Empirically Testing the Neurocognitive Model of Insomnia

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Abstract

The Neurocognitive Model of insomnia proposes that, through conditioned arousal, individuals with insomnia may experience heightened cortical arousal leading to increased sensory processing of external stimuli and sleep state misperception. This thesis provides a novel contribution to the literature by utilising auditory stimuli to examine the propositions of the Neurocognitive Model as a method of both eliciting and measuring the effects of cortical arousal. Firstly, the effect of noise on sleep was observed within the habituated home environment, evidencing that NREM sleep may be more susceptible to increased arousal through noise comparative to REM. Furthermore, traits typically associated with insomnia showed relationships with sleep disturbance due to noise, indicative that noise may increase cortical arousal. Secondly, the administration of novel noise in a non-habituated laboratory environment was utilised to raise cortical arousal levels in good sleepers to directly test the propositions of the Neurocognitive Model. Results demonstrated that noise altered both subjective and objective sleep along with creating a misperception of sleep onset, albeit not associated with explicit memory of noise stimuli. Finally, utilising individuals assumed to be experiencing heightened cortical arousal (insomnia) and good sleepers, words were administered during sleep onset periods to directly assess the processing and misperception components of the Neurocognitive model. Results demonstrated that both explicit and implicit recognition for words presented during sleep was greater for individuals with insomnia, yet this did not associate with a misperception of sleep. Therefore, this thesis proposes that the Neurocognitive Model could be a model of the effects of raised cortical arousal on sleep of which the two outcome pathways are the processing of auditory stimuli and sleep state misperception. Future research may wish to continue to examine the role of cortical arousal in the context of the Neurocognitive Model as a potential mechanism of sleep state misperception in those with insomnia and vulnerable good sleepers.

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List of Abbreviations

AASM	American Academy of Sleep Medicine
A-I-E	Attention-Intention-Effort
AIS	Athens Insomnia Scale
ANOVA	Analysis of Variance
BIS	Bergen Insomnia Scale
C3/4	Central
CAP	Cyclic Alternating Pattern
CBTI	Cognitive Behavioural Therapy for Insomnia
CCTV	Close Circuit Television
COI	Childhood Onset Insomnia
CVLT	California Verbal Learning Task
DSM-IV-TR	The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revisions
DSM-V	The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
ECG	Electrocardiogram
EEG	Electroencephalography
EMG	Electromyography
EOG	Electro-occulography
F3/4	Frontal

FIRST	Ford Insomnia Response to Stress Test
FP1/2	Pre-Frontal
GAL	Galanin
HADS	Hospital Anxiety and Depression Scale
Hz	Hertz
ICD-10-CM	International Classification of Diseases, Tenth Revision, Clinical Modification
ICSD-2	The International Classification of Sleep Disorders, Second Edition
ISI	Insomnia Severity Index
LAMF	Low Amplitude Mixed Frequency
LC	Locus Coeruleus
LDT	Laterodorsal Tegmental Nucleus
M1/2	Mastoid
MSLT	Multiple Sleep Latency Task
N3	Stage 3 and Stage 4 combined
NREM	Non Rapid Eye Movement
NWAK	Number of Awakenings
O1/2	Occipital
P3/4	Parietal
PPT	Pedunculopontine Tegmental Nucleus

PSAS	Pre-Sleep Arousal Scale
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
PSRS	Perceived Stress Reactivity Scale
PSS	Perceived Stress Scale
R&K	Rechtschaffen and Kales
RDC	The Research and Diagnostic Criteria
REM	Rapid Eye Movement
S1/N1	Stage 1
S1/N2	Stage 2
S3	Stage 3
S4	Stage 4
SCN	Suprachiasmatic Nucleus
SD	Standard Deviation
SDQ	Short Insomnia Questionnaire
SE	Sleep Efficiency
SOL	Sleep Onset Latency
SORC	Stimulus-Organism-Response-Consequence
SSS	Stanford Sleepiness Scale

SWA	Slow Wave Activity
TIB	Time in Bed
TMN	Tuberomammillary Nucleus
TSP	Total Sleep Period
TST	Total Sleep Time
VAS	Visual Analogue Scale
VLPO	Ventrolateral Preoptic Nucleus
WASO	Wake After Sleep Onset
WHO	World Health Organisation
WNSS	Weinstein's Noise Sensitivity Scale
α	Alpha
Ω	Ohm
μV	Micro Volts
χ^2	Chi-Squared

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Declaration

I declare that the work contained in this thesis has not been submitted for any other award and that it is all my own work. I also confirm that this work fully acknowledges opinions, ideas and contributions from the work of others.

Any ethical clearance for the research presented in this thesis has been approved. Approval has been sought and granted by the Faculty of Health and Life Sciences Ethics Committee between March 2011 and December 2012.

Name:

Signature:

Date:

Chapter 1: Classification, Symptomology, Prevalence, and Burden of Insomnia

1.0 Introduction

This chapter aims to introduce the classification and symptomology of insomnia concerning the change from normal sleep to disordered sleep. It will discuss the various diagnostic criteria used to define insomnia along with associated symptoms, prevalence, and the socio-economic burden of this sleep disorder in the general population, alluding to the importance of studying insomnia for providing evidence towards scientific theory, clinical diagnosis, and treatment practices.

1.1 Defining the Normal Sleep Process

Although the methods to measure sleep are well established, the reasons as to why we sleep are not as clear. Sleep is not a static process involving the absence of consciousness, but a dynamic process of changing electrical activity in the brain and physiological functioning (Carskadon & Dement, 1994). Three key theories have been proposed as to the function of sleep.

1.1.1 Theories of the Purpose of Sleep

The restoration and repair theory states that sleep is essential for the body to restore physiological and mental functioning. It is theorised that maximal cell division and protein synthesis occur during sleep, specifically during slow wave sleep (Adam & Oswald, 1984; Scheving, 1959; Tung, Takase, Fornal, & Jacobs, 2005). Following strenuous exercise, the proportion of slow wave sleep increases comparative to baseline indicative of a physiological response through sleep to promote cellular repair and growth (Browman, 1980; Shapiro, Bortz, Mitchell, Bartel, & Jooste, 1981). However, sleep does not just consist of slow wave sleep, in adults the majority of the

sleep period consists of lighter stages of sleep (Carskadon & Dement, 1994), suggesting that sleep may not just be needed for restoration and repair given the existence of other stages.

The evolutionary theory of sleep states that sleep is an adaptive process stemming from the evolutionary design of a species (Webb, 1974). The rest and activity cycle is a means of conserving energy and the cycle affords the safety of the animal by ensuring wake occurs during the most hazardous period of the day. Support for this theory relates to comparative research between the sleep of animals, evidencing that animals with fewer predators sleep longer whereas prey animals have shorter sleep periods (Webb, 1974). However, this may only be a residual evolutionary trait in human: as a species, we no longer have threats by animal predators.

The consolidation theory of sleep suggests that sleep is required for memory consolidation and can improve retention of newly-learned information (for review see Stickgold, 2005). This consolidation of memory has been shown to be dependent on the properties of sleep: declarative memory consolidation is said to occur in non-rapid eye movement sleep (NREM), and procedural memory consolidation in rapid eye movement sleep (REM) (Plihal & Born, 1997; Smith, 2001). However, Smith (2001) argues that although declarative memory is said to occur during NREM sleep (specifically slow wave sleep), it could be that NREM sleep occurs at the same time as an unknown process involved in memory and thus this unknown process is masked. Again, as with the restoration and repair theory, given that in adults it is the lighter stages of NREM sleep that constitutes the majority of the total sleep period (Carskadon & Dement, 1994), it is possible that the theory that sleep provides a memory consolidation mechanism is not the only explanation for the requirement to sleep.

Regardless of the theory of the purpose of sleep, what is clear is that sleep is an essential process, be it for cognitive or physiological requirements, and that a

disruption of the normal sleep process may have severe negative consequences to both physiological and psychological functioning.

1.1.2 Polysomnographically Defined Sleep

The sleep process can be defined by distinct electrophysiological properties and physiological characteristics. In general, sleep in humans is thought to represent a state of reduced sensory information processing, most likely to occur in a supine position with eyes closed (Carskadon & Dement, 1994). The measurement of sleep via the electrical activity of the brain and body is known as polysomnography (PSG) which encompasses electroencephalography (EEG) to measure brain neuronal activity, electromyography (EMG) to measure muscle tone, and electro-occulography (EOG) to measure eye movement. These recordings are used to classify sleep into two distinct states: non-rapid eye movement sleep (NREM) and rapid eye movement sleep (REM). The brain cycles between these two states throughout the sleep period approximately every 90-100 minutes in adults (Carskadon & Dement, 1994). Sleep is commonly thought to involve the brain in a resting state, with only autonomic functions essential for life activated. However, this is actually only the case for NREM sleep, as during REM sleep the brain is in a highly active state, displaying similar electrophysiological properties to wake, but with muscle atonia (paralysis) (Dement, 1958).

The categorisation of electrical activity in the brain can be divided into two further broadly defined sleep measures: sleep continuity and sleep architecture, (see

Table 1.1 for a breakdown of variables relating to these measures). Sleep continuity refers to the overall amount of sleep versus wake over the sleep period, including measures such as time taken to fall asleep and the number of awakenings. Sleep architecture refers to the classification or staging of the sleep EEG (wake, stages 1 to 4, and REM) within the overarching NREM and REM categories, and examines the quantification and distribution of these stages over the sleep period.

Table 1.1 Measures of sleep continuity and sleep architecture.

Sleep Continuity	
Measure	**Description**
Time in Bed (TIB)	The time from the point the individual enters bed to the time the individual gets out of bed for the final time.
Total Sleep Period (TSP)	The time from lights out to the final awakening.
Total Sleep Time (TST)	The total time spent asleep during the night.
Sleep Onset Latency (SOL)	The time taken to fall asleep as determined by stage 1 onset via PSG or subjective estimation.
Wake After Sleep Onset (WASO)	The time spent awake once sleep onset has occurred until the final awakening.
Number of Awakenings (NWAK)	The number of awakenings during the night. PSG determines an awakening to be 2 epochs (30 seconds) of wake or movement.
Sleep Efficiency (SE)	The ratio of TST to TIB (calculated by: (TST/TIB)*100)
Sleep Architecture	
Measure	**Description**
Latency to Stage	The time taken to the first epoch of a stage.
% of Stage	The distribution of the sleep stages over the whole sleep period.
Number of Stage changes	The total number of times epochs change from stage to another over the course of the sleep period.

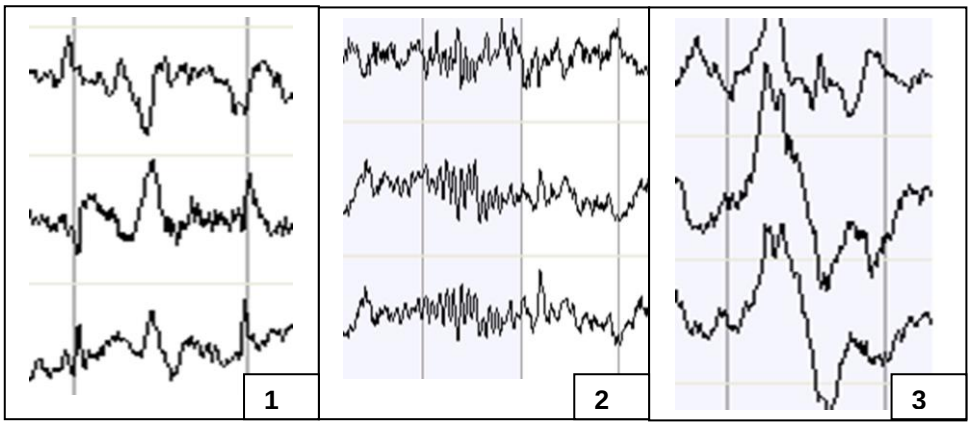
The EEG recording is traditionally measured in 20-30 second periods (epochs) and these can be categorised (scored) into stages dependent on the frequency and amplitude of the electrical waveforms along with the presence or absence of various electrical markers within the waveform.

NREM sleep constitutes 75% of sleep in a night using the Rechtschaffen and Kales (R&K) scoring criteria(Rechtschaffen & Kales, 1968b). Stage 1 sleep (S1) is the transitional state between wake and sleep and is characterised by a slowing of the EEG; from bursts of activity in the alpha frequency (8-12 Hz), to slower, low amplitude mixed frequency activity (LAMF) with bursts in the theta (4-8 Hz) range. Slow rolling eye movements, circular rotations of the eye with closed eyelids, also accompany this alongside the appearance of vertex waves in the EEG (sharp negative waves, Figure 1.1). Although this is defined as sleep, an individual still has a low arousal threshold and can be easily woken; hence this stage is sometimes defined as drowsiness as opposed to true sleep (Carskadon & Dement, 1994). An arousal is defined as a short duration (less than 30 seconds) alteration in the EEG whereby there is an increase in the frequency of the electrical activity in the alpha or theta range which is associated with an increase in muscle tone, preceded by 10 seconds of continuous sleep (Halasz, Terzano, Parrino, & Bodizs, 2004). S1 constitutes about 2%-5% of the nights' sleep and, due to the transitional nature of this stage, an increase in S1 can be judged as an indication of more fragmented, disturbed sleep (Carskadon & Dement, 1994).

Stage 2 sleep (S2) is defined as the official onset of sleep, comprising approximately 45% to 55% of total sleep in young adults (Carskadon & Dement, 1994). During S2 a more significant stimuli is required to elicit an arousal. The EEG activity is similar to that of S1 in terms of LAMF and theta bursts with the absence of slow rolling eye movements. S2 is further characterised by two distinct EEG elements occurring within

the EEG: spindles and K complexes, Figure 1.1. A spindle is a spontaneous burst of high frequency activity with oscillations of 12-16 Hz occurring over at least 0.5 seconds to a maximum duration of 2 seconds (Gibbs & Gibbs, 1964; Rechtschaffen & Kales, 1968b). Spindles have no maximal amplitude and can be further categorised into slow or fast spindles depending on the frequency of the oscillations (Andrillon et al., 2011). A K complex is a negative linear wave followed immediately by a positive spike (a depolarisation and a hyperpolarisation of the neurons) and can occur spontaneously or evoked by internal or external stimuli. Unlike the spindle, there is no maximal frequency for the K complex, but criteria states it must reach minimum amplitude of ±75µV and the negative/positive wave must occur over a period of at least 0.5 seconds (Rechtschaffen & Kales, 1968b).

Figure 1.1: EEG showing various grapheoelements of sleep 1) Vertex wave 2) Spindle 3) K complex (in a 27-year-old male on the baseline night of the study within Chapter 5)



As S2 transitions into stages 3 (S3) and 4 (S4), the frequency of the EEG decreases into the delta range (0-4Hz), and the amplitude of the waveform increases to at least 75µV. The distinction between S3 and S4 is based on the percentage of slow wave activity (SWA) within an epoch: S3 must contain at least 20% (but less than 50%) SWA and S4 requires over 50% of the epoch to contain SWA. Stage 3 is thought to comprise between 3% to 8% of the total sleep period in a healthy young adult and stage 4 comprises approximately 10% to 15% (Carskadon & Dement, 1994).

More recently the American Academy of Sleep Medicine (AASM) formed a task force to reclassify the R&K scoring criteria for SWS renumbering NREM stages as N1-3 with N3 (also known as Slow Wave Sleep) being an amalgamation of the R&K S3 and S4 (Iber, 2007). Within this thesis, when analysis of sleep architecture with a separation of stages 3 and 4 is needed (Chapter 4) R&K criteria are employed, whereas when overall continuity is required and an amalgamation of stages 3 and 4 is applied (Chapter 5), AASM criteria are used.

REM sleep constitutes the remaining 25% of sleep and is categorised by higher frequency EEG activity and rapid eye movements. During REM sleep, there is also a loss of muscle tone (atonia), with only the muscles of the eyes, cardiac, and respiratory processes remaining active. The sleep cycle consists of a repeating pattern between NREM and REM sleep throughout the night, with the sleep stages transitioning from wake and lighter sleep to deeper sleep, then back to lighter sleep and then to REM. In addition, as the sleep period progresses, the distribution of the sleep stages change; the first third of the night being predominately NREM (S2) and SWS, and the last third constituting mainly REM and NREM (S2) sleep (Carskadon & Dement, 1994).

1.1.3 Actigraphically Defined Sleep

An alternative way to measure sleep objectively is through Actigraphy. Actigraphy is the assessment of body movements through a wrist-based device using an inbuilt accelerometer that counts activity levels into epochs, an Actiwatch. Whilst PSG is considered the traditional “gold-standard” objective measure of sleep, actigraphy affords non-invasive ambulatory recording of sleep at a cheaper cost than PSG. Actigraphy records measures of activity, which is analysed to classify periods with activity as wake and inactivity as sleep. This allows measures of sleep continuity and some indices of sleep architecture to be calculated (sleep fragmentation).

Actigraphy is useful to estimate various aspects of sleep timing, specifically as a screening method to investigate underlying sleep rhythm (circadian) disorders (Morgenthaler et al., 2007). However, it is suggested to be less appropriate to use in individuals who have long periods of motionless awakenings during the night, as could be the case for insomnia, as the lack of activity may be classified as sleep, thus resulting in an inaccurate sleep report (Sadeh, 2011a; Sadeh & Acebo, 2002).

1.1.4 Subjectively Defined Sleep

Although measuring sleep through PSG is the gold standard for sleep research, the practice parameters suggested by the American Academy of Sleep Medicine state that it is not necessary for a diagnosis of insomnia due to the varying and subjective nature of the disturbance to sleep experienced in insomnia (Chesson et al., 2000; Littner et al., 2003). Due to this, and the costs associated with PSG, the subjective report of insomnia is suggested to be primarily viewed through sleep diaries which can be supplemented with various psychometric questionnaires (Chesson et al., 2000) with a diagnosis formed using clinical nosologies.

1.1.4.1 Measuring Sleep through Sleep Diaries

Sleep diaries are source reports completed daily by the participant to collect subjective sleep quality and quantity measurements. An example of this is the Pittsburgh Sleep Diary, which has been validated in both good and poor sleepers of all ages and correlates with indices of sleep obtained using actigraphy (Monk et al., 1994). More recently, a standardised sleep diary (the Consensus Sleep Diary) has been created to facilitate comparative consistency across the research community (Carney et al., 2012). Carney and colleagues (2012) suggest specific criteria to be included in the sleep diary pertaining to measurements of sleep continuity, including SOL, NWAK, WASO TST, TIB, and SE, along with measures pertaining to sleep quality and feeling of wellness upon awakening. By comparing subjective measures to PSG (Table 1.1)

affords the ability to conduct comparative analysis between subjectively and objectively defined sleep.

1.1.4.2 The Measurement of Sleep and Diagnosis of Insomnia through Psychometric Questionnaires

Certainly, a drawback of using a sleep diary to ascertain sleep timing and quality is the number of days that an individual may need to complete the diary to get a true representation of sleep, thus requiring a certain level of commitment from the participant/patient. Short, single-administration instruments can supplement the conventional sleep diary.

Psychometric questionnaires can be used to assess the quality of sleep with the most widely used being the Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). It is an 18-item questionnaire comprising a range of questions focussing on sleep quality, quantity and perceived sleep disturbance. The questionnaire is designed to give a numerical output of between 0 and 21, is an extremely well validated measure for assessing sleep disturbances over the course of the previous month, and a cut-off score of >5 has been validated to correctly identify 88.5% of responders with clinical sleep disturbances (Buysse et al., 1989). The PSQI has a high level of internal consistency (Chronbachs α of 0.8) (Buysse et al., 1989; Carpenter & Andrykowski, 1998), as well high test-retest reliability at 0.87 (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002; Beck, Schwartz, Towsley, Dudley, & Barsevick, 2004). The PSQI is not a measure of a specific sleep disorder per se, but enables the categorisation of good versus poor sleepers, showing a good sensitivity and specificity at distinguishing good sleepers from those with primary insomnia (Backhaus et al., 2002).

Specific questionnaires can be used to directly asses sleep for the symptoms of insomnia (for example, the Athens Insomnia Scale (AIS: Soldatos, Dikeos, &

Paparrigopoulos, 2003), the Bergen Insomnia Scale (BIS: Pallesen et al., 2008), and the Short Insomnia Questionnaire (SDQ: Violani, Devoto, Lucidi, Lombardo, & Russo, 2004). However, the most widely used within the literature is the Insomnia Severity Index, (ISI: Morin, 1993) which asks respondents to rate various perceptions of sleep quantity and quality over the course of the past two weeks. The score can either used on a continuum or quantified into four categories: no clinically significant insomnia, sub-threshold insomnia, clinical insomnia (moderate severity) and clinical insomnia (severe). The ISI shows excellent internal consistency and test-retest reliability (0.74 to 0.90) (Bastien, Vallieres, & Morin, 2001; Savard, Savard, Simard, & Ivers, 2005) and a cut off level of 8 has been shown to successfully identify 68% of respondents with insomnia (Savard et al., 2005). The ISI has also been shown to be able to determine treatment efficacy, with a reduction in score post treatment (either pharmacological or psychological), mirroring the improvements seen in both subjective and objective sleep (Bastien et al., 2001).

Although the ISI provides a quantification of the severity of the perceived sleep problem, with inferences made as to the clinically significant levels of sleep disturbance, it does not provide a diagnosis of insomnia. The Brief Insomnia Questionnaire, developed as a tool for the American Insomnia Survey (Kessler et al., 2010), has been shown to be a valid tool in the identification of primary insomnia in accordance with the different diagnostic nosologies (as discussed in the following section).

Although offering a quick quantifiable scale, single-administration questionnaires may not adequately capture the persistence of insomnia (Vallieres, Ivers, Bastien, Beaulieu-Bonneau, & Morin, 2005). Whereas an insomnia diagnosis is made upon the basis of a clinical interview through a subjective report of sleep disturbance, quantitative measures may be needed to determine true severity. A comprehensive way to

determine sleep disruption would be subjectively, through sleep diaries, and objectively through PSG and Actigraphy, supplementing this with questionnaires.

1.2 Diagnostic Criteria for Insomnia

Insomnia defines a period of poor sleep quantity or quality resulting in distress and functional deficits. In general, insomnia encompasses difficulties initiating, maintaining, or having sufficiently restful sleep, that results in social and occupational functioning impairments over the course of 3 nights a week for at least a month (Roth, 2007). Four of the key nosologies (three diagnostic texts and one research criteria) are discussed within this section, highlighting the different diagnostic pathways.

1.2.1 International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM)

The ICD-10-CM (The World Health Organization, 2013) is a set of clinical classifications for various diseases provided by the World Health Organisation (WHO). The ICD-10-CM classifies insomnia into two categories, organic and non-organic, dependent on the probable developmental factor that led to the presence of insomnia. Insomnia of an organic nature occurs as the result of another condition: be that a mental or behavioural disorder, a concurrent sleep disorder, or substance use, whereas, non-organic insomnia occurs independently of a co-morbid factor.

The diagnostic criteria for research of the ICD-10-CM states that, for a diagnosis of non-organic insomnia, there must be difficulties in the initiation of sleep, the maintenance of sleep, or that sleep is reported as poor quality occurring for three or more nights a week over the course of at least a month. The interim period of 0-1 months is termed transient insomnia and, as it is said to be initiated by a period of life stress, falls under the category of a stress disorder. In addition to sleep disturbances, there must be an impact upon the individuals daily functioning resulting in distress. However, the ICD-10-CM does quantify normal sleep and how disrupted the report of

disrupted sleep should be for a diagnosis of insomnia. This limitation is noted within the ICD-10-CM, which states that other diagnostic manuals should be sought for greater specificity of diagnosis. Used mainly as a clinical text, this broadness is advantageous to clinicians as referral of individuals with potential insomnia to appropriate healthcare services may be easier, however, this would be a disadvantage for researchers attempting to accurately record insomnia prevalence or typology based on the manifestation of the sleep disturbance.

1.2.2 The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V)

The DSM-V (American Psychiatric Association, 2013) was released in the summer of 2013 with several changes to the classification of insomnia from the DSM-IV-TR (American Psychiatric Association, 2000). Therefore, the majority of previous research has been conducted using the DSM-IV-TR that categorises insomnia according to the presence or absence of co-morbid disorders, similar to the categorisation set forth in the ICD-10-CM. Again, primary insomnia encompasses difficulties initiating or maintaining sleep (characterised by frequent awakenings with an inability to return to sleep), early morning awakenings, or non-restorative sleep without the presence of a co-morbid condition. The symptoms must occur for three or more nights a week, persist for more than a month, and be associated with clinically significant distress. This disturbance to sleep occurs alongside daytime impairments, such as fatigue or deficits to social/occupational functioning, and must not be due to voluntary sleep restriction through social or occupational constraints (American Psychiatric Association, 2000).

The newly released DSM-V has removed the categorisation of insomnia by comorbid factors - thus there is no distinction between primary or secondary insomnia, resulting in the diagnosis of a general “insomnia disorder”. This occurred through on-going debate in the scientific community suggesting that, although insomnia can occur secondary to a co-morbid condition, insomnia often persists when the co-morbidity has

been successfully treated indicating that the co-morbidity may have acted as a perpetuating factor in the development of insomnia (to be discussed in the following chapter) rather than a causative factor (Harvey, 2001b; Lichstein, 2006; McCrae & Lichstein, 2001). Certainly, in the case of depression, it is argued that, in a study of 86 patients, 70% of those with insomnia and depression saw alleviation or reduction of their depression symptoms once sleep had been improved, evidencing that insomnia may not be a secondary diagnosis (Morawetz, 2003).

The predominant complaint in an insomnia disorder remains an overall dissatisfaction with sleep quantity or quality, which must be accompanied by difficulties initiating or maintaining sleep, non-restorative sleep or early morning awakenings. Furthermore, the sleep disturbance must be present for over three months, rather than one, and the interim period defined as acute insomnia (0-1 months) and transitional/sub-acute insomnia (1-3 months). The DSM-V also provides a list of eight items that constitute a significant impairment in daytime functioning, at least one of which must be experienced, including; fatigue, daytime sleepiness, cognitive impairment (e.g. memory), mood disturbance (e.g. irritability), behaviour changes, impaired occupational functioning, impaired social functioning, or negative impact on family/caregiver functioning. In addition to the exclusion of sleep disturbances as a result of inadequate opportunity for sleep (i.e. occupational and social constraints), exclusion is also made for sleep disturbances that occur through natural age related changes (which may include changes in sleep structure, such as frequent awakenings).

Whilst the DSM-V provides a greater specificity in the diagnosis of an insomnia disorder, similar to the ICD-10-CM, again there is no mention of quantifiable measures (for example, how long must sleep onset latency be to constitute a problem). That said, the DSM-V does suggest scales to use for severity criteria; mild, moderate and severe disturbance to sleep.

1.2.3 The International Classification of Sleep Disorders, Second Edition, ICSD-2

Like the DSM-V, the International Classifications of Sleep Disorders (Second edition) (ICSD-2) diagnostic and coding manual classifies insomnia upon difficulties initiating or maintaining sleep, waking too early, or has non restorative or poor quality sleep despite adequate opportunity and circumstances for sleep (American Academy of Sleep Medicine, 2005). Additionally, one of nine symptoms that relate to daytime impairment due to sleep difficulty must be experienced including; fatigue, cognitive difficulties, social/vocational dysfunction, mood disturbances, daytime sleepiness, motivation reductions, proneness for errors at work/driving, somatic disturbances including tension headaches, or concerns or worries about sleep.

