LeBlanc et al., 2009; Morin et al., 2009). Furthermore, results suggest that in populations where disrupted sleep is a re-current experience, stress reactivity could be a particularly salient aspect of daytime functioning.

Despite growing evidence for increased reactivity to stress in poor sleeping samples, a review by Meerlo and colleagues (2008) highlighted several studies which did not observe change in autonomic function in poor sleep. Other studies reported sympathetic nervous system activation increased beyond the level seen during wakefulness or autonomic activity scarcely elevated from sleep. The authors explain that autonomic responding to stress is dependent upon several factors which include physical demands, cognitive activation and emotional responding. They emphasise the importance of using appropriate tests of stress which induce not only physiological arousal but realistic concern over the ability to cope effectively.

In general it is not feasible to perform experimental studies which inflict severe stress on human subjects which means there are limited studies informing us of stress reactivity in poor sleep. The studies which have used acute, mild stress manipulations have exclusively focused upon nocturnal aspects of poor sleep as dependent measures (e.g. time taken to fall

asleep). Psychosocial stressors such as the threat of giving a speech upon awakening from a nap suggest that increased cognitive and physiological arousal disrupt normal sleep processes (Gross & Borkovec, 1982; Tang & Harvey, 2004; Hall, Buysse, Reynolds, Kupfer and Baum, 1996). However, the validity of these studies is questionable given the underlying assumption that delivery of a speech is equally threatening to all participants. In Gross and Borkovec (1982), for example, the researchers did not verify whether or not the manipulation actually increased arousal.

Studies where the stressor is anticipated permit individuals to commit extra cognitive effort to coping. Timeframes are known allowing for rationalisation and planning which may attenuate the stress response (Lazarus & Folkman, 1984). Undoubtedly these studies provide insight into cognitive aspects of induced stress (i.e. worry) which interfere with nocturnal aspects of insomnia (i.e. SOL), however, daytime autonomic reactivity (and associated anxiety) in response to acute stress has not been examined.

A reliable determinant of sympathetic activation is physical exercise which increases both heart rate and blood pressure. Many studies have looked at autonomic activity associated with high and low intensity physical exercise (e.g. treadmill or bicycle) in sleep deprivation ranging from one to three days. There is a general consensus that physical stress of this type does not significantly affect physiological response to the challenge (Martin, 1988). However, these studies crucially revealed that cognitive and emotional perceptions of physical challenge are dramatically altered in sleep deprivation (Meerlo et al., 2008). In one study, time to exhaustion was reduced by 20% despite monetary incentives to perform (Martin & Chen, 1984), and other studies have revealed lower tolerance of exercise and perceived exertion despite normal physiological activation (Martin, Bender & Chen, 1986; Martin, 1981). Novel, candidate paradigms for the investigation of stress reactivity in poor sleep should, therefore, induce mood related change (e.g. state anxiety) as well as reliably increase key autonomic systems.

Within anxiety research, inhalation of CO_2 enriched air is a well-established and successful experimental model of anxiety which produces cognitive and physiological symptoms consistent with generalised anxiety (Seddon et al., 2011). This model translates effectively between animals and humans, where 10% CO_2 inhalation triggers freezing behaviour, reduced activity in an open-field test and greater fear conditioning in rodents and 7.5% CO_2 inhalation causes hypervigilance to threat in humans as measured by erroneous eye movements towards threat on the antisaccade task (Zieman et al., 2009; Garner, Attwood, Baldwin, James & Munafò, 2011). The model is considered to be a reliable and

valid method for investigating cognitive, behavioural and physiological aspects of anxiety disorder.

Twenty minute inhalation of 7.5% CO_2 enriched air (92.5% normal air) reliably induces self-reported state anxiety (tension and worry) in healthy individuals (Bailey et al., 2006; Bailey et al., 2009). This method also increases heart rate and blood pressure (Bailey, Argyropolous, Kendrick & Nutt, 2005; Bailey, Dawson, Dourish & Nutt, 2011; Garner et al., 2011). Based on these properties, this model has been differentiated from models which produce 'panic' like effects (e.g. 35% CO_2 model; Esquivel, Schruers, Kuipers, & Griez, 2002), and instead is considered a model of generalised anxiety. This model, therefore, is particularly appropriate for investigating stress reactivity in insomnia symptoms because the effects of inhalation involve a reliable increase in perceived state anxiety with simultaneous increases in autonomic responsivity and because insomnia symptoms are highly comorbid and bi-directional with generalised anxiety symptoms (Roth et al., 2006).

The 7.5% CO_2 model of anxiety has been used primarily for the assessment of pharmacological interventions for anxiety (see Bailey et al., 2011 for a review). In this study we respond to the call in Harvey, Gehrman and Espie (2013) for the investigation of stress reactivity in populations vulnerable to sleep disruption. The 7.5% CO_2 inhalation model was used to test whether insomnia symptoms in a healthy population are associated with increased autonomic arousal and self-reported state anxiety following acute stress in an experimental model of anxiety.

Method

Participants. Fifty six participants were recruited through Psychobook, an online service provided by the University of Southampton. Mean age of the sample was 20.42 years (*SD*=3.0) and 69.1% were female. Consistent with safety protocols for CO_2-challenge screening involved general physical and mental well-being checks based on questions from the Mini International Neuropsychiatric Interview (Sheehan et al., 1998). This was initially done via phone consultation a week before participation and then followed up at the time of testing. Exclusion criteria were: recent use of medication (eight weeks excluding paracetamol, aspirin, and contraceptive pills), pregnancy, risk of respiratory condition, risk of cardiovascular disease, risk of psychiatric illness, being under or over weight (i.e. BMI 18-28), history of drug dependence and recent alcohol consumption (checked via breath test). The study was reviewed and approved by the Ethics Committee at the School of Psychology, University of Southampton, UK.

Design. Participants completed measures after inhaling 7.5% CO_2 enriched air (21% O_2; balance N2) and normal air in a repeated measures design. Participants were blind to inhalation condition (CO_2 versus air) and inhalation order was counterbalanced across participants.

Measures

Physiological Measures. Omron-M6 monitoring devices (Medisave, UK) provided measures of heart rate, systolic blood pressure (SBP) and diastolic blood pressure (DBP). Electrocardiography with twin electrodes attached to the wrists measured heart-rate variability with a sample rate of 100Hz (MP150-amplifier and AcqKnowledge 3.8.1. software, Biopac Systems, Goleta, CA). Output for heart-rate variability used the standard deviation of peak-to-peak intervals and sympathetic-vagal system activation ratio and mean heart rate during recording.

Self-report Measures. Consistent with studies described in previous chapters, participants completed the following: STAI-T, (a = .84), the state version of the Spielberger State Trait Anxiety State (SSAI, a= .88), PSQI (a= .77), ISI (α=.88) and ACS (a=.80) – see Chapter Two for details.

Procedure. Participants attended a single test session where trait measures of anxiety, sleep quality, insomnia symptoms and attentional control were completed. Baseline levels of self-reported state anxiety and autonomic arousal (SBP, DBP and heart rate) were measured. Participants then put on an oro-nasal facemask before inhaling either CO_2 enriched air, or normal air for 20 minutes. Participants completed the second inhalation following a 30 minute rest period. This allowed any residual effects from the first inhalation to subside. Twenty four hours after the experiment participants were contacted for the opportunity to discuss any adverse effects of inhalation – none were reported. [9]

Results

CO_2-inhalation was successful in increasing both autonomic arousal and self-reported state anxiety (see Table 4.1). Systolic blood pressure, diastolic blood pressure and heart rate significantly increased from baseline to post CO_2 inhalation, and were significantly greater following CO_2 inhalation compared to air. State anxiety was greater following CO_2 inhalation compared to both baseline and post-air inhalation.

[9] Bonferroni correction for multiple comparisons involves adjusted significance level p<.006 (based on eight comparisons between sleep measures and outcome variables).

Table 4.1

Anxiety, blood pressure and heart rate (Mean and SD) at baseline and following 7.5% CO_2 and normal air inhalation

Measures	Baseline	Air	7.5% CO2	ANOVA F (1,52)
SSAI	32.80 (6.81)[a]	34.71 (7.06)[b]	47.45 (11.13)[c]	78.89*** n^2_p= .61
Systolic BP	115.04 (10.99)[a]	114.71 (9.70)[a]	126.94 (15.11)[b]	39.19*** n^2_p=.44
Diastolic BP	70.23 (8.30)[a]	73.19 (8.17)[b]	76.46 (11.58)[c]	10.76*** n^2_p=.17
HR	72.71 (11.92)[a]	72.04 (12.88)[a]	86.65 (19.02)[b]	38.77*** n^2_p=.43

Note. When comparing conditions within the same row, different superscripts (e.g. a,b,c) indicate where a significant difference was observed between condition mean values, e.g. (SSAI was significantly increased in air condition compared to baseline, and significantly increased in CO_2 condition compared to air).Differences were computed using post-hoc t-tests. $p<.05$. ***$p<.001$.

Thirty three percent of the sample were classified as 'poor sleepers' on the PSQI and 31.4% were classified as experiencing insomnia symptoms on the ISI. These variables had distributions that departed significantly from normality, therefore, non-parametric tests of association were performed. Shown in Table 4.2, poor sleep quality and insomnia symptoms were not associated with increases in physiological arousal and self-reported state anxiety despite successful induction of acute stress (see Appendix K for associations between basal autonomic activity and self-report measures). Both nightime and daytime symptoms of insomnia were associated with reduced anxiety following CO_2 relative to air inhalation. Daytime symptoms of insomnia were associated with a reduced effect of CO_2 on heart rate variability. Poor sleep quality (PSQI quality subscale) was also associated with reduced effect of CO_2 on state anxiety and trends were observed in the same direction between systolic blood pressure increase, GPSQI and PSQI subscales (disturbance, latency, duration). No associations were found between trait anxiety, attentional control and increase in physiological arousal and state anxiety. Strong associations were observed between sleep measures and trait anxiety, but not between sleep measures and attentional control.

Table 4.2

Descriptive statistics for self-report measures and Spearman's Rho correlations with difference scores between air and CO_2 conditions.

			Gas condition difference					
Measure			STAI-T	ACS	SSAI	SBP	DBP	HR
	M	*SD*						
ISI total	6.16	5.51	.46**	-.21	-.39**	.07	.07	-.28 (p=.06)
ISI daytime	3.63	3.45	.45**	-.22	-.33*	-.00	.06	-.38*
ISI nighttime	2.53	2.38	.42**	-.22	-.36*	.11	.07	-.19
GPSQI	5.83	3.56	.34*	-.06	-.19	-.07	.05	-.22
PSQI quality	1.15	.77	.45**	-.05	-.31*	-.01	-.05	-.24
PSQI disturbance	1.15	.44	.37**	-.18	-.15	-.02	.07	-.22
PSQI latency	1.52	1.05	.22	.05	-.11	-.08	.03	-.04
PSQI duration	.25	.67	-.13	.03	-.16	-.10	-.10	-.09
PSQI efficiency	.94	1.19	.12	-.08	-.16	.01	.05	-.08
PSQI day dysfunction	.85	.71	.29*	-.18	-.05	-.00	.20	-.24
STAI-T	32.70	6.16		-.19	.05	.01	-.07	-.15
ACS	48.64	7.49			.06	-.05	-.15	.14

Note. PSQI medication omitted due to null scoring. *N*: SSAI=56, SBP=54, DBP=54, HR=54, ISI=51, PSQI=51, STAI-T=56, ACS=56. * $p<.05$, $p<.01$**

Discussion

This is the first study to investigate associations between insomnia symptoms and responsiveness to an acute stressor using the 7.5% CO_2 challenge. It was predicted that insomnia symptoms would be positively associated with increased reactivity to CO_2 inhalation as measured by autonomic indices (systolic blood pressure, diastolic blood pressure, and heart rate variability) and by self-report state anxiety. The CO_2 challenge successfully increased self-report anxiety and autonomic arousal compared to both baseline and post-air-inhalation, consistent with previous studies (e.g. Bailey et al., 2005; Bailey et al., 2006). However contrary to predictions, insomnia severity (ISI) and poor sleep quality (PSQI quality subscale) were associated with a blunted (rather than enhanced) response to CO_2-challenge.

It is difficult to interpret the unexpected negative associations observed between sleep measures, state anxiety and autonomic arousal. In Chapter Two (Study One) impaired alertness was revealed to be associated with poor sleep quality. Although speculative, it is possible that reduced alertness, (a consequence of acute sleep loss) is associated with a

blunted response to CO_2 challenge via interference of the processing of stress cues. The subjective experience of sleepiness and compensatory effort to overcome attentional lapses might override the natural effects of CO_2 challenge on autonomic function. In the same way, insomnia symptoms could also affect the accurate processing and appraisal of somatic cues used to determine current mood, e.g. my heart is racing, therefore I must be anxious. Alternatively, poor sleepers may not experience/interpret increased autonomic activity as synonymous with state anxiety. If sleep debt results in rapid increases to HR and BP, poor sleepers may be desensitised to the effects created by the CO_2 challenge (i.e. they are habituated to sensations related to rapid increases to HR and BP), reporting lower levels of anxiety.