Fulfilling the above criteria results in a diagnosis of insomnia and, following this, the ICSD-2 has 11 subcategories from which a differential and more expanded diagnosis can be made depending on the probable causal factor or symptomology present. The ICSD-2 classifies primary insomnia (as under the previous edition ICSD-R) into the following six categories; adjustment insomnia, psychophysiological insomnia, idiopathic insomnia, paradoxical insomnia, inadequate sleep hygiene, and behavioural insomnia of childhood (Thorpy, 2012). The latter two (inadequate sleep hygiene and behavioural insomnia of childhood) will not be discussed within the scope of this work and neither will the additional five categories defined as secondary insomnia; insomnia due to mental disorder, insomnia due to substance abuse, insomnia due to medical condition, insomnia unspecified, and, physiological insomnia. As will be discussed further in subsequent chapters, the role of conditioned arousal is to be examined within insomnia and therefore, childhood onset insomnia will not be examined as the ICSD-2 states the disturbance to sleep is most likely due to a physiological alteration from birth. Furthermore, both secondary insomnia and inadequate sleep hygiene are due to external factors, be that a co-morbid condition or an unsuitable sleep environment, and therefore will not be examined within this thesis.

1.2.3.1 Adjustment Insomnia

Prior to the second edition of the ICSD, the ICSD-R, similar to the DSM-V, classified insomnia by symptom duration: acute period (less than 1 month), sub-acute (1 and 6 months), and chronic insomnia (6 months or more). Within the second edition, the acute and sub-acute periods have been incorporated into adjustment insomnia: insomnia lasting no more than three months. Adjustment insomnia, also known as transient insomnia, focuses on a stressful life event that drives sleep to become disturbed, as the ICD-10-CM, and there are said to be minimal to no effects of this sleep loss on occupational or social functioning. Additionally, it is proposed that, based upon an amalgamation of the ICD-10-CM, DSM-V, and the ICSD-2 nosologies, the acute period should stem from 3 days to 3 months and present with quantifiable disruption to sleep (SOL and/or WASO of >30 minutes), following a significant perceived alteration in the individuals quality of life (Ellis, Gehrman, Espie, Riemann, & Perlis, 2012). It is argued that adjustment insomnia is morphologically distinct to the chronic phase of insomnia, with differences in responses on biological, psychological and psychometric measures and is therefore a distinct form of insomnia as opposed to a purely transitional state (Ellis & Cropley, 2002; Rodenbeck & Hajak, 2001; Taylor, Espie, & White, 2003).

1.2.3.2 Psychophysiological Insomnia

Psychophysiological insomnia is also known as stress induced, or learned insomnia. The ICSD-2 suggests that stress creates a period of heightened cognitive and somatic arousal that results in sleep disturbances and thus a greater perceived pressure to compensate for the anticipated daytime consequences of sleep loss. It is suggested that, as sleep is an autonomic function, the more effort employed by the individual to initiate sleep, the more elusive sleep becomes (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006). The increased efforts to sleep results in behaviour modifications, perceived by the individual to aid sleep, which are sleep preventing and

thus a conditioned arousal forms to the bedroom environment. Unlike the DSM-V, there is no set definition of how many days a week that a sleep disturbance must be present for, but individuals experience a remitting state whereby, during periods of low stress, sleep returns to normal only for disordered sleep to return when psychological demands are high.

The ICSD-2 states that approximately 15% of those with insomnia fall under the psychophysiological subtype. Additionally, it occurs in the first instance in young adults and gets progressively more severe due to a cyclic interaction between arousal and poor sleep. A key component of psychophysiological insomnia is conditioned arousal to the bedroom, therefore individuals with psychophysiological insomnia are often able to sleep better away from their usual environment. In a laboratory setting this is evidenced as the “reversed first night effect” whereby sleep will be improved for the first monitored night then deteriorate over subsequent nights (Frankel, Coursey, Buchbinder, & Snyder, 1976; Hauri & Olmstead, 1989).

The ICSD-2 states that there will be evidence of alterations to both sleep continuity and architecture, with SOL and WASO over 30 minutes, an increase in stage 1 sleep at the expense of slow wake sleep, yet coupled with normal daytime alertness (as determined through a Multiple Sleep Latency Test). As this subtype is said to be due to heightened arousal, it is also suggested that individuals may have greater heart rate variability and basal metabolic rate (measures of somatic arousal) comparative to good sleepers indicative of a continuous physiologically aroused state.

1.2.3.3 Idiopathic Insomnia

The word idiopathic means “without a cause” hence, unlike psychophysiological insomnia, this subtype is believed to have no instigating source and is present from childhood. Due to this, the ICSD-2 proposes that causation may lie in

miscommunications in the sleep promoting areas of the brain, through either genetic or congenital abnormalities.

Studies have documented a genetic basis for insomnia (although not necessarily differentiating sub-types) and other sleep disturbances (for review see Barclay & Gregory (2013). Population-based and twin studies evidenced that individuals with primary insomnia (not specifically idiopathic) may have a familial history, indicative of a heritable component (Bastien & Morin, 2000; Dauvilliers, Maret, & Tafti, 2005; Watson, Goldberg, Arguelles, & Buchwald, 2006; Wing et al., 2012). This has been shown to associate with higher levels of insomnia severity, anxiety, and basal levels of arousal (Beaulieu-Bonneau, LeBlanc, Merette, Dauvilliers, & Morin, 2007). In common with psychophysiological insomnia, objective disruptions in sleep continuity are also characteristic of idiopathic insomnia; however, alterations also occur to the overall sleep architecture. Additionally, it is suggested that individuals who have had insomnia from childhood experience a greater severity of disruptions to sleep, with greater alterations in the EEG, and a significant reduction in eye movements during REM sleep, comparative to individuals who first experienced insomnia in adulthood (Hauri & Olmstead, 1980). Evidence indicates that idiopathic insomnia may be due to a dysfunction in the circadian system, as the administration of melatonin (the circadian rhythm “sleep” hormone) has shown to be an effective treatment (Smits et al., 2003). In addition, Barclay and Gregory (2013) suggest that specific genes should now be isolated for their role in sleep propensity and initiation, such as genes that drive the molecular biological rhythm, which may also provide evidence towards the development of idiopathic insomnia.

Due to the suggested biological cause of idiopathic insomnia and that the detriment to sleep occurs relentlessly across most nights (Espie, Barrie, & Forgan, 2012), the ICSD-2 states that diagnosis is not related to either duration or severity of the sleep

disturbance. In addition, due to the lifelong persistence of this subtype, the ICSD2 suggests that it is difficult to observe a case that is not co-morbid with another disorder. Studies have shown that children with idiopathic insomnia are more likely to exhibit depression and anxiety type symptoms than those without (Ivanenko, Barnes, Crabtree, & Gozal, 2004; Liu et al., 2007). As such, the ICSD-2 affords a diagnostic classification of idiopathic insomnia in light of a co-morbid condition as long as the disturbance to sleep is not better defined as a symptom of the co-morbid condition, such as insomnia through neurological alterations.

1.2.3.4 Sleep State Misperception (Paradoxical Insomnia)

Unlike psychophysiological and idiopathic insomnia, individuals with sleep state misperception show no detriments in objectively recorded sleep but report a severe loss of sleep subjectively. It is thought that the misperception between subjective and objective sleep may be due to an inability to accurately gauge the sleep/wake state, argued to be through an inability to perceive sleep "as sleep" when awoken during the night, leading to a negatively skewed perception of WASO (Borkovec, Lane, & VanOot, 1981; Dorsey & Bootzin, 1997).

Whereas the ICSD-R provides a quantitative value for PSG defined normal sleep (a TST of over 6.5 hours with a SOL of less than 20 minutes), the revised ICSD-2 states that, for a diagnosis of sleep stat misperception, the subjective report of sleep should be 1.5 times larger than what occurs objectively (Borkovec, Grayson, Obrien, & Weerts, 1979; Dorsey & Bootzin, 1997). In addition, the detriments to daytime function, as defined under a general insomnia diagnosis, need to be present but at a less severe level than expected when assessed alongside the reported sleep disturbances. The report of insomnia must be present for at least one month and the subjective sleep reports show severe insomnia with many nights when sleep was not attained. The individual must also report a near constant awareness of environmental stimuli

throughout the night, a pattern of conscious thoughts, or rumination whilst in bed attempting sleep. It is suggested that this subtype accounts for around 10% of those with a formal diagnosis of primary insomnia (Coleman et al., 1982; Edinger & Krystal, 2003).

However, the diagnosis of sleep state misperception as a distinct subtype of insomnia is controversial, as it is suggested by some that time estimation discrepancies are not a unique entity, but apparent in all those with an insomnia disorder and good sleepers (Feige et al., 2008; Fichten, Creti, Amsel, Bailes, & Libman, 2005; Rioux, Tremblay, & Bastien, 2006; Tang & Harvey, 2005). Additionally, if sleep state misperception is calculated using subjective against objective values (calculated as: (Subjective Value / Objective Value)*100), it has been shown that no significant differences in the degree of the misperception sleep between those with insomnia or those with another dyssomnia (sleep disorder of quantity or quality) (Edinger & Fins, 1995). Moreover, individuals with insomnia are equally likely to underestimate as overestimate sleep, therefore it is argued that a diagnosis of sleep state misperception must be made upon the size of the discrepancy between subjective and objective sleep (Manconi et al., 2010; Trajanovic, Radivojevic, Kaushansky, & Shapiro, 2007). Furthermore, although an assumption of sleep state misperception is that no physiological differences in sleep between these individuals and good sleepers exists, when physiological arousal is examined, individuals who misperceive sleep experience a significantly higher basal metabolic rate (Bonnet & Arand, 1997). This suggests that rather than sleep state misperception being a distinct subtype of insomnia, it may be a variant of psychophysiological insomnia.

1.2.4 The Research and Diagnostic Criteria (RDC)

In response to the lack of standardisation between diagnostic manuals and the lack of a manual specifically for research, the Research and Diagnostic Criteria (RDC) for

insomnia was developed via a working group of the American Academy of Sleep Medicine (AASM) (Edinger et al., 2004). Utilising existing literature to examine the presence of insomnia subtypes, the AASM provided a standardised set of research related diagnostic criteria. Although not technically a diagnostic manual, the RDC employs a similar format to the ICSD-2 using subtypes based on the morphology of the insomnia, along with a broad overall diagnosis of an insomnia disorder.

Under the RDC, an insomnia disorder encompasses issues surrounding the quality or quantity of sleep despite adequate opportunity and circumstances for sleep. Additionally, as the ICD-10-CM, DSM-V and ICSD-2, this results in detriments to psychosocial and occupational functioning and must persist over a minimum of one month. Additionally, unlike the DSM-V, there is a requirement that, unless the insomnia can be shown to be occurring independently (in which case then a diagnosis of primary insomnia can be given), there must be no history or current complaint of any other sleep disorder, medical condition, or psychiatric disorder.

For psychophysiological insomnia, the RDC is similar to that of the ICSD-2, requiring at least one form of conditioned arousal or learned sleep disruptive behaviour present such as; anxiety surrounding sleep, excessive/intrusive thoughts while attempting sleep, or extension of time in bed. The inability to sleep must persist during the day regardless of tiredness. Although adjustment/acute insomnia is not mentioned as a diagnostic subtype, for a diagnosis of psychophysiological insomnia there must be disturbances to sleep for at least a month, and the term acute psychophysiological insomnia is used for a duration of disturbed sleep under 4 weeks. The nomenclature of acute psychophysiological insomnia can be used as long as the above criteria are satisfied, as RDC states that learned behaviours and arousal take time to develop and form a cyclic pattern - poor sleep results in compensatory behaviours, increased arousal and thus further poor sleep. There is no causational event said to be the

initiating factor in the disturbance to sleep, although one would expect this to be similar to the ICSD-2 with a stressful life event driving conditioned arousal.

The RDC and ICSD-2 texts are similar regarding the classification of idiopathic insomnia; albeit the former applies a greater stringency within the diagnostic criteria. As idiopathic insomnia is thought to have a genetic or developmental origin, the symptoms must be present in childhood with an age of onset before 10 years, thus it is also known as child onset insomnia (COI). Unlike the methods of assessment proposed by the DSM-V and ICSD-2, COI is diagnosed through objective sleep disruption via PSG, and as such insomnia with a co-morbid sleep, medical and mental health conditions are excluded. The RDC working group literature review suggests that, idiopathic insomnia accounts for 30% of individuals who currently have insomnia.

Concerning paradoxical insomnia, the RDC states that, as this subtype is not due to a learned/conditioned response to sleep loss, there is no basis for an acute period and therefore there is no transition period from good to poor sleep. In addition, there must be an extreme subjective report of sleep loss, with little or no sleep reported on most nights, present for at least one month and a mismatch must occur between the subjective report of sleep and what is seen objectively: with objective recordings indicating almost normal sleep with a TST >6hrs, SE >85%, and SOL <20 minutes. To avoid controversies surrounding the quantification of sleep state misperception as a distinct phenotype (Edinger & Krystal, 2003), the RDC suggests a potential role for high frequency EEG intrusion, specifically in the beta and gamma range, as a causative factor resulting in the misperception of sleep, a point which is to be discussed throughout this thesis (Krystal, Edinger, Wohlgemuth, & Marsh, 2002; Maloney, Cape, Gotman, & Jones, 1997; Perlis, Merica, Smith, & Giles, 2001; Perlis, Smith, Andrews, Orff, & Giles, 2001).

1.3 Prevalence

Due to the different criteria employed by the various diagnostic manuals, the exact prevalence of insomnia is difficult to define as the broader diagnostic criteria of the DSM-V would undoubtedly result in a higher prevalence rate than would be derived using the highly specific ICSD-2. This is highlighted by the results of the America Insomnia Survey where, of 10,094 adults, the prevalence of insomnia was 22.1% using the DSM-IV-TR guidelines, 3.9% using the ICSD-2, and 14.7% using the RDC (Roth et al., 2011). To account for these differences, Roth et al. (2011) proposed an all-encompassing diagnosis combining the criteria used within the three diagnostic manuals resulting in an insomnia prevalence of 23.6%.

Prevalence rates also vary considerably between countries; for example in France the prevalence is 19% (Leger, Guilleminault, Dreyfus, Delahaye, & Paillard, 2000), Norway 11.7% (Pallesen et al., 2001), Italy 27.6% (Ohayon & Smirne, 2002), Japan 21.4% (Kim, Uchiyama, Okawa, Liu, & Ogihara, 2000), and 36.8% in the UK (Morphy, Dunn, Lewis, Boardman, & Croft, 2007). Again, these prevalence rates should be viewed with comparatively, as these studies did not employ a standardised diagnostic criterion for the diagnosis of insomnia. Furthermore, it is proposed that factors other than the differences in diagnostic criteria may be account for discrepancies in prevalence rates. A study conducted on members of the French population indicated that if mental health state was accounted for, the prevalence of insomnia dropped from 12.7% to 1.3% (Ohayon, 1997), however, once the implementation of the DSM-V becomes widespread the diagnosis of secondary insomnia will become obsolete, thus prevalence rates for primary insomnia may increase.

The duration of the period over which the participant is asked to evaluate their insomnia has also been suggested to be a possible confound between studies assessing the prevalence of insomnia (Ohayon, 2002). Although insomnia is said to persist over the course of a month, there is an element of night-to-night variability with

some nights seeing a remission of the disordered sleep. Ohayon (2002) suggests that there are four distinct ways to interpret and classify prevalence studies: those that assess insomnia symptoms regardless of duration experienced; those that assess insomnia symptoms with daytime consequences; those that assess general dissatisfaction with sleep quantity or quality; and those that use an insomnia diagnosis under the various diagnostic manuals. When utilising those four grouping variables, Ohayon (2002) showed that prevalence rates differed substantially: at around 35% for insomnia symptoms, 9%-15% for individuals with insomnia and daytime deficits, 8%-18% for individuals experiencing a form of dissatisfaction with sleep, and 6% for those that fulfil a clinical insomnia diagnosis.

1.3.1 Difference in Insomnia Prevalence by Gender

The prevalence of insomnia varies through gender: women are more likely to report insomnia symptoms (with or without daytime consequences) or dissatisfaction with sleep than men (Ohayon, 2002). Additionally, females are approximately twice as likely to report diagnostically (DSM-IV-TR/ICSD-2) relevant insomnia than males (Johnson, Roth, Schultz, & Breslau, 2006; Leger et al., 2000; Ohayon, 1997; Ohayon & Sagales, 2010; Ohayon & Smirne, 2002; Pallesen et al., 2001). Ohayon (2002) states that women are four times more like to experience insomnia symptoms than men are, increasing to seven times more likely after the age of 45. Moreover, women may be more biologically predisposed to insomnia as they are more likely to experience insomnia when hormones are fluctuating such as during menstruation or pre/post child birth (Miller, 2004).

1.3.2 Differences in Insomnia Prevalence with Age

The National Institutes of Health and various epidemiological studies state that the prevalence of insomnia increases with age (Kim et al., 2000; Leger et al., 2000; National Institutes of Health, 2005; Ohayon & Smirne, 2002; Roth et al., 2011). Certainly, aspects of normal ageing, such as frequent awakenings, have been

implicated in models theorising the development of insomnia (Fichten & Libman, 1991). Moreover, changes to the circadian system due to ageing may result in the presence of alterations in sleep (difficulty initiating sleep, maintaining sleep, and early morning awakenings) that fall under the diagnosis of insomnia (Pallesen et al., 2001). However, this is suggested to not be representative of a true diagnosis of insomnia as these disturbances of sleep are often reported without feelings of daytime impairments, therefore not fulfilling the DSM-V, ICSD-2 or RDC criteria for insomnia (Pallesen et al., 2001). Furthermore, social inactivity and associated napping during the day may be the cause of sleep dissatisfaction and insomnia type sleep disturbances in ageing. Indeed, a large multi-country population study demonstrated that when social activity and satisfaction with social life were controlled for, ageing was not predicative of insomnia. Thus social activity may be a protective factor against insomnia in the elderly (Ohayon, Zulley, Guilleminault, Smirne, & Priest, 2001).

1.3.3 Insomnia Incidence, Persistence and Remittance

The National Institutes of Health provides a standard consensus on the prevalence rate for insomnia suggesting that, although the prevalence for people suffering a sleep disorder may be close to 30%, once inclusion of daytime dysfunction and chronicity is accounted for, the number of people suffering chronic primary insomnia is around 10% (National Institutes of Health, 2005).

Regardless of the difficulty in identifying a robust prevalence rate of insomnia, insomnia has high incidence and persistence rates. Several studies have indicated an incidence rate of around 10%, a 1 year persistence rate of 50%, a remission rate of 50%, and a high relapse rate of around 30% (Jansson-Frojmark & Linton, 2008; LeBlanc et al., 2009; Morin et al., 2009; Morphy et al., 2007). These figures indicate that even if the prevalence rate of clinically significant insomnia is just 10%, the high persistence and relapse rates show the clinical and research relevance of continuing research for this sleep disorder.

1.4 The Societal Cost of Insomnia

The cost to the individual and society due to insomnia is vast. Insomnia not only has monetary costs to the state/health providers (LeBlanc et al., 2009) but also cost to employers due to the impact of insomnia on occupational functioning and health and safety implications.

The direct costs of insomnia are measured through the monetary costs to the health system resulting from clinician time, prescription costs and costs of sleeping medications. Individuals with insomnia are suggested to utilise the healthcare system (with physician visits, hospitalization and medications) more often than good sleepers (Leger, Guilleminault, Bader, Levy, & Paillard, 2002; Wade, 2010). The National Institute for Health and Care Excellence states that between 2005 and 2006, there were 4.7 million prescriptions for Z-type sleeping drugs (Zopiclone, Zolpidem and Zaleplon) at a cost of £13.5 million, and for Benzodiazepines, not including the widely used anxiolytics Diazepam or Lorazepam, there were 5.1 million prescriptions at a cost of £8.8 million (National Institute for Health and Care Excellence, 2007). Within the USA, the direct costs of insomnia was estimated at $13 billion annually, with the total direct cost for treatment of insomnia alone at $1.8 billion (Martin, Aikens, & Chervin, 2004).

The indirect cost of insomnia refers to economic cost resulting from the associated morbidity of insomnia, such as loss of productivity, work place accidents, and absenteeism from work (Walsh, 2004). A study conducted within the Québec region of Canada found that by categorising individuals with insomnia into those experiencing the insomnia syndrome (have a formal diagnosis of insomnia) and those experiencing issues in the quality and quantity sleep but not fulfilling full diagnostic criteria and/or taking sleep medication (i.e. subsyndromal), a better judgement of societal costs can be extrapolated (Daley, Morin, LeBlanc, Gregoire, & Savard, 2009). Compared to good

sleepers and subsyndromal insomnia, those with syndromal insomnia reported greater absenteeism and decreased productivity due to the daytime consequences of insomnia. When all participants were examined, it was estimated that over a year, days lost from work and reduced productivity due to the symptoms of insomnia cost the region $6 billion.

The American Insomnia Survey, conducted between 2008-2009, indicated a strong association between the number of days out of work and broadly defined insomnia (insomnia with co-morbidities such as cardiovascular disease), suggesting that individuals with insomnia are more likely to experience greater number of days of sick from work than good sleepers (Hajak et al., 2011). Furthermore, employees with insomnia are indicated to be absent from the workplace for approximately 3 more days per year than good sleepers and cause significantly higher costs to employers through sick leave and short term disability payments (Kleinman, Brook, Doan, Melkonian, & Baran, 2009). Moreover, it is proposed that presenteeism, being present at work but unproductive, could equate to higher costs to the employer (Bolge, Doan, Kannan, & Baran, 2009; Kessler et al., 2011) evidenced by a loss of up to 70% in workplace productivity from those with severe insomnia (as defined using the ISI) comparative to a good sleeping individual (Sarsour, Kalsekar, Swindle, Foley, & Walsh, 2011). Due to daytime consequences of insomnia (cognitive and motor dysfunctions), individuals with insomnia are posited to have an increased risk for having serious road accidents comparative to good sleepers (Leger, Massuel, & Metlaine, 2006). Furthermore, it is suggested that the direct and indirect costs are over $1000 greater over the course of six months for those with untreated insomnia than those that are currently being treated (Ozminkowski, Wang, & Walsh, 2007).

1.5 Morbidity Associated with Insomnia

Another indirect cost of insomnia pertains to the effect of associated secondary conditions that impact on the individual's quality of life. Certainly, individuals with insomnia often report feelings of fatigue, reduced performance, worry, difficulty in concentration, irritability, and depression (Carey, Moul, Pilkonis, Germain, & Buysse, 2005; Moul et al., 2002). Furthermore, Carey et al. (2005) noted, via a focus group with insomnia patients, that individuals felt the severity and impact of their symptoms to be misunderstood by their partners, family and caregivers, evidencing a negative cost to social functioning as a consequence of insomnia.

Insomnia has also been identified as having an impact on both mental and physical health. The DSM-IV states that disturbances to the natural sleep pattern is a symptom of both depression and anxiety (Neckelmann, Mykletun, & Dahl, 2007), Indeed, the links between insomnia, anxiety and depression are well established, sleep disturbances not only indicate the risk of relapse into depressive states (Perlis, Giles, Buysse, Tu, & Kupfer, 1997), but also, insomnia increases the risk of developing depression at a later stage two-fold (Baglioni et al., 2011; Riemann & Voderholzer, 2003). Whereas anxiety is most likely to precede or occur concurrently with insomnia, insomnia is most likely to be a predictor of depression (Ohayon & Roth, 2003). The mechanism by which insomnia relates to depression is yet to be determined, but it is suggested that fragmentation of sleep from an increased number of awakenings may be a causal factor (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005), along with alterations in REM sleep (posited to have a role in emotional memory regulation) (Baglioni & Riemann, 2012; Staner, 2010). This relationship will also add to the direct cost of insomnia as determined through the utilisation of the health care system.

As is the case for psychophysiological insomnia, highly stressful periods are associated with disturbances to sleep and directly impact on the afflicted individual's quality of life (Carey et al., 2005; Cheng & Lee, 2011; Sutton, Moldofsky, & Badley, 2001; Theobald,

2004). When controlling for severe depression, a study of 1053 individuals found that those with severe insomnia, classified using DSM-IV criteria, reported significantly lower quality of life and general health (Léger, Scheuermaier, Philip, Paillard, & Guilleminault, 2001). In addition, insomnia has been shown to be associated with heightened perception of somatic symptoms, such as increased pain sensitivity (Léger et al., 2001; Tang, Wright, & Salkovskis, 2007; Wilson, Eriksson, D'Eon, Mikail, & Emery, 2002). Insomnia also reported to associates with alterations in immune functioning, suggesting that individuals with insomnia may have a decreased immune responsive state compared to good sleepers (Irwin, Clark, Kennedy, Christian Gillin, & Ziegler, 2003; Savard, Laroche, Simard, Ivers, & Morin, 2003). Moreover, people with insomnia may experience an increased inflammatory state, as indicated through higher basal levels of the inflammatory cytokine, interleukin-6, suggestive of an increased risk for inflammatory or cardiovascular disorders (Burgos et al., 2006). Again, further evidence towards the impact insomnia has on health and the potential impact to both quality of life and health care utilisation.

1.6 Conclusions and Relation of this Chapter to the Research Questions

Insomnia is a disorder of sleep resulting in issues initiating, maintaining, or gaining sufficiently restful sleep, which in turn, results in a negative impact to one's social or occupational functioning. Broadly, insomnia is a perceived perturbation of the normal sleeping process for a period of at least 3 nights per week over the course of at least 3 months. With a prevalence of up to 30%, it is unsurprising that there is a large associated economic burden in terms of societal costs and the impact on the afflicted individual's psychosocial and occupational functioning.

Although the clinical definition for insomnia as a sleep disorder is well known, vulnerability factors are lesser known and this is what will be explored within this thesis in regards to the Neurocognitive Model (discussed in the following chapter). Insomnia

is a complex disorder with a lack of standardisation between various coding nosologies; as such this thesis will employ the definition of primary insomnia as defined using the DSM-V. Furthermore, the disturbance of sleep will be examined for sleep state misperception, therefore this thesis will employ the methodology suggested within the ICSD-2 and RDC for defining the disparity between subjective and objective sleep.

As the following chapter will discuss, a night of poor sleep may initiate a cascade of events that lead to a vulnerable good sleeping individual to develop insomnia, and this disturbance to sleep initiates a cyclic response resulting in a persistent state of insomnia. The negative consequences that insomnia is suggested to have on occupational and social functioning and health (both mental and physical), are well documented, thus evidencing the high clinical and social relevance for the continuation of study into the development, and potential treatment, of insomnia.

Chapter 2: The Neurocognitive Model: Applications to Environmental Noise

2.0 Introduction

Environmental noise is known to impact on sleep, the World Health Organization (WHO) reports that noise of 35 decibels or more is sufficient to disturb sleep, and thus issued a European directive stating that nocturnal environmental noise should not exceed 40 decibels (World Health Organisation Europe, 2009). It has been shown that noise during sleep can have a negative impact on subjective sleep, by creating a negative perception of sleep quality (Kuroiwa, 2002), and creating a general sense of annoyance during the sleep period (Aasvang, Engdahl, & Rothschild, 2007). Moreover, nocturnal noise has also been shown to cause disruptions to objective sleep: affecting both the structure of sleep by increasing the amount of light sleep at the expense of deep sleep, and by increasing arousals, therefore affecting measures of both sleep continuity (Griefahn, 2002; Griefahn, Marks, & Robens, 2006) and sleep architecture (Basner, Glatz, Griefahn, Penzel, & Samel, 2008; Basner & Samel, 2005). Furthermore, noise during the night has been shown to affect physiological functioning as measured through altered cardiac response and partial blunting of the cortisol awakening response (Carter, Hunyor, Crawford, Kelly, & Smith, 1994; Waye, Clow, Edwards, Hucklebridge, & Rylander, 2003).

Alterations of both subjective and objective sleep occur in insomnia, as such it is important to examine whether the vulnerability to disruption of sleep by noise contributes to the persistence of insomnia. As noise is a potential disrupter to sleep, this thesis will consider whether noise can be applied to existing models of insomnia that chart its developmental course, specifically the Neurocognitive Model (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). This chapter aims to explore the models of

insomnia that propose increased levels of arousal as a potential mechanism by which sleep is disturbed culminating in exploration of the Neurocognitive model and how auditory stimuli may be used to test the propositions of this model in subsequent empirical chapters.

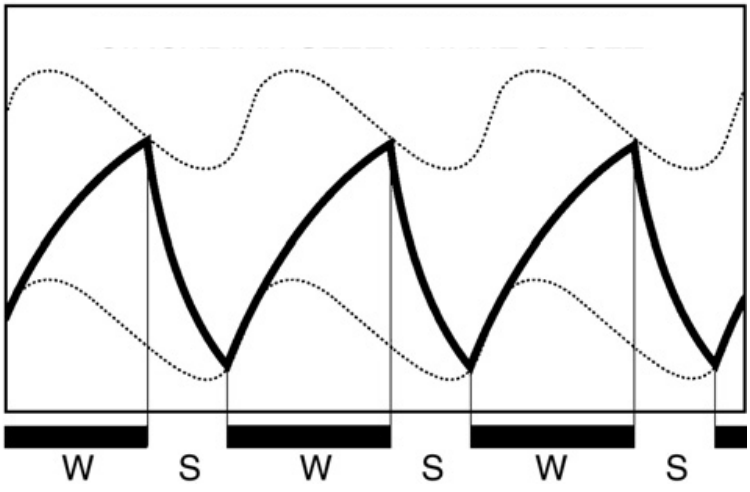
2.1 Models of Normal Sleep Processes

Before the models of insomnia can be discussed, the two fundamental models of normal sleep need to be defined: the two process model of sleep (Borbely, 1982) and the Sleep Switch/Flip-Flop model (Saper, Chou, & Scammell, 2001).

2.1.1 Two Process Model of Sleep Regulation

The two process model of sleep defines the dynamic nature of sleep documenting how the sleep wake cycle can adjust in response to an individual's need for sleep through two distinct processes: process S, the sleep homeostat (a measure of sleep drive), and process C, or the phase of the circadian system, Figure 2.1 (Borbely, 1982).

Figure 2.1: The sleep wake cycle with the waxing and waning of process S (the solid black line), influenced by the thresholds of process C (shown by the dotted line) adapted from Beersma & Gordijn (2007).



Process S is representative of the body's homeostatic drive for sleep and therefore varies in response to prior time spent awake: increasing during wake, as sleep debt accumulates, and decreasing during the night as sleep is replenished. This drive is

regulated by an upper and lower threshold, suggested to be related to the circadian phase (process C) (Beersma & Gordijn, 2007). Process S increases in line with the duration of wakefulness until the upper threshold is reached at which point sleep onset is most likely to occur, therefore, this upper threshold can be defined as the sleep transition threshold. As sleep is attained, process S decreases until it reaches a lower threshold, whereby it is most likely that wake will occur and therefore, this lower threshold could be defined as the sleep cessation threshold.

The circadian system (the internal 24hr biological rhythm) is influenced by external "zeitgebers" (time givers) that entrain the primary internal biological clock located in the suprachiasmatic nucleus (SCN) in the brain (Aschoff, 1965). The SCN is influenced by environmental non-photic factors such as temperature and food but the key influence on the SCN is light (photic) (for review see Mistlberger & Skene,2005). Light acts upon the SCN via melanopsin containing, non-visual, retinal ganglion cells within the eye that have direct axonal projections to the SCN via the retinohypothalmic pathway (Hattar, Liao, Takao, Berson, & Yau, 2002). Within the SCN, light causes the release of an excitatory neurotransmitter, glutamate which signals the transcription and translation of the clock genes (the *Period* and *Cryptochrome* genes) (Rosenwasser, 2009). The transcription/translation of clock genes instigates a negative feedback loop, generating the near 24hr daily oscillations seen in human circadian physiology, such as core body temperature rhythms, cortisol secretion, and the waxing and waning levels of the "sleep" hormone melatonin. Therefore process C can be defined as the individuals biological timing (circadian phase).

Process C is represented within the two-process model as two parallel circadian oscillations (Figure 2.1). These thresholds are not finite levels defining the time of sleep onset/offset, but rather the point at which the body is maximally ready to sleep or wake. Process S can rise beyond the upper threshold with extended wakefulness, but sleep duration and quality will be modulated by the level of the lower threshold. Circadian

desynchrony is the process of uncoupling process S from process C. By extending the wake rest cycle to 28hrs, process S is forced to extend beyond the thresholds of process C, which has been shown to result in variations of total sleep time and sleep onset latency depending on process C (Dijk & Czeisler, 1995). This suggests that although sleep can occur due to the drive from process S, independent of the circadian system, in the absence of the relationship between the two processes, sleep can become altered. Within the context of the two process model, vigilance induced wakefulness and/or alterations to the sleep drive system (whereby process S takes too long to reach the upper threshold or decreases too rapidly during sleep) may all lead to insomnia due to the uncoupling of the circadian system and the homeostatic sleep drive (Beersma, 2002).

2.1.2 The Sleep Switch/ Flip-Flop Model of the Hypothalamic Control of Sleep

The role of the SCN as the body's internal clock is well-established; however, the methods by which sleep is driven are less clear (Economo, 1930; Edgar, Dement, & Fuller, 1993). Numerous studies have hypothesised that sleep is under the control of a wake promoting brain region and an opposing sleep promoting brain region. Lesion studies have identified that the wake promoting area of the brain is located within the posterior lateral hypothalamus (Lin, Sakai, & Jouvet, 1994) and that the ventrolateral preoptic (VLPO) nucleus promotes sleep (Gaus, Strecker, Tate, Parker, & Saper, 2002).

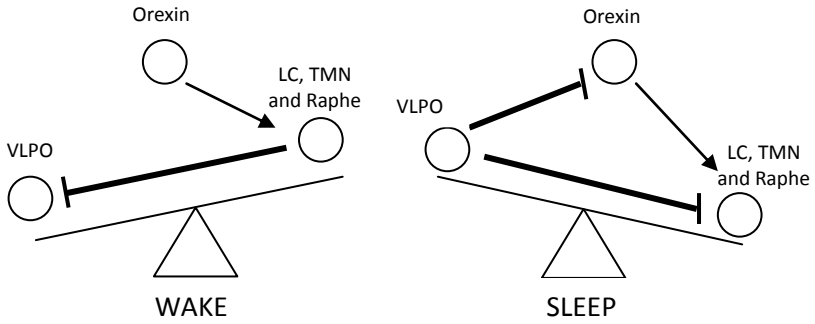
The wake promoting region is regulated by the ascending arousal system with aminergic projections from the raphé nucleus (serotonin), the locus coeruleus (noradrenaline), and the tuberomammillary nucleus (histamine) diffusing throughout the forebrain and cortex and cholinergic projections from the laterodorsal tegmental nuclei and the pedunculopontine tegmental nuclei to the thalamus (Magoun, 1952). The arousal system begins with the cholinergic nuclei based in the upper pons and the

raphé and locus coeruleus in the lateral hypothalamus. Lesions to this area have been shown to inhibit arousal and cause a narcoleptic-type state in rats (Gerashchenko et al., 2001).

During wake, the neurons of the arousal system are highly active and fire rapidly, thus an aroused state is exhibited through the release of excitatory neurotransmitters, however, during sleep, these neurons have a slower firing rate (Vanni-Mercier, Gigout, Debilly, & Lin, 2003). Studies have shown that the VLPO area of the hypothalamus has neuronal projections to all major areas of the ascending arousal system which are sleep active, expressing the fos neuropeptide (a marker for neuronal activation) and the inhibitory neurotransmitters galanin (GAL) and GABA (Sherin, Shiromani, McCarley, & Saper, 1996). Lesions to the VLPO result in prolonged wakefulness and insomnia-like symptoms (such as difficulties initiating and maintaining sleep) suggest that it is this brain region that inhibits the monoaminergic projections of the arousal system (Lu, Greco, Shiromani, & Saper, 2000).

The flip-flop model (Saper et al., 2001) proposes that the sleep/wake state is a feedback loop between the inhibitory VLPO and the ascending arousal system, hence sleep is under hypothalamic regulation. As both the wake and sleep promoting centres have neuronal projections that inhibit one another, it raises the question of how sleep and wake states are regulated. Alternatively, Saper and colleagues (2001) suggest a fragmented flicking pattern within one system as seen in Figure 2.2 (Saper et al., 2001).

Figure 2.2: The flip-flop model of sleep regulation or the sleep switch point adapted from Saper, Scammell, & Lu (2005). LC, locus coeruleus; TMN, tuberomammillary nucleus; VLPO, ventrolateral preoptic nucleus.



The flip-flop model of sleep regulation suggests that both the sleep promoting VLPO and the wake promoting arousal system are governed by orexin/hypocretin secreting neurons located in the peri-fornical area of the lateral hypothalamus (Saper et al., 2005). Orexin is known to be an excitatory neurotransmitter and the neurons that secrete orexin are located within many areas of the brain including the ascending arousal system (de Lecea et al., 1998). Orexin binds through G-coupled protein receptors and, although there are connections between the orexin neurons and the VLPO, the VLPO does not have receptors capable of binding orexin (Marcus et al., 2001). According to Saper and colleagues (2005), the presence of orexin stabilises the sleep/wake system and a seesaw system is created. During wake, orexin neurons are active and release excitatory neurotransmitters that in turn activate the ascending arousal system. As the arousal system is active, the histaminergic and noradrenergic neurons inhibit the VLPO and thus wake is stable. Whereas during sleep, the VLPO becomes active releasing inhibitory neurotransmitters (GAL and GABA) resulting in the inhibition of both the orexin neurons and those of the ascending arousal system, thus sleep is stable.

In the case of insomnia, this model suggests that any dysfunction in the neurotransmitter release, as explained by a lesion study, could result in a reduction of

the stability of the sleep/wake state (Lu et al., 2000). If the switch is unstable, arousal systems could remain switched on, in an active state, leading to impaired sleep or a vulnerability to environmental intrusions to sleep, a point which is discussed later in relation to the Neurobiological model of insomnia (Buysse, Germain, Hall, Monk, & Nofzinger, 2011).

2.2 The Fundamental Models of Insomnia

Two key models are widely considered fundamental for defining the processes that lead to the development and maintenance of insomnia; the stimulus control model of insomnia (Bootzin, 1972), and the Cumulative or 3P model of insomnia (Spielman, 1986).

2.2.1 Stimulus Control Model of Insomnia

The stimulus control model of insomnia states that people with insomnia can develop a conditioned arousal state which leads to associations between the bedroom and the bedroom routine not being a suitable environment for sleep (Bootzin, 1972). To treat insomnia, Bootzin (1972) states that a strong re-conditioning response to sleep in the bedroom environment (i.e. individuals' must only go to bed when sleepy and use the bedroom for sleep alone) is needed to counteract the developed conditioned arousal occurring in the bedroom. Furthermore, distracting items such as televisions and laptops must be removed from the room to decondition the bedroom as a place of wakefulness and enforce the feeling of sleepiness and rest within the bedroom.

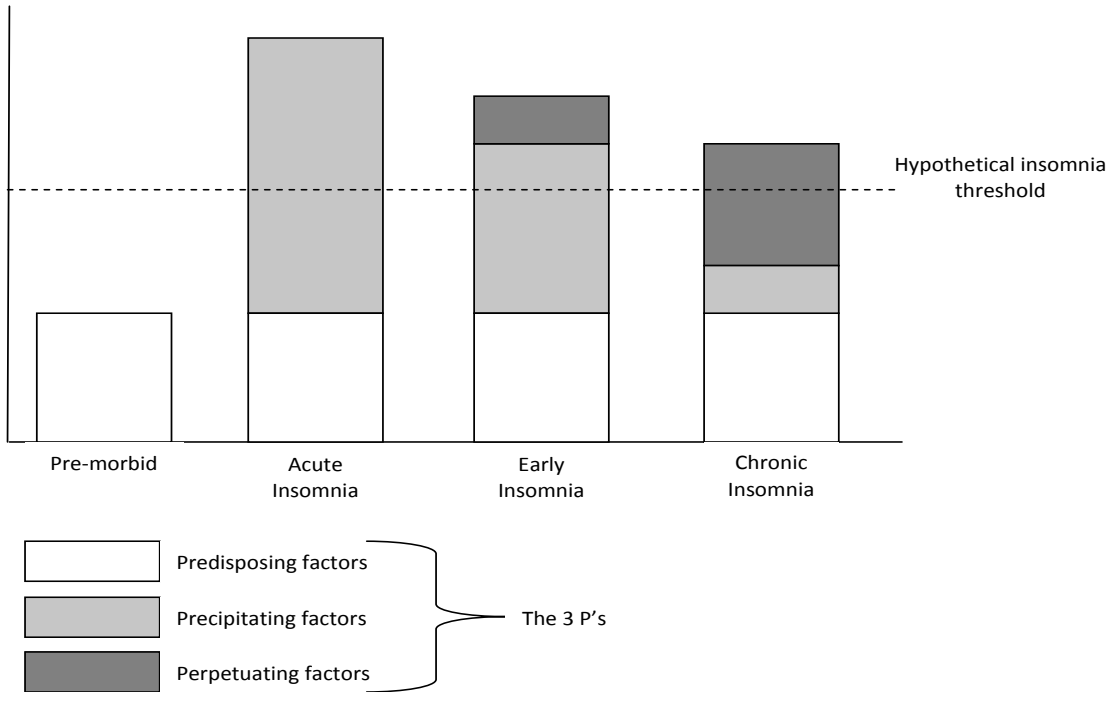
This model proposes that people with insomnia may misinterpret or not be aware of the internal cues for sleepiness, and thus require retraining of the associations between the bedroom environment and the internal physiological feelings of sleepiness. Only entering the bed when sleepy enforces this association to the internal cues for sleep, whereas leaving the bed if sleep is not achieved aids to weaken the association between the feeling of alertness and wake in the bedroom (Sloan et al., 1993). This

process initiates a restriction of time in bed, which has been shown to effectively alleviate insomnia symptoms (Spielman, Saskin, & Thorpy, 1987). Although this model does not necessarily chart the progression of insomnia, it was one of the first models to associate an arousal state with the persistence of insomnia.

2.2.2 The Cumulative/3P Model of Insomnia

The 3P model of insomnia is one of the only models to chart the progression of insomnia from a pre-morbid state to a chronic, severe condition (Spielman, Caruso, & Glovinsky, 1987). In 1986, Spielman first described three factors that are suggested to act in a cumulative fashion to reach and exceed a hypothetical insomnia threshold,

Figure 2.3: Graphical representation of the 3P model showing how the 3 factors accumulate successively in the progression of insomnia from a pre-morbid state to chronic insomnia adapted from Spielman, Caruso, et al., (1987).



The first factor suggests that some individuals may have a higher vulnerability to developing insomnia (an insomnia predisposition) than others. Predisposing factors represent the pre-insomnia state, as these factors alone are not sufficient to trigger an insomnia episode. These predisposing factors are exacerbated by a precipitating factor, usually a stressor, surpassing the individuals' threshold for insomnia resulting in

an acute insomnia phase. The final factor comprises perpetuating factors (i.e. learned sleep preventing behaviours) that maintain the insomnia severity over time, even in the absence of the factors associated with the initial precipitating event.

2.2.2.1 Predisposing Factors

All individuals have a level of predisposition to insomnia, these factors are not causative of insomnia, but rather increase the individual's vulnerability, raising an individual closer to a hypothetical insomnia threshold. As discussed in Chapter 1, evidence regarding idiopathic insomnia suggests a genetic predisposition to insomnia, with studies indicating that those who have insomnia are more likely to have a familial history (a genetic predisposition) (Bastien & Morin, 2000; Dauvilliers et al., 2005; Watson et al., 2006; Wing et al., 2012). Spielman (1986) proposed that a history of poor sleep can act as a predisposing factor of insomnia, this is evidenced by a recent study charting the natural history of poor sleep finding that, after an average follow-up duration of 7.5 years, 17% of individuals classified as poor sleepers had gone on to experience chronic insomnia (Fernandez-Mendoza et al., 2012).

Individuals with insomnia who have a familial history of insomnia are also more like to experience higher levels of anxiety and basal levels of arousal than those without (Beaulieu-Bonneau et al., 2007), which in turn suggests that arousal and anxiety levels are also predisposing factors. As discussed in Chapter 1 within the context of psychophysiological insomnia, a period of life stress is suggested to be a causative agent in the development of insomnia. Although this could be considered within the precipitating dimension of insomnia according to Spielman's model, studies have shown that people with a greater reactivity to stress could be predisposed to developing insomnia (Drake, Richardson, Roehrs, Scofield, & Roth, 2004), and that there is a heritable component to stress induced sleep loss (Drake, Scofield, & Roth, 2008). It has also been suggested that whilst the number of stressful events that an

individual experiences would be considered a precipitating factor, an individual may exhibit an inadequate coping mechanism to deal with stressful events, which can raise an individuals' predisposition to developing insomnia (Morin, Rodrigue, & Ivers, 2003).

Along with stress reactivity and coping skills, prior mental health history may also be a predisposing factor. Certainly it has been shown that insomnia is often co-morbid with anxiety and depression and these mental health issues correlate with the severity and duration of insomnia (Ohayon & Roth, 2003). Moreover, it has been demonstrated that individuals that show high internalisation in depression are at a higher emotional arousal state, which again could increase an individuals' predisposition to insomnia (Kales, Caldwell, Preston, Healey, & Kales, 1976).

Personality traits may also be construed as a predisposing factor in the development of insomnia. Introversion is linked to higher levels of cortical arousal and has been suggested to be associated with objective insomnia (Dorsey & Bootzin, 1997; Killgore, Richards, Killgore, Kamimori, & Balkin, 2007). A population based study of 464 good sleepers found that higher arousal, poor self-reported mental health, and lower extroversion scores led to a higher risk for developing insomnia over the course of three years (LeBlanc et al., 2009). In addition, high levels of perfectionism have been associated with insomnia (Lundh, Broman, Hetta, & Saboonchi, 1994) with measures relating to self-doubt about actions, parental criticism, and concerns over mistakes, significantly greater in those with insomnia comparative to good sleepers (Vincent & Walker, 2000).

2.2.2.2 Precipitating Factors

Precipitating factors are thought to be stressful events, be that psychosocial, environmental, behavioural, or physiological which can act as triggers for the transition from normal sleep to an acute phase of insomnia. Certainly, the predisposition to insomnia seems to be highly related to coping styles and vulnerability to stress. It is the

interaction between the precipitating factor and the predisposing factor that results in the initial period of sleep loss, for example a period of greater psychosocial stress coupled with a poor ability to cope with said stress (Âkerstedt, 2006).

A study of 327 clinical patients at a sleep disorders centre found that negative issues surrounding health, family and work/school were significant precipitating stressful life events in the development of insomnia (Bastien, Vallieres, & Morin, 2004). Furthermore, Bastien and colleagues (2004) evidenced that the nature of the perceived precipitating event correlated with the age at which insomnia began: work/school commitments correlated with early onset of insomnia (<30 years of age) and health issues correlated with later onset of insomnia (>30 years of age). Moreover, it has been indicated, in a study of 816 workers, that the presence of psychosocial work stress was related to the development of sleep problems within a six-month period (Linton, 2004). Negative life events, such as divorce (Ribet & Derriennic, 1999), and bereavement (Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010; Monk, Germain, & Reynolds, 2008) have also been suggested as precipitating factors in insomnia. In conditions such as post-traumatic stress disorder (PTSD), where disturbances to sleep are experienced (Germain, Buysse, Shear, Fayyad, & Austin, 2004), insomnia can persist even when the PTSD has been treated (Zayfert & DeViva, 2004), suggesting that PTSD could be the precipitating factor and that the persistence of insomnia is explained by perpetuating factors that surpass the occurrence of the precipitant.

2.2.2.3 Perpetuating Factors

Whereas the precipitating factor results in the reaching of the insomnia threshold and an acute period of insomnia, the presence of perpetuating factors maintains insomnia when the impact of the precipitating factor has ceased.

Perpetuating factors are thought to be learned negative cognitions and behaviours developed during the acute period of insomnia in response to the initial sleep loss and

maladaptive compensatory behaviours which results in a preoccupation with sleep and dysfunctional beliefs about the negative consequences of sleep. These behaviours include the dissociation of the bed as a sleep environment and maladaptive sleeping habits, such as extension of time in bed, as discussed in the stimulus control model of insomnia (Bootzin, 1972). Arousal, both physiological and psychological, occurs prior to sleep, increasing cognitive activity and an inability to down-regulate arousal prior to sleep. Worry prior to sleep, intrusive thoughts, and rumination are all posited as being perpetuating factors and have been shown to increase sleep onset latency along with leading to poorer subjective sleep quality (Thomsen, Yung Mehlsen, Christensen, & Zachariae, 2003).

2.3 Supporting Arousal Models of Insomnia

Whereas Spielman, et al. (1987) examined the developmental course of insomnia, the following models assume the acute period of insomnia has passed and examine the perpetuating factors associated with the development of a chronic insomnia state.

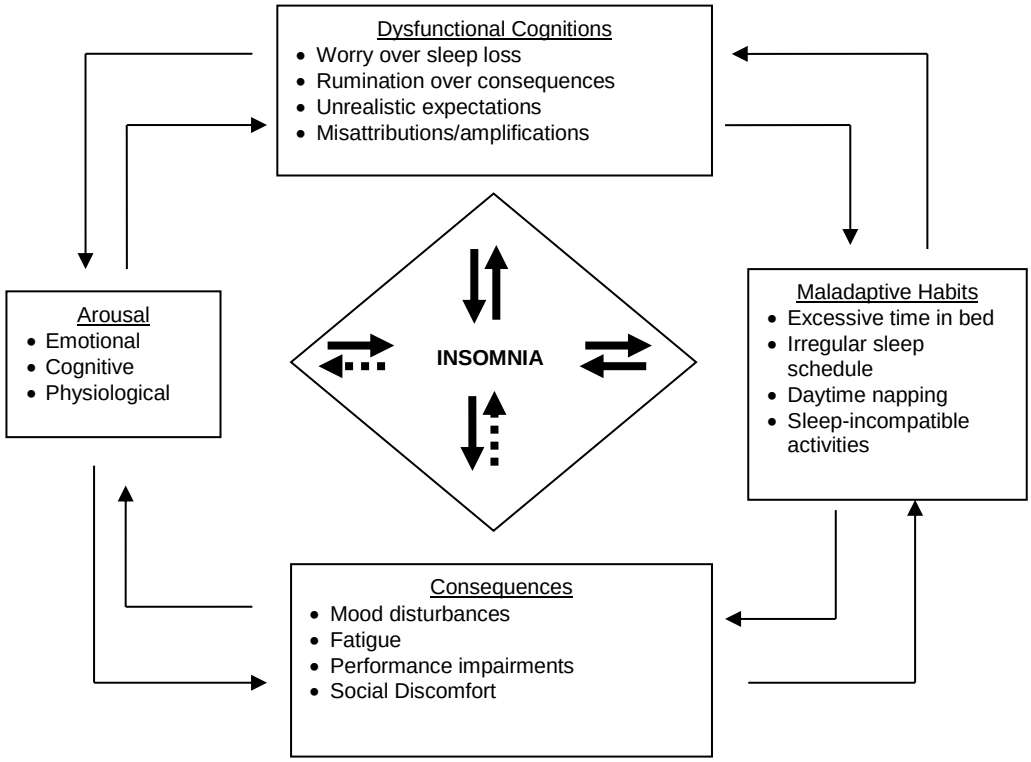
Of the models discussed here, all use a concept of conditioned arousal, be that cognitive, somatic, or cortical in nature, which results in the maintenance of insomnia. A general higher basal state of arousal comparative to good sleepers has been shown to be evident in people with psychophysiological insomnia, and to a lesser extent, those with sleep state misperception, as shown by a higher metabolic level (Bonnet & Arand, 1995; Bonnet & Arand, 1997). This indicates that individuals with insomnia may be continually at a higher arousal level than their good sleeping counterparts. Evidence shows that although people with insomnia show an equal number of daily stressors comparative to good sleepers, people with insomnia rate the impact of minor life stressors and the intensity of major negative life events higher than that of good sleepers, suggesting a greater sensitivity to life related arousal events (Morin et al., 2003).

2.3.1 Microanalytical Model

The Microanalytical model of insomnia is an integrative view of insomnia based upon the stimulus-organism-response-consequence (SORC) model (Morin, 1993). Utilising the principles of operant conditioning, the model implies that following a stimulus, the organism will dictate the response and then modify future behaviour towards a repeated experience of that stimulus (see Figure 2.4.). The Microanalytical model does not provide a framework to identify one single causative factor that leads to a cascade of responses, but rather identifies four key elements (arousal, maladaptive habits, dysfunctional cognitions, and consequences) that inter-relate in the maintenance of insomnia. The model is cyclic, with the four factors having both a role in the development of insomnia and occurring as a direct consequence of insomnia.

This model suggests that emotional, cognitive and physiologic arousal occur pre-sleep as a consequence of not sleeping and feed into the propagation of the insomnia, eventually only partially being driven by the disorder. The model proposes that individuals can be predisposed to insomnia by having a higher level of basal arousal, as Spielman's (1986) 3P model. With regard to the SORC model and following Bootzin's (1972) stimulus control model, the bedroom can become the arousing stimuli along with apprehension and worry about trying to sleep - reinforcing this arousal. Following the initial sleep loss, the individual can develop dysfunctional cognitions regarding sleep, such as unrealistic expectations of sleep need and a distorted perception of the consequences of sleep loss, thus exacerbating the perception of the sleep problem. Therefore, dysfunctional cognitions are perpetuating factors in the progression of an insomnia disorder and, as in the SORC model the response is described as a consequence to sleep loss, Morin (1993) suggests that these include mood disturbances, fatigue and performance impairments.

Figure 2.4: A Microanalytical model of insomnia adapted from Morin (1993) with sold arrows indicating a strong directional relationship and the dotted arrows indicating a weaker directional relationship.



2.3.2 Integrative Model of Insomnia

Further to Morin's Microanalytical model (1993), Lundh and Broman (2000) proposed an integrative model suggesting that insomnia is a product of the relationship between sleep-interpreting and sleep-interfering processes. Figure 2.5 depicts how the two processes interact leading to the development of insomnia, with the sleep interfering processes depicted within the four boxes at the top of the diagram (arousability, stimulus-arousal associations, behavioural and cognitive strategies, and interpersonal relations) and the three boxes below representing sleep-interpreting processes (dysfunctional attributions of sleep, perfectionism, and dysfunctional beliefs about sleep). The sleep-interfering processes cause arousal whereas the sleep-interpreting processes result in the dysfunctional appraisal of sleep.