Interestingly, however, insomnia symptoms were not associated with basal levels of autonomic activity. Given that previous research suggests elevated basal autonomic activation following sleep deprivation (Irwin et al., 1999) which is compounded by additional stressors (Meerlo et al., 2008), the explanations offered above may not sufficiently account for our findings. Levels of poor sleep and insomnia symptoms in this sample were lower than levels reported in Chapter Two and Chapter Three. Although increased autonomic activity was successfully induced by the CO_2 challenge, sleep debt in this sample may not have been sufficient to induce over-arousal typically associated with insomnia.

The validity of results is further questioned by the negative direction of (non-significant) trends observed between arousal indices, anxiety and attentional control. Previous work established reliable positive associations between trait anxiety and dependent measures on the CO_2 challenge (e.g. Garner, Attwood, Baldwin & Munafò, 2012) which informed predictions for the current study. It is, therefore, unsurprising that sleep measures were unrelated to autonomic indices given the absence of well-known associations between anxiety and CO_2 challenge (and that acute insomnia symptoms are closely associated with anxiety). Furthermore, levels of trait anxiety (STAI-T) did not predict increases in self-reported state anxiety (STAI-S) between air and CO_2 conditions despite the latter being a transient emotional state highly predisposed by the former trait (Spielberger et al., 1983).

The absence of well-established associations between self-report measures (PSQI, ISI with STAI-T, ACS) could suggest insufficient natural variation in these constructs, disguising any important associations in this population. The strict exclusion criteria for the study may have reduced the contribution of factors such as affect dysregulation and current stress which are important to the complaint of insomnia and are associated with autonomic

over-arousal (Riemann et al., 2010).[10] However, all self-report measures in this study had good internal validity despite a healthy, well-screened sample.

A larger sample size may be required for the detection of any true associations between sleep quality, insomnia symptoms, anxiety and arousal indices following CO_2 inhalation in a well-screened sample. Aside from these considerations, our results also question whether the CO_2 challenge is an effective model of stress reactivity in this population. Ellis and colleagues (2012) suggest that stressors which are not truly psychosocial in nature and do not elicit coping and appraisal processes are less likely to be effective analogues for understanding the way in which arousal characterises insomnia. Whilst this is a reasonable observation, the CO_2 model of anxiety overcomes several limitations of other stress paradigms previously used in sleep research (e.g. speech threat). The CO_2 model reliably creates real-time situational stress that individuals are unable to consciously overcome. Furthermore, this model benefits from creating both the subjective experience of increased anxiety/stress and the autonomic correlates which surpasses previous experimental stressors.

In conclusion, this was a novel first study of the predicted associations between insomnia symptoms and reactivity to an acute stress manipulation using the 7.5% CO_2 model of anxiety. In this sample, we did not find insomnia symptoms to be associated with increased responsiveness to inhalations of CO_2 as measured by autonomic monitoring and self-reported state anxiety. Replication studies within poor sleeping samples will further improve our understanding of the relationship between insomnia and stress reactivity.

[10] Analysis of variance performed on PSQI scores, (F (2,245) =6.14, p<.01), ISI scores (F (1,163)= 8.39, p<.001) and STAI-T scores (F(2,248) =12.38, p<.01) revealed significantly lower levels of poor sleep and trait anxiety in this study compared to Chapter Two and Three.

Chapter Five

Insomnia Symptoms and Risky Decision Making

In the final study of this thesis, we focused upon a particularly important aspect of cognitive function in poor sleeping young adults; risky decision making. Twenty four hours of total sleep deprivation is associated with sub-optimal decision making and a tendency to take risks (Venkatraman, et al., 2007). Whilst individuals with insomnia symptoms also report impaired decision-making as a key aspect of daytime dysfunction (Roth & Ancoli-Israel, 1999), there is a paucity of research investigating whether this involves risk-taking. Moreover, tentative explanations for risky decision-making in sleep deprivation (e.g. increased sensitivity to reward, over-optimism) (Venkatraman, Huettel, Chuah, Payne & Chee, 2011) appear incompatible with established features of insomnia (e.g. negative affect). However, the important distinctions between cognitive dysfunction associated with sleep deprivation versus insomnia have been discussed in previous chapters and are known to also be highly relevant when considering decision-making processes (Shekleton et al., 2010).

Young adults make decisions with important consequences across educational, financial and social domains. Increased incidence of reckless and intoxicated driving, illicit substance use, unprotected sex and antisocial behaviour are well-established risky behaviours in adolescence (Arnett, 1992), which for many may escalate into early adulthood (Douglas et al., 1995). Given that risky behaviour is a feature of young adulthood and that insomnia is increased in this population, it is important to establish whether risky decision-making is directly associated with poor sleep.

Risky decision making has not yet been investigated in insomnia despite being extensively researched within sleep deprivation studies. Equivocal evidence of acute sleep deprivation on decision making processes (Van Dongen, 2012) may be due to methodological issues with the tasks used. The popular Iowa Gambling Task (IGT; Bechara, Damasio, Damasio & Anderson, 1994) is purported to mimic real-life decision-making under conditions of uncertainty, rewards and punishment. In this task participants are required to continuously draw cards from four choice piles. The expected value (EV) of two decks result in high immediate reward but long-term overall losses, whilst the other two decks have low immediate reward but long-term overall gains. In this task participants should learn over time to choose cards from the decks with long-term gains rather than those yielding immediate reward (Bechara et al, 1994). Task performance, therefore, has typically been calculated using a simple difference score between the number of cards

drawn from the advantageous decks versus the disadvantageous decks. Importantly, the reward/punishment schedule is ambiguous meaning that participants are unable to easily calculate net gains and losses to guide decisions. Instead, heuristic decision-making processes are required (Dunn, Dalgleish & Lawrence, 2006). Emotion based biasing signals experienced in the body are believed to guide decision-making when participants are presented with multiple choices (somatic-marker hypothesis; Damasio, 2004 as cited in Dunn et al., 2006). Ultimately decision-making involves both higher-reasoning (e.g. logical cost-benefit analysis) and 'marker signals' – the latter being particularly important for guiding how rewarding or punishing an action may be when situations are complex and detailed analysis is not possible (Damasio, 2004 as cited in Dunn et al., 2006).

Forty nine and a half hours of continuous, monitored sleep deprivation has been related to worsened performance on the IGT compared to baseline (Killgore et al., 2006). In this study participants chose high-risk strategies, demonstrated reduced concern with negative consequences when faced with high rewards, and a lack of learning for the negative consequences when choosing between decks of cards. These findings have been replicated (e.g. Killgore, 2007; Harrison & Horne, 2000, Pace-Schott et al., 2011) and extended to suggest that decision making under uncertainty increases for gains but decreases for losses following sleep deprivation (Killgore, Grugle & Balkin, 2012). These results are in contrast to rested individuals who typically shift preference towards 'safe' decks as the task progresses showing learning of the underlying paradigm with appropriate behavioural adjustment (Bechara et al., 2005).

Evidence from this task, and variants of it, has been used to suggest that sleep deprivation disrupts the optimal balance between the 'hot' affective system and the 'cold' rational-analytical system when attributing value to respective wins and losses. The idea that emotion becomes increasingly influential in decision making following sleep deprivation is well supported (Killgore, 2006; 2011) but there is less clear evidence for the nature of this interaction. Neuroimaging research suggests that sleep deprivation causes a processing bias towards the prospect of reward (Venkatraman et al, 2007; Venkatraman et al., 2011) which may manifest in a sense of false optimism for winning (McKenna et al., 2007) despite awareness of the likelihood of adverse consequences to behaviour (Venkatraman et al, 2007; Venkatraman et al., 2011; Pace-Schott., Nave, Morgan & Spencer, 2012).

The IGT, therefore, has been valuable for demonstrating that decision making processes are altered in sleep deprivation and that individuals are willing to take greater immediate risks than when well-rested. However, this paradigm has significant limitations

(see Dunn et al., 2006 for a detailed review) highlighted by several studies where healthy individuals do not demonstrate the basic predicted behaviour believed to be promoted by the task properties (see McKenna et al., 2007).

The IGT provides a very complex and indirect assessment of risk taking, such that the computed 'difference score' between advantageous and disadvantageous decks may be too crude to capture decision-making preferences (Chiu & Lin, 2007). For example, healthy volunteers have adopted preferred strategies involving preference for one disadvantageous deck and one advantageous deck (e.g. O'Carroll & Papps, 2003, Furnie & Tunney, 2006). As attention to long-term gains does not provide an adequate explanation for decision-making on this task, recent studies have begun to disentangle and quantify the contribution of both immediate gain/loss net frequency, and long-term outcome (Hortsmann, Villringer & Neumann, 2012).

Other limitations of the IGT include its development for use within brain-injured populations, potentially restricting its sensitivity to detecting the type of decision-making impairment characteristic of sleep deprivation. Due to multiple variables involved in the task it is difficult to determine the underlying impaired cognitive mechanism responsible for a pattern of behavioural responding. Indeed, the IGT task intrinsically recruits multiple cognitive functions, including non-executive processes (Jackson et al, 2013). Furthermore, there is an intrinsic ceiling effect where individuals who learn underlying contingencies early on are penalised later in the task. Finally, there are other valid and deserving explanations for results on this task, e.g. individual differences in working memory efficiency (see Dunn et al., 2006 for a comprehensive critique of IGT methodology).

Recent research has highlighted the importance of applying sufficiently sensitive measures of executive performance to poor sleeping populations, as discussed at length in Chapter Two. For example, studies using a modified Sternberg task (working memory) and a numerosity discrimination task (Tucker et al., 2010) revealed that despite acute sleep deprivation impairing global performance, analysis of component processes led to a different interpretation of results. In the Sternberg task, working memory scanning efficiency and proactive interference inhibition remained unaffected and in the numerosity task non-decision making processes showed degradation (encoding, response execution). Evidently, other decision-making tasks are required in order to support risky decision making in poor sleeping populations.

The Balloon Analogue Risk Task (BART) (Lejuez et al., 2002) is a computerised behavioural measure of risk taking. In this task each click of the mouse increases the on-

screen balloon size, which offers greater reward. Participants must decide their threshold for risk-taking by opting to either 'cash-out' before the balloon bursts or to further increase the balloon size in order to gain greater rewards. Contrary to their initial predictions, Killgore, Kamimori and Balkin (2011) found participants to display reduced behavioural inhibition or poorer acuity in establishing a profitable threshold which resulted in increased risk taking. Interestingly, this behavioural evidence was in contrast to previous work reporting no change or reduced risk taking on the BART following sleep deprivation (Acheson et al., 2007; Killgore, 2007; Killgore et al, 2008) and was also in contrast to self-reported risk taking which showed modest decline following sleep deprivation and self-reported impulsivity which remained stable across the experiment (Killgore et al, 2011).

Overall, there has been significant variability in findings across all studies using the BART in sleep deprivation (Womack, Hook, Reyner & Ramos, 2012). The contribution of motor inhibition function is unclear and the effort required to repeatedly key press for increases in balloon size may lead to motivation confounds which disguise the propensity to risk-take (Killgore, 2012; Acheson et al., 2007). Indeed, in Killgore et al. (2011), increased risk taking was revealed only after 77 hours of sleep deprivation: a time period selected in order to overcome motivation confounds. Furthermore, given the discrepant findings between the IGT and the BART, convergent validity of the BART has been called into question (Womack et al., 2012). To date, no study has investigated the convergent validity of the BART with the IGT in sleep deprivation and therefore it is difficult to establish the reason for inconsistent findings in this area.

Venkatraman and colleagues (2007) used both a behavioural measure of risk taking and neuroimaging to investigate risk taking following 24 hours of total sleep deprivation. Decision making was based upon explicitly presented odds with uncertain yet probabilistic outcomes. This a departure from previously discussed decision-making tasks where probability and value contingencies were not explicitly presented. Three gamble types (certain/high risk/low risk) in two possible pairings (Low risk/certain) (High risk/certain) were presented. The risky gamble consisted of a choice between two rewards associated with paired probabilities of 25% and 75% or 50% and 50% where differing hypothetical monetary rewards were associated with different 'risk' levels. Interestingly, sleep deprivation was not associated with increased risk-taking observed behaviourally by way of experimental wheel choice. Despite this, neuroimaging evidence suggested an increased expectation of winning on high-risk gambles (increased activation in the right nucleus accumbens) and reduced disappointment in response to losses (selectively attenuated

response in the anterior insula). The results of this study suggest a tendency for risk-taking, however, the lack of behavioural evidence calls into question the appropriateness of this task to characterise the effects of sleep on maladaptive risky behaviour.

The current study is the first to investigate whether risky decision making observed in sleep deprivation is also a daytime correlate of those with naturally disturbed sleep and insomnia symptoms. Self-reported poor sleep quality (over the past month) and insomnia (over the past two weeks) were related to decision-making under risk using the Risky Choice Task (Fairchild et al., 2009). This task is broadly similar to that of Venkatraman et al. (2007), but benefits from the ability to systematically profile decision making across many trial types which differ in expected value (EV) thus allowing for identification of conditions under which risk is taken. Furthermore, 'framing trials' in this task provide an assessment of the influence of emotional factors in decision-making (Gonzalez, Dana, Koshino, & Just, 2005) where intuitive responses over-come mathematical considerations (Kahneman and Frederick, 2007). Healthy participants demonstrate risk-aversion when comparing possible gains (preferring smaller certain gains compared to larger more risky gains), and risk-seeking when comparing possible losses, (avoiding more certain but smaller losses) (Fairchild et al., 2009).

Decision making under risk on the RCT is different to decision making under conditions of uncertainty (e.g. the IGT), in that participants are aware of the likely consequence of their choices. Furthermore, unlike the IGT, earnings are explicitly updated following each decision which should prompt behaviour that is consistent with the long term goal (earning of maximum points). On the IGT, successful learning of and adherence to an underlying paradigm is key to optimal performance meaning that results may be interpreted in different ways, e.g. insensitivity to reward/punishment, risk-taking preference, anhedonia, (Dunn et al., 2006). The RCT, however, is a purer measure of risk-taking propensity given that conscious decisions are made on a full set of information.

Predictions for this study were based on several factors. Although there is evidence for risky decision making in sleep deprivation, sleep debt associated with insomnia symptoms is generally less severe. Accordingly, impairment to decision-making processes may be more subtle. In addition, poor sleepers are able to temporarily overcome the effects of sleep debt during motivating laboratory sessions. Despite these considerations there is good evidence to suggest executive control impairment in poor sleeping populations (see Chapter Two for a review), and sound rationale for risky-decision making in particular. The RCT has the recommended properties (novelty, unpredictability, risk) to overcome some of

the limitations associated with tasks that have previously failed to reveal executive control impairment in this population. Furthermore, risky decision making under conditions of uncertainty may more closely approximate the type of cognitive challenge experienced in the day-to-day life of poor sleeping young adults.

It was hypothesised that insomnia symptoms are associated with increased risky decision-making where a gamble is made despite the probability of an unfavourable outcome. A secondary, exploratory hypothesis is that the 'framing effect' observed in healthy individuals will be less influential in poor sleepers (i.e. they will not reliably chose a certain, smaller gain over the opportunity to obtain larger, albeit uncertain gains). As a secondary analysis, the uniqueness of this relationship beyond the effects of anxiety (and self-report deficits in cognitive control) was assessed. This was important because trait anxiety is associated with impulsivity (reflected by a lack of inner restraint under perceived stressful and time pressured situations) (Schaefer, Esposito-Smythers & Riskind, 2012),

Method

Participants. Ninety two undergraduate students at the University of Southampton were recruited via an online advertisement and received course credit for participation; mean age = 20.31 years (*SD*= 4.13); 80.43% females. Written informed consent was obtained from all participants and the study was reviewed and approved by Ethics Committee at the School of Psychology, University of Southampton, UK.

Self-report Measures.

Consistent with previous chapters, participants completed the following: PSQI (*a*= .73), ISI (*a*=.84), STAI-T, (*a* = .93), SSS, ACS (*a*=.79) and CFQ (*a* =.92) – see Chapter Two for details.[11]

Risky Choice Task

We used a modified version of the original decision making task, the Risky Choice Task (RCT: Rogers et al., 2003) developed by Fairchild et al. (2009). Participants were required to make a decision between two wheels presented to them on- screen. Both wheels showed the number of points available (gains and losses) and the relative probability of each outcome (as each wheel was made up of segments that communicated this information). A 4 second response window is designed to elicit an intuitive response to the odds presented. As such, this task is considered to measure the 'risk taking' of an individual based on a short

[11] MEQ scores (*M*=46.40, *SD*=8.40, intermediate type)

processing time rather than the ability to accurately calculate probabilities per se. Figure 4.1 shows a schematic of a typical trial sequence on the RCT.

Excluding framing trials, a 'control' wheel was presented on all trials, and provided a 0.5 chance of gaining 10 points and a 0.5 chance of losing 10 points (*Figure 5.1*, left wheel). The experimental wheel was presented simultaneously and varied systematically in terms of the amount available to be won (80 or 20), the amount available to be lost (80 or 20), and the probability of winning (.75 or .25). Altogether, there were eight different trial types differing in relative EV. Expected values were calculated for each decision in order to assess the more profitable wheel choice for each trial type (See Table 5.1). For example, in Figure 5.1 the left wheel has an EV of 0 (.5 x 10 +.5 x-10), whereas the right wheel has an EV of +5 (.25 x 80 +.75 x -20). The difference between these two choices in expected value (delta EV) is +5 in favour of choosing the experimental gamble. Thus the different trials ranged in expected value from -55 to +55. For clarity Table 5.1 specifies the probabilities and delta EVs for each of the eight trial types in this study.

In addition, two framing trials were included in which both wheels yielded the same expected value but one yielded a certain moderate gain or loss (+40 or -40), whereas the other could lead either to zero or to a large gain or loss (+80 or -80). These trials were designed to measure risk aversion. Healthy individuals typically show moderate risk aversion when comparing possible gains (opting for the certain +40) but conversely show a tendency towards risk seeking when comparing possible losses, opting for the uncertain option on the loss framing trial (Tversky & Kahneman, 1981).

The positive framing trial (0- frame, see Trial type 9 in Table 5.1) involved one wheel with a certainty of losing 40 points and the other wheel with a .5 chance of losing 80 and a .5 chance of losing 0. The negative framing trial (0+ frame, see Trial type 8 in Table 5.1) involved one wheel guaranteed to deliver 40 points and another wheel with a .5 chance of gaining 80 and a .5 chance of gaining 0.

In total, there were 10 trial types presented in a pseudorandom order across four blocks made up of 20 trials (see Table 5.1, and see Appendix L for schematics). An example schematic of each trial type can be found in Figure 5.1. A short break was provided between blocks; the overall task lasted approximately 20 minutes. The wheels (control, experimental/risky) appeared randomly on either the left or right hand side of the screen with participants indicating their choice using a mouse. Participants began each block with 100 points and were instructed to win as many points as possible. Feedback was given after each trial and an updated points total was displayed for two seconds before the next trial.

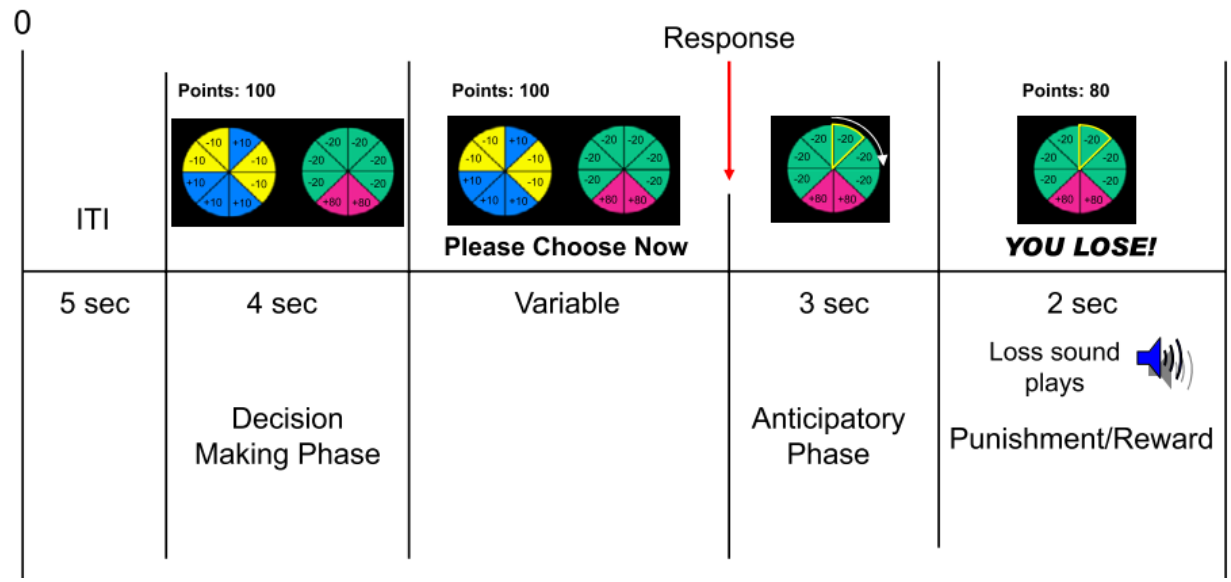


Figure 5.1. Schematic trial sequence of the modified Risky Choice Task. Available options shown in roulette wheel format. The 'control gamble', with an expected value of 0 (.5 X10 + .5 X -10), is shown on the left, while the experimental gamble, with an expected value of +5 (.75 X -20 + .25 X 80) is shown on the right. Following response selection, a highlight spins around the wheel, gradually becoming slower and slower until it lands on one of the eight wedges. Following this anticipatory phase, verbal and auditory feedback about the outcome (gain or loss) is provided. The revised points total is also displayed (i.e. "Points: 80.")

Table 5.1
Ten trial types of the Risky Choice Task, including the balanced 'framing' trials on which the expected value of each wheel is the same, but one wheel has a certain outcome (either + 40 or -40) and one has an uncertain outcome (a .5 chance of winning or losing 80, and a .5 chance of receiving 0). Pr (Gain) indicates the probability of a gain.

Trial type	Experimental gamble			Control gamble			Difference in expected value (ΔEV)between gambles	Mean % of time (sd) experimental wheel chosen
	Pr(Gain)	Gain	Loss	Pr (Gain)	Gain	Loss		
2	0.25	20	-80	0.50	10	-10	-55	1.36 (.05)
4	0.25	80	-80	0.50	10	-10	-40	5.43 (.11)
6	0.25	20	-20	0.50	10	-10	-10	4.21 (.09)
3	0.75	20	-80	0.50	10	-10	-5	76.77 (.28)
0	0.25	80	-20	0.50	10	-10	+5	29.08 (.28)
7	0.75	20	-20	0.50	10	-10	+10	100.00
5	0.75	80	-80	0.50	10	-10	+40	98.64 (.05)
1	0.75	80	-20	0.50	10	-10	+55	100.00 (.02)
9	0.50	80	0	1.00	40	0	0 (+ frame)	50.27 (.30)
8	0.50	0	-80	0.00	-40	0	0 (- frame)	84.83 (.21)

Results

Table 5.2 shows descriptive statistics for all self-report measures and Pearson's correlations between self-report measures. Fifty eight percent of the sample were classified as 'poor sleepers' on the PSQI and 51.5% experienced insomnia symptoms (81.36% "subthreshold insomnia," 18.64% "mild clinical"). Mean cognitive failures, attentional control and current sleepiness scores fell within expected ranges for this population. Insomnia symptoms were positively associated with: trait anxiety, current sleepiness, poorer attention control and increased cognitive failures. Both the ISI and PSQI questionnaires suggested the importance of daytime dysfunction associated with insomnia as well as the underlying nocturnal problems in this population.[12]

Table 5.2
Sample characteristics and associations between self-report measures

Measure			SSS	ACS	CFQ	STAI-T
	M	*SD*				
ISI total	8.00	4.46	.25*	-.33**	.36***	.48**
ISI daytime	5.02	2.91	.27**	-.39***	-.37***	.54***
ISI nighttime	2.98	2.05	.16	-.21	.26*	.27**
GPSQI	6.56	3.13	.24**	-.33***	.39***	.52**
PSQI quality	1.22	.67	.30**	-.28**	.30**	.32***
PSQI disturbance	1.25	.48	.21*	-.28**	.37***	.52***
PSQI latency	1.56	.87	.18	-.10	.14	.22*
PSQI duration	.28	.64	-.01	-.09	.19	.23*
PSQI efficiency	.90	.67	.08	-.24*	.30**	.42**
PSQI meds.	.18	.51	.09	-.03	.08	.08
PSQI day dysfunction	1.17	.68	.24*	-.46***	.46***	.54***
SSS	3.00	.80		-.27**	.31**	.35**
ACS	48.12	8.81			-.64**	-.57**
CFQ	45.70	14.46				
STAI-T	39.99	10.54				

Note. N: RCT=92, ISI=92, PSQI=87, SSS=92, ACS=86, CFQ=92, STAI-T-=88. *p<.05, **p<.01, ***p<.001.

Decision to choose experimental wheel, by trial type. The primary dependent measure was the percentage of trials in which the experimental wheel was chosen. Wheel

[12] MEQ scored were significantly associated with poor sleep quality, insomnia symptoms and performance on trial types 2, 4 and 6 (See Appendix M for analysis).

preference significantly differed across probability/value contingencies as shown by a main effect of trial type (F (3.48, 320.38)= 539.93, p<.001, with Greenhouse-Geisser correction). As previously reported in Fairchild et al. (2009), participants preference for choosing the experimental wheel was not entirely consistent with objective probability because in this context decisions are based on both probability, on value (e.g. incurring a large loss even if highly unlikely) and non-rational influences including heuristics and biases (Gigerenzer & Todd, 1999) (See *Figure 5.2*).