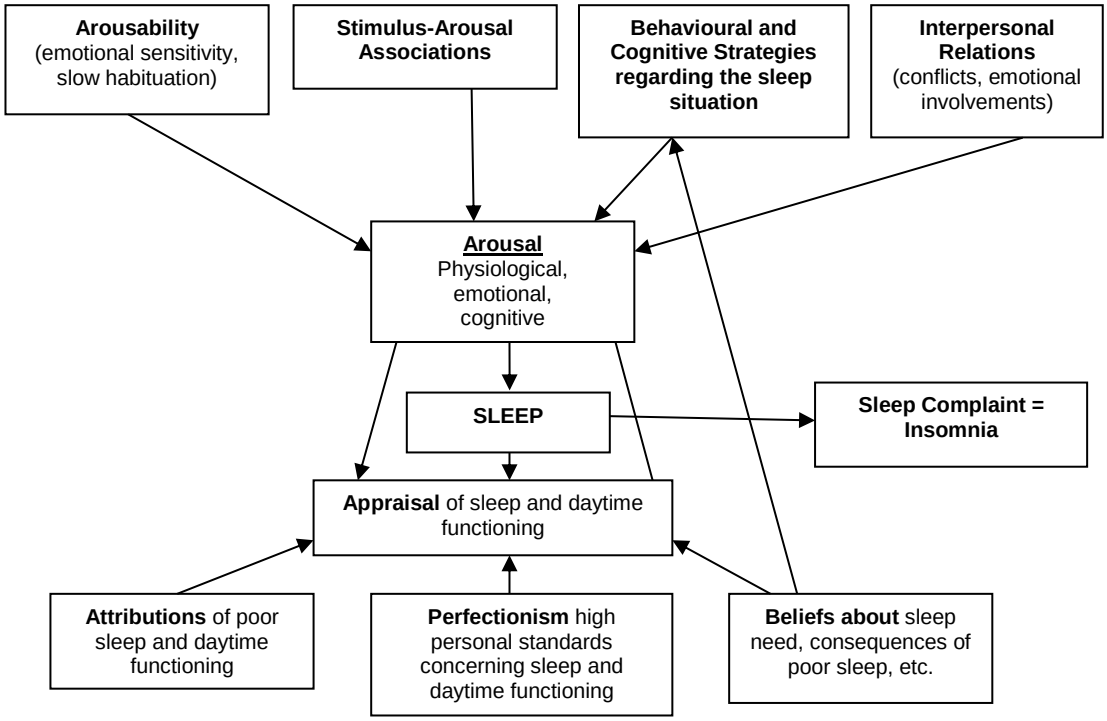
Sleep interfering factors relate to a high basal state of arousal or an inability to habituate to a stressful situation. Under Spielman's (1986) model this could be described as a predisposing factor. Lundh and Broman (2000) also acknowledge the propositions within Bootzin's stimulus control model (1972) as being interfering factors. As the sleep problems persist, individuals will associate the bedroom with arousal rather than sleep and thus the room becomes sleep interfering as behavioural and cognitive mechanisms for coping with perceived sleep loss are adopted. Uniquely, interpersonal and family relationships are included in the final sleep interfering factor. It is reported that people with insomnia are also more vulnerable to the effects of emotional burden than good sleepers with personality traits, such as a tendency to worry, said to act as sleep interfering processes (Lundh, Broman, & Hetta, 1995).

Sleep-interpreting processes are said to disrupt normal sleep through an increase in the appraisal of sleep and perceived daytime consequences of poor sleep. Lundh et al. (1995) suggest that, as insomnia is not akin to sleep deprivation, when assessed through objective measures insomnia can occur entirely subjectively, as in sleep state misperception. As such, the nature of the disturbance to sleep through insomnia cannot be based on sleep interference alone. Attributions of poor sleep and reduced daytime function, as well as dysfunctional beliefs about the consequences of poor sleep all feed into a negative appraisal of sleep resulting in sleep-interfering arousal prior to sleep. Similar to Spielman (1986), Lundh and Broman (2000) suggest that people with insomnia may have a higher sense of perfectionism, thus high personal standards surrounding sleep ability and the need to function highly during the day (Lundh et al., 1994). However, it has been suggested that although people with insomnia show higher levels of perfectionism through self-doubt, they do not show higher personal standards, suggesting that it is the doubt in one's ability to sleep rather than the struggle to get a perceived good night's sleep that relates to poorer sleep appraisals (Vincent & Walker, 2000). Moreover, it has been indicated that perfectionism may not

be a single predisposing factor to insomnia but requires associated emotional distress, a sleep-interfering component, as the association between high perfectionism and insomnia becomes non-significant if emotional distress is accounted for (Jansson-Fröjmark & Linton, 2007). That said, a recent review suggests that more longitudinal data is needed in the role of personality as a predisposing/perpetuating factor or consequence of the sleep disorder (van de Laar, Verbeek, Pevernagie, Aldenkamp, & Overeem, 2010).

Sleep-interfering and sleep-interpreting processes result in cognitive, physiological and emotional arousal that interfere with the normal sleep process and in turn maintain insomnia. A range of physiological measures have been shown to be elevated in people with insomnia, compared to good sleepers, such as heart rate variability, body temperature, vasoconstriction and the number of body movements during sleep (Bonnet & Arand, 1998; Monroe, 1967). The same has been found for measures of cognitive arousal such as pre-sleep worry, negative thoughts and rumination prior to sleep (Carney, Harris, Moss, & Edinger, 2010; Harvey, 2001a; Harvey & Greenall, 2003; Jansson-Frojmark & Norell-Clarke, 2012). Emotional arousal is also included within Lundh and Broman's (2000) Integrative model viewed within a cognitive arousal framework, as sleep-related rumination tends to be of a negative valence (Baglioni et al., 2010; Bélanger, Morin, Gendron, & Blais, 2005). The drawback of this model however, is that no mention is made as to which of the sleep-interfering factors or the appraisal of sleep result in which form of arousal (be that physiological, cognitive or cortical) or whether there are relationships between the differing types of arousal.

Figure 2.5: Insomnia viewed as an interaction between sleep-interfering and sleep-interpreting processes, adapted from Lundh & Broman (2000). Factors that lead to an arousal state are sleep-interfering whereas those that lead to the appraisal of sleep are sleep-interpreting.



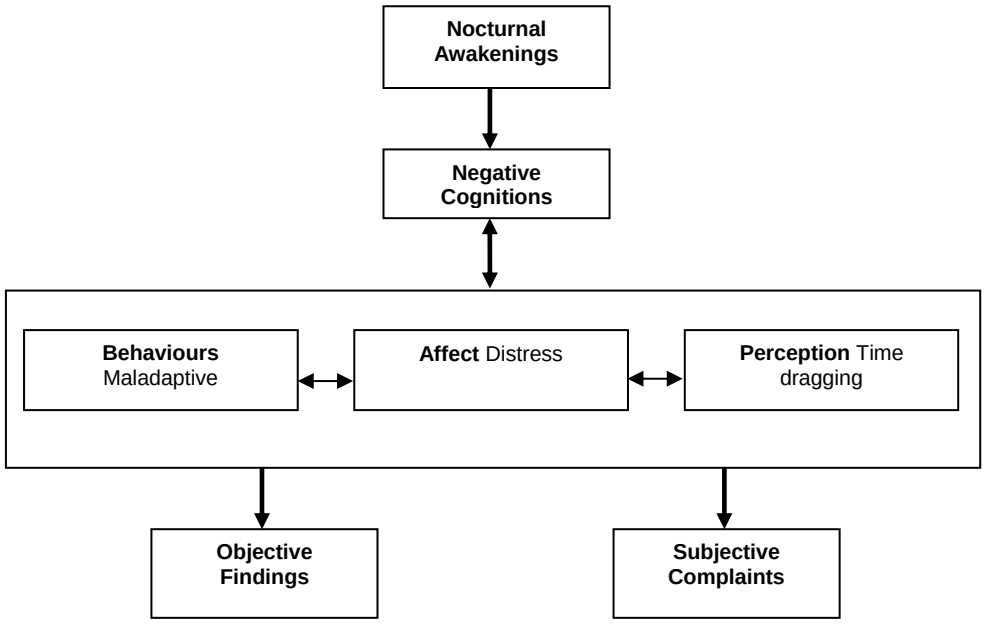
2.3.3 A Model of Chronic Insomnia in the Elderly

Fichten and Libman's (2001) model of insomnia is the only model to date that focuses specifically on late-life insomnia (Fichten & Libman, 1991; Fichten et al., 2001) (Figure 2.6). This model suggests that, although a natural by-product of aging is more fragmented sleep with frequent and prolonged awakenings (Webb, 1982), not all elderly individuals develop insomnia. Within the model the number of nocturnal awakenings are a predisposing factor in the development of insomnia in the elderly. The authors propose that some individuals engage in negatively toned cognitive activity during these nocturnal extended periods of wakefulness, ruminating on issues of the day as well as becoming preoccupied with the sleep loss and apply more effort to try to achieve sleep.

It is suggested that people with insomnia experience an overestimation of sleep onset latency and the duration of wake during the night (Tang & Harvey, 2004a). Studies

have shown that there is no difference in the ability to correctly estimate time between good sleeping individual's and people with insomnia, suggesting that there must be other factors resulting in the overestimation of wakefulness (Rioux et al., 2006). However, in contrast, it is also reported that during nocturnal awakenings, people with insomnia may perceive a longer wake duration (Fichten et al., 2005). This perception of time dragging can result in distress, which as Lundh and Broman (2000) suggest, could be a form of sleep-interfering arousal. Also contributing to distress, as Bootzin's (1972) stimulus control model suggests, maladaptive sleeping habits or behaviours that are incompatible with sleep, can result in poor sleep or the worry about a perceived detriment to daytime functioning. These three factors (Behavioural, Affective, and Perceptual) are said to result in the subjective complaints of insomnia along with the objective alterations in sleep (Fichten et al., 2001).

Figure 2.6: Adapted from Fichten et al., (2001) showing how prolonged nocturnal wakefulness, as occurring naturally as a product of aging, can result in insomnia through objective changes in sleep coupled with subjective complaints about sleep.



2.3.4 Psychobiological Model

The psychobiological model (Espie, 2002) suggests that normal sleep is an automated biological process and states, with reference to the two process model of sleep regulation by Borbely (1972), that both endogenous and exogenous factors can influence the homeostatic drive for sleep (García-García & Drucker-Colín, 1999). In addition, Espie (2002) proposes that the homeostatic drive for sleep is adaptable, responding to these internal and external factors, to manage and maintain sleep regulation and the normal sleep processes.

The model suggests that there is a default mode for good sleep and that variations in sleep wake patterns are accounted for by plasticity and automaticity surrounding the sleep homeostat and circadian system (shown as the grey square surrounding and "protecting" the inner square in Figure 2.7). The plasticity component refers to the ability of the sleep wake system to respond to psychological and environmental challenges whilst retaining good sleep. On the other hand, the automaticity component is a natural, involuntary response by the individual that adjusts to sleep associations and assumptions/expectations about sleep.

Espie (2002) suggests that there are four potential disrupters of the normal sleep process whereby the automaticity and plasticity of the sleep system employs involuntary defensive properties to aid the protection of sleep. Sleep-related stimulus control identifies a good sleeper's innate ability to recognise the biological cues of sleep and thus prepare adequately for rest by initiating an internal de-arousal process. In agreement with the stimulus control model, (Bootzin, 1972), the psychobiological model suggests that people with insomnia may have associated the bedroom with wake behaviours, have irregular sleep habits, and undergo activities in-bed, such as reading, that are incompatible with sleep. It is also proposed that individuals engage in negative processes before bed that cause physiological arousal, such as the intake of stimulants, like nicotine, known to correlate with poorer sleep quality (Phillips & Danner,

1995). Using voice recorded documentation of subjective reasoning during sleep disturbances, it has been shown that intrusive thoughts, while attempting sleep, prolong sleep onset latency (Wicklow & Espie, 2000). This suggests that whilst in bed, individuals with insomnia may be experiencing rumination of the day, potentially leading to a cognitively aroused state that impairs sleep.

Furthermore, Espie (2002) suggests that the longer the individual remains in bed the more cognitive worry there is surrounding sleep and therefore more effort is applied by the individual to try to attain sleep. This increased effort to control sleep is detrimental to the plasticity of sleep, as sleep is viewed as an autonomic function in this model, and therefore cannot be controlled by the individual, the greater the effort employed by the individual to sleep, the more elusive sleep becomes (Broomfield & Espie, 2005). Moreover, to attain sleep, an individual must become de-aroused both cognitively and physiologically, and these two processes run in parallel: the more effort employed to sleep, the higher the physical arousal the individual experiences.

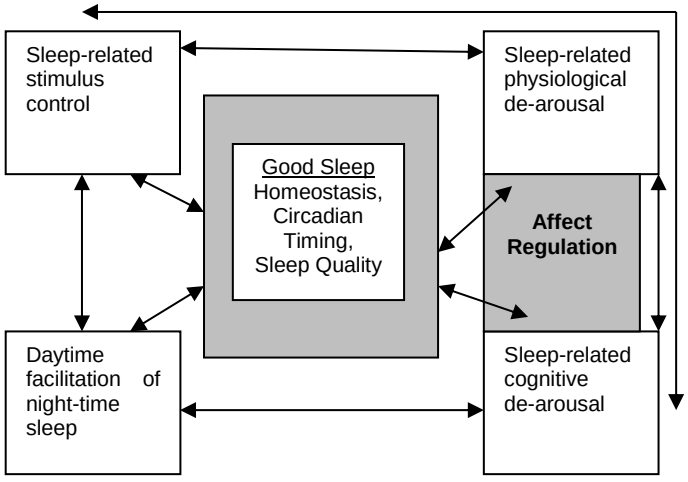
Good sleeping individuals are thought to put less blame on sleep for fatigue experienced during the day, and are less likely to exhibit compensatory patterns for poor sleep, such as daytime napping or increasing time spent in bed. Therefore, the model suggests that good sleepers employ actions during the day that aid facilitation of sleep through maintenance of the sleep drive and, to a lesser extent, learned sleep-related arousal. On the other hand, individuals with insomnia are more likely to be attuned to feelings of fatigue during the day and subsequently employ compensatory behaviours, such as napping. The attention-intention-effort (A-I-E) pathway (Espie et al., 2006) documents the transition of insomnia from an initial sleep problem to a persistent state. The A-I-E pathway suggests that if the individual has an impaired response in plasticity to a night of poor sleep the individual may become attuned to the sleep problem by employing more attention to the physiological signs of sleepiness and sleep onset.

Affect regulation (shown in the grey box in Figure 2.7), represents the interaction between both physiological and cognitive arousal processes. In those with good sleep, this is neutral as the individual puts little conscious effort into sleep, whereas negative emotions arising from increased sleep effort affect the stability of the sleep system in poor sleepers. Additionally Espie (2002) suggests that good sleep processes and cognitions surrounding sleep aid and reinforce the protection of the sleep system:

"…good sleep begets good psychobiological preparation for sleep, which begets good sleep" (Espie, 2002 pp228).

In good sleepers, it is the ability of the plasticity and automaticity surrounding the sleep homeostatic system to adapt, which protects their good sleep. Espie (2002) also suggests that when an individual experiences a precipitating stressful event, the related arousal inhibits the plasticity and automaticity of the sleep system and thus sleep is impaired. This model does not directly include the precipitating or predisposing events that could lead to this impairment of the sleep system. Both physiological and cognitive arousal are said to occur during the day and thus it is a failure to de-arouse prior to sleep as opposed to a general arousal state occurring at sleep onset that is implicated to be the perpetuating factor for developing insomnia.

Figure 2.7: The Psychobiological model of good sleep, adapted from Espie (2002).



2.3.5 A Cognitive Model of Insomnia

In the cognitive model of insomnia (Harvey, 2002), it is suggested that the beliefs held by an individual about the negative consequences of sleep loss can exacerbate and, if not already present, instigate a disruption to sleep, Figure 2.8. In addition, similar to Fichten and Libman's model (1991), a distorted perception of a reduced sleep quality or daytime functioning leads to an actual disruption to sleep.

Unlike the previous models, the cognitive model examines the interaction between both the day and night with regard to perpetuating factors in the maintenance of insomnia. A distorted perception of reduced sleep quality or poor daytime functioning exacerbates negatively toned cognitive activity through worries regarding the effect of sleep loss. This activity is said to trigger coping and control behaviours, such as increasing time in bed to try to achieve the believed "8 hours" sleep (sleep extension, resulting in an increase in sleep effort that, as Broomfield and Espie (2005) suggest, can make sleep more elusive). The implementation of compensatory behaviour is coupled with safety behaviours employed during both the day and night to attempt to achieve good sleep, such as imagery and thought control. It has been shown that people with insomnia often employ worry or reappraisal as safety behaviours to control negative intrusive thoughts in an attempt to suppress them (Harvey, 2001a), and the attempted suppression of sleep related negative thoughts during sleep onset leads to a perceived longer sleep latency and poorer sleep (Harvey, 2003). Attempts to control rumination, which has been shown to be independent of negative mood, relates with increased sleep latency and poorer perceived sleep quality (Thomsen et al., 2003).

Due to these coping and safety behaviours, an individual becomes both cognitively aroused and distressed, experiencing both cognitive arousal and physiological arousal through a heightened autonomic response. A physiological difference has been noted between self-reported poor sleepers and good sleepers, with the former demonstrating a higher core body temperature, higher heart rate, more body movements during the

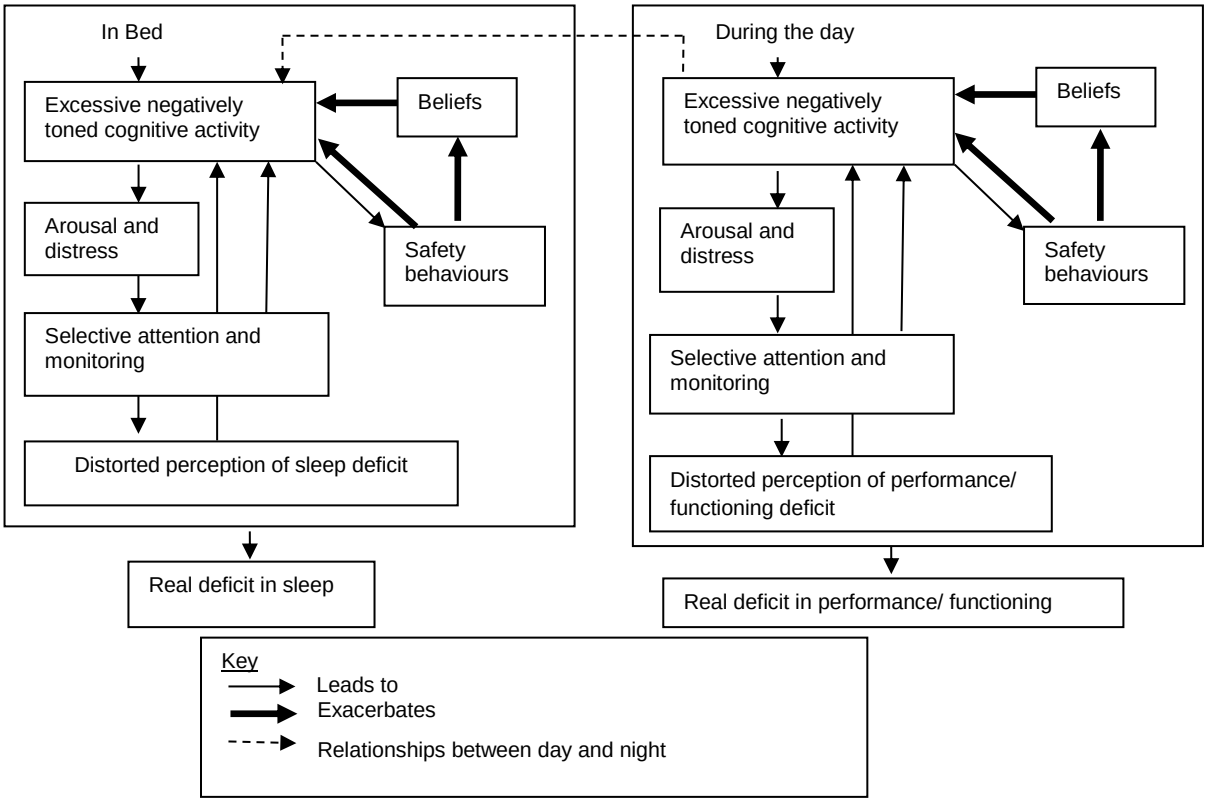
night and a higher mean number of vasoconstrictions (Monroe, 1967) indicative of heightened autonomic arousal persisting during both the day and the night. Although not an insomnia paradigm, it has been demonstrated that the threat of a stressful task, using the speech task paradigm, can instigate changes in heart rate variability in those with good sleep, resulting in poorer sleep quality (Hall et al., 2004). Therefore, it could be suggested that if individuals with insomnia are under heightened threat due to dysfunctional beliefs surrounding perceived sleep loss, this could lead to heightened physiological stress that will, in turn, negatively affect sleep. As Harvey (2002) suggests, the longer these perceived deficits are present for increases the likelihood of forming a real deficit.

The model further states that arousal during the sleep onset process promotes selective attention and monitoring of the environment to ascertain sleep latency or for stimuli that may delay sleep onset, along with monitoring for the physiological sensations of sleep. An example of environmental monitoring through the monitoring of a clock within the bedroom which can lead to longer perceived sleep latencies and even exacerbate pre-sleep worry (Tang, Schmidt, & Harvey, 2007). People with insomnia are proposed to have a selective attention bias for sleep related cues, and particularly those of a negative valence such as clock (Woods, Marchetti, Biello, & Espie, 2009), a process that is fundamental in the psychobiological model, (Espie, 2002). Furthermore, there may be an attention shift in these individuals with insomnia, applying more attention towards sleep and potential sleep disruptors and thus resulting coping safety behaviours to the believed sleep deficit (Harvey, Tang, & Browning, 2005). Individuals with insomnia have been shown to exhibit a heightened sensitivity to the environment during sleep and excessively monitor for the physical sensation of sleeping such as relaxation to indicate sleep onset (Harvey, 2000) as well as documenting significant disturbance to sleep onset through environmental noises (Wicklow & Espie, 2000).

The cognitive model is proposed to be cyclic as arousal and selective monitoring lead to an increasing distorted perception of a sleep deficit. Furthermore, perceived deficits in daytime performance following a night of poor sleep are proposed to exacerbate negative cognitive activity regarding daytime functioning and this builds towards the evening as sleep draws near. Moreover, it is reported that people with insomnia may have a distorted fear of the impact of sleep loss and monitor for the negative effects of sleep loss during the day, further exacerbating the false perception of poor sleep quality (Harvey & Greenall, 2003). This results in a continuing cycle whereby the perceived deficit results in insomnia symptoms and a true sleep/performance deficit. Again, the longer an individual cycles through daytime and night-time arousal, the greater the detriment to actual sleep and daytime performance, which is in agreement with the ICSD-2's diagnosis of psychophysiological insomnia: the longer an individual has psychophysiological insomnia, the worse the detriment to sleep becomes (American Academy of Sleep Medicine, 2005).

Further to the cognitive model, it has been shown that this perceived deficit in sleep can be corrected by showing individuals the differences between their subjective sleep, from a sleep dairy, and their objective sleep, from actigraphy (Tang & Harvey, 2004a). It has also been shown that self-reported symptoms of insomnia correlate with self-reported detriments in daytime functioning (Ustinov et al., 2010).

Figure 2.8: A cognitive model of Insomnia applied to the night and day adapted from Harvey (2002).



2.3.6 The Neurobiological Model

Unlike the previous arousal models, the neurobiological model of insomnia is unique in that it suggests that cognitive alterations exhibited in insomnia are possibly a result of a dysfunction in the wake promoting areas of the brain (Buysse et al., 2011). The model suggests that insomnia may result from persistent wake activity in the brain during NREM sleep, which may be resulting in the activation of both the wake and sleep promoting brain areas simultaneously.

This model expands upon the widely accepted flip-flop model using the concept of the "sleep switch" process in the maintenance of stable sleep and wake (Saper et al., 2005). However, this model proposes a role for localised sleep (distinct localised sleep promoting brain regions), with these distinct groupings of neurons driving sleep or wake states (Hobson, Lydic, & Baghdoyan, 1986). These neural assemblies (groups of

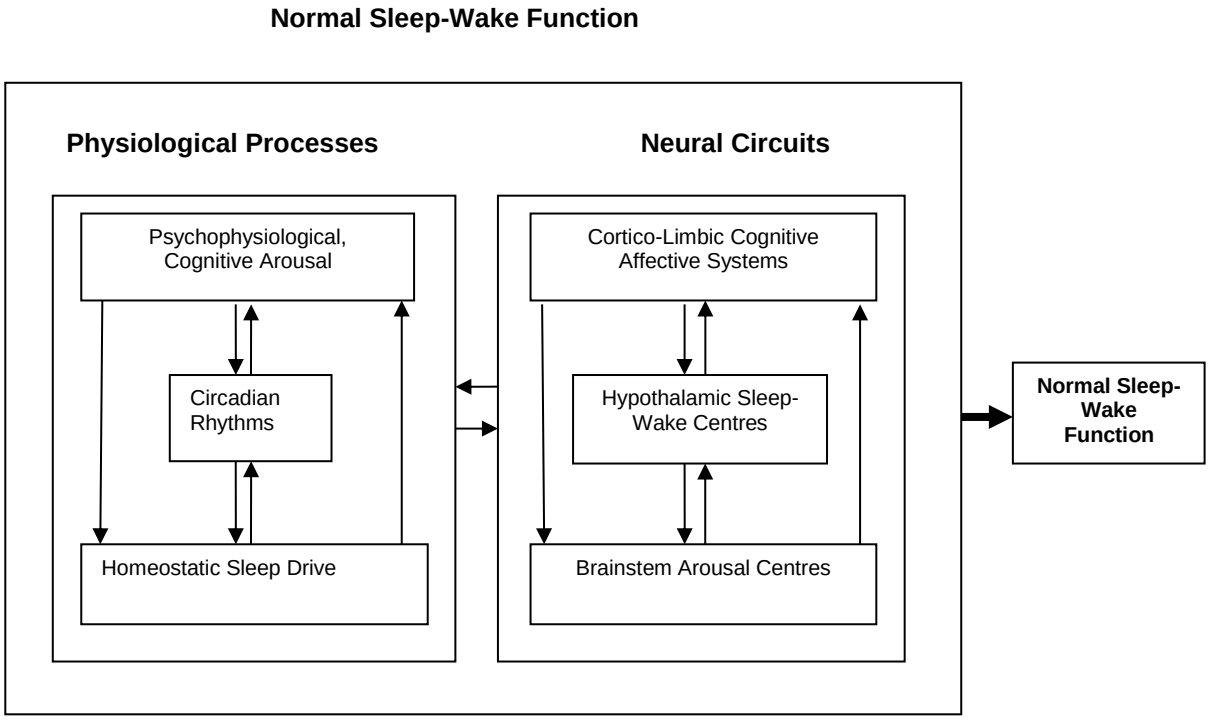
neurons) are made of neural columns which have been shown, in animal models, to display distinct sleep-like functional states (Rector, Topchiy, Carter, & Rojas, 2005), forming local sleep areas (Rector, Schei, Van Dongen, Belenky, & Krueger, 2009). It is thought that, through sleep promoting mediators such as adenosine and tumour necrosis factor α, these columns can transition from a wake like state to a sleep like state independent of other neuronal columns or the greater brain region (Basheer, Strecker, Thakkar, & McCarley, 2004; Churchill et al., 2008; Huang, Urade, & Hayaishi, 2007). Furthermore, it is thought that these individual neuronal assemblies can influence other areas to transition into sleep-like or wake-like states via neurotransmitter release or electrical activity, and it is the proportion of assemblies in this sleep-like state compared to wake-like state that propagate whole brain sleep (for review see Krueger et al., 2008).

The neurobiological model, like the psychobiological model, is a model of good sleep processes, proposing a complementing interplay between neural circuitry and responses in the physiological aspects of sleep, which in turn feedback on the neural circuitry (Figure 2.9). The model states that, in the case of insomnia, the cortico-limbic cognitive-affective system can become aroused which in turn causes activation of the brain stem arousal centres. This arousal of the neuronal assemblies results in a dysfunction in the sleep-wake switch itself, located in the VLPO influencing the physiology of the individual presenting as cognitive arousal. There may also be impairment of the sleep homeostatic drive and the circadian system, which is exacerbating the physiological aspect of insomnia by altering the sleep thresholds underpinning the drive to wakefulness. Whereas insomnia is not associated with significant structural brain differences comparative to good sleeping individuals (Spiegelhalder et al., 2013), it has been shown that individuals with insomnia may have abnormal activation (decreased blood flow) of the central nervous system (basal ganglia) during NREM sleep comparative to those with good sleep, (Smith et al., 2002).