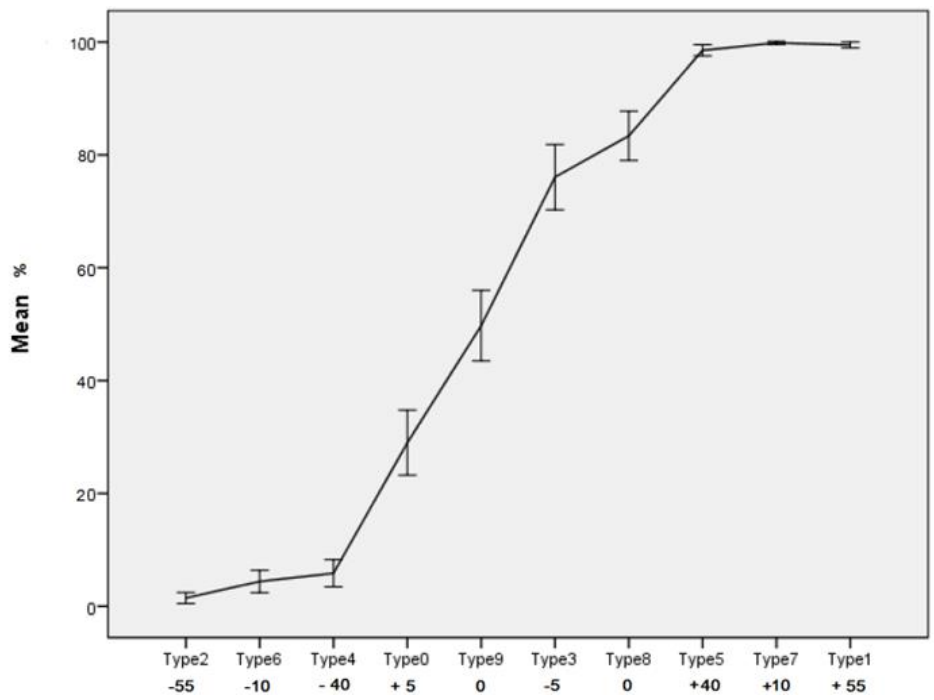


Figure 5.2. Percentage of time participants gambled on RCT as a function of trial type, ordered according to preference found in Fairchild et al. (2009).

Correlations were run on each individual trial type with all self-report measures.[13] Results are displayed in Table 5.3. Both sleep quality (PSQI) and insomnia symptoms (ISI), were associated with poor decision making on trials involving negative expected values and where the probabilistic outcome was most explicit. Poor sleep quality and insomnia severity were not related to decision making on trials with little difference in EV between the two wheels i.e. those involving lower risk and greater cognitive effort.

The PSQI subscales indicated that nocturnal symptoms of poor sleep (efficiency, duration, quality) were most strongly related to choice of the experimental wheel on trials where the decision to gamble was likely to result in a loss; trial types 6 (ΔEV=-10) and 4

[13] Bonferroni correction involved adjusted significance level p<.003 (based on primary analysis between two sleep measures and 20 dependent measures).

(ΔEV=-40) and trends for trial type 2 (ΔEV=-55). The ISI subscales revealed further moderate associations between risky decision making (trial types 6 and 4) and concern about daytime dysfunction associated with insomnia. Interestingly, poor sleep quality (disturbance, quality) was also weakly associated with reduced choice of experimental wheel on trials where the likely outcome was a large win (trial type 5: ΔEV = +40; trial type 1: ΔEV = +55).

Secondary analyses were run in order to determine the extent to which relationships between poor sleep, insomnia symptoms and risky decision making could be explained by anxiety and current sleepiness. Partial correlations performed between a) poor sleep, b) insomnia severity and decision-making on trial type 4 (EV=-40) revealed that associations decreased to non-significance when controlling for current sleepiness (SSS) and trait anxiety (PSQI: r=.05, p=.66; ISI: r=.18, p=.10). Partial correlations performed on trial type 6 (EV=-10) controlling for trait anxiety revealed a unique association between sleep quality and risk taking, (r=.24, p=.03) but not between insomnia and risk taking (*r*=.17, *p*=.12). No associations were found between decision making on the RCT, attentional control and cognitive failures.

Table 5.3

Correlations between RCT trial type and self-report measures

	Trial type, delta EV									
Measure	2	6	4	0	9	3	8	5	7	1
	-55	-10	-40	+5	0+	-5	0-	+40	+10	+55
ISI total	.17	.27*	.27*	.03	.01	.05	-.03	.07	-	-.13
ISI nighttime	.09	.18	.21*	-.12	.01	.14	-.04	.09	-	-.13
ISI daytime	.20	.28**	.26*	.13	-.01	-.02	-.01	.04	-	-.04
GPSQI	.19 (p=.07)	.31**	.17	.09	.00	-.02	-.01	.09	-	-.16
PSQI quality	.04	.16	.22*	-.07	-.10	.09	.02	.14	-	-.22*
PSQI disturbance	.17	.15	.11	.19	-.08	.13	.05	.21*	-	-.04
PSQI latency	.06	.14	.12	.07	.08	-.12	.07	.05	-	-.03
PSQI duration	.21*	.21*	.12	.01	-.10	-.06	-.20	.03	-	-.12
PSQI efficiency	.22*	.35**	.02	.07	-.03	.04	-.13	.02	-	-.15
PSQI meds.	.01	.10	.05	-.04	.01	-.02	.02	-.01	-	-.06
PSQI day dysfunction	.11	.16	.12	-.15	-.02	.11	.16	.02	-	-.05
STAI- T	.30***	.29**	.23*	.18	.12	.02	.11	.09	-	-.03
SSS	.07	.03	.31**	.07	.14	-.07	.03	.07	-	.14
ACS	-.08	-.11	-.09	.13	-.06	-.13	-.03	-.05	-	.05
CFQ	.12	.12	.14	-.06	-.03	.10	.15	.13	-	-.04

Note. On trial type 7 experimental gamble chosen 100% of the time. *p*<.05*, *p*<.01**, *p*<.001***.

Gains only or Losses only trials (framing trials). A paired samples t-test on the framing trials (8 and 9) confirmed that the way in which the EVs were presented had an effect on wheel choice in the sample as a whole. Participants were more likely to choose the experimental wheel on trial type 8 (M=0.84, SD=.21) in which the outcomes were framed in a negative context (concerning losses) than on trial type 9 in which outcomes were framed in a positive context (concerning gains) (M=0.50, SD=0.30) (t (92)= 10.01, p<.001, d=1.04).This was despite delta EVs between the wheels being equal (0) on these trials. No associations were observed between self-report measures of insomnia or sleep quality and these framing effects (ISI: r= .03, p=.79, GPSQ1: r=.01, p=.90).

Discussion

In a sample of young, healthy volunteers insomnia symptoms (ISI and PSQI) were associated with unprofitable decision-making, and risk-taking on the RCT. Evidence was observed that poor sleepers demonstrate increased gambling behaviour on trials with unfavourable outcomes (i.e. with high, negative EVs) regardless of the value of potential winnings. Secondary analyses suggested that the effects of insomnia symptoms on risk taking on unfavourable trials were partially mediated by trait anxiety.

Insomnia symptoms were associated with increased risk taking on trials in which choosing the experimental wheel was likely to result in losses (i.e. maladaptive choices). These results are consistent with previous behavioural and neuroimaging findings of altered valuation of reward and punishment following sleep deprivation, resulting in a shift towards risky decision making for potential profit (Killgore et al., 2012; Venkatraman et al., 2007). However, where previous studies have failed to reveal behavioural evidence for greater risk-taking in sleep deprivation, here this is demonstrated in relation to insomnia symptoms using an appropriately sensitive gambling paradigm.

The RCT successfully discriminated the conditions under which poor sleepers decided to risk-take. Consistent with expectations that poor sleepers maintain good cognitive performance through motivation and compensatory effort, the profile of decision making in those with poor sleep quality and insomnia symptoms was broadly similar to that of the sample as a whole. In fact, poor sleep was not associated with decision making on more difficult trials where the differences between the control and experimental wheels were small (e.g. Trial Type 0 with a delta EV or +5). Instead, poor sleep was associated with risky (or non-profitable) decision making on trials in which choosing the experimental wheel was clearly the unfavourable (or favourable) option. These results confirm that participants with insomnia symptoms engaged with the task demands and performed

adequately but yet made informed, high-risk decisions. Measures of cognitive control (ACS and CFQ) were unrelated to RCT performance suggesting that the ability to regulate attention was unrelated to performance on this task. This could be because of the motivating and incentive-based properties of the task.

Results further imply a unique association between poor sleep quality (PSQI subscale) and decreased gambling behaviour where the likelihood of a win was high. However, these results should be interpreted with caution. This association may suggest that poor sleep impairs the processing of rewards *and* punishments, such that participants are less affected by the experimental contingencies where potential gains and losses are manipulated along with their relative probability. However, these associations were not observed when controlling for multiple comparisons. Furthermore, the ceiling effect is recognised in trials with high positive EVs (i.e. trial type 5, 7 and 1) where the sample as a whole chose the experimental wheel over 98% of the time. Therefore, the meaningfulness of these observations is questioned.

Despite previous research demonstrating an association between trait anxiety and risk aversion (e.g. Maner et al, 2007), we found trait anxiety to be associated with greater risk taking. This has not been found in previous studies using risky decision making tasks (e.g. Leland & Paulus, 2005). Specifically, trait anxiety appeared to explain the relationship between poor sleep and risk taking on the trial with the highest, negative EV. Current sleepiness accounted for the most variance in outcome on the second most unfavourable trial type within the regression models for both insomnia symptoms and poor sleep quality. Poor sleep quality itself was uniquely associated with increased gambling behaviour on the third most risky trial type. These results suggest that poor sleep quality and insomnia symptoms, trait anxiety and current sleepiness play a role in gambling behaviour on the RCT but predominantly only on trials with high EVs.

Within the sleep deprivation literature, risk taking has been explained as an imbalance between a 'cold' rational-analytical system involving slower, intentional information processing and a 'hot' intuitive- experiential system involving faster, emotion based processing (Epstein, Pacini, Denes-Raj & Heier, 1996). Sleep deprivation has been argued to strengthen the initial emotion- based response or 'gut feeling' which occurs under conditions of uncertainty and results in an impulsive urge or desire to gamble despite awareness of the likely disadvantageous outcome (Starcke & Brand, 2012). This explanation may also be relevant to the observation here of risk-taking in those with insomnia symptoms. However, our findings suggest that *unprofitable* decision making (for both gains

and losses) may characterise insomnia symptoms rather than over-confidence in the likelihood of winning, increased sensitivity to the prospect of immediate reward (McKenna et al., 2007) and disregard regard for the negative consequences (Harrison & Horne, 1998; 2000).

As predicted, insomnia symptoms were not associated with the framing effect (i.e. the difference between the positively and negatively-framed balanced trials) suggesting that emotionally leading cues involved in the task did not unduly influence decision-making. Associations were not observed between attentional control or cognitive failures and decision making on the RCT suggesting that these traits were not influential in performance of the task. The motivating and engaging nature of the RCT may have accounted for a lack of association between these measures.

Results suggest that insomnia symptoms are associated with unprofitable and risky decision making when high, negative EVs are involved. Whilst poor sleeping individuals perform similarly to the sample as a whole on the more cognitively demanding trials (indicating engagement and ability to process contingencies appropriately), different decisions are made under explicit risk. Although speculative, it is possible that negative processing biases in insomnia and anxiety (Harvey, 2002; Mathews & MacLeod, 1994) operate such that individuals perceive an almost inevitable loss on trials with clearly unfavourable IVs. Accordingly, poor sleepers 'cut their losses' and take the chance on a potential win. The control wheel may be perceived as valueless despite being the safer option, especially if the value fails to meet personally meaningful amounts. Risk-aversion on trials with a high probability of gain could result from a negatively biased appraisal of the likelihood of losing. However, this explanation is limited because it is unclear why negative emotion processing biases associated with insomnia symptoms would selectively effect decision making processes on high EV trials. It is worth considering that unmeasured variables may also have influenced the results, for example cognitive impulsivity (suggested by the partially mediating role of trait anxiety) and sensation-seeking. Individuals with a preference for risk-taking may make apparently 'rational' choices based on their arousal and interest level (Zuckermann, 1994). This may be especially likely because real money was not at stake in this task (Dunn et al., 2006).

This study is believed to be the first to investigate associations between risk-taking and insomnia symptoms. The exploratory nature of this research suggests the importance of risky decision making in poor sleeping young adults but certainly requires replication in order to establish the reliability of these results. Beyond the consideration of other variables

which may affect performance on the RCT, it is important to investigate whether a profile of unprofitable decision-making (for gains and losses), based on full sets of information, is also a feature of those with Insomnia Disorder. It is likely that the profile of risk-taking behaviour changes across the developmental trajectory of insomnia such that risk-aversion characterises chronic insomnia, commonly accompanied by elevated levels of anxiety and mood disturbance. It is also important to further investigate the mechanisms which promote risky, unprofitable decision making in those with insomnia symptoms. The extent to which explanations for risky decision-making in sleep deprivation are relevant to insomnia is very unclear.

This study supports the use of the RCT as a sensitive measure of risky decision-making in poor sleeping populations and overcomes limitations of the IGT by explicitly presenting *complete* value and probability contingencies and prompting rapid and intuitive responses to the presented information. This study provides strong evidence for risky decision making associated with insomnia symptoms in a sample of healthy young adults. Risky choices were made predominantly when trials involved high probability of loss, however, we tentatively provide evidence that poor sleep is also associated with risk-aversion under favourable conditions. These results are important because risk-taking is an existing trait within young adults, particularly in relation to driving, sexual behaviour, drug and alcohol use (Douglas et al., 1995). Acute sleeplessness may compound an existing tendency to risk-take, bringing about severe and aversive consequences. An increased understanding and awareness of the effects of insomnia symptoms on decision-making processes is essential in order to promote personally advantageous and safe behaviour under conditions of risk.

Chapter Six

General Discussion

Review of thesis aims

The purpose of the studies described within this thesis was to improve understanding of daytime dysfunction in a healthy population where insomnia symptoms are naturally elevated and where the risk of chronic insomnia is increased (Buyssse et al., 2008). This work is highly relevant given the recent drive to improve conceptualisation, diagnosis and treatment of acute insomnia (Ellis et al., 2012) in order to improve quality of life and to prevent chronic insomnia development. Once chronic, insomnia tends to be unremitting and variable in presentation over time, detrimental to quality of life, economically costly and increases risk for psychiatric and physical ill-health (Matteson-Rusby, Pigeon, Gehrman & Perlis, 2010). Through identifying key mechanisms which promote dysfunctional cognitive (and behavioural) responses to sleep disturbance, effective and early preventative treatment and/or education can be more seriously considered. This is essential given that insomnia is a steadily-increasing global public health complaint (Calem et al., 2012).

This thesis focussed upon cognitive aspects of daytime dysfunction associated with insomnia which is important in a sample of healthy, high-achieving young adults. Furthermore, an aim was to explore attentional control as a mechanism which may be susceptible to dysregulation given its established role in anxiety disorder (Bishop, 2009), and given that acute insomnia follows an acute stress-response (Harvey, Gehrman & Espie, 2014). Consequently we selected novel experimental paradigms (validated within anxiety literature) which provide the opportunity to expand current knowledge but also to address limitations of previous studies in the field of sleep and cognition.

Associations were examined between poor sleep (objective aspects of overall sleep quality) over the past month, insomnia symptoms (both nocturnal aspects and daytime cognitive aspects) over the past two weeks, and subjective (attentional control, cognitive error proneness) and objective measures of cognitive performance (and physiological responsiveness). A cross-sectional design was suitable for this novel programme of research as it encouraged the collection of data from a large number of participants, a requisite for revealing potential markers in sub-clinical levels of insomnia. Furthermore, this approach allowed us to estimate prevalence of poor sleep and insomnia symptoms in this population: an under-reported yet valuable indicator of the risk for chronic insomnia development. Finally, we responded to the need to provide convergent self-report measures of cognitive

performance in conjunction with objective performance measures within insomnia research (Buysse et al., 2006), using measures of cognitive control (ACS, CFQ) to explore associations with sleep measures.

Summary of thesis findings

Four studies examined whether poor sleep and insomnia symptoms are associated with aspects of daytime dysfunction, and specifically:

1) Impairment to attentional control as measured by the Attention Network Task (ANT) (alerting, orienting, executive control), response inhibition as measured by the Stop Signal Paradigm (SSP), and switching of attention as measured by the Switching of Attention Task.(SAT)
2) Frequency and valence of thought intrusions at baseline and following a period of self-referential worry.
3) Autonomic responsiveness (blood pressure and heart rate variability) and self-report subjective anxiety following a novel stress-induction procedure, i.e. the 7.5% CO_2 experimental model of anxiety.
4) Risky decision making on a gambling task where value and probability contingencies are explicit.

In Study One (Chapter Two) we selected established and sensitive measures of attentional control which are well validated within anxiety research in order to determine the extent to which poor sleep is associated with cognitive dysfunction (i.e. attentional lapses, executive control deficits). Study Two (Chapter Three) examined the phenomenon of thought intrusions using the thought intrusions task. Thought intrusions are associated with impoverished attentional control and are a central feature of 'worry' (and anxiety). Study Three (Chapter Four) used a healthy volunteer model of stress induction (known to model GAD symptoms) in order to investigate autonomic symptoms and self-reported state anxiety associated with insomnia symptoms. Finally, Study Four (Chapter Five) profiled decision making across a range of value-probability contingencies in order to establish whether, and the circumstances under which (i.e. related to lower-level or higher level cognitive processing), insomnia symptoms are associated with risky choices on a gambling task.

Characterising insomnia symptoms in young healthy adults

Across three of our studies, poor sleep quality over the past month and insomnia symptoms over the past two weeks were elevated such that the average participant was currently a 'poor sleeper' and/or was experiencing 'sub-threshold' levels of insomnia

severity (over 50% of the sample could be defined by at least one of these categories). This finding was entirely consistent with previous reports in healthy young adult populations (e.g. Lund et al., 2010). Our two measures of sleep were strongly correlated across studies and, consistent with previous work (e.g. Benitez & Gunstead, 2012), daytime aspects of insomnia were most closely associated with subjective aspects of poor sleep, i.e. 'sleep quality' and 'daytime dysfunction' whereas nighttime aspects of insomnia correlated consistently with all PSQI subscales. Elevated scores on both measures indicates both underlying sleep difficulty (e.g. SOL, disturbance) and daytime cognitive consequences (e.g. believing that others notice the daytime consequences of sleeplessness) of insomnia symptoms in this population. This was evident in the contribution of both 'nighttime' and 'daytime' components of the ISI and the 'daytime dysfunction' subscale of the PSQI to associations with performance measures.

These results confirmed that the two sleep measures were appropriate for detecting important dimensions of unsatisfactory sleep in this population. As previously discussed in the literature review, acute insomnia can take different forms and can be initiated by different profiles of stress reactivity (Ellis et al., 2012). Accordingly, whilst some individuals may currently be experiencing disturbed sleep without acknowledged daytime dysfunction, others may be acutely aware of the impact upon performance, as reported in Taylor et al. (2013). Furthermore, at any time, stress experienced by participants may be predisposing, precipitating or perpetuating in insomnia. Therefore, by using both a measure of poor sleep quality (emphasis on nocturnal symptoms) and a measure of insomnia symptoms (poor sleep and perceived daytime impairment and concern over impairment) the different presentations of acute insomnia symptoms were captured.

The distribution of poor sleep quality scores are broadly consistent with Taylor et al.'s (2013) comprehensive investigation of insomnia in a sample of 1,039 young adults where 42.9% has significant sleep problems. The importance of assessing poor sleep *and* insomnia was highlighted in this study by 26.9% of participants meeting DSM-5 criteria without reporting 'insomnia', 9.5% meeting criteria for chronic insomnia and 6.5% reporting insomnia who did not satisfy *all* DSM-5 criteria. Across the chapters in this thesis (with the exception of Chapter Four where screening criteria may have eliminated important variation in sleep variables) there is sufficient evidence to suggest that university students are an 'at risk' population for the development of more persistent sleep disorder. The content of current worry identified in Chapter Three invariably involved salient life stressors (e.g. work deadlines, finances) aligning our findings with Lund et al. (2010) who revealed that

perceived stress (more than erratic sleep schedule, alcohol, drug use, exercise frequency, or electronics use) provided the most explanatory power for poor sleep in healthy young adults.

Associations between poor sleep and current sleepiness. The measure used to assess current sleep propensity (SSS) did not correlate strongly with poor sleep quality and insomnia symptoms (nor with a propensity for sleep phase delay as measured by the MEQ). With poor sleep quality correlations were: r=.27* (Chapter Two), r=.31** (Chapter Three), r=.24** (Chapter Five) and with insomnia symptoms correlations were: r=.28** (Chapter Three), r=.25* (Chapter Five). Given that sleep deprivation results in fundamental attentional dysregulation (i.e. attentional 'lapses'), we predicted that sleep measures would be reliably associated with current sleepiness and that current sleepiness would be further associated with impaired cognitive performance. However, sleepiness was unreliably associated with daytime and nocturnal aspects of poor sleep and with objective performance measures.

These findings may be due to several factors. Firstly, a mean SSS score was computed for each participant based upon repeated administrations of the scale, however, there is inevitable variability in the conditions under which measures are completed. For example, variables such as motivation, stimulation, fatigue and boredom may have been more or less important given the type of task performed and the amount of time passed in the test session (Johns, 2010). Nevertheless, this was considered in the methodology where the SSS was completed immediately before task performance and immediately following task performance.

Current sleepiness has been considered as multi-faceted (Kim & Young, 2005), encompassing environmental, physical and emotional factors and thus we are cautious in interpreting this measure as a reflection of pure sleep debt. Previous research involving young adults has also failed to establish the SSS as a reliable indicator of daytime performance deficits associated with sleep debt (MacPhee, 2009). Herscovitch and Broughton (1981) first highlighted the use of the SSS as an adjunct tool for assessment of attentional deficits in insomnia, emphasising that it is not a substitute for objective measurement of individual performance relating to impaired alertness.

Despite these considerations, current sleepiness was consistently elevated in this population. This is consistent with this age group (Lund et al., 2010) and with the presentation of acute insomnia where underlying sleep debt is more profound than in

clinical samples (Ellis et al., 2012) who report low levels of sleepiness and take longer to fall asleep on the MSLT than controls (Stepanski et al., 1988).

Associations between poor sleep quality and anxiety. We found reliable, positive associations between insomnia symptoms (and poor sleep quality) and trait anxiety. Whereas sleep problems were elevated across studies (with the exception of Chapter Four), trait anxiety scores fell within the typical range for healthy individuals. These findings confirm that in a healthy student population, sleep problems are a primary issue and disrupted sleep is a normal response to psychosocial stressors (Espie et al., 2006). The primary nature of daytime dysfunction in insomnia symptoms is demonstrated by positive associations with impaired objective performance observed in the absence of (or despite) associations between anxiety and performance (using measures validated in anxiety research) This suggests that the effects of poor sleep upon daytime dysfunction are not dependent upon anxiety levels but are important in themselves, consistent with the reconceptualisation of insomnia as a 24 hour condition with its own pathology (Benitez & Gunstad, 2012). Although we did not find evidence of anxiety as a moderator between insomnia symptoms and daytime dysfunction, it is possible that in a population with elevated levels of trait anxiety, performance deficits are compounded by poor sleep (Lundh & Broman, 2000). In Study Three state anxiety was a key dependent measure found to be negatively associated with poor sleep quality (PSQI subscale) and insomnia severity. This finding was difficult to explain, however the sample contained healthier sleep profiles on average when compared to Studies One, Two and Four. It is possible that there is a threshold for sleep disturbance (not reached in this study) which results in increased responsivity to threat and perceived state anxiety.

Associations between poor sleep and cognitive control. In 'Recommendations for a standard research assessment of insomnia' (Buysse et al., 2006) it was stated that 'the lack of consistency of findings in the literature prevents a recommendation for any single…cognitive measure of performance in insomnia.' The validation of suitable self-report measures of cognitive function in insomnia was highlighted. Given the importance of stress reactivity (and associated anxiety) to the experience of acute insomnia symptoms, we proposed in Chapter One how the ACS and CFQ may be sensitive measures of cognitive decline (particularly executive control) in sleep loss due to their validated psychometric properties in related populations (e.g. subclinical anxiety).

With the exception of Chapter Four, results showed that self-reported attentional control and the propensity to make cognitive errors were moderately associated with poor

sleep quality and insomnia symptoms (in addition to associations with anxiety). Findings were broadly consistent with the notion that subjective aspects of poor sleep (e.g. quality, daytime dysfunction) most closely associate with cognitive control (as measured by the ACS and CFQ) (Benitez & Gunstad, 2012). The ACS and CFQ were also highly correlated with self-reported trait anxiety emphasising the close association between sleep report and anxiety in this population.

Interestingly, despite self-report measures indicating executive control impairment associated with poor sleep, subjective measures of cognitive control did not reliably correlate with objective measures of attention and cognition, e.g. no associations between ACS and performance in Chapter Two, Four and Five. Given the observed associations between poor sleep and objectively measured cognitive control, these results suggest that the ACS and objective performance measures were tapping different underlying processes involved in attentional control. This may be surprising given that the ACS purports to measure attentional focusing and shifting which are key components of the Attention Network Task. However, comparing subjective and objective cognitive failures in poor sleeping healthy young adults, MacPhee (2009) concluded that there is no association between these forms of measurement. In the case of the CFQ, this was explained by the fact the measure is considered to have a general, one factor structure (Broadbent et al., 1982) which cannot be expected to correlate with the highly specific conditions of neuropsychological performance tests.