A brain region that, as indicated by Saper et al. (2005), is essential for the regulation of the sleep/wake state.

This model is supported by the cage exchange stress model of insomnia (Saper et al., 2001) where insomnia like sleep states are observed in male rats when transferred to a novel cage during the night that had previously contained another male rat. When the arousal from the limbic system was shut off through experimental lesions, the rats returned to “normal” sleep in the switched cage. Using imaging techniques, research has shown that poor sleepers may be experiencing an increased level of metabolism within the brain, through glucose utilisation comparative to good sleepers which persists during wake, indicative of a general state of arousal within the individual (Nofzinger et al., 2004). Although only cognitive arousal is directly suggested in this model, the activation of the neuronal circuitry and the cortico-limbic system is suggestive of a general cortical arousal. Indeed the activation of neuronal columns to a wake-like state during sleep may suggest why people with insomnia experience an intrusion of high frequency EEG during normal sleep (Krystal et al., 2002; Merica, Blois, & Gaillard, 1998).

Figure 2.9: Proposed Normal Sleep-Wake function adapted from Buysse, et al., (2011).



2.4 The Neurocognitive Model

Whereas the previous insomnia models state that arousal can lead to and/or exacerbate insomnia, the Neurocognitive Model specifically includes cortical arousal, formed over the course of poor sleep through conditioned cognitive and somatic arousal, as the primary developing factor for sleep state misperception and ultimately an actual deficit to sleep. The Neurocognitive Model of insomnia (Perlis, Giles, Mendelson, et al., 1997) is based upon the principles of the 3P model (Spielman, 1986) and indicates how subjective-objective discrepancies in sleep can arise through the perpetuating factor of extended time in bed and related neurocognitive factors (Figure 2.10) (Riemann et al., 2010). Evidence suggests that people with insomnia often report longer sleep latencies, more awakenings and shorter sleep times, resulting in a lower sleep efficiency rating, which is not always evident in objective sleep recordings (for example see Bonnet & Arand, 1997).

The Neurocognitive Model assumes that the insomnia threshold (i.e. Spielman, 1986) has been met, be that through predisposing/precipitating biopsychosocial factors such as responding to stress and personality factors, or through a co-morbid psychiatric problem (for example depression) and that a disruption to sleep is present. Upon this disruption, maladaptive sleep behaviours are initiated by the individual to cope with the current sleep loss, such as the extension of time in bed, and these are said to be the perpetuating factors that establish neurocognitive arousal. This arousal is said to be present during the day and at sleep onset, be that cognitive or somatic, which interact resulting in cortical arousal that persists throughout the night. Cortical arousal is said to be evidenced by the presence of high frequency EEG that is seen to intrude into the sleep of some individuals with insomnia (Krystal et al., 2002; Perlis, Merica, et al., 2001; Perlis, Smith, Andrews, et al., 2001; Spiegelhalder et al., 2012). In turn, it is proposed that this leads to neurocognitive alterations through increased sensory and information processing during sleep and sleep onset, which results in the formation of memories to external stimuli skewing the perception of sleep "as sleep" upon appraisal of sleep the following morning.

The Neurocognitive Model focuses on three levels of evidence that the perception of sleep is altered in insomnia. Firstly, when asked to rate whether they were asleep or awake when awoken from polysomnography (PSG) defined sleep, people with insomnia tend to report wake more frequently than good sleepers, both during the night and during daytime naps, (Borkovec et al., 1981; Mercer, Bootzin, & Lack, 2002). It is suggested that although both good sleepers and people with insomnia are able to perceive awakenings following a consolidated block of sleep, people with insomnia tend to perceive consecutive awakenings as continuous wake, disregarding the sleep period between periods of wake (Coates et al., 1983; Knab & Engel, 1988). Additionally, people with insomnia may take longer to perceive that sleep has occurred, as when examining subjective feelings of being asleep, individuals with insomnia

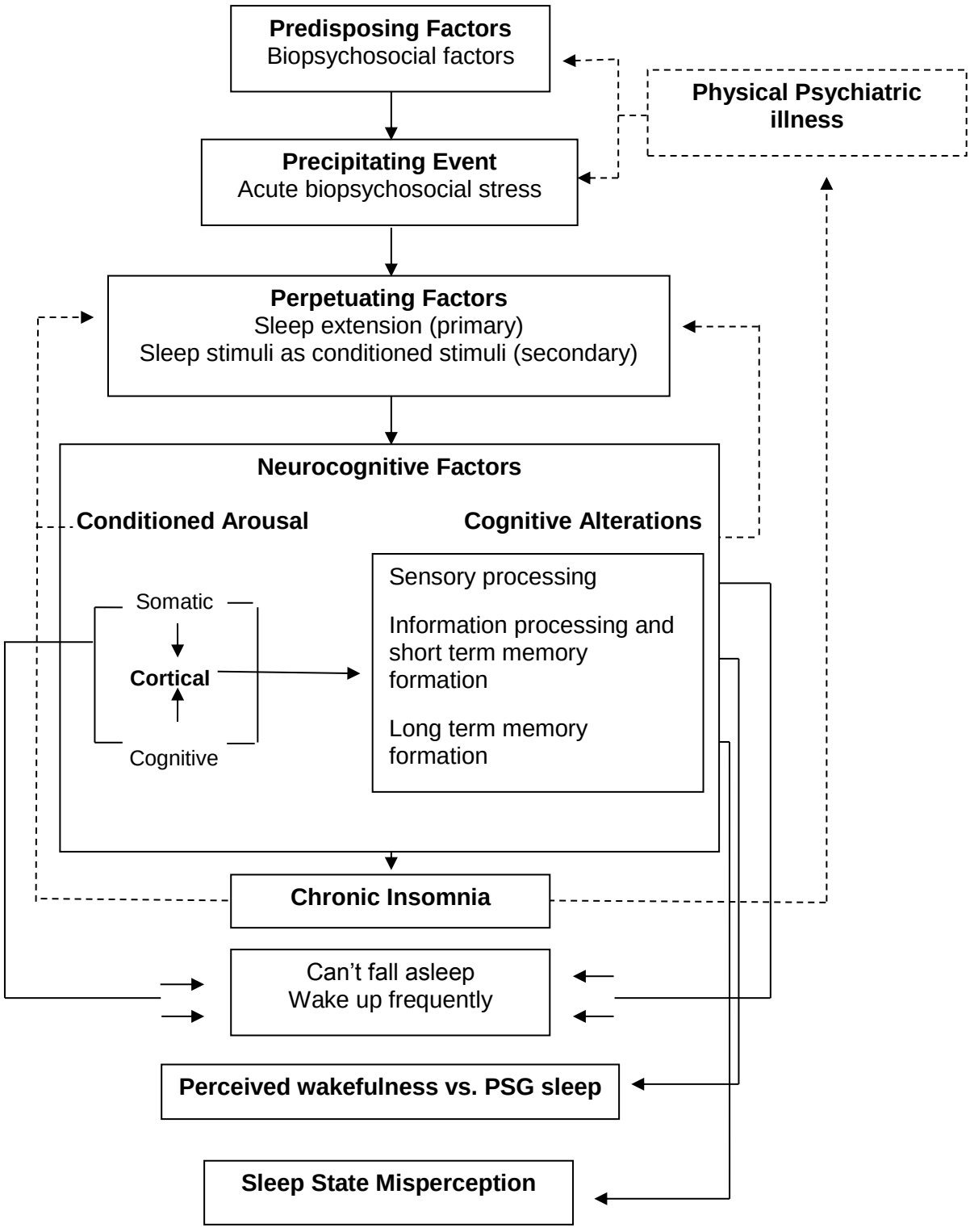
indicate that they had started falling asleep 15 minutes post onset of stage 2 sleep (Hauri & Olmstead, 1983).

Another component proposed by the Neurocognitive Model is the discrepancy reported between subjective and objective sleep measures in some individuals with insomnia (sleep-state misperception). This difference between subjective and objective sleep perception is not dictated by gender (Voderholzer, Al-Shajlawi, Weske, Feige, & Riemann, 2003) and, although there is minimal objective sleep disturbance, individuals with sleep state misperception still report daytime cognitive impairments attributed to perceived sleep loss (Rosa & Bonnet, 2000). Although evidence suggest that people with insomnia are equally likely to overestimate as to underestimate subjective sleep, this too indicates an inability to correctly perceive sleep as "sleep" (Vanable, Aikens, Tadimeti, Caruana-Montaldo, & Mendelson, 2000). Furthermore, this disparity between subjective and objective sleep is thought not to be a result of an inability to accurately gauge time, as individuals with insomnia have been shown to have no difference, comparative to good sleepers, on the time estimates during the day and prior to sleep (Tang & Harvey, 2005).

The final component of the model regards the disparity between measures of sleep improvement seen during the use of some sleep inducing pharmaceuticals, whereby a subjective improvement of sleep is often beyond the improvement seen in the objective sleep. Evidence suggests that benzodiazepine hypnotics have a more definitive impact on the subjective-objective discrepancy of sleep as opposed to objective sleep alone (Mendelson, 1995a; Mendelson & Maczaj, 1990) but do not cause the same subjective-objective discrepancy (a positive misperception of sleep) in good sleepers (Mendelson, 1995b). The subjective benefits of benzodiazepine use in insomnia is surprising as a meta-analysis indicates that, on average, the medication decreases objective sleep onset latency by 4.2 minutes compared to a decrease in the subjective sleep onset estimate of 14.3 minutes (Holbrook, Crowther, Lotter, Cheng, & King, 2000).

Additionally, Holbrook et al., 2000 also reported an increase in objective total sleep time with benzodiazepine use. However, this may not be an equal extension of sleep across all stages, but rather an increase in stage 1 and 2 sleep (light, higher frequency sleep) at the expense of stage 3 (deep, low frequency sleep) with some indication of a supersession of REM sleep (reviewed in Morin & Wooten, 1996). This subjective-objective sleep discrepancy in treatment efficacy is not present with the non-benzodiazepine category of hypnotics, however; following withdrawal from treatment, people with insomnia tend to overestimate poor sleep (Kryger, Steljes, Pouliot, Neufeld, & Odynski, 1991; Sivertsen, Omvik, Pallesen, Nordhus, & Bjorvatn, 2009).

Figure 2.10: The Neurocognitive Model of insomnia, adapted from Perlis, Shaw, Cano, & Espie (2011)



2.4.1 Neurocognitive Factors

2.4.1.1 Conditioned Cognitive and Somatic Arousal

Conditioned arousal can be classified into two sub-types: cognitive (of the mind) and somatic (of the body). The Neurocognitive model does not explain when increased arousal occurs, be that diurnal, nocturnal, or both, but rather, conditioned arousal occurs due to maladaptive sleep habits associated with an extension of time in bed, resulting in a self-perpetuating cycle of somatic and cognitive arousal.

Higher levels of somatic arousal are implicated in primary insomnia: including increased metabolism and heart rate in individuals at sleep onset and throughout sleep (Bonnet & Arand, 1997, 1998; Bonnet & Arand, 2000, 2003; Hall et al., 2004; Nofzinger et al., 2004). As suggested by the Neurobiological model (Buysee, et al., 2001), this could be indicative of the body remaining in a wake-like state, thus opposing the natural physiology of sleep. It is also suggested that, although process C (the circadian phase) remains intact, alterations can occur within the internal biological rhythms, such as the attenuated secretion of melatonin (Hajak et al., 1995) and increases in the amplitude of free cortisol (Rodenbeck, Huether, Rüther, & Hajak, 2002; Vgontzas et al., 2001). However, in a similar group of participants with insomnia, when sleep was only marginally disturbed (as observed by PSG) compared to the good sleeper controls, only nocturnal plasma melatonin was found to be altered (attenuated) in those individuals with insomnia (Riemann et al., 2002). This is also indicated with sleep/fatigue related inflammatory cytokines and interleukins, with greater levels of interleukin(IL)-6 observed in people with primary insomnia comparative to good sleepers (Burgos et al., 2006). Additionally, Tumour Necrosis Factor (TNF) -α has been shown to have an altered secretion pattern in individuals with insomnia, shifting towards daytime secretion, as opposed to being secreted maximally during slow wave sleep (Vgontzas et al., 2002). However, these changes that are indicative of a physiologically aroused state have been shown to only significantly associate with a

subjective detriment to sleep as opposed to alterations seen objectively through PSG, so may only be relevant to those with the specific insomnia subtype, sleep state misperception (Riemann et al., 2002; Vgontzas et al., 1998).

Somatic arousal is said to occur in conjunction with cognitive arousal which, as mentioned in the above models, includes worry, rumination, intrusive thoughts and environmental monitoring (Harvey, 2001a; Harvey, 2003; Harvey et al., 2005; Semler & Harvey, 2004; Tang & Harvey, 2004b; Tang, Schmidt, et al., 2007). Just as Harvey (2000) suggested, people with insomnia may experience negatively toned cognition, which in turn leads to selective attention to sleep, and thus a failure to sleep can heighten arousal through worry and, as Espie (2002) suggests, increased sleep effort. Compared to good sleepers, it is suggested that poor sleepers experience excess cognitive arousal both during the day and night (Ong, Carde, Gross, & Manber, 2011). In addition, the process of somatic arousal is said to exacerbate cognitive arousal as altered levels of bodily arousal could impair the subjective experience of feeling "sleepy". If an individual is somatically aroused, then it can be assumed that sensations of sleepiness will not be present or blunted, thus leading to a cognitive focus on falling asleep, which is shown to impair sleep (Ansfield, Wegner, & Bowser, 1996; Broomfield & Espie, 2005).

The Neurocognitive Model states that the interaction between somatic and cognitive arousal during sleep onset could be termed cortical arousal. Cortical arousal is suggested to be both somatic in nature, as determined through alteration in sleep EEG, and cognitive, as it is suggested to result in alterations in memory. This arousal transcends sleep onset and persists into NREM sleep.

2.4.1.2 Cortical Arousal and High Frequency EEG

Cortical arousal is associated with a state of heightened wakefulness within the brain whilst an individual with insomnia is asleep or in bed attempting to attain sleep.

Following the theory of local sleep, it is suggested that for the brain to fully initialise sleep, sufficient neuronal columns must enter a sleep like state (Rector et al., 2009). Evidence using event related potentials (ERPs) suggests increased arousal responses to deviant tones throughout the night and upon awakening in people with insomnia compared to good sleepers, providing support for the presence of a cortical hyperarousal (Bastien, St-Jean, Morin, Turcotte, & Carrier, 2009). Additionally, high frequency EEG activity during sleep in insomnia is suggested to be associated with cortical arousal and an inability of the neuronal columns to enter a sleep state (Merica et al., 1998).

Using power spectral analysis, the Neurocognitive Model suggests that cortical arousal in primary insomnia can be observed via the distribution of high frequency EEG. Along with the classical staging for sleep as REM and NREM, sleep can also be categorised into distinct bands based on the power spectra of the EEG waveform. Sleep onset is seen as the transition from the higher frequency bands of alpha (8-12 Hz) to lower frequencies of theta (4-8 Hz) and delta (0-4 Hz) (Hori, 1985; Zeitlhofer et al., 1993). It has been suggested that higher frequency waveforms are associated with wakefulness and attention (Pulvermüller, Birbaumer, Lutzenberger, & Mohr, 1997) and cognition (Kaiser & Lutzenberger, 2005). High frequency EEG in the beta (12-30 Hz) and gamma (25-100 Hz) range has been shown to associate with poorer subjective sleep quality, indicating that cortical arousal may be altering how individuals are perceiving sleep (Perlis, Smith, Andrews, et al., 2001). Certainly, the presence of increased high frequency EEG during NREM sleep has been found to be present in those with subjective but not objective insomnia, further suggesting that high frequency EEG may alter, or at least be associated with, perceptions of sleep continuity (Krystal et al., 2002).

High frequency, beta, EEG activity has been shown to be present prior to stage 2 sleep onset in those with insomnia that experience difficulties initiating sleep (Freedman,

1986; Perlis, Merica, et al., 2001). Evidence suggests that beta activity is inversely related to delta activity and that, although high frequency EEG occurs mainly in stage 1 and REM sleep in both good sleepers and those with insomnia, a higher relative power is found in those with insomnia (Perlis, Kehr, et al., 2001). Moreover, evidence suggests that beta EEG activity is also higher during stage 2 sleep in those with primary insomnia (Spiegelhalder et al., 2012). A discriminant analysis of the spectral EEG at sleep onset identified that, using the rate of change between beta and delta activity during the first three minutes of the transition period from wake to the first NREM period, individuals with insomnia experienced higher beta EEG activity and lower delta activity comparative to controls (Merica & Gaillard, 1992). Mercia and Gaillard (1992) also indicated that if judgements of an insomnia diagnosis are made using this method, the ratio of beta to delta activity can correctly identify insomnia 75% of the time.

The Neurocognitive Model suggests that it is at the wake to sleep transitions during NREM sleep as well as the initial sleep onset period where cortical arousal is at its highest and it is during this period that there is an increased vulnerability to cognitive alterations.

2.4.1.3 Cognitive Alterations

There are three possible cognitive alterations that are said to occur as a product of enhanced cortical arousal; enhanced sensory processing, increased information processing and short-term memory formation, and long-term memory formation (Perlis, Giles, Mendelson, et al., 1997). These cognitive alterations may then lead to objective disturbances in sleep, as determined through sleep initiation and maintenance problems, along with sleep state misperception.

The enhanced sensory processing component of the Neurocognitive Model suggests that individuals may be sensitive to perturbation of sleep by environmental stimuli

around the time of sleep onset (Perlis et al., 1997). Through the cognitive model of insomnia, Harvey (2000) suggests that people with insomnia experience increased monitoring and awareness of environmental stimuli, and self-reports from people with insomnia by Wicklow and Espie (2000) suggests that individuals with insomnia report auditory environmental stimuli as disrupters to sleep onset more frequently than good sleepers. Further, it is suggested that people with insomnia have deficiencies in sensory gating, that is the ability to filter sensory information during sleep as measured through evoked K complexes in response to a sensory stimulus (Jahnke et al., 2012). People with insomnia have been shown to have a diminished response or absence of K complexes during sleep (Hairston, Talbot, Eidelman, Gruber, & Harvey, 2010), in particular during stage 2 sleep (Milner, Cuthbert, Kertesz, & Cote, 2009). This suggests that individuals may experience enhanced sensory processing during sleep, and that people with insomnia may be more prone to sensory intrusions during sleep onset and stage 2 sleep (Milner et al., 2009). Moreover, evidence suggests that people with insomnia experienced higher levels of attention, and reduction in sensory gating, to stimuli at sleep onset than good sleepers (Yang & Lo, 2007). With regards to high frequency EEG, a study of individuals with fibromyalgia indicated that, when compared to healthy individuals with good sleep, they experienced a greater amount of alpha and exhibited greater arousals to stimuli (auditory) presented during the sleep onset period (Perlis, Giles, Bootzin, et al., 1997). This is suggestive of a relationship between high frequency EEG and the ability to protect or gate the sleep from sensory perturbations during the sleep onset period.

The second cognitive alteration is said to occur in increased information processing and short-term memory formation at sleep onset and wake to sleep transitions after arousals from NREM sleep (Perlis, Giles, Mendelson, et al., 1997). The Neurocognitive Model states that information processing during sleep onset can blur the distinction between wake and sleep and result in sleep state misperception. It has been

demonstrated that, within a sample of good sleepers, forced awakenings from stage 2 sleep resulted in around 38% of individuals reporting a false perception of being awake prior to the forced awakening, and that these individuals also reported sensations of awareness of the external environment, (Weigand, Michael, & Schulz, 2007). This may demonstrate a potential vulnerability present in some good sleepers to sensory processing at sleep onset which may alter the ability to perceive sleep “as sleep”. With regards to people with insomnia, it is suggested that such individuals may perceive sleep onset later than good sleepers with reports of sleep onset occurring after 15 minutes of uninterrupted stage 2 sleep (Hauri & Olmstead, 1983). Moreover, people with insomnia may misperceive the period before an awakening from sleep as wake and have a diminished sleep/wake discrimination comparative to good sleepers (Knab & Engel, 1988; Mercer et al., 2002). Concerning cortical arousal, it has been suggested that high frequency EEG, specifically in the alpha and beta range, correlates negatively with the perception of sleep (Perlis, Giles, Bootzin, et al., 1997; Perlis, Smith, Andrews, et al., 2001).

Finally, it is suggested that, due to the cortical arousal, individuals with insomnia may form long-term memories of stimuli occurring during sleep onset and following arousals during sleep that results in an inability to discriminate sleep from wake upon appraisal of the sleep period the following morning. When presented with memory tasks during the sleep onset period using auditory stimuli, following a short 10 minute sleep post presentation, good sleepers showed deficits in memory performance relating to the stimuli suggestive of a natural amnesia to environmental stimuli during this onset period (Portnoff, Baekeland, Goodenough, Karacan, & Shapiro, 1966; Wyatt, Bootzin, Anthony, & Bazant, 1994). Additionally, analysis of the sleeping EEG during the 10 minute sleep opportunity post stimuli showed a positive association between the beta EEG power and the recall of presented stimuli (Wyatt, Bootzin, Allen, & Anthony, 1997). The Neurocognitive Model suggests that, as beta EEG activity is associated

with memory formation in good sleepers; increased cortical arousal in individuals with insomnia may facilitate the formation of memories for environmental stimuli that occur during sleep onset. Evidence suggests that when presented with word blocks (consisting of nouns) at sleep onset and at three sleep onsets following a forced awakening from NREM sleep, individuals with insomnia exhibit greater total word recognition and number of words recalled at initial sleep onset than good sleepers (Perlis, Smith, Orff, Andrews, & Giles, 2001).

2.5 Environmental Noise as a Model of Cortical Arousal

The key component of insomnia is the presence of a heightened level of arousal with the above models describing the effect of this arousal on the natural sleep process. The above models all differ depending on the nature of the arousal event. Firstly, the models differ upon the time at which arousal occurs, specifically whether it is due to a failure to de-arouse prior to sleep or whether it is persists through the day and night. Secondly, the models differ in terms of how the arousal forms, whether it is a conditioned implicit arousal as a learned behaviour or a physiological abnormality. Finally, the models differ in terms of the type of arousal present, i.e. whether the arousal is of a cognitive, somatic, or cortical nature. Reviews of arousal in insomnia have suggested that there is a case for a general hyperarousal state within individuals with insomnia that is characterised by heightened physiological, autonomic, cortical and cognitive arousal (Bonnet & Arand, 2010; Riemann et al., 2010).

It has been suggested that people with insomnia are more susceptible to environmental perturbations to sleep and actively monitor for anything that could impact on sleep onset (Semler & Harvey, 2004). Furthermore, individuals with insomnia are more likely to report environmental perturbations at sleep onset than good sleepers (Wicklow & Espie, 2000). If this is applied to the Neurocognitive Model then active monitoring of the environment at sleep onset could result in attention to environmental stimuli that in turn could be processed, resulting in memory formation whilst impairing the perception of

sleep as sleep. As Perlis and colleagues (1997) highlighted in their study with individuals with fibromyalgia, even if sounds cannot be recalled, that is not to say that long term memory formation has not occurred as subjective sleep is still altered, indicating possible implicit memory formation (Perlis, Giles, Bootzin, et al., 1997). Therefore, one could ask whether noise that does not wake an individual and cannot be recalled explicitly upon awakening, creates cognitive distortions implicitly and affects the perception of sleep continuity.

2.5.1 Application of Environmental Noise to Sleep and the Neurocognitive Model

The World Health Organisation (WHO) have acknowledged that noise pollution is a hazard, stating that night time noise can lead to significant decrements in health and wellbeing, presumably by adversely affecting sleep. Guidelines suggest that a level of traffic noise of 35 decibels or more is sufficient to cause arousals in sleep and subjective sleep disturbance, with increments in noise level increasing the risk of adverse health effects, hence the implementation of requiring nocturnal environmental noise to remain below 40 decibels (World Health Organisation Europe, 2009). Further to this, the WHO released a directive calculating the burden of disease from environmental noise and suggested that extrapolation from current noise maps of the European Union Member States, the Disability-Adjusted-Life-Years (DALYs) due to sleep disturbance from night time noise could be in the range of 0.5-1.0 million years for the EU population (World Health Organisation Europe, 2011).

Noise at night is recognised as a substantial health risk and a European Union Directive 2002/49/EC states that union members must monitor and act to reduce noise at night (European Parliament Council, 2002). However, current guidelines only suggest the monitoring of transportation noises during the hours of 23:00 to 07:00, which is proposed to be insufficient for determining the risks of sleep-based noise disturbances (Basner, Müller, & Griefahn, 2010). It has been suggested that, to

correctly quantify the disturbance to sleep from noise, individual differences in sensitivity need to be identified, as those with a higher vulnerability will perceive greater levels of sleep disturbance (Heinonen-Guzejev et al., 2000; Lercher & Kofler, 1996). This vulnerability to noise could be seen as a predisposing factor to insomnia and certainly has been implicated in individuals with insomnia (Wicklow & Espie, 2000). The Hypertension and Exposure to Noise near Airports (HYENA) taskforce investigated levels of perceived annoyance to noise across several European communities, confirming that the higher annoyance for sounds at night related mainly to aircraft and traffic sounds (Babisch et al., 2009).

Noise could be posited as creating a physiological arousal response in good sleepers, as environmental noise has been suggested to affect heart rate variability as well as causing alterations in the cortisol awakening response (Graham, Janssen, Vos, & Miedema, 2009; Waye et al., 2003); measures suggested by the Neurocognitive Model to relate to somatic arousal. Moreover, noise disturbance is seen to have a dose response effect with louder noises causing more disturbance (Öhrström, 1995) and the effect of noise disturbance is increased if the stimuli is a combination of different sounds (Lee, Shim, & Jeon, 2010). This suggests that that there may be a habituation effect in the protection of sleep when the noises are of the same type, and that, implicitly, sleeping individuals may be able to distinguish the change in noise, thus resulting in increased disturbance. Evidence suggests that transportation noises can result in alterations in objective sleep as well as alterations in perception of sleep quality, with the former responding to relatively low levels and the latter showing higher subjective decrements as noise levels increase (Griefahn et al., 2006). Within good sleepers there is a proposed subjective habituation to noise, however objectively there is no apparent habituation effect (Kuroiwa, 2002).

In support of the Neurocognitive Model, a review article suggests that noise may be processed by an individual during sleep, hence the alterations seen in subjective sleep

quality (Muzet, 2007). Although subjective habituation is said to occur to environmental noise, intermittent and continuous noise affect objective sleep in different ways with the former increasing the lighter sleep stages at the expense of deep sleep and the latter causing a decrease in REM sleep (Eberhardt, Ståle, & Berlin, 1987). If noise is creating an increase in the lighter stages of sleep, it could be assumed that this could be resulting in an increased vulnerability for experiencing higher frequency EEG during sleep as the time spent in the delta frequency of deep sleep is reduced. Moreover intermittent tones administered to good sleepers during the night, removes any habituation effect of the noise on sleep can result in a misperception of sleep onset (the subjective SOL is perceived as longer than the objective SOL) (Townsend, Johnson, & Muzet, 1973).