In addition, self-report measures of attentional control capture every-day, spontaneously occurring aspects of daytime impairment which are contextually meaningful to the individual. In the laboratory, however, a range of confounds including motivation and task features (e.g. colour, sound) may disguise or override these natural deficits in performance. Another possible explanation is that associations between self-reported poor sleep and attentional control were largely explained by trait anxiety whereas objective performance primarily indexed sleep-related impairment. Of note, the CFQ was a more reliable measure of the propensity to make cognitive errors, e.g. associations observed with errors of commission on the SSP which reflects underlying motor function. A detailed analysis of the factor structures of the ACS and CFQ was beyond the scope of this thesis, however, results suggest the importance of acknowledging the differences between objective and subjective measurement of cognitive control in insomnia symptoms.

What has been learnt about daytime dysfunction associated with poor sleep in young adults?

Overall, the results of these studies suggest that healthy young adults experience both poor sleep and daytime cognitive aspects of insomnia and display mild to moderate cognitive impairment across important areas of functioning. We provide evidence for attentional dysfunction (both non-executive and executive), negative thought intrusions and distractibility and unprofitable decision making associated with insomnia symptoms in healthy young adults. These results are consistent with the limited, previous research into daytime cognitive dysfunction in poor sleeping healthy individuals where it was reported that performance is impaired independently of anxiety and depression (Benitez & Gunstad, 2012).

Attention. As highlighted in the literature review, the state of research into acute insomnia is such that we do not know whether non-executive cognitive deficits are most profound in this population or whether deficits also exist in executive control (e.g. in cognitive flexibility and response inhibition). Some researchers argue strongly that sleep deprivation should exert selective effects upon the prefrontal cortex and thus higher-level cognition is impaired as a result (Horne, 2013). Others argue that sleep deprivation primarily affects non-executive processes (e.g. maintenance of attention) which, in turn affects the efficiency of executive control (Tucker et al., 2010). Chapter Two was designed to discriminate between these processes using the ANT, SSP and SAT.

Associations were confirmed between poor sleep quality and impaired 'alerting' on the ANT which is entirely consistent with attentional lapses following sleep deprivation. However, attentional dysregulation did not extend to 'orienting' and in fact participants with higher scores on subscales of the PSQI showed greater orienting to task-salient stimuli. These results are interesting because unlike sleep deprivation studies reporting impairment across all three attentional networks (including executive control) (e.g. Jugovac & Cavallero, 2012) participants were able to maintain an adequate level of performance despite associations between poor sleep and decreased alertness. Although hyper-sensitive orienting is a feature of chronic insomnia (e.g. Espie et al., 2006), the underlying reason for this observation is likely to be different between the two populations. Whereas chronic insomnia involves hypervigilance for sleep-related and threat-relevant stimuli, it is possible that our study captured motivational effects whereby participants recruited extra cognitive effort in order to overcome sleepiness and maintain performance. This is most likely given the laboratory environment and the sample characteristics (i.e. psychology undergraduates)

(Schmidt, Richter, Gendolla & van der Linden, 2010). It is, therefore, understandable that we did not find executive control impairment associated with insomnia symptoms.

Importantly, however, this study does not dismiss the possibility of executive control dysfunction as a feature of insomnia symptoms. To the contrary, consistent and strong associations between self-report measures of sleep and cognitive control suggest that supervisory functions are impaired, affecting cognition and goal-orientated behaviour in day-to-day life. Compensatory effort may have obscured any 'real' effect of sleep debt upon executive control. Furthermore, ecological validity of the tasks used is questionable and a lack of sensitivity to the type of cognitive conflict considered problematic by poor sleepers may in part underlie null results.

Thought intrusions. For the first time, it was observed that insomnia symptoms are significantly associated with increased thought intrusions during an attentional focus task, and negative intrusions in particular. This observation was made in the absence of an association between trait anxiety and negative thought intrusions, and further supports the growing evidence that poor sleep independently escalates attentional and emotional dysregulation. Following a period of focused attention upon a current worry, associations between poor sleep and thought intrusions remained, however greater variance in this association was accounted for by trait anxiety. This suggests that poor sleep sets a vulnerability to thought intrusions which is further weakened by the presence of worry and associated anxiety. This study was the first to apply an objective measure of on-line thought intrusions to the investigation of daytime dysfunction in acute insomnia and supports the use of the thought intrusions task in future studies of poor sleep.

Risky decision making. Using the Risky Choice Task (Fairchild et al., 2009) we demonstrated that poor sleep and insomnia symptoms were uniquely associated with a tendency to choose the experimental wheel when explicitly presented odds were unfavourable. Participants with insomnia symptoms (expressing concern about daytime impairment associated with insomnia) also showed *risky* decision making (i.e. choosing the experimental wheel when a high number of points were likely to be lost). Results further suggested that poor sleep is associated with risk-aversion and unprofitable decision making. Participants with poor sleep were less likely to choose the experimental wheel despite an explicitly favourable probabilistic outcome (however, the meaningfulness of these results was cautioned).

The results were observed despite good overall performance on this task (which involves a range of value/probability contingencies) in poor sleepers. This suggests that

whilst participants were motivated and engaged to make profitable decisions overall (and on trials involving more cognitive effort), they disregarded losses for the opportunity to profit when unfavourable outcomes were highly probabilistic. Overall, insomnia symptoms and poor sleep were associated with non-profitable and risky decision making (for both small, 20, and large, 80, winnings). Evidence for an association between poor sleep and risk-aversion on trials with highly favourable outcomes could reflect a further change in the meaningfulness of value/probability contingencies in the extreme ranges. As well as suggesting potential dysfunction within executive control mechanisms, this study confirmed the appropriateness of investigating risky decision making in poor sleeping populations using the RCT.

Stress reactivity. There was no evidence to support the hypothesis of increased stress reactivity in poor sleep using the 7.5% CO_2 model of anxiety. However, as discussed in Chapter Four, poor sleep has previously been shown to increase autonomic and emotional reactivity to psychosocial stressors. Explanations for the observed results could involve poor sleepers as desensitised to the physiological effects induced by the CO_2 challenge. However, there were also significant limitations to the study design including lower levels of insomnia symptoms (and anxiety) compared to other chapters, possibly related to strict screening criteria which may limit the natural variability in sleep which is found in this population. In light of this, and the psychophysiological nature of insomnia, it remains possible that increased autonomic reactivity may yet be established as a reliable feature of the complaint which was not revealed here.

Results in context

To what extent have we provided evidence of daytime impairment in young adults with insomnia symptoms? Sufferers complain of difficulty concentrating, learning, and remembering information, difficultly completing everyday tasks, disorganised thought and increased distractibility (Kyle et al., 2010). Our finding of impaired alerting associated with poor sleep quality suggests that a diminished and variable state of alertness (experienced as poor concentration) affects the initial throughput of information which may in turn disrupt information processing and learning. Consequently, the effects of poor sleep may significantly affect academic success, particularly during a stressful period (e.g. Ahrberg, Dresler, Niedermaier, Steiger & Genzel, 2012), or individuals may compensate for this deficit which in turn negatively impacts mood and quality of life (Kyle et al., 2010).

Increased distractibility and negative thought intrusions are consistent with reports that "I can't think straight, I can't think the same…you find you've read a page and you

have no clue what it said, you have to go back and reread it" (Kyle et al., 2010, p.7). Negative thought intrusions are known to increase negative mood, increase risk for psychiatric disorder and detrimentally affect cognitive processes such as working memory (Brewin & Smart, 2005). As such, thought intrusions may be an important, but currently under-acknowledged aspect of acute insomnia symptoms.

Finally, unprofitable decision making in the form of gambling for high gains given unfavourable odds and non-gambling given favourable odds associated with poor sleep suggests that individuals may make detrimental choices despite having sufficient information upon which to base decisions. This is a particularly important observation in young adults given that education, financial and social decisions can have significant effects upon behaviour, e.g. intoxicated driving, substance misuse, gambling etc. (Arnett, 1992). Results are consistent with reports of irritability and mood-instability (Kyle et al., 2010) associated with irrational decision-making following sleep loss (Anderson & Dickenson, 2010).

Implications for understanding acute insomnia symptoms.

Chapter One involved an extensive review of theoretical models and research literature important to acute insomnia, which has received surprisingly limited research attention until very recent years. At the time of writing there is no validated model of acute insomnia development or maintenance, however, the psycho-bio-behavioural model (Harvey et al., 2014), detailed in Chapter One, provides a useful future framework for addressing the deficit in our knowledge and understanding of the condition.

The programme of research detailed in this thesis contributes to our understanding of daytime dysfunction in insomnia symptoms by acknowledging attentional dysregulation (perceived and objectively measured) as an important aspect of the complaint over and above reported levels of anxiety. This is in contrast to models of chronic insomnia outlined in Chapter One which focus heavily upon sleep-related attentional bias as a perpetuating factor in insomnia development/maintenance and assume a fundamental role of anxiety in daytime dysfunction. Whereas clinical models highlight sleep-related intrusive thought and selective attention to sleep-related material as important features of the disorder, we highlight dysregulation to broader attentional networks as a feature of poor sleep.

Results suggest that poor sleep alone confers a vulnerability for attentional dysregulation in the acute stages of sleeplessness, identifying a therapeutic target for early intervention. In the same way that 'excessive negatively toned cognitive activity' may underpin the disorder of chronic insomnia, we suggest that spontaneous negative thought

intrusions and increased distractibility may worsen the experience of poor sleep. However, in contrast to clinical models where thought content is focussed primarily around sleep and the consequences for daytime function, we demonstrated that the content of negative thoughts centres around current concerns and stressors. Consistent with this, whereas a distorted perception of sleep debt and daytime performance contributes to the maintenance of chronic insomnia (e.g. Harvey, 2002), we revealed performance deficits in healthy individuals despite the likely confounds which facilitate superior levels of performance during testing session, e.g. motivation, social desirability, awareness of testing timeframes (Dorrian & Dinges, 2003). This suggests that young adults experience daytime dysfunction associated with insomnia despite the fact that some may not acknowledge the impairment or attribute impairment to inadequate sleep (Taylor et al., 2010).

We also found unprofitable decision making associated with poor sleep was characterized by high risk decisions (this association was partly mediated by trait anxiety on some trial types). This feature of poor sleep could further escalate symptoms if alterations to mood and anxiety followed comparatively frequent exposure to unfavourable outcomes. Therefore, we offer attentional dysregulation, distractibility and unprofitable and risky decision making as important aspects of daytime dysfunction in poor sleep.

Limitations of thesis, future work

This programme of research recruited primarily female undergraduate psychology students from the University of Southampton. Our results, therefore, are important for healthy, high achieving young adults but may be less applicable to other poor-sleeping populations. Given the female bias in psychology undergraduate study, we did not look at gender differences in daytime dysfunction which may be an interesting area for future work. On the other hand, insomnia has a higher incidence in females, though females are no more likely than males to complain of daytime dysfunction associated with sleep (Léger et al., 2010).

A student population is further characterised by rather unique aspects of sleep behaviour, e.g. students are able to sleep in/nap, can plan their sleep schedule and can decide to miss important daytime activities where dysfunction may be most likely to manifest. As reported in Tucker et al. (2010), such unhelpful and sleep-incompatible behaviours can ultimately render individuals vulnerable to chronic sleep difficulties, anxiety and depression. But it is because of factors such as caffeine use, compensatory effort and incentives that uncovering daytime dysfunction in this population is so challenging. Basal level of performance is often disguised, particularly for laboratory testing sessions. Measures

such as the PSQI and ISI do not differentiate between weekday and weekend sleep patterns and 'average' scores may disguise important aspects of sleep complaint in this population (and may also disguise associations with daytime performance). Furthermore, there can be considerable intra-individual differences in sleep in this population, where it is not uncommon for individuals to voluntarily stay up for 24 hours once a month (i.e. 20% experience total sleep deprivation), yet other times insufficient sleep over several days causes anxiety and worry related to daytime function (Lund et al., 2010). These aspects of sleep variation cannot be reflected in the self-report measures but may differentially affect performance in the laboratory.

It is possible that in order to better profile daytime dysfunction in healthy young adults, greater control is required over variables such as daytime sleep opportunity, circadian factors and caffeine or stimulant intake. It would be valuable for the development of theoretical models of acute insomnia to investigate the strategies used by this population to maintain performance and how these may exacerbate an existing sleep problem. Furthermore, by effectively counter-acting these strategies in the laboratory the type of dysfunction experienced by sufferers on a day-to-day basis may be uncovered

Due to large sample sizes and practical considerations within this programme of research, time of day testing was variable and self-selected (testing slots were offered at intervals between 9.30 am and 15.00 pm in all studies). Where possible circadian influences were assessed through self-report measures, (e.g. MEQ assessed 'morningness-eveningness,' and SSS measured current sleepiness). However, the build-up rate of cumulative neurobehavioural deficits following several nights of sleep restriction has been reported as largest at 08.00am, following which point it becomes increasingly smaller, especially between 16.00 and 20.00 (Mollicone, Van Dongen, Rogers & Dinges, 2008). While it is possible that our testing window may have precluded the strongest circadian influences, future studies would benefit from more careful control of this factor.

Future research could also test the same participants on all performance measures (i.e. all studies) in order to reduce variability of individual differences in sleep profiles, circadian factors and time of testing. This approach would enable direct comparisons between performance measures although it would also reduce the generalisability of results. In addition, it would be beneficial to measure state anxiety at time of performance testing. In the current programme of research trait anxiety was a key variable of interest because of the bi-directional nature of association between trait anxiety and insomnia symptoms. Although state anxiety increases as a function of trait anxiety under situational stress (Meijer, 2001),

future studies could measure this transient state specifically in order to increase understanding of the association between sleep and anxiety during performance testing (participants may be more anxious at certain times than at others).

The decision to conduct cross-sectional research using a correlational design was theory driven and most appropriate given the paucity of published work into insomnia symptoms and daytime cognitive dysfunction. It was important to be able to reveal trends *across* a population where poor sleep and insomnia symptoms come and go and where repeated episodes can eventually lead to a chronic problem with sleep (Ellis et al., 2012). Given that cut-off scores on the sleep measures have not been established for this population, group comparisons were considered too restrictive and could impose categories that may be unstable and highly changeable. However, the cross sectional nature of this work means that causality cannot be inferred from the data. This means it is possible that a third variable may have accounted for associations between insomnia symptoms and daytime dysfunction (e.g. cognitive impulsivity). In relation to modelling the development of chronic insomnia, only longitudinal data can truly unpick the predisposing, precipitating and perpetuating factors involved. This thesis suggests important aspects of daytime function which could act alone or interact with emotional aspects of the complaint (as proposed in cognitive models) to increase insomnia symptoms.

Although acute insomnia symptoms are the most common form of sleep disturbance in young adults, there should be some caution when comparing our results to studies where DSM-5 criteria have been met. There may be an important line between those who experience acute sleep loss and sleep-related concerns and those who report aspects of insomnia but are non-complaining (Tucker et al., 2010). In part, we addressed this issue by including both the PSQI and the ISI, which were highly correlated. However, given large sample sizes we did not include an interview which may have provided additional information such as longevity of sleep complaint, and the number of affected nights per week. Despite this, our measures specified a period of past two weeks (ISI) and one month (PSQI), thus capturing symptom profiles across this timeframe only. A further caution is that within our ostensibly non-clinical sample, scores from a small number of participants indicated the presence of a potential clinical sleep disorder. We were unable to clarify whether these cases were of clinical importance and retained them in order to keep the natural heterogeneity of poor sleepers in this random, unselected population, consistent with a previous study using similar methodology (Benitez & Gunstad, 2012).

A diagnosis of insomnia is made on the basis of self-report information and therefore it was considered most appropriate to assess sleep complaints in a large healthy population using questionnaire measures. The information provided by these questionnaires profiled perceived sleep patterns and behaviour over the past two weeks to one month which is a valid approach to assessment (Buysse et al., 2006). However, objectively measured sleep (assessed the night before testing) is a key variable predicative of cognitive performance in the laboratory. In this programme of research perceived insomnia symptoms was the key variable of interest and therefore sleep was not objectively measured due to practical considerations of time, expense and additional burden upon participants. However, the inclusion of actigraphy and/or sleep diaries within future studies would be an important step in further characterising insomnia symptoms in this population and would address a current limitation that daytime dysfunction reported through the ISI and PSQI cannot be confidently attributed to genuinely disturbed sleep.

Specifically, actigraphy (measuring rest-activity pattern) would enable researchers to assess sleep the night before testing in the laboratory session using outcome measures of mean/SD of sleep onset and offset, SOL, number of awakenings, wake after sleep onset, total sleep time and sleep efficiency. These recordings may reveal stronger associations with daytime cognitive performance and stress reactivity than was found using self-report inventories reflecting cumulative sleep debt. Orff et al. (2007), for example, found prospectively measured sleep (sleep diaries) and objectively measured sleep (polysomnography) to be associated with severity of daytime complaint but only objectively measured sleep parameters were associated with neuropsychological testing performance. Objective measurement of sleep the night before testing, therefore, would help to clarify the relationship between subjective and objective sleep reports in this population and how these relate to daytime performance in the laboratory. In addition, sleep diaries completed on a nightly basis (e.g. for a week before testing) are recommended as best-practise methods for assessing insomnia symptoms in order to minimise retrospective reporting bias when assessing the past month of sleep and to rule out circadian rhythm disorder prior to performance testing (Buysse et al., 2006) . However, diary measures should be used as an adjunct to the ISI and PSQI when considering insomnia symptoms because they do not routinely assess emotional factors such as distress and dysfunction associated with the complaint.

As previously discussed, self-report measures of cognition did not associate reliably with performance measures. It seems important to improve the congruence between subjective

and objective measures of cognitive performance through the development of measures (similar to the ACS and CFQ) which are multi-faceted and informative about the circumstances promoting cognitive dysfunction in insomnia symptoms. As highlighted throughout this thesis, the deficits associated with different forms of insomnia (acute, short-term, chronic) can be divergent and objective measures must be sufficiently sensitive to the nature of the complaint under investigation. Therefore, subjective measures of cognitive function should be equally effective at detecting impairment in poor sleeping populations and should strive to discriminate between general and context dependent dsysfunction.

Across all studies in this thesis correlations between measures of sleep and performance are modest (e.g. accounting for up to 12% of variance). Therefore, results should be interpreted with caution and future studies should replicate our findings to further support this advancement in the understanding of daytime dysfunction in insomnia symptoms. Given the tendency for student populations to engage in behaviours known to escalate insomnia (Tucker et al., 2010), they are at risk for future sleep and mental health problems making it important to establish the reliability of attentional dysfunction, intrusive thought and risky decision making as daytime features of the complaint. As discussed in Benitez and Gunstad (2012) an increased awareness of the types of cognitive deficits associated with insomnia will guide clinical decision making about the cause of observed dysfunction in healthy populations. Furthermore, this will minimise the likelihood of misattributing deficits to pathological conditions and will promote consideration of transient, reversible aspects of the complaint.

The research presented in this thesis suggests the appropriateness of using the RCT and the thought intrusions task (which are novel to sleep research) for use in healthy poor sleeping populations. These tasks could also be used in experimental studies comparing DSM-5 defined insomnia, insomnia symptoms and normal sleepers. The profiling of cognitive dysfunction across these groups will be particularly valuable for understanding the development of acute insomnia and for developing effective early intervention methods for chronic insomnia.

Implications for early intervention for insomnia and concluding comments

Only very recently has acute insomnia been considered a condition worthy of clinical recognition and targeted intervention (Ellis et al., 2012). In order to consider appropriate intervention and treatment options it is essential to understand cognitive and emotional aspects of daytime dysfunction. This thesis has highlighted important avenues for further research which will help increase understanding of the consequences of insomnia symptoms

and aid in the development of theoretical models of acute insomnia. Currently, a lack of specificity in diagnostic criteria for acute insomnia restricts the ability to efficiently identify and address treatment-worthy cases for research and clinical practise. It is also difficult to determine the point at which 'normal' sleep disruption becomes dysfunctional for the individual which is a critical step in establishing the earliest point at which treatment should realistically be delivered (Ellis et al., 2012). Indeed, help-seeking behaviour for insomnia is triggered by the perception that daytime functioning is impaired in an important and distressing way for the individual (Morin et al., 2006).

In healthy, poor sleeping populations improved awareness about indicators and effects of acute insomnia could help prevent the development of sleep-focussed worry, maladaptive coping strategies and safety behaviours such as those highlighted in Harvey's model (2002). In student populations in particular, not only do many individuals meet criteria for insomnia (with and without awareness), but also compensate for sleep debt in ways known to exacerbate the problem (Tucker et al., 2010). This is consistent with the embarrassment surrounding insomnia and the belief that one should be able to cope with it alone (Kyle et al., 2010). Currently, despite a distinct lack of evidence supporting the usefulness of sleep hygiene in the treatment of insomnia (Stepanski & Wyatt, 2003), this remains the principle advice from general practitioners who feel under-skilled to manage the complaint (Everitt, McDermott, Leydon, Yules, Baldwin & Little, 2014).

The work presented in this thesis shows that important aspects of daytime function are impaired in healthy poor sleepers. In light of this, it is possible that some CBT-I principles typically applied to clinical cases could be of value at a much earlier stage. There is some evidence that CBT-I is effective for insomnia of less than one month (Ellis et al., 2012), although currently the programme is not tailored for acute insomnia where coping skills related to stressful life events are emphasised as important. Whilst it is unfeasible to deliver CBT-I for the large number of sufferers with insomnia symptoms, through improved understanding of daytime dysfunction in acute insomnia GPs and health practitioners may be better equipped to recommend appropriate self-help CBT-I techniques, e.g. management strategies for intrusive thought. On the other hand, given the evidence for more generalised dysregulation to emotional and cognitive systems in acute insomnia, skill in acceptance based techniques such as mindfulness may be preferable to CBT-I, or may increase the effectiveness of CBT-I (Ong, Sharipo & Manber, 2009) by fostering an accepting and non-judgemental approach to insomnia symptoms associated with psychosocial stress.

The investigation of daytime dysfunction associated with insomnia symptoms must be pursued in order to improve understanding of and outcomes in acute insomnia, in turn preventing the development of chronic insomnia. In this programme of research, attentional dysregulation (executive and non-executive), negative thought intrusions and unprofitable, risky decision making were found to be correlates of insomnia symptoms in a healthy, non-clinical population where psychological, neuropsychological and medical conditions are not prevalent.

Appendix A

PITTSBURGH SLEEP QUALITY INDEX

INSTRUCTIONS:
The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, what time have you usually gone to bed at night?

   BED TIME _____

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

   NUMBER OF MINUTES _____

3. During the past month, what time have you usually gotten up in the morning?

   GETTING UP TIME _____

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)

   HOURS OF SLEEP PER NIGHT _____

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you . . .

a) Cannot get to sleep within 30 minutes

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

b) Wake up in the middle of the night or early morning

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

c) Have to get up to use the bathroom

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

d) Cannot breathe comfortably

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

e) Cough or snore loudly

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

f) Feel too cold

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

g) Feel too hot

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

h) Had bad dreams

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

i) Have pain

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

j) Other reason(s), please describe_____

_____

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

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

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

Very good _____

Fairly good _____

Fairly bad _____

Very bad _____

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

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

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

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

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

No problem at all _____

Only a very slight problem _____

Somewhat of a problem _____

A very big problem _____

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

No bed partner or room mate _____

Partner/room mate in other room _____

Partner in same room, but not same bed _____

Partner in same bed _____

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

a) Loud snoring

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

b) Long pauses between breaths while asleep

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

c) Legs twitching or jerking while you sleep

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

d) Episodes of disorientation or confusion during sleep

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

e) Other restlessness while you sleep; please describe_____

_____

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

Appendix B

Insomnia Severity Index

The Insomnia Severity Index has seven questions. The seven answers are added up to get a total score. When you have your total score, look at the 'Guidelines for Scoring/Interpretation' below to see where your sleep difficulty fits.

For each question, please CIRCLE the number that best describes your answer.

Please rate the CURRENT (i.e. LAST 2 WEEKS) SEVERITY of your insomnia problem(s).

Insomnia Problem	None	Mild	Moderate	Severe	Very Severe
1. Difficulty falling asleep	0	1	2	3	4
2. Difficulty staying asleep	0	1	2	3	4
3. Problems waking up too early	0	1	2	3	4

4. How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern?

Very Satisfied	Satisfied	Moderately Satisfied	Dissatisfied	Very Dissatisfied
0	1	2	3	4

5. How NOTICEABLE to others do you think your sleep problem is in terms of impairing the quality of your life?

Not at all Noticeable	A Little	Somewhat	Much	Very Much Noticeable
0	1	2	3	4

6. How WORRIED/DISTRESSED are you about your current sleep problem?

Not at all Worried	A Little	Somewhat	Much	Very Much Worried
0	1	2	3	4

7. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?