As for the mechanisms under which noise perturbations could be applied to the Neurocognitive Model, cognitive alterations through cortical arousal suggest that people with insomnia experience insufficient sensory gating manifesting as increased sensory processing at sleep onset. A recent review of sleep microstructure in insomnia has suggested that individuals with insomnia experience more spontaneous K complexes and a reduction in the density of sleep spindles comparative to good sleepers (Feige et al., 2013), although Bastien and colleagues suggest that this does not associate with a reduction in sleep stability (Bastien, St-Jean, Turcotte, Morin, Lavallee, & Carrier, 2009; Bastien, St-Jean, Turcotte, Morin, Lavallee, Carrier, et al., 2009). That said, one study examined sleep stability within a hospital ward and found that good sleepers who had more spindles experienced greater sleep protection: thus sleep was maintained in the face of arousal from environmental noise (Dang-Vu, McKinney, Buxton, Solet, & Ellenbogen, 2010). Therefore, as the Neurocognitive Model suggests, sensory processing may be increased in insomnia; a noisy sleep environment may result in a greater disruption to sleep and therefore result in a higher risk of sleep state misperception through increased information processing,

comparative to good sleepers. As noise during sleep has the ability to create sleep onset misperception in good sleepers, coupled with cortical arousal and the neurocognitive alterations proposed by the Neurocognitive Model, it is plausible that low-level environmental noise at night may be perpetuating the misperception of sleep seen in insomnia and potentially create an initial acute period of insomnia in vulnerable good sleepers.

2.6 Thesis Rationale

The rationale of this thesis is two-fold. Firstly, this thesis will examine the effect of environmental noise as a measure of raising cortical arousal in both the home environment and within a controlled laboratory setting in good sleeping participants. This will culminate in determining whether noise can be used in the testing of the Neurocognitive Model. Auditory stimuli will be used to determine whether increased cortical arousal affects information processing during sleep through the ability to explicitly or implicitly recognise said auditory stimuli. As such, this thesis will explore the constructs of the Neurocognitive Model examining whether increased cortical arousal is a vulnerability factor in the development of insomnia that influences alterations in sleep perception. Therefore, auditory stimuli will be used as both a method of eliciting and measuring cortical arousal through the propositions of the Neurocognitive Model. The testing of the Neurocognitive Model of insomnia, will not only constitute a novel contribution to insomnia theory by providing evidence towards the processes involved in the development of sleep state misperception but also evidence towards clinical theory regarding the treatment of insomnia, as if noise is shown to be applicable to the Neurocognitive Model, then treatment for insomnia may need to include noise control as well as investigating for the presence of cortical arousal. Therefore, this thesis has the following research questions that will be addressed across three empirical chapters:

Chapter Three

This chapter broadly aims to observe the impact noise has on sleep in an ambulatory setting and examine what factors allow some individuals to habituate to a noisy sleep environment. As the Neurocognitive model proposes that specific time points during sleep may be susceptible to cortical arousal, this study will aim to examine whether there is a differential effect of sleep stage on the impact of noise on sleep. Furthermore, the Neurocognitive model suggests that an outcome of enhanced sensory processing is a disturbance to both subjective and objective sleep therefore findings from this study will be applied to the propositions of the Neurocognitive model regarding potential memory processing of noise that occurs during sleep and whether this associates with skewed perception of sleep quality. To further this, three distinct research questions are asked:

1a. Is there a differential effect of sleep stage (NREM or REM) on the impact of noise on sleep within participants' homes?

1b. Are there vulnerability traits which predict the ability by some individuals to habituate to noise in the sleep environment?

1c. Does the recollection of noise during the night associate with a discrepancy between subjective and objective sleep?

Chapter Four

This chapter will expand on the previous chapter by affording greater control of study variables through moving from an ambulatory to a laboratory setting thus providing a novel non-habituated environment with the aim of eliciting increased cortical arousal in good sleepers. The study in Chapter 4 aims to examine the cortical arousal component of the Neurocognitive Model and the impact that this may have on the perception of sleep continuity through processing to memory of environmental noise. To this extent,

environmental noise will only be administered during sleep (i.e. not played during wake periods during the night) and participants will be unaware that noise is to be played during sleep. To further this, four distinct research questions are asked:

2a., What is the impact of increased cortical arousal on subjective and objective sleep and what comparisons can be made to the constructs of the Neurocognitive model?

2b., Are there a personality traits that make individuals more vulnerable to sleep disturbance by noise in the laboratory i.e. inhibiting the ability to habituate to the novel bedroom environment?

2c. Does noise during sleep result in a change in the discrepancy between subjective and objective sleep comparative to a night without noise?

2d. Can good sleepers explicitly recall noise administered during sleep and does this associate with an increase in sleep state misperception?

Chapter Five

Chapter 5 will move from examining noise during sleep to investigating the effect of noise (words) administered during wake to sleep transitions as a result of heightened cortical arousal. This chapter will directly test the Neurocognitive Model comparing good sleepers with those with insomnia on sleep perception in awakenings (short term memory), explicit and implicit recognition of the noise stimuli (long term memory), and perception of sleep continuity. Therefore, three distinct research questions are asked:

3a. Is there a difference between good sleepers and those with insomnia in regard to sleep perception (short-term memory) following a natural awakening from NREM sleep (as determined by 60 seconds of wake)?

3b. When administering noises (words) during wake to sleep transitions in NREM, is there a difference between good sleepers and those with insomnia on measures of

explicit and implicit memory recognition for the noises (long term memory) immediately upon awakening the next morning?

3c.Does explicit and implicit memory of noise presented during wake to sleep transitions associate with a disparity between subjective and objective sleep regardless of whether the individual is a good sleeper or has insomnia (levels of cortical arousal?

Chapter 3: An observation of the impact of environmental noise at home on sleep

3.0 Introduction

Environmental noise during the night is a well-known source of sleep disturbance of which the World Health Organisation (WHO) has set specific guidelines on the control and level of noise in urban areas highlighting the impact it can have on population health (World Health Organisation Europe, 2009, 2011). Transportation noises (car, air, and rail) have been evidenced to cause alterations in both objective and subjective sleep continuity. Detriments to objective sleep continuity are said to occur similarly at both lower and higher loudness levels of noise whereas a dose response effect is said to occur for the effect on subjective sleep, with increasing loudness associating with a worsening of the perception of sleep (Griefahn et al., 2006). The number of noise events during the night has been shown to correlate with worsening perceptions of sleep quality along with relating to feelings of daytime tiredness in people who self-report a high sensitivity to environmental noise (Öhrström, 1995). Additionally, an increase in body movements, and thus awakenings, as determined through actigraphy, has been shown to associate with noise levels during sleep (Öhrström, 1995; Öhrström, Björkman, & Rylander, 1990).

It is suggested that although a subjective habituation to noise can occur, as determined by perceived improvements in sleep over concurrent nights, the disruption of noise to objective sleep (polysomnography) remains stable (Kuroiwa, 2002) indicating that although individuals may perceive themselves to have slept better, actual sleep is still disturbed - a positive misperception of sleep. Moreover, environmental noise has been suggested to create alterations in the autonomic nervous and neuroendocrine system, as evidenced by changes in cardiac measures (Graham et al., 2009) and alterations in

the cortisol awakening response (Waye et al., 2003). Waye et al. (2003) showed that salivary cortisol levels in good sleepers are attenuated at 30 minutes post awakening following nights where a low frequency noise was administered, and these levels were shown to associate with perception of sleep quality. Lower awakening cortisol levels are found in those with insomnia, and these levels also associate with worsening subjective report of sleep quality (Backhaus, Junghanns, & Hohagen, 2004). Therefore, the study by Waye et al. (2003) may indicate that noise can be used to test the propositions of the Neurocognitive model using a sample of good sleepers due to the potential impact noise has on the subjective report of sleep.

A review by Muzet (2007) suggested that some individuals may be able to process noise during sleep. Furthermore, a report by the World Health Organisation (2011) indicates that the burden of noise may be measured through noises implicitly affecting sleep. As the Neurocognitive Model proposes that memory processing of information at the transition to sleep in individuals with insomnia may be recognised the following morning both explicitly and implicitly (Perlis, Giles, Mendelson, et al., 1997), the evidence provided by Muzet (2007) and WHO (2011) supports the notion that noise may be a suitable method to test the Neurocognitive model.

The overall aim of this chapter is to examine whether noise can be used to test the Neurocognitive model of insomnia by observing the impact ambulatory noise has on both subjective and objective sleep measures in the habituated bedroom environment along with examining various personality traits that may result in vulnerability for noise based sleep disturbance through heightened cortical arousal. Furthermore, this study aims to examine differential effects of noise dependent on sleep stage using the first and last 90 minutes of sleep as the Neurocognitive Model posits that it is NREM sleep is the most vulnerable to cortical arousal. Therefore, four questions were proposed with the following four hypotheses tested within this chapter:

1. As the Neurocognitive Model proposes that it may be NREM sleep and sleep to wake transition points that are more susceptible to the effects of heightened cortical arousal, this study aims to assess differential effects sampling period regarding the impact of noise on sleep: it is hypothesised that noise within the first 90 minute sampling period will result in more objective wake than sleep.
2. The Neurocognitive Model follows from the 3P model in regards to perpetuating insomnia factors, therefore do vulnerability traits which are said to be measures of stress and arousal associate differentially with the presence of noise and the sleep/wake state for the first or last 90 minute sampling period: it is expected that participants that are able to sleep in the presence of noise (habituated) may have sleep protective traits, such as lower levels arousal during sleep, and that the impact of these traits will be apparent within the NREM dominant sampling period.
3. The Neurocognitive Model proposes that an outcome of heightened cortical arousal is a disturbance to subjective sleep therefore this study seeks to observe the impact environmental noise on subjective sleep in the habituated home and are relationships differentially associated with NREM or REM sleep once the ability to recall noises during the night is controlled for. To assess relationships between nocturnal noise and subjective sleep: it is anticipated that a greater disruption to subjective sleep will be associated with noise occurring in the first sampling period (NREM) when the number of noises recalled as disturbing sleep are controlled for.
4. Finally, the Neurocognitive Model proposes that the ability to recall external stimuli occurring during sleep may result in a misperception of sleep. Therefore, this study aims to observe whether the discrepancy between subjective and objective sleep associates differentially with noise during sleep for the first or last 90 minute sampling period if the recollection of noise during the night is controlled for: it is expected that a greater disparity between

subjective and objective sleep will associate with noise occurring in the first sampling period (NREM) when the number of noises recalled as disturbing sleep are controlled for.

3.1 Methodology

3.1.1 Design

Using a 10 night protocol (7 night baseline to determine good sleeper status, 3 observational study nights), an ambulatory study was employed using an observational design to examine the overall effect of environmental noise on sleep within the home environment. A correlational methodology was employed to examine associations between sleep/wake states in the presence/absence of environmental noise and to examine measures of individual differences in stress, arousal and psychopathology that may afford sleep protection from environmental noise perturbations.

3.1.2 Participants

An opportunity sample of self-reporting good sleepers was obtained from the University of Northumbria through word of mouth and an email and poster campaign. Additional participants were sought using an email campaign from a public database sample held at the sleep laboratory. To be initially eligible for the study, participants had to be aged between 23 and 60, and live within their own, or rented accommodation. Participants were able to flat share but they were excluded if they lived in shared accommodation such as university halls of residence or sheltered housing as this was deemed to be an unusually noisy environment. In addition, participants were required to have a stable sleep wake pattern during the three study nights (Monday to Wednesday) with sleep stability assessed using a pre-study baseline sleep diary.

Participants were not excluded for sharing the bed with a partner or if the bed partner had a sleep problem (for example snoring). In such instances, additional consent was obtained from the partner for the noise recording over the three observational nights.

Furthermore, exclusion was not made if participants had children unless their children co-habited the parental bedroom on a regular basis. This data was gathered at initial screening and no participants were excluded for these reasons.

In total, eighteen self-reporting good sleeping participants were recruited to the study, of which nine were male and nine were female, however two were excluded due to incomplete data leaving sixteen participants with an equal mix of males and females and an average age of 27.5 years (*SD* = 4.27 years).

At initial screening, participants were excluded if they presented with a current complaint of any sleep disorder, anxiety or depression (self-reported and using the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983)), had a current medical problem, or were taking medication that is reported to interfere with sleep. Participants were also excluded if they had a history of severe head injury, had a hearing disorder, or were employed in a sector that required rotating shift work. Two of the 16 participants (13%) reported a history of a sleep problem (insomnia), however neither was excluded as they did not experience a current subjective complaint and therefore did not fall under DSM-V criteria for insomnia disorder.

A demographic questionnaire was administered to gather participants' education level (appendix B), employment status, drug habits, and living arrangements. All participants had up to UK A-level equivalent qualifications with 14 (88%) of the participants having at least an undergraduate degree qualification. Participants were not excluded if they drank alcohol or smoked, however they were asked to abstain from drinking alcohol on the three study nights. All but one of the participants had drunk alcohol at some point during their lifetime but not during the study and half had smoked tobacco although all had quit or were social smokers.

Of the 16 participants, 12 (75%) were in full time work, three (19%) were students (post-graduate level) and one (6%) was currently interning. Three (19%) participants

had a bed partner and 13 (81%) participants reported sleeping alone. Two participants (13%) had children in the household. Of the participants, two (13%) had pets (cats) and these were allowed to enter the bedroom at will. Eight (50%) of the participants lived in a flat whereas the remaining eight (50%) participants lived in houses. All bedrooms had windows and nine (56%) were described as facing a road and seven (44%) as facing a garden or a yard. All but two participants shared the property with at least one other person, be that children, partner, or housemates.

3.1.2.1 Consent and Ethical Considerations

The Ethics Committee at the Faculty of Health and Life Sciences, Northumbria University, UK granted ethical approval for this study. Prior to taking part in the research study, all participants had to indicate their informed consent for the study including noise recording of their bedroom environment. Participant remuneration was £20 on full completion of the three-night observational study.

3.1.3 Procedure

To investigate the role of environmental noise on sleep, a ten day ambulatory study was conducted within participants' own homes, Figure 3.1. The protocol consisted of an initial eligibility screening using an in house questionnaire (as defined in section 3.1.4.1) followed by 7 nights screening for sleep stability via a sleep diary (section 3.1.4.2) and actigraphy. Once baseline procedures were completed, participants returned to the laboratory to complete a series of questionnaires to measure potential noise vulnerability traits including measures of arousal, stress, and noise sensitivity. These were not used as exclusion criteria but rather to describe the population taking part in the study and to examine whether these could have a role in the effect of noise on sleep. In addition, participants received the materials (actiwatch, sleep diary, and sound recorder) needed to commence the three-day observational protocol.

Figure 3.1: Protocol schematic. [a] Sleep diary was adapted from screening to include the ability to document nocturnal noise disturbances

Assessment Schedule	-7	-6	-5	-4	-3	-2	-1	0	1	2	3
Study Consent											
Eligibility Screening											
Screening Sleep Dairy											
Actigraphy (1 min epochs)											
Psychometric Testing											
Observational Sleep Diary[a]											
Actigraphy (15s epochs)											
Noise Recording											
Study Debrief											

3.1.3.1 Three night Observational Study

During the observational nights, participants continued to wear an actiwatch with the sampling rate reduced to 15-second epochs to allow a greater specificity for observing the effect of noise on sleep. For three nights, participants started the recording device from the point of attempting sleep (lights out) whilst simultaneously pressing a marker button on the actiwatch to confirm the time of sleep initiation as thus providing a sync point between the two devices. Participants were requested to place the recording device as close to their head as possible during the study without it being on the bed or floor (bedside tables were stated as being preferable).

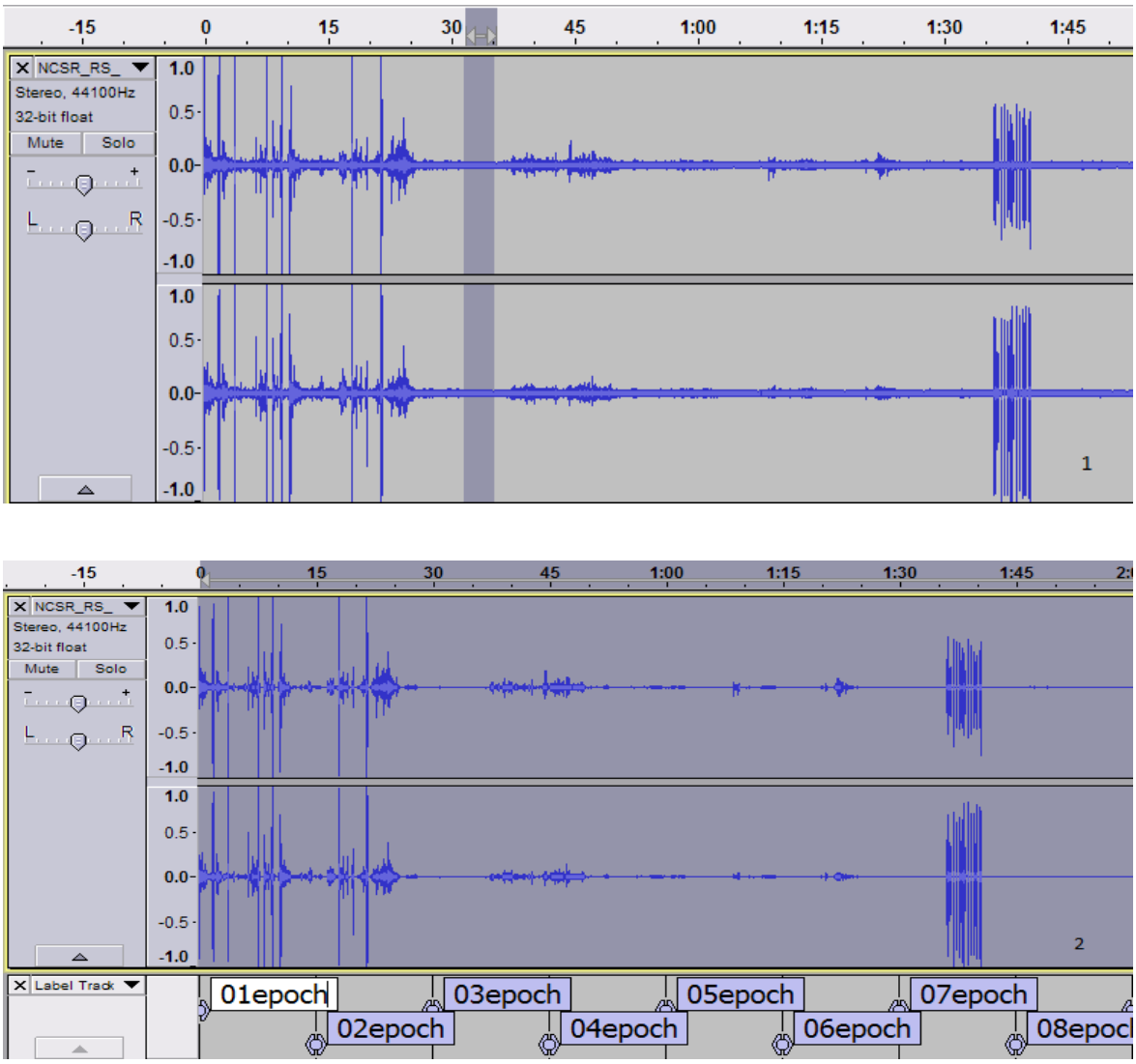
Upon awakening, participants were advised to end the recording by switching off the device and simultaneously press the marker on the actiwatch. This allowed the creation of a matched recording window between the two devices for analytical purposes. Following this, participants were instructed to complete the adapted study sleep diary, which included the Stanford Sleepiness Scale along with an area to document any noises perceived to have disturbed sleep. This was repeated over the three

observational study nights. On the fourth day, participants returned all equipment and materials to the Northumbria Centre for Sleep Research whereby they received a full study debrief and remuneration.

3.1.3.1.1 Analytic Strategy for the Noise Recording

The environmental noise recording was downloaded from the recording device to a desktop PC in an MP3 format. Recordings were then converted to WAV format and imported into Audacity, version 2.0.3 (Audacity Team, 1999-2012). To create an objective count of noises during the night, Audacity was used to view the waveform of the recording and then, using the selection tool, a period of silence at the start of the recording was selected as a baseline. The whole recording had this baseline noise level removed, resulting in a measure of distinct noise peaks, Figure 3.2 As this study aimed to examine the first sleep onset period and the sleep period directly prior to the final awakening, to compare the periods of NREM and REM sleep, only the first 90 minutes of sleep and last 90 minutes of sleep were examined. Using the label function within the Audacity program, markers were placed on the noise recording at 15 second intervals, resulting in epochs of the same length as those generated from the actigraphy. As participants were requested to indicate using the actiwatch marker system when the noise recording had started, epochs from the noise recording (Figure 3.2) and the sleep-wake profile from the actiwatch (Figure 3.3) could be simultaneously compared.

Figure 3.2: Screenshots of the Audacity program and the nightly environmental noise recording, both the raw data (1) with the baseline silence highlight and the prepared data (2) post noise extraction showing noise spikes above baseline silence.



The sleep/wake profile generated from the actigraphy provided an epoch-by-epoch view of the whole night with each epoch recorded as wake (W) or sleep (S) (Figure 3.3). Using the markers placed on the recording by the participants, the sleep/wake profile and the noise recording were matched and then sleep/wake state and presence/absence of environmental noise was recorded for each epoch by highlighting the epoch if noise was present. Therefore, each 90-minute section contained 360 x 15 second epochs matched between the actigraphy and noise recording. This res

resulting in four potential classifications of an epoch; wake with noise, wake with silence, sleep with noise, and sleep with silence.

Figure 3.3: Example of the sleep wake profile generated from the actiwatch. W represents Wake and S represents Sleep with each row being 5 minutes in duration made up of 20 x 15s epochs

Time	Epoch No																			
00:25	01 to 20	W	W	W	W	W	W	W	W	W	W	S	S	W	W	W	W	W	W	W	W
00:30	21 to 40	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W
00:35	41 to 60	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W	W
00:40	61 to 80	W	W	W	S	S	S	S	S	W	W	W	W	S	S	W	W	S	S	S	S
00:45	81 to 100	S	S	W	W	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S

3.1.4 Apparatus and Materials

3.1.4.1 Screening Questionnaire

A general in house health-screening questionnaire was administered to participants prior to inclusion into the study, appendix A, B and C. This questionnaire assessed participants' health, both mental and physical, as well as screening for the symptoms of various sleep disorders including sleep apnoea, insomnia and restless legs syndrome. Included in this screening questionnaire was a demographics questionnaire to gather participants age, height, weight and information about their household including number of pets, number of dependents, type of accommodation, and whether they had a bed partner.

3.1.4.2 Sleep Diary

A standard sleep diary (Carney et al. (2012) was completed by the participants for one week prior to partaking in the observational part of the study. This included measures of sleep continuity (SOL, NWAK, WASO, TST, TIB, and SE), and measures of sleep

quality using four point Likert scales (“my sleep was”). This diary was used to ascertain a stable sleep wake pattern (going to bed and waking at consistent times during the working week), see appendix D.

Following inclusion into the study, the diary was used over the course of the three observational nights and modified to allow the recording of any noises the participants reported hearing during the night using participant free recall along with including the Stanford Sleepiness Scale (SSS) (Hoddes, Dement, & Zarcone, 1972).

3.1.4.2.1 The Stanford Sleepiness Scale (SSS)

The SSS is a state measure of immediate sleepiness using a 7 item Likert scale from 1 (feeling active, vital, alert, or wide awake) to 7 (no longer fighting sleep, sleep onset soon, having dreamlike thoughts) and has been shown to correlate with measures of performance (r=0.68) and exhibits a test re-test reliability of 0.88 (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973). The SSS has been shown to be sensitive to changes in alertness after partial (40%) sleep deprivation in good sleepers (Herscovitch & Broughton, 1981).

3.1.4.3 Questionnaires

3.1.4.3.1 Pre-Sleep Arousal Scale (PSAS)

The PSAS is a 16 item scale comprising two sub-scales of 8 items each relating to intensity of feelings of cognitive and somatic arousal immediately prior to sleep (Nicassio, Mendlowitz, Fussell, & Petras, 1985). Respondents are asked to rate on a scale of 1 (not at all) to 5 (extremely) how intensely they experience a series of symptoms as they fall asleep. Therefore, for each sub- scale, a score from 8 to 40 can be achieved. This scale has shown excellent internal consistency in both good sleepers and people with insomnia with a Chronbach’s α of 0.8 (Nicassio et al., 1985). Higher scores on both sub-scales indicate that the responder experiences higher levels of either somatic or cognitive arousal prior to sleep, and scores on the PSAS have been

shown to associate with subjective measures of sleep continuity (higher scores correlate with a worsening in subjective sleep continuity) (Nicassio et al., 1985). As such, this measure was used to examine if those who experience higher pre-sleep arousal experience greater sleep disturbance in the presence of noise.

3.1.4.3.2 Hospital Anxiety and Depression Scale (HADS)

The HADS is a 14 item scale (7 items for anxiety and 7 items for depression) asking respondents about their experience of depressive or anxiety symptoms over the past week on a four point scale of 0 (never) to 3 (frequently) (Zigmond & Snaith, 1983). The sub-scale score ranges from 0-21 with a score of 8-10 indicating possible depression/anxiety and a score of over 11 indicating probable depression/anxiety (Snaith, 2003; Zigmond & Snaith, 1983). Internal consistency of the HADS is indicated by a good Chronbach's α of 0.7 in "normal" controls and up to around 0.9 in clinical samples (Bjelland, Dahl, Haug, & Neckelmann, 2002; Johnston, Pollard, & Hennessey, 2000). The recommended exclusion cut of level for research is suggested to be a score of over 10/11 on either the depression or anxiety subscale which is said to result in a specificity of around 0.9 (Brennan, Worrall-Davies, McMillan, Gilbody, & House, 2010).

The HADS was used as a clinical exclusion for this study. Due to the reported inconsistencies with the HADS at distinguishing between the constructs of anxiety and depression, it has been suggested that the HADS is rather a measure of emotional distress (Cosco, Doyle, Ward, & McGee, 2012). Within this present study, responses from the HADS scale were collapsed into one overall score. With regards to a clinical cut off, as it is suggested that a score of over 11 indicates probable anxiety or depression (Snaith, 2003), for the combined subscales a cut off value of 22 was used, no participants were excluded on this basis.

3.1.4.3.3 Ford Insomnia Response to Stress Test (FIRST)

The FIRST asks how likely the respondent is to experience sleeplessness in the context of nine stressful situations, such as having a bad day at work (Drake et al., 2004). The responder answers on a Likert-style scale from 1 (not likely) to 4 (very likely) for each scenario giving an overall rating of 9-36 with higher scores indicating a higher likelihood by the participant to experience problems sleeping following a stressful situation. This gives a score relating to the individuals sleep reactivity to stress. This measure has been shown to have good internal consistency (Chronbach's alpha = 0.8), and shows a significant correlation ($r=0.24$ $p<0.05$) with the multiple sleep latency test (MSLT), a measure of sleepiness (Drake et al., 2004). As the FIRST measures individuals' insomnia response to stress, or rather a vulnerability for sleep to be altered due to a stressful event, this study will examine whether higher scores are associated with a greater disturbance of sleep due to the presence of noise, with nocturnal noise a potential environmental stressor or a mechanism to increase cortical arousal.

3.1.4.3.4 Perceived Stress Scale (PSS)

The PSS is a 14 item Likert-style questionnaire relating to feelings and thoughts over the past month (Cohen, Kamarck, & Mermelstein, 1983). Responders are asked to rate their response, on a scale of 1 (never) to 5 (very often), to a range of positive (e.g., in the last month, how often have you dealt with irritating life hassles) and negative (e.g., in the last month, how often have you felt nervous and stressed) scenarios. This gives a score of 0 to 70 relating to total perceived stress with higher scores indicating greater perceived life stress. This scale has good internal consistency (0.8) and test-retest reliability was shown to be 0.85, two days post study (Cohen et al., 1983). Similar to the FIRST, this scale was administered to examine whether participants who had greater levels of perceived stress, and therefore may be at a higher level of arousal, experience a greater disturbance to sleep by noise.