Not at all Interfering	A Little	Somewhat	Much	Very Much Interfering
0	1	2	3	4

Appendix C

STAI-T

Please read each statement below and then circle the number that best describes how you *generally* feel. There are no right or wrong answers so do not spend too long on any one statement.	ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
1. I feel pleasant……..	1	2	3	4
2. I feel nervous and restless……	1	2	3	4
3. I feel satisfied with myself……...	1	2	3	4
4. I wish I could be as happy as others seem to be……..	1	2	3	4
5. I feel like a failure……	1	2	3	4
6. I feel rested……..	1	2	3	4
7. I am "calm, cool and collected"……	1	2	3	4
8. I feel that difficulties are piling up so that I cannot overcome them……	1	2	3	4
9. I worry too much over something that doesn't really matter……	1	2	3	4
10. I am happy……	1	2	3	4
11. I have disturbing thoughts……	1	2	3	4
12. I lack self-confidence……	1	2	3	4
13. I feel secure……..	1	2	3	4
14. I make decisions easily……	1	2	3	4
15. I feel inadequate……	1	2	3	4
16. I am content……	1	2	3	4
17. Some unimportant thought runs through my mind and bothers me……	1	2	3	4
18. I take disappointments so keenly that I can't put them out of my mind….	1	2	3	4
19. I am a steady person……	1	2	3	4
20. I get in a state of tension or turmoil as I think over my recent concerns and interests……	1	2	3	4

Appendix D

ACS
Please rate each of the following statements using the scale:

1=almost never; 2=sometimes; 3=often; 4=always

1. It's very hard for me to concentrate on a difficult task when there are noises around.	1	2	3	4
2. When I need to concentrate and solve a problem, I have trouble focusing my attention.	1	2	3	4
3. When I am working hard on something, I still get distracted by events around me.	1	2	3	4
4. My concentration is good even if there is music in the room around me.	1	2	3	4
5. When concentrating, I can focus my attention so that I become unaware of what's going on in the room around me.	1	2	3	4
6. When I am reading or studying, I am easily distracted if there are people talking in the same room.	1	2	3	4
7. When trying to focus my attention on something, I have difficulty blocking out distracting thoughts.	1	2	3	4
8. I have a hard time concentrating when I am excited about something.	1	2	3	4
9. When concentrating I ignore feelings of hunger or thirst.	1	2	3	4
10. I can quickly switch from one task to another.				
11. It takes me a while to get really involved in a new task.	1	2	3	4
12. It is difficult for me to coordinate my attention between the listening and writing required when taking notes during lectures.	1	2	3	4
13. I can become interested in a new topic very quickly if I need to.	1	2	3	4
14. It is easy for me to read or write while I'm also talking on the phone.	1	2	3	4

15. I have trouble carrying on two conversations at once.	1	2	3	4
16. I have a hard time coming up with new ideas quickly.	1	2	3	4
17. After being interrupted or distracted, I can easily shift my attention back to what I was doing before.	1	2	3	4
18. When a distracting thought comes to mind, it is easy for me to shift my attention away from it.	1	2	3	4
19. It is easy for me to alternate between two different tasks.	1	2	3	4
20. It is hard for me to break from one way of thinking about something and look at it from another point of view.	1	2	3	4

Appendix E

CFQ

The following questions are about minor mistakes which everyone makes from time to time, but some of which happen more often than others. We want to know how often these things have happened to your in the past 6 months. Please circle the appropriate number.

		Very Often	Quite often	Occasion-ally	Very rarely	Never
1.	Do you read something and find you haven't been thinking about it and must read it again?	4	3	2	1	0
2.	Do you find you forget why you went from one part of the house to the other?	4	3	2	1	0
3.	Do you fail to notice signposts on the road?	4	3	2	1	0
4.	Do you find you confuse right and left when giving directions?	4	3	2	1	0
5.	Do you bump into people?	4	3	2	1	0
6.	Do you find you forget whether you've turned off a light or a fire or locked the door?	4	3	2	1	0
7.	Do you fail to listen to people's names when you are meeting them?	4	3	2	1	0
8.	Do you say something and realize afterwards that it might be taken as insulting?	4	3	2	1	0
9.	Do you fail to hear people speaking to you when you are doing something else?	4	3	2	1	0
10.	Do you lose your temper and regret it?	4	3	2	1	0
11.	Do you leave important letters unanswered for days?	4	3	2	1	0
12.	Do you find you forget which way to turn on a road you know well but rarely use?	4	3	2	1	0
13.	Do you fail to see what you want in a supermarket (although it's there)?	4	3	2	1	0

14.	Do you find yourself suddenly wondering whether you've used a word correctly?	4	3	2	1	0
15.	Do you have trouble making up your mind?	4	3	2	1	0
16.	Do you find you forget appointments?	4	3	2	1	0
17.	Do you forget where you put something like a newspaper or a book?	4	3	2	1	0
18.	Do you find you accidentally throw away the thing you want and keep what you meant to throw away – as in the example of throwing away the matchbox and putting the used match in your pocket?	4	3	2	1	0
19.	Do you daydream when you ought to be listening to something?	4	3	2	1	0
20.	Do you find you forget people's names?	4	3	2	1	0
21.	Do you start doing one thing at home and get distracted into doing something else (unintentionally)?	4	3	2	1	0
22.	Do you find you can't quite remember something although it's "on the tip of your tongue"?	4	3	2	1	0
23.	Do you find you forget what you came to the shops to buy?	4	3	2	1	0
24.	Do you drop things?	4	3	2	1	0
25.	Do you find you can't think of anything to say?	4	3	2	1	0

Appendix F

SSS

The Stanford Sleepiness Scale is a quick and easy way to assess how alert you are feeling.
Discover your own pattern of alertness by recording your “degree of sleepiness” at different times throughout the day.

Using the 7-point scale below pick what best represents how you are feeling and note the corresponding number on the chart below.

Degree of Sleepiness	Scale Rating
Feeling active, vital, alert, or wide awake	1
Functioning at high levels, but not fully alert	2
Awake, but relaxed; responsive but not fully alert	3
Somewhat foggy, let down	4
Foggy; losing interest in remaining awake; slowed down	5
Sleepy, woozy, fighting sleep; prefer to lie down	6
No longer fighting sleep, sleep onset soon; having dream-like thoughts	7
Asleep	X

Appendix G

STAI-S

Please read each statement below and then circle the number that best describes how you feel *right now*. There are no right or wrong answers so do not spend too long on any one statement.	ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
1. I feel pleasant	1	2	3	4
2. I feel nervous and restless	1	2	3	4
3. I feel satisfied with myself	1	2	3	4
4. I wish I could be as happy as others seem to be	1	2	3	4
5. I feel like a failure	1	2	3	4
6. I feel rested	1	2	3	4
7. I am "calm, cool and collected"	1	2	3	4
8. I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
9. I worry too much over something that doesn't really matter	1	2	3	4
10. I am happy	1	2	3	4
11. I have disturbing thoughts	1	2	3	4
12. I lack self-confidence	1	2	3	4
13. I feel secure	1	2	3	4
14. I make decisions easily	1	2	3	4
15. I feel inadequate	1	2	3	4
16. I am content	1	2	3	4
17. Some unimportant thought runs through my mind and bothers me	1	2	3	4
18. I take disappointments so keenly that I can't put them out of my mind	1	2	3	4
19. I am a steady person	1	2	3	4
20. I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

Appendix H

Table A1.

Pittsburgh Sleep Quality Index sleep parameters pooled across studies

PSQI	Mean	Min	Max	SD
Time to bed (hours, mins)	24.30	22.50	04.20	1.24
Sleep onset latency (mins)	34.18	5.00	150.00	26.79
Rise time (hours, mins)	09.21	06.00	12.00	1.32
Actual sleep (hours, mins)	7.31	4.50	10.00	1.28

Appendix I

Table A1.

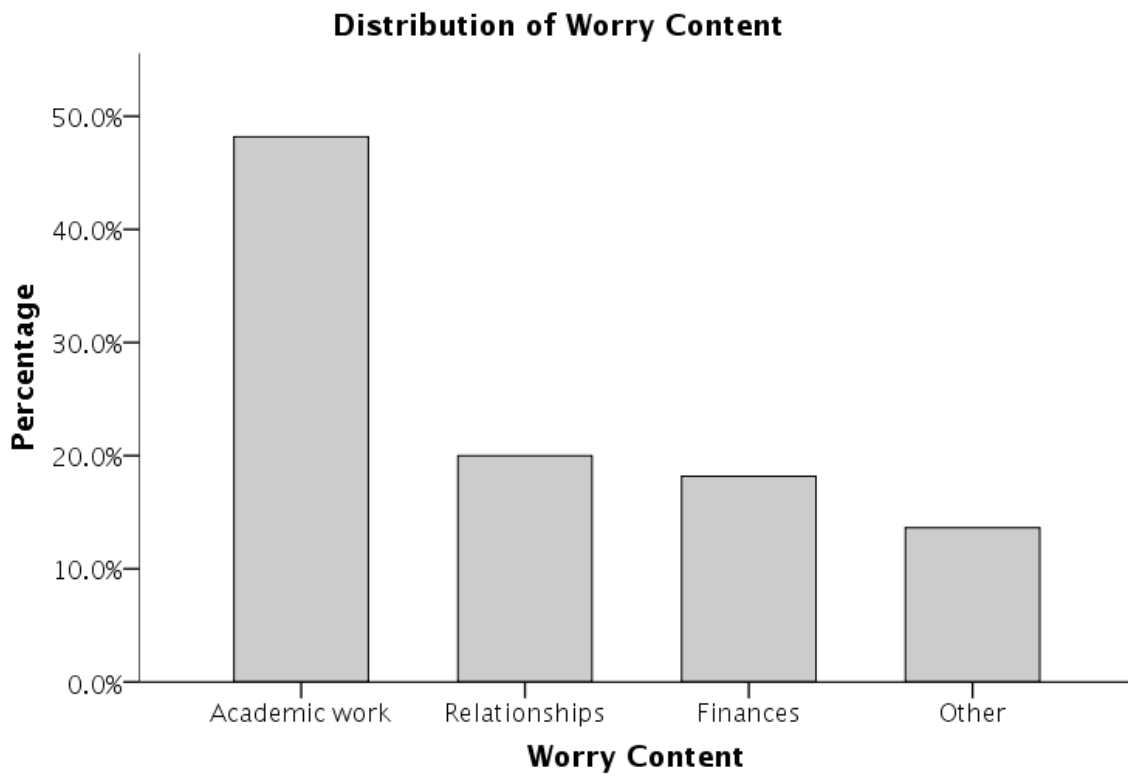
Correlations between self-report measures and errors made on the SAT

	Error			
Measure	SAT side	SAT dir.	SAT S side	SAT S dir.
GPSQI	-.25*	-.17	-.18	-.15
PSQI quality	-.09	-.03	-.02	.00
PSQI disturbance	-.20	-.08	-.19	-.16
PSQI latency	-.20	-.06	-.14	-.07
PSQI duration	-.26*	-.22	-.16	-.13
PSQI efficiency	-.26*	-.21	-.28	-.16
PSQI meds.	-.02	.08	.06	-.14
PSQI day dysfunction	-.21	-.19	-.13	.21
SSS	-.10	-.07	-.07	.01
ACS	.00	-.07	.13	-.09
CFQ	-.06	-.01	-.09	-.05
STAI-T	-.04	-.07	-.05	.11

Note. *p<.05

Appendix J

Chapter Three.
Bar graph showing categories of 'current worry' identified by participants in the thought intrusions task.



Appendix K

Chapter Four.

Table A4

Spearman's Rho associations between sleep measures and basal autonomic activity and state anxiety.

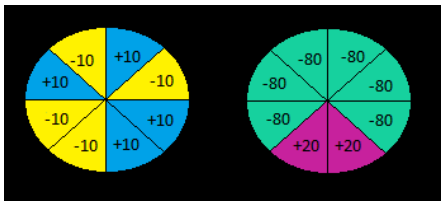
	SSAI	SBP	DBP	HR
ISI	.15	-.29*	-.27 (p=.05)	-.07
GPSQI	.20	-.18	-.18	-.10
STAI-T	.64**	-.08	.01	.15
ACS	-.20	.01	-.00	-.13

Note. *p<.05, **p<.01

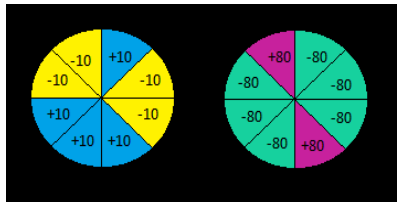
Appendix L

Chapter 5. Example schematics of all trial types included in the Risky Choice Task.

Type 2.



Type 4.

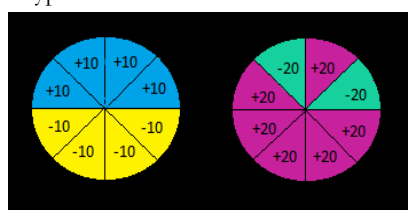


Type 6.

Type 3.

Type 0.

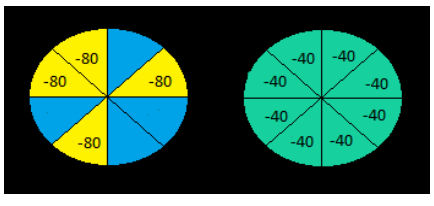
Type 7.



Trial 5.

Type 1.

Trial 9.



Trial 8.

NB. Control wheel presented on the left and the experimental wheel on the right for ease of comparison. During the task, the wheels were presented at random on the right and left of the screen.

Appendix M

Chapter Five.

Table A5.

Correlations between 'morningness-eveningness' (MEQ) scores and choice of the experimental wheel on the Risky Choice Task.

Measure	Trial type, delta EV									
	2	6	4	0	9	3	8	5	7	1
	-55	-10	-40	+5	0+	-5	0-	+40	+10	+55
MEQ	-.03	-.44**	-.21*	-.25*	.05	.06	-.17	-.06	-	-.02

*p<.05, **p<.01.

Table A5. shows that a tendency towards 'eveningness' (i.e. phase delay) is associated with unprofitable and risky decision making on trial types 6 and 4. In partial correlations, controlling for MEQ scores, poor sleep quality (PSQI) remained associated with the decision to choose the experimental wheel on trial type 6 (r=.22*, p<.05). Insomnia symptoms remained associated with the decision to choose the experimental wheel on trial type 4 (r=.23, p<.05), but not 6 (r=.19, p=.07), when controlling for MEQ.

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