3.1.4.3.5 Perceived Stress Reactivity Scale (PSRS)

The PSRS is a 23 item scale used to measure the stress response of the respondent to a range of stressful scenarios (Schlotz, Yim, Zoccola, Jansen, & Schulz, 2011). The scale is split into five sub-scales and one overall scale of total stress reactivity. The scale demonstrates good internal consistency (0.9) and has been shown to have good test re-test reliability (0.8) (Schlotz et al., 2011). The PSRS total score ranges from 0 to 46 with higher scores indicating a higher subjective reactivity to stressful situations. As this study uses a small sample, only the total score for the scale will be used to examine whether high scores associate with an increase in sleep disturbance by noise.

3.1.4.3.6 Weinstein's Noise Sensitivity Scale (WNSS)

The noise sensitivity scale comprises 21 Likert-style items where responders reply on a 0 to 5 scale with regard to how strongly they agree to a statement relating to noise (I am more aware of noise than I used to be) (Weinstein, 1978); the higher the score, the more sensitive the individual is to noise. This scale has been shown to have excellent internal consistency (Chronbach's $\alpha = 0.86$) and retest reliability (0.87) based on a sample of 213 university students (of which 178 completed the retest-reliability 4 weeks post study) (Zimmer & Ellermeier, 1999). This present study will assess whether a predisposition to noise sensitivity results in greater disturbance of sleep by noise and sleep misperception.

3.1.4.4 Noise Recording Device

Nocturnal noise within the participants' sleep environment was recorded using a high quality noise recorder (Olympus Linear PCM Recorder LS-3) placed on a bedside table (or similar height object near the bed). The recording was made using the in-built, high sensitivity, three directional microphone system set to record on a wide frame in MP3 format at 320kbps. Participants were instructed in how to start and end recordings on the device.

3.1.4.5 Actigraphy

Wrist based actigraphy (Cambridge Neurotechnology) was used throughout the study, with participants instructed to wear an actiwatch on their non-dominant wrist at all times with the exception of when it would be at risk of getting wet. Participants were instructed to use the in-built marker system on the watch to record a time stamp of when the watch was off the wrist along with to pressing the marker when getting into when exiting the bed. All analysis of raw data was conducted using Actiwatch Activity and Sleep Analysis 7 program, version 7.4.3 from Cambridge Neurotechnology.

Actigraphy was used during the seven-day screening period prior to inclusion into the three night observational part of the study. In this instance, 1-minute epochs (32 Hz) were used to corroborate reports from the sleep diary pertaining to the habitual sleep schedules of the participants and to acclimatise participants to the equipment. During the three-day observational part of the study, participants continued to wear the actiwatch as during the baseline period however the sampling epoch was changed from epochs of one minute to epochs of 15 seconds (the highest sensitivity possible on the device).

As Actigraphy uses a piezoelectric accelerometer that registers a voltage change when movement occurs and creates a count up of these events. At epochs of 1 minute, the algorithm within the sleep analysis program defines wake when the activity count reaches over 40. By reducing the epoch window to 15s for the observational segment of the study, the number of counts needed for a classification of wake is also reduced (to 10 counts), providing greater specificity and the smallest possible analysis window.

Once in bed with the lights out preparing for sleep, participants were instructed to synchronise the marker system and the noise-recording device by pressing the in-built actiwatch marker as they began to record the environmental noises.

3.1.5 Statistical analyses

All statistical analyses were completed using SPSS for windows version 20. Chi square analysis was used in the first instance to examine differences in the frequencies of the four possible epoch classifications (wake with noise/silence; and sleep with noise/silence) within and between the two 90 minute sampling periods. The numbers of epochs in each of the four possible classifications were averaged over the three nights for each participant. These counts from each participant were summed resulting in analysis on the average total frequencies from all participants for each 90-minute sampling period.

Following this, the four classifications were grouped on whether they were an expected outcome, a hit, or an unexpected outcome, a miss. Hits were classified as epochs of wake when a noise was present and sleep when there was silence. Conversely, misses were classified as epochs of wake when there was silence and sleep when there was noise. The percentage of epochs that were classified as hits or misses was calculated for each separate sampling period (i.e. the first or last 90 minutes of the sleep recording).

Paired t-tests were conducted for the average percentage of hits and misses between the first and last 90-minute periods. This was followed by a mixed ANOVA across the two 90-minute sampling periods for each of the four possible classifications (wake with noise/silence; and sleep with noise/silence) to examine potential differential effects of noise dependent on sampling period.

Bivariate Pearson's correlations were used to identify potential relationships between the percentage of the number of hits and misses for each sampling period. Relationships were also examined between the percentage of the number of hits and misses for each sampling period and both subjective sleep continuity and the discrepancy between subjective and objective sleep whilst controlling for the number of

noises recalled in the morning. This was to assess whether noise can be used within the constructs of the Neurocognitive Model by examining if the recollection of noise associates with a disruption to sleep only in the first 90 minute sampling period (indicative of NREM sleep).

Subjective-Objective discrepancy values were calculated using the method within the paper by Edinger and Fins (1995), whereby subjective misperception was calculated as a percentage of the observed sleep: Subjective value/ Objective value)*100. As this method employs a percentage value, the directions of the discrepancy become apparent in the score; <100 is a greater objective value, 100 is no difference, and >100 is a higher subjective value.

3.1.5.1 Missing Data

With regards to the pairing of the actigraphy and noise recording, if the participant forgot to hit the marker to indicate the beginning and the end of the recording period, the wake and sleep times from the sleep diary were used as a substitute (out of 96 recording period marker points, 8 (8%) were substitutes from the sleep diary). One participant's data was missing from the baseline actigraphy analysis due to a recording error. This participant remained in the study as the baseline actigraphy was only used to corroborate the sleep diary and as training for the observational part of the study.

3.2 Results

3.2.1 Associations between Sleep/Wake State and the Presence or Absence of Environmental Noise

There was a significant interaction between the sleep/wake state and presence/absence of noise in each of the 90 minute sampling periods. The analysis indicated that for both the first 90 ($\chi^2(1,5768)=49.84,p<0.01$) and last 90 ($\chi^2(1,5774)=64.22,<0.01$) minute sampling periods, there were small (Phi's test) but significant associations between sleep state and the presence or absence of

environmental noise (Table 3.1 and Table 3.2). Using odds ratios, under both conditions the condition of wake with noise was 1.78 times more likely than sleep with noise within the first 90 minutes sampling period and 2.18 times more likely in the last 90 minutes sampling period.

Table 3.1: Cross tabulation of the counts of wake/sleep epochs that were associated with noise or silence as an average of the first 90 minutes of sleep from the 3 experimental nights. **= p < 0.01 Adjusted standardized residuals appear in parentheses below group frequencies.

First 90 Minutes of Sleep				
	Environmental Noise			
Sleep State	**Noise**	**Silence**	**χ2**	**Φ**
Wake	534	245	49.84**	-0.09
	(4.3)	(-5.0)		
Sleep	2748	2241		
	(-1.7)	(2.0)		

Table 3.2: Cross tabulation of the counts of wake/sleep epochs that were associated with noise or silence as an average of the last 90 minutes of sleep from the 3 experimental nights. **= p < 0.01 Adjusted standardized residuals appear in parentheses below group frequencies.

Last 90 Minutes of Sleep				
	Environmental Noise			
Sleep State	**Noise**	**Silence**	**χ2**	**Φ**
Wake	733	134	64.22**	-0.11
	(3.8)	(-6.3)		
Sleep	3509	1398		
	(-1.6)	(2.7)		

3.2.2 Differential effects of sampling period in the reaction of sleep to noise

3.2.2.1 Overall Differential Effect of Noise within each Sampling Period

The four classifications for each of the epochs can be categorised dependent on their expected outcome, in this case a true positive would be classed as wake when a noise was present and a true negative would be sleep when there is silence, with both these classified as hits. On the other hand, misses would be false positives, wake when there is no noise, and false negatives, sleep when there is noise. The percentage of epochs classified as hits and misses was calculated for each sampling period, therefore the percentage of epochs classified as hits or misses are complimentary for each sampling period (they add up to 100%). To examine the differential effects of noise within each of the sampling periods paired t-tests were conducted for the average percentage of hits and misses between each of the 90-minute sampling periods (Table 3.3). There were significant differences between the percentage of hits and misses between the two sampling periods, with the first 90 minute period having a significantly higher percentage of hits ($t(15)=2.701$, $p<0.05$) yet a significantly lower percentage of misses than the last 90 minute sampling period ($t(15)=-2.701$, $p<0.05$). Indicating that it is the period of sleep said to contain predominately NREM sleep (first 90 minutes) that is most susceptible to awakenings/movement due to noise.

Table 3.3: Paired t-tests between the two sampling periods (first and last 90 minutes) on the percentage of hits (wake with noise and sleep with silence) and misses (wake with silence and sleep with noise)

Average percentage of either hits or misses	Sampling period						
	First 90 minutes		Last 90 minutes				
	Mean	SD	Mean	SD	t	df	p
Hits	48.08	16.41	36.91	15.92	2.701	15	0.016
Misses	51.92	16.41	63.09	15.92	-2.701	15	0.016

3.2.2.2 Differential Effect of Sampling Period

A mixed ANOVA was conducted between the two 90 minute sampling periods and average percentage of each of the four possible epoch classifications (wake with sleep or silence and noise with sleep or silence), Table 3.4. Due to a significant Mauchly's test of sphericity, a Greenhouse-Geisser correction was applied. The ANOVA indicated that there was no significant effect between sampling periods (first 90 minutes vs last 90 minutes) on the distribution of the percentages of each of the four epoch classifications, $F(1,30)=2.358$, $p=0.135$. However there was a significant effect of epoch classification within each of the two sampling periods ($F(1.27,38.03)=70.32$, $p<0.01$), that is if the sampling periods are ignored, then there is a significant difference between epoch classifications. Furthermore, there was significant interaction between sampling period and the epoch classifications ($F(1.27,38.03)=4.514$, $p<0.05$). Indicating that the distribution of noise and the sleep disturbance was equal over the two sampling periods yet there was a differential distribution of the four epoch classifications within each sampling period. This suggests that there are differential properties affording or inhibiting protection of sleep from noise between NREM and REM sleep.

Table 3.4: Averages for a mixed ANOVA between the two sampling conditions using each of the four possible sleep/wake conditions

	Average percentage of each potential epoch classification							
	Wake with noise		Wake with silence		Sleep with noise		Sleep with silence	
Sampling Period	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
First 90 minutes	9.26	6.79	4.25	4.17	47.67	18.99	38.83	17.67
Last 90 minutes	12.70	6.46	2.29	1.88	60.81	17.35	24.21	18.51

3.2.3 Relationships between Noise during Sleep and Measures of Sleep, Stress and Arousal

All questionnaires were initially checked for internal consistency within the sample using a test of Chronbach's α, Table 3.5. No significant relationships were observed between the questionnaire measures and the percentage of hits and misses during the first 90 minute sampling period (Table 3.6). However, relationships were observed between the questionnaire measures and the percentage of hits and misses during the last 90 minute sampling period (Table 3.7). Scores on the somatic subscale of PSAS and the FIRST showed significant positive relationships with the percentage of epochs classified as hits ($r(14)=0.512$, $p<0.05$ and $r(14)=0.506$, $p<0.05$ respectively) and significant negative relationships with the number of epochs classified as misses ($r(14)=-0.512$, $p<0.05$ and $r(14)=-0.506$, $p<0.05$ respectively). As both pre-sleep somatic arousal (precipitating/perpetuating factor) and the propensity to experience sleep disturbances when exposed to stress (predisposing factor) are known influences in the development of insomnia, results indicate that only the period of sleep thought to be predominately REM sleep is vulnerable to disturbance of sleep by noise through these factors.

Table 3.5: Average participant response to a variety of questionnaires and the internal consistency of the questionnaire within this sample. PSAS= Pre-Sleep Arousal Scale, FIRST= Ford Insomnia Response to Stress Test, PSS= Perceived Stress Scale, PSRS= Perceived Stress Reactivity Scale, and WNSS= Weinstein's Noise Sensitivity Scale.

	Mean	SD	Chronbach's α
PSAS somatic subscale	9.81	2.20	0.613
PSAS cognitive subscale	18.69	5.30	0.803
FIRST	19.13	5.73	0.874
PSS	20.00	7.28	0.877
PSRS total	18.19	8.42	0.889
WNSS	75.44	18.70	0.890

Table 3.6: Bivariate Pearsons correlations showing relationships between the average percentage of hits and misses for the first 90 minutes and scores on the questionnaires. PSAS= Pre-Sleep Arousal Scale, FIRST= Ford Insomnia Response to Stress Test, PSS= Perceived Stress Scale, PSRS= Perceived Stress Reactivity Scale, and WNSS= Weinstein's Noise Sensitivity Scale.

Average percentage of either hits or misses in the first 90 minute sampling period	Questionnaires					
	PSAS Somatic	PSAS Cognitive	FIRST	PSS	PSRS Total	WNSS
Hits	0.190	0.335	0.444	0.096	0.108	0.467
Misses	-0.190	-0.335	-0.444	-0.096	-0.108	-0.467

Table 3.7: Bivariate Pearsons correlations showing relationships between the average percentage of hits and misses for the last 90 minutes and scores on the questionnaires. PSAS= Pre-Sleep Arousal Scale, FIRST= Ford Insomnia Response to Stress Test, PSS= Perceived Stress Scale, PSRS= Perceived Stress Reactivity Scale, and WNSS= Weinstein's Noise Sensitivity Scale. * p<0.05

Average percentage of either hits or misses in the last 90 minute sampling period	Questionnaires					
	PSAS Somatic	PSAS Cognitive	FIRST	PSS	PSRS Total	WNSS
Hits	**0.512***	0.154	**0.506***	0.144	0.362	0.330
Misses	**-0.512***	-0.154	**-0.506***	-0.144	-0.362	-0.330

3.2.4 Relationships between Noises Recalled and Sleep

The average recall of noises per night across the three-night observation was less than one noise (mean of 0.92, *SD* 0.92) and the average recall rate ranged from no noises to a maximum of 2.67 noises averaged across all three nights.

3.2.4.1 Relationships between Noise during Sleep and Subjective Sleep Continuity

To examine whether noise during sleep influences subjective sleep quality a partial Pearsons correlation was performed for each 90 minute sampling period (Table 3.8 and Table 3.9) controlling for the average number of noises recalled the following

morning. Significant correlations were only found within the first 90 minute sampling period for both the percentage of hits and misses for total sleep time, with the percentage number of hits showing a negative relationship ($r(14)=-0.524$, $p<0.05$) and the number of misses showing a positive relationship ($r(14)=0.524$, $p<0.05$). Indicating that only the period thought to consist of predominately NREM sleep showed a relationship with noise during sleep and perceived disruption to total sleep time.

Table 3.8: Partial Pearsons correlations showing relationships between the average percentage of hits and misses for the first 90 minutes and average measures of sleep continuity whilst controlling for the number of noises recalled. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, SE = Sleep Efficiency * p<0.05

Average percentage of either hits or misses in the first 90 minute sampling period	Average subjective sleep continuity				
	SOL	**NWAK**	**WASO**	**TST**	**SE**
Hits	-0.082	0.123	0.206	**-0.524***	-0.181
Misses	0.082	-0.123	-0.206	**0.524***	0.181

Table 3.9: Partial Pearsons correlations showing relationships between the average percentage of hits and misses for the last 90 minutes and average measures of sleep continuity whilst controlling for the number of noises recalled. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, SE = Sleep Efficiency

Average percentage of either hits or misses in the last 90 minute sampling period	Average subjective sleep continuity				
	SOL	**NWAK**	**WASO**	**TST**	**SE**
Hits	0.152	0.095	0.142	0.184	-0.147
Misses	-0.152	-0.095	-0.142	-0.184	0.147

3.2.4.2 Relationships between Noise during Sleep and the Discrepancy between Subjective and Objective Sleep

Using the methodology defined by Edinger and Fins (1995) the discrepancy between subjective sleep (calculated through the sleep diary) and objective sleep (gathered through actigraphy) was formulated, see section 3.1.5.

Table 3.10 shows the average discrepancy between subjective and objective sleep measures over the three observational nights. Participants within this study were able to correctly estimate their sleep onset latency, total sleep time, and sleep efficiency, as indicated through mean values of close to 100. However, participants were unable to accurately estimate their wake after sleep onset, perceiving it to be significantly shorter than what was observed via actigraphy, $t(15)=6.917$, $p<0.01$.

A partial Pearsons correlation was performed between the average percentage of hits and misses for each sampling period and the discrepancy between subjective and objective sleep whilst controlling for the average number of noises recalled post sleep. No significant relationships were found for the first 90 minute sampling period (Table 3.11) or the last 90 minute sampling period (Table 3.12).Despite minimal alterations in the perception of sleep, in terms of WASO, the misperception of sleep did not associate with noise presence during the sampling period if the number of noises recalled post sleep was controlled.

Table 3.10: The average discrepancy between subjective and objective measures of sleep continuity over the three observational nights. SOL = Sleep Onset Latency, WASO = Wake After Sleep Onset, TST = Total Sleep Time, SE = Sleep Efficiency

	Mean Subjective	Mean Objective	Mean Discrepancy	SD
SOL (min)	11.73	19.17	88.81	105.58
WASO (min)	7.45	49.06	13.33	31.03
TST (min)	417.81	407.50	103.01	10.95
SE	87.71	85.67	102.65	10.96

Table 3.11: Partial Pearsons correlations showing relationships between the average percentage of hits and misses for the first 90 minutes and the average discrepancy between subjective and objective measures of sleep continuity whilst controlling for the number of noises recalled. SOL = Sleep Onset Latency, WASO = Wake After Sleep Onset, TST = Total Sleep Time, SE = Sleep Efficiency

Average percentage of either hits or misses in the first 90 minute sampling period	Average discrepancy between subjective and objective sleep			
	SOL	WASO	TST	SE
Hits	-0.401	0.236	-0.191	-0.174
Misses	0.401	-0.236	0.191	0.174

Table 3.12: Partial Pearsons correlations showing relationships between the average percentage of hits and misses for the last 90 minutes and the average discrepancy between subjective and objective measures of sleep continuity whilst controlling for the number of noises recalled. SOL = Sleep Onset Latency, WASO = Wake After Sleep Onset, TST = Total Sleep Time, SE = Sleep Efficiency

Average percentage of either hits or misses in the last 90 minute sampling period	Average discrepancy between subjective and objective sleep			
	SOL	WASO	TST	SE
Hits	0.062	0.131	-0.153	-0.133
Misses	-0.062	-0.131	0.153	0.133

3.3 Discussion

In regards to environmental noise and insomnia, evidence suggests that people with insomnia may actively monitor for environmental stimuli that could impact sleep (Semler & Harvey, 2004). Indeed, individuals with insomnia have been shown to frequently report environmental noises as a potential sleep disturbance and annoyance whilst attempting to sleep (Wicklow & Espie, 2000). The aim of this chapter was to observe the effect of environmental noise during the night on measures of sleep within self-reported good sleeping participants' own homes and to examine whether noise can

increase cortical arousal and as thus be utilised as method to explore the propositions of the Neurocognitive Model. Relationships between noise during the night, sleep continuity (subjective and objective), and measures of stress, arousal, and noise sensitivity were examined along with relationships between the ability to recall noise stimuli during the night and sleep state misperception. There was evidence that individuals were more likely to wake during noise than to remain asleep and individuals who awoke with noise where more likely to report a worsening of subjective sleep continuity (shorter TST). Furthermore, traits pertaining to an insomnia type response to stress (FIRST) and somatic arousal prior to sleep (PSAS) were associated with potential inability to habituate to noise (i.e. the ability to sleep when environmental noise is present).

3.3.1 Associations between Nocturnal Noise and Sleep State

The first hypothesis proposed that noise during the night would be more likely to associate with wake than sleep, with the first 90 minute sampling period being most disturbed by noise. Findings from this study indicates that noise within the home is sufficient to disturb sleep as indicated by significant associations between time spent awake in the presence of noise, and time spent asleep in the absence of noise confirming the first hypothesis and supporting the guidelines on the detriment of nocturnal noise stipulated by WHO (2009). As this measure to assess objective sleep utilised actigraphy, this study replicated the findings in the seminal work by Öhrström (1995) which assessed the relationship between body movements and noise interruptions. As body movements are interpreted by the actigraphy software as an awakening, the results from this present study could be interpreted alternatively as the more noise is present in the environment, the more body movements that occur during sleep, and thus a sleep environment with excess noise may results in a greater risk for sleep fragmentation in vulnerable individuals.

As the sampling periods were separated into the first and final 90 minutes of the total sleep period, findings from this study indicated that noise might not affect sleep unilaterally, as shown by the results of a mixed ANOVA highlighting a significant interaction between classification of the epochs and the sampling period. As the sampling periods were selected on the basis that they would differ in terms of their sleep characteristics these findings suggest that the impact of environmental noise on sleep may be dependent on the stage or depth of sleep, and it is NREM sleep that is the most vulnerable. However, due to the measures employed, this needs to be interpreted tentatively as actigraphy is a measure of rest/active cycles of which sleep/wake states can be interpreted. As thus, in order to fully address the hypothesis that noise would have a differential effect on sleep dependent on the structure of sleep, a more robust technique would be required, specifically the utilisation of polysomnography to categorise sleep architecture within the two sampling periods.

3.3.2 Relationships between Nocturnal Noise during Sleep and Measures of Stress, Arousal, and Noise Sensitivity

This study proposed that vulnerability traits specific to insomnia surrounding noise sensitivity and arousal may affect how noise influences sleep. Wicklow and Espie (2000) evidenced that environmental noise is reported by people with insomnia (i.e. assumed to be at a higher level of cortical arousal) as a significant disrupter to sleep. This present study expanded on the findings by Wicklow and Espie (2000) by examining associations between vulnerability to noise during sleep and measures of stress and arousal in a good sleeping cohort.

In this study, only the second 90 minute sampling period indicated significant associations with measures that are vulnerability factors for insomnia. The somatic subscale of the PSAS had a significant positive relationship with the percentage of epochs classified as hits and a significant negative relationship with the percentage of epochs classified as misses. This suggests that high levels of somatic arousal prior to

sleep may result in more wake when noise is present and more sleep when there is silence occurring when sleep is at an endogenously higher frequency (last 90 minute sampling period assumed to constitute mostly REM and stages N1 and N2). Although the latter may seem spurious, as it suggests that higher levels of somatic arousal correlate with sleep when the environment is silent, this result is accompanied by a significant negative correlation between the somatic subscale of the PSAS and the percentage of epochs classified as sleep with noise, as included under the percentage of epochs classified as misses. Therefore, higher levels of perceived somatic arousal prior to sleep may be a vulnerability trait for disturbed sleep from environmental perturbations, those individuals with higher levels of somatic arousal may be unable to protect sleep from environmental noise.

However, this result is slightly surprising in its singularity as, although somatic arousal during the pre-sleep period is implicated in insomnia, so too has cognitive arousal, of which there was no significant relationships apparent (Harvey, 2000; Harvey, 2001a; Wuyts, De Valck, Vandekerckhove, Pattyn, Bulckaert, et al., 2012). Additionally, it is the one key proposition of the Neurocognitive Model that somatic arousal and cognitive arousal interact resulting in an increase in cortical arousal (Perlis, Giles, Mendelson, et al., 1997). It is this interaction which ultimately leads to subjective detriments to sleep due to external sensory processing (hypothetically environmental noise) during sleep through the effects of raised cortical arousal.

Furthermore, scores on the FIRST also showed a significant positive relationship with the percentage of epochs classified as hits and a significant negative relationship with the percentage of epochs classified as misses, for the last 90 minute sampling period. Again, this suggests that the more vulnerable an individual is to experiencing insomnia when they experience a heightened period of life stress, the more epochs will be associated with sleeping when there is silence (hits) as opposed to sleeping through noise (misses). Again, the FIRST provides a measure of vulnerability to increased

cortical arousal; therefore, this finding could be interpreted as individuals who have an increase in cortical arousal upon experiencing or anticipating a stressful event may have an inability to habituate to sleeping in a noisy environment.

Therefore, the hypothesis that vulnerability traits that relate to increased arousal are implicated within the Neurocognitive model will be associated with disturbance of sleep by noise can only be partially confirmed by this study. Although it was found that measures of somatic arousal and insomnia type responses to stress correlate with the impact of noise on sleep, well-known precipitating insomnia factors surrounding stress and perceived stress did not. What it does highlight, is the role of potential vulnerability factors surrounding cortical arousal and its influence on sleep protection, therefore affirming that noise may be used in the testing of the Neurocognitive model.

3.3.3 Relationships between Nocturnal Noise and Subjective Sleep

Based upon the propositions of the Neurocognitive Model it was hypothesised that a greater number of noise events during sleep or wake would be associated with a worsening subjective report of sleep continuity when the number of noises recalled in the morning was controlled for. The Neurocognitive Model states that it is the processing of external stimuli to memory that results in disparities between the subjective sleep reports and objective measures of sleep when sleep is appraised upon waking in the morning. Moreover, this processing is said to occur primarily during NREM sleep onset periods due to the endogenous presence of higher frequency EEG, with beta EEG activity shown to be present prior to stage 2 sleep onset (Freedman, 1986; Perlis, Merica, et al., 2001) and at a higher level throughout stage 2 sleep (Spiegelhalder et al., 2012) in those with primary insomnia. This was supported by the findings of this present study whereby relationships were found between subjective sleep and noise presence during the night within the first 90 minute sampling period

only. As the first 90 minutes of sleep are suggested to be indicative of mostly NREM it would appear supportive of the propositions of the Neurocognitive model.

However, significant correlations were only found between TST and the percentage of epochs classified as hits (a negative correlation) and the percentage of epochs classified as misses (a positive correlation). Therefore, this would seem to suggest that good sleepers who are able to sleep in the presence of noise may have recalled fewer noises in the morning and thus had a better perception of their total sleep time. Although this may indicate that nocturnal environmental noise may not be used to test the subjective detriments to sleep through memory encoding, as the Neurocognitive Model suggests, it may also indicate that, as this study employed self-reported good sleepers, habituation to the bedroom environment negates any effect of increased cortical arousal through noise.

3.3.4 Relationships between Nocturnal Noise and the Discrepancy between Subjective and Objective Sleep

It was expected that the higher the percentage of noise occurring during sleep would alter the perception of sleep continuity creating a disparity between the subjective report of sleep and what was determined objectively via actigraphy when the number of noises recalled in the morning was accounted for. The Neurocognitive model suggests that heightened levels of arousal during the night, observed as higher frequency EEG activity (Perlis, Merica, et al., 2001), could be in part responsible for environmental stimuli penetrating the unconscious state and result in a failure of the normal amnesia of sleep onset to the external environment (Perlis, Smith, Orff, et al., 2001). This ability to recall environmental stimuli that occurred during sleep onset is said to impair the ability to perceive sleep “as sleep” resulting in a misperception of sleep continuity.

Despite relationships being observed between noise and subjective sleep, no relationships were found between noise during the sleep period and the discrepancy between subjective and objective sleep when the number of noises recalled upon

awakening were accounted for. When the discrepancy between subjective and objective sleep was examined, it was found that, on average, participants reported better or the same on sleep measures pertaining to sleep continuity when compared to that obtained objectively through actigraphy. Specifically, within this study, participants reported a shorter WASO than what was found through actigraphy, indicative of a positive sleep state misperception (Trajanovic et al., 2007).

Again, this suggests that participants in this study may not be responding to noise within their sleep environment through habituation. Therefore, the hypothesis that the recollection of noise during sleep would associate with a discrepancy between subjective and objective sleep cannot be addressed within this study as there was a minimal discrepancy found in the participants. Furthermore, participants recalled very few noises having disturbed their sleep during the night, so although noise was shown to associate with wake, during these wake periods noise may not have been encoded to memory. This is further suggestive of a protection mechanism present in good sleepers and supportive of work highlighting the amnesia properties for verbally presented information during sleep onset (Wyatt et al., 1997; Wyatt et al., 1994).

3.3.5 Limitations and Future Considerations

The main limitation of this study is the lack of control inherent in an ambulatory study. That is, environmental and behavioural factors, such as temperature, light and electronic devices in bed that are incompatible with good sleeping behaviour could not be accounted for. Furthermore, state variables related to sociodemographic factors, such as areas of low income and deprivation, are likely to impact on some individuals sleep through increased stress and arousal and were not accounted for within this study.

In addition, the noise equipment used did not quantify the volume of incoming noise, and due to time constraints the valence of the individual noise events were not

analysed but rather collapsed in to 15s epochs. However it is generally accepted that the impact of noise may in fact be a dose response effect involving a combination of a number of noise events, the valance of noise combinations and the loudness level (Basner, Müller, & Griefahn, 2010; Björkman, 1991; Lee et al., 2010; Lercher & Kofler, 1996; Öhrström, 1995; Öhrström et al., 1990). Therefore, future research may wish to account for this by using decibel meters within the rooms as well as calculation of the valance of the noise to assess for distinction between natural environmental noises (e.g. weather), mechanical noises (e.g. car), or perhaps noises specifically related to sociodemographic factors (e.g. crime and locality).

Another key limitation is the use of actigraphy as an objective measure of sleep. The determination of wake/sleep is made through the presence or absence of activity, and although activity in the actigraphy output has been shown to correlate with noise events (Öhrström, 1995), actigraphy has been shown to have limited reliability in the assessment of awakenings in people with sleep disorders such as insomnia where a body movement does not proceed an arousal event (Sadeh, 2011b). A solution to this would be to use ambulatory polysomnography as an objective means of assessing sleep. Certainly research now indicates that sleep microstructure, most notably sleep spindles, have a protective role on the sleeping brain from noise intrusions (Dang-Vu et al., 2010). In addition, components of the EEG (the Cyclic Alternating Pattern) have been suggested to be better markers of arousal and thus sleep misperception than conventional number of awakenings (fragmentation index) (Feige et al., 2013). All of these parameters, along with a definitive measure of cortical arousal via high frequency EEG activity, cannot be identified by actigraphy and require polysomnography.

Finally, the ability of an individual to habituate to noise in the sleep environment was not measured, an ability that may be influenced by factors such as the length of time that an individual had resided in their current accommodation. As these participants were a good sleeping cohort, it is likely that if an individual had experienced disturbed

sleep due to nocturnal ambulatory noise, their sleep may have already habituated to this and thus affords a protection to subsequent similar noise events. Certainly it has been suggested that although arousals do occur individuals who are disturbed by noise actively attempt to protect sleep when presented with noise, through mechanisms such as shutting windows (Schuemer-Kohrs, Griefahn, Mehnert, Schuemer, & Moehler, 2000). Furthermore, it has been shown that only those that are sensitive to noise may show alternations to sleep when presented with noise during the night (Öhrström et al., 1990). By examining the mechanisms by which good sleepers habituate to environmental noise may provide isolation of mechanisms that are attenuated or absent in individuals with insomnia.

3.3.6 Conclusions

This study indicated minimal associations with noise and sleep, although wake is more likely to occur in the presence of noise rather than silence. It provided an original contribution to the literature by the examination of traits that may act as vulnerability factors that inhibit habituation to noise beyond measures of noise sensitivity, although in this instance minimal significant relationships were found. The lack of significant results may be due to the nature of this study not being sensitive enough to identify changes in sleep due to its ambulatory nature and use of actigraphy as the objective sleep measure. Alternatively, the lack of significant findings may evidence the protective mechanisms that are present in good sleeping individuals due to habituation to the bedroom environment negating the effect of raised cortical arousal: a response by the plasticity of sleep present in good sleepers as proposed by Espie (2002). In light of this, the following chapter will assess the effect of sound on sleep employing novel stimuli in a non-habituated environment (a sound attenuated sleep laboratory) as a means of raising cortical arousal and utilising polysomnography as the objective sleep measure. As previous studies highlight that the number of sound events can influence

disturbance of sleep along with loudness of the auditory stimuli, these factors will be controlled within an experimental paradigm.

In summary, although significant results were few, associations between noise and the potential fragmentation of sleep (increased wake/body movement) supports the guidelines and directives set out by WHO (2009) which state that environmental nocturnal noise is sufficient to disturb sleep and thus needs to be monitored by local authorities (World Health Organisation Europe, 2011). The associations surrounding scores on the somatic subscale of the PSAS and the FIRST suggests a potential vulnerability to cortical arousal in good sleepers that can influence the plasticity of sleep and sleep protection afforded through habituation to the bedroom. Certainly the FIRST has been suggested, along with being a measure of vulnerability to insomnia, to be associated with sleep interruptions via stress related arousal (Drake et al., 2004), therefore the ability to sleep in the presence of noise may be an ability lacked in those with insomnia through the presence of high frequency EEG intrusion into NREM sleep, as the Neurocognitive Model proposes. Furthermore, although environmental noise is suggested to be a disrupter to sleep through monitoring prior to sleep, the presence of environmental nocturnal noise may be an important sleep disrupter throughout the whole sleep period, by raising cortical arousal. It is plausible that there may be a differential effect of sleep stage regarding the impact of noise on sleep once memory processing is accounted for, although again, limitations of actigraphy use do not allow further expansion of this within this study.

Seminal work suggests that habituation to environmental noise during the night can occur subjectively but not objectively (Öhström & Björkman, 1988). Moreover, there is a differential effect of noise on sleep dependent on the frequency of noise events: intermittent noise is said to increase lighter sleep at the expense of deep sleep whereas continuous noise is said to decrease REM sleep (Eberhardt et al., 1987). This suggests that by removing habituation, noise may increase cortical arousal during

sleep. Furthermore, pulses of tones used as the noise stimuli are said to result in a subjective objective discrepancy within the sleep onset latency, with sleep onset latency subjectively reported longer than objectively reported further - indicating the discrepant effect of noise on sleep (Townsend et al., 1973). This further suggests that noise may result in heightened cortical arousal as, through the propositions of the Neurocognitive Model, increased cortical may result in memory formation of said auditory stimuli, which in turn alters the perception of sleep “as sleep”.

The following chapter will try to address some of the limitations suggested by this present study by examining the impact of noise on sleep whilst in a novel non-habituated laboratory setting using PSG as the objective method to measure sleep. The use of PSG affords a greater specificity for recording sleep and allows the examination of both sleep continuity and sleep architecture. Moreover, the live viewing of the participants sleep state will allow noise to only be administered to participants during sleep, as opposed to wake, and therefore achieve a representation of the impact of noise on sleep alone. Furthermore, conducting the study within a laboratory will allow greater control of the environmental noise stimuli presented along with negating the effect of habituation afforded within participants own homes.

Chapter 4: The Impact of Environmental Noise on Sleep

4.0 Introduction

Insomnia is categorised by a disturbance to the normal sleep processes and, moreover, individuals with insomnia often report an awareness of environmental noise or noise in the bedroom environment as potential issues inhibiting the timely onset of sleep (Ellis, Hampson, & Cropley, 2002; Wicklow & Espie, 2000). As indicated in Chapter 3, some individuals may be vulnerable to awakenings by noise through measures that are perpetuating factors in insomnia. It is these factors that inhibit the protection against cortical arousal that is afford through the habitation of the bedroom environment. The Neurocognitive Model proposes that it through the processing of environmental stimuli during sleep onset and NREM sleep an overly negative perception of sleep can occur. Therefore, if noise during the night raises cortical arousal and result in, not only alterations to subjective and objective sleep in good sleepers, but also a misperception of sleep, then this would serve to support the propositions of the Neurocognitive Model.

Chapter 3 evidenced that noise is present in all individuals homes during the night and numerous studies indicate that traffic and aircraft noise during the night can lead to poor quality sleep, both subjectively and objectively, characterised by disruptions to both sleep continuity and architecture (Babisch et al., 2009; Basner et al., 2010; Griefahn, 2002; Griefahn, Brode, Marks, & Basner, 2008; Griefahn et al., 2006; Lercher & Kofler, 1996; Öhrström, 1995; Pirrera, De Valck, & Cluydts, 2010). Noise throughout the night not only has implications for sleep, but has also been shown to affect both the autonomic nervous system, as indicated by raised cardiac response to noise stimuli (Griefahn et al., 2008) and the circadian system, through alterations in the cortisol awakening response following noise during the night (Waye et al., 2003): both potential

measures of increased somatic arousal. As shown in Chapter 3, scores on the somatic subscale of the PSAS associated with a lower percentage of misses (with sleep in the presence of noise included in this) over the three observational nights, suggestive of a potential vulnerability trait for sleep disturbance by noise via an inhibition of protection against cortical arousal afforded through habituation to the bedroom.

The effect of noise on sleep is dependent on the nature, frequency and loudness of the noise events with evidence suggesting that noise has a dose-response effect on sleep: increasing numbers of noise events, with increasing volume, results in greater disturbances to both sleep continuity and architecture (Basner & Samel, 2005; Öhrström, 1995). The administration of noise during sleep has been suggested to be somewhat analogous to a model of transient insomnia with subjective and objective sleep showing equal detriments that increase in a linear fashion with the increase in noise loudness (Terzano, Parrino, Fioriti, Orofiamma, & Depoortere, 1990). Furthermore, Terzano et al, (1990) indicated the application of noise, in this instance continuous white noise, can alter sleep architecture along with sleep continuity, by reducing the amount of REM and SWS. In addition, intermittent and continuous noise affects objective sleep in different ways; with the former increasing the lighter sleep stages at the expense of deep sleep and the latter causing a decrease in the amount of REM sleep obtained (Eberhardt et al., 1987). This provides further support that a noisy sleep environment may be a vulnerability factor in insomnia by increasing the levels of cortical arousal, and thus noise can be applicable to the testing of the Neurocognitive Model.

Seminal work suggests that although individuals may habituate to environmental noise subjectively, that is after time subjective sleep will stop showing a detriment after a night of noise; this element of habituation is does not occur when objective sleep is measured i.e. objective sleep does not improve when faced with a series of nights when noise is present (Öhström & Björkman, 1988). However, when the noise

disturbance is intermittent, for example through pulses of tones rather than continuous tones, individuals seemingly cannot habituate and experience both subjective and objective detriments to sleep (Townsend et al., 1973). More specifically, Townsend et al., (1973) showed that pulsatile noise may also result in a disparity between the subjective and objective report of SOL, with the perception of SOL longer than what is observed via PSG. The ability to habituate to noise during sleep may be a protective mechanism which differentiates good sleepers from individuals with insomnia. If we are to test the Neurocognitive Model using environmental stimuli, we would expect that habituation may involve the ability to gate stimuli (the sensory processing component of the Neurocognitive Model) through the good plasticity of the sleep system (Espie, 2000) which may consequently reduce memory formation of the noise during sleep.

Although apparent habituation effect to noise perturbations within subjective and objective sleep have been shown in good sleepers, this effect does not extend to physiological functions, as the cardiac response to noise during sleep does not attenuate with subsequent nightly exposures, indicative of an inability to autonomically habituate to noise (Griefahn et al., 2008). Autonomic hyper-arousal is also said to be present in individuals with insomnia (Bonnet & Arand, 1998; Covassin et al., 2011; Nofzinger et al., 2004) and somatic arousal, such as that of the autonomic system, is said to interact with cognitive arousal leading to an increase in cortical arousal. Therefore, this could suggest that even if protective mechanisms are in place surrounding sleep which allows objective habituation to a noisy environment, cortical arousal may still be created through the somatic effect of noise on sleep, which as the Neurocognitive Model proposes, will result in a misperception of sleep.

Noise studies have been extensively used to elicit a transient model of sleep onset and maintenance insomnia, thus allowing researchers to examine the efficacy of sleep inducing pharmaceuticals under such conditions (Barkin, 2007; Bettica et al., 2012; Okuma & Honda, 1978; Parrino, Boselli, Spaggiari, Smerieri, & Terzano, 1997; Stone

et al., 2002). A recent study focused on continuous traffic noise during sleep, at approximately 50 decibels over the course of seven days to examine the effects of gaboxadol (a sleep inducing pharmaceutical) on sleep (Dijk et al., 2012). The placebo group (did not receive the sleep inducing drug) was found to have significantly disturbed subjective and objective sleep due to the noise protocol, with no members of the placebo group fully habituating to the noise by the end of the seven nights. This highlights how noise during the night could create a possible transitional, maintenance insomnia phenotype in good sleepers. As habituation did not occur, one could infer that the novel noise and sleep environment removed sleep protection via habituation and raised cortical arousal through the impact of noise on sleep. The Neurocognitive Model proposes that cortical arousal, proposed to occur through noise, creates a disparity between subjective and objective sleep through increased sensory processing of the noise stimuli which is contradictory to the study by Dijk et al., (2012) as both objective and subjective sleep were impaired by noise. However, a study examining the effect of noise volume provides results more consistent with the expectations of the Neurocognitive Model (Eberhardt et al., 1987). Eberhardt and colleagues (1987) demonstrated that as the noise gets quieter, from 55 to 45 decibels, a differential effect of noise on sleep is apparent: with the former resulting in awakenings and the latter resulting in the increase in light sleep at the expense of deep sleep. This suggests that nocturnal noise around, or slightly above, the WHO guidelines for environmental noise, may be sufficient to disrupt the normal sleep architecture of an individual without necessarily causing an awakening and altering sleep to a point which may be endogenously susceptible to cortical arousal. The higher spectral frequencies, seen in lighter sleep, have been associated with the development of sleep state misperception in individuals, and certainly EEG alterations to N1 and N2 have been indicated in those with insomnia (Krystal et al., 2002; Perlis, Merica, et al., 2001; Perlis, Smith, Andrews, et al., 2001; Spiegelhalder et al., 2012).

Analysis of the study within Chapter 3 highlighted that noise during the night is present for most individuals yet the vulnerability to noise based sleep intrusions as a predictor for developing insomnia through increased cortical arousal has been overlooked. As noise is a well-researched potential sleep disrupter, this chapter again, aims to test the propositions of the Neurocognitive Model using nocturnal noise. It was also evidenced in Chapter 3 that good sleepers may have a vulnerability to noise, and that this may occur during NREM sleep however, due to the limitations of actigraphy and that the nature of noise was not controlled, the results were not able to provide empirical testing of the Neurocognitive Model regarding sleep misperception and memory processing of the auditory stimuli.

This chapter aims to use a range of common nocturnal noises administered at the level defined by WHO that local governments must ensure environmental noise levels to do not surpass (40 decibels). This chapter aims to expand on the study within Chapter 3 by allowing greater control of the application of noise to the propositions of the Neurocognitive Model by moving from an ambulatory to a laboratory setting. Using a range of environmental noises will allow not only an examination of potential vulnerability to a disparity between subjective and objective sleep, but also determination whether explicit memory recall associates with sleep state misperception, as the Neurocognitive Model proposes. To this extent, environmental noise will only be administered during sleep bouts and participants will be unaware that noise is to be played during sleep.

The Neurocognitive Model proposes that this altered perception of sleep continuity comparative to what is observed objectively is due to the encoding to long term memory of sensory stimuli that have occurred during wake to sleep transitions (including the initial sleep onset) and possibly during NREM sleep as a whole. Currently, theory surrounding memory and sleep state as role for memory consolidation which occurs during sleep (for review see Stickgold, 2005) yet there is little data

surrounding processing during sleep onset. It is documented that individuals with insomnia can experience higher frequency EEG activity during sleep, most notably in the beta and gamma frequency range (Perlis, Smith, Andrews, et al., 2001). This high frequency EEG activity is said to be representative of a higher cortical arousal state influenced by the interaction of conditioned cognitive and somatic arousal. High frequency EEG activity is said to be present in N1 and N2 at higher level in those with insomnia comparative to good sleepers (Spiegelhalder et al., 2012). The Neurocognitive Model states that it is this increase in the frequency of the EEG, as a bio-marker of cortical arousal, that is resulting in the misperception of sleep through an impairment of the normal amnesia to external stimuli that is said to occur in good sleepers. It has been shown that when presented information during sleep onset, good sleepers struggle to recall the information following a brief (5 minute) period of stage 2 (N2) sleep (Portnoff et al., 1966; Wyatt et al., 1994). Furthermore, when quantitative analysis was conducted on the sleeping EEG in the period between stimuli presentation and recall, a positive association was identified between the presence of beta EEG power and the ability to recall the presented stimuli (Wyatt et al., 1997). Whereas in insomnia, evidence suggests that, when presented with auditory stimuli (word nouns) during wake to sleep transitions (i.e. sleep onset and at three forced NREM awakenings), those with insomnia correctly recall more word stimuli than good sleepers (Perlis, Smith, Orff, et al., 2001).

Therefore, the use of a non-habituated environment noise should act as sufficient method to raise cortical arousal, removing the potential protection mechanisms observed in Chapter 3. As thus, if an increase in cortical arousal results in an ability to recall/recognise stimuli occurring during sleep and if a disparity occurs between subjective and objective sleep, noise is a sufficient method to examine the propositions of the Neurocognitive Model. Therefore to examine the impact of increased cortical

arousal on sleep in a non-habituated environment , four hypotheses tested within this chapter:

1. Furthering the findings of Chapter 3, this study aims to conduct a more thorough examination of the impact of noise on sleep, using a within subjects design (night of silence versus night of noise) in an environment that should negate habituation effects (the novel noises and the sleeping environment). This will allow examination of the impact on subjective and objective sleep and what comparisons can be made to the constructs of the Neurocognitive Model: as previous work indicates, if habituation is not occurring then it is anticipated that noise will disrupt both subjective and objective sleep, as Dijk et al., (2012).
2. Furthermore, to examine whether noise in this protocol can be used to test the constraints of the Neurocognitive Model, this study will assess the impact of low-level noise on the perception of sleep and whether, comparative to a night of silence, a disparity occurs between the subjective report of sleep and what is observed through PSG: if the Neurocognitive Model is correct regarding sensory processing leading to sleep state misperception and as noise during sleep is said to lead to an increase in cortical arousal (intrusion of higher frequency EEG) (Eberhardt et al., 1987), it is anticipated that individuals will experience a discrepancy between subjective and objective sleep measures.
3. Chapter 3 indicated a potential association between measures of insomnia as a response to stress and arousal before sleep (somatic) and the ability to sleep in the presence of noise, this study aims to examine whether vulnerability factors that are directly measures of arousal and/or measures that contribute to a heightened level of somatic or cognitive arousal contribute to an increase in sleep state misperception following low-level nocturnal noise in an environment that participants are not used to, therefore removing habituation effects: As the Neurocognitive Model proposes that it is an increase in cortical arousal (that is

due to the interaction of both cognitive and somatic arousal) that results in the processing of information (in this study to be tested using noise) that alters the perception of sleep continuity, it is anticipated that vulnerability factors that pertain to increased arousal will correlate with an increase in them misperception of sleep from a night of no noise to a night that is interrupted by noise.

4. Finally, the Neurocognitive Model proposes that the perception of sleep is altered due to the ability to recall environmental stimuli (to be tested within this present study using environmental noise) that has occurred during sleep, thus resulting in memory encoding where there should have been none altering the perception of “sleep” as sleep. As the typology of noise occurring during the night was not controlled or documented during Chapter 3, memory encoding beyond a sense of noise disturbance by the participants was not possible. Within this present study, the type and frequency of noise is controlled and therefore will examine whether, using low-level nocturnal noises, recollection of said noise associates with a skewed perception of sleep continuity: it is anticipated that the ability to either freely recall noises presented during the night or an ability to recognise noise that were presented during the night (as indicators of sensory processing) will associate with a discrepancy between the subjective report of sleep and what is observed through PSG after a night of noise.

4.1 Methodology

4.1.1 Design

To test the Neurocognitive Model using noise as an external stimulus, a three-night (consisting of an adaptation night, baseline night, and experimental night) within subject design was implemented within the environmentally controlled conditions of the Northumbria Centre for Sleep Research (NCSR), Northumbria University. Firstly, using

repeated measures analysis, this study implemented a novel noise protocol that, unlike the transient insomnia noise models such as that implemented by Dijk et al., (2012), used environmental noise at the WHO threshold to assess the effect of low-level nocturnal noise on numerous subjective and objective sleep measures. Secondly, sleep vulnerability traits (as indicative of the participants basal arousal state) were measured to assess relationships with subjective-objective discrepancy on various indices of sleep. Finally, variables pertaining to explicit memory recall and recognition of noises administered during the night were measured to examine correlational relationships between memory processing and sleep state misperception, testing the memory formation aspects of the Neurocognitive Model of insomnia.

4.1.2 Participants

An opportunity sample of good sleeping participants was recruited from Northumbria University via word of mouth and poster campaigns. Using a screening questionnaire (see appendix A), participants were excluded at the initial eligibility screening stage if they had a sleep disorder, had a history or current complaint of anxiety or depression, had a current medical problem, or were taking medication deemed to interfere with sleep. Participants were also excluded if there was a history of severe head injury, had a hearing disorder or were employed in a sector that required rotating shift work. Exclusion for circadian rhythm disorders or irregular sleep wake schedules was completed using a combination of a baseline two-week sleep diary and actigraphy use.

In total, nineteen self-reporting good sleeping participants (as determined through a subjective sleep diary) were recruited to the study, of which 11 were male and 8 were female. One male was removed from analysis due to error in the EEG recording leading to a total sample of 10 males and 8 females with an average age of 23.29 years ± 3.16 years (range 19-30 years).

From a demographic questionnaire (see appendix B) 4 participants stated that they currently smoked, 5 were in active paid work, 1 was unemployed, and the rest were full time students at varying levels. All participants had completed at least 13 years of education (UK A-levels/Scottish Highers/BTEC award).

4.1.2.1 Consent and Ethical considerations

Ethical approval was granted by the Faculty of Health and Life Sciences ethics committee at Northumbria University. Prior to partaking in the research study, all participants indicated their informed consent for the study including overnight video monitoring. Additionally participants were requested to sign a disclosure form confirming that they acknowledged that while participating in the study they would not drive or undertake any activities that may become dangerous if drowsy. Participant remuneration was £150 for the study on a pro-rata basis over the three nights.

4.1.3 Procedure

The protocol involved two weeks of baseline sleep monitoring by sleep diary and actigraphy, followed by three nights within the NCSR, Figure 4.1. The protocol was of a fixed schedule with an adaptation recording on night one, baseline recording on night two and the experimental noise protocol occurring on night three. This fixed order was chosen rather than counterbalancing to eliminate the potential risk of residual effects of the noise on the subsequent night's sleep. Based upon previous research demonstrating that anticipating noise disturbance during the night is sufficient to disturb sleep (Wuyts, De Valck, Vandekerckhove, Pattyn, Exadaktylos, et al., 2012), participants were not made aware that noises were to be played to them during the study. Furthermore, to avoid a potential bias concerning sleeping patterns and workload (potential for cognitive arousal and rumination to tasks in the day ahead on the night before), all study nights were conducted during the week on a Monday (adaptation), Tuesday (baseline), and Wednesday (experimental) nights.

Following induction into the study, habitual bedtimes and wake times for the study overnights were calculated using an average of the 10 weekdays from the two-week sleep diary. Participants were instructed to keep a stable sleep-wake pattern across the working days, which were corroborated using actigraphy; weekends were not used in the determination of habitual times for the study, as again, the study was conducted on week nights and habitual times were calculated for a standard working week for each participant. Following this baseline period, participants returned to the NCSR for overnight PSG studies. Participants were advised not to consume alcohol or nap (corroborated using Actigraphy) at any point during the overnight study period and to refrain from caffeinated substances from 6pm onwards.

All participants arrived at the NCSR at 9pm and, prior to going to sleep, participants were free to read or watch television with the exception of the first night whereby paper based psychometric questionnaires were completed. An hour prior to habitual bedtime, participants were advised to get ready for bed, following which electrodes were applied to the scalp and face (with electrodes applied to the anterior tibialis muscles on both legs on the first night in order to screen for movement disorders such as restless leg syndrome) using the international 10-20 system (section 4.1.2.4). Participants also wore in-ear headphones on all nights to allow adaptation to sleeping with headphones in but were not advised as to the purpose of the headphones. Once in bed, bio-calibrations were performed to ensure the accuracy of the EEG recording and to provide an indication of individual EEG markers for the external scorers when conducting the staging of sleep, particularly alpha waves and REM. Bio-calibration began once the EEG recording had begun and involved 30 seconds of quiet wake with eyes open, 30 seconds of quiet wake with eyes closed, followed by a series of eye movements (look left, right, up, and down), and finally a series of EMG checks (grit teeth, yawn, smile, and purse lips). After bio-calibrations had been completed, the lights were turned out (at habitual time) and participants were requested to sleep and

not to sit in bed and read. Mobile phones and other electronic devices were left outside of the bedroom during the overnight study. The study was conducted in noise-attenuated rooms, with blackout blinds to ensure minimal light intrusion during the night (room at <5 Lux). Participants slept and woke at habitual times determined through the 10 weekdays from the baseline sleep diary. On all mornings, a post sleep questionnaire was administered and, following a provided optional breakfast, participants left approximately 90 minutes after habitual wake time. On the first and second mornings participants continued to wear the actiwatch throughout the day to ensure compliance to the protocol.

This procedure was repeated for all three nights, but during the third and final night, the noise protocol was administered. On the final morning, participants completed a memory task, testing both recall and recognition of the noises administered during the previous night. As the participants were not aware that noises were to be played to them, a measure of hearing ability could not be conducted prior to testing and thus screening for hearing difficulties took place post study on the final morning. To assess hearing level, participants were required to verbally respond to a 1000 Hz tone played at approximately 40 decibels (the loudness level used during the study). As long as they stated that they could hear it, the data was included in the study. No participant's data was excluded due to hearing issues.

4.1.3.1 Noise Protocol

Thirty nocturnal noises along with an inter-stimulus noise (white noise obtained through the program Audacity v2.0) were administered at approximately 40 decibels via in-ear bud style headphones (manufactured by NoiseMAGIC), held within the ear by surgical tape.

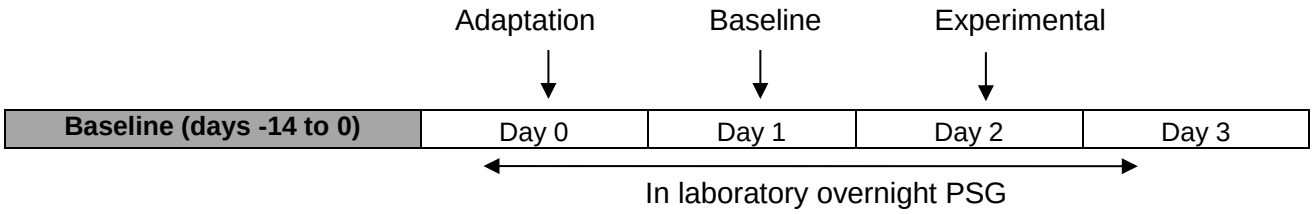
Noises were administered via a set playlist through Microsoft Windows Media Player on an HP Compaq 6005 mini-tower computer. The headphones attached to the

computer through a 10m audio extension cable (3.5mm gold plated) allowing administration of noises to the participant throughout the experimental night. As participants were not aware noises were to be played during the study, headphones were worn on all three nights.

Each noise was organized in 100 second blocks consisting of a repeating pattern of 3s of noise followed by 7s of quiescence. This pattern was repeated three times to form a 300s block (i.e. 30 repetitions of the same noise). Between each 300s block, 100s of inter-stimulus noise (white noise) was played followed by a 100s of quiescence. Upon completion of this 500s block, the next noise stimulus would commence from the set of thirty in a predefined order to ensure all participants experienced the same type of noise from sleep onset, see appendix E. Upon completion of the entire playlist (30 noise blocks of 500s the playlist would repeat until one hour prior to habitual wake time.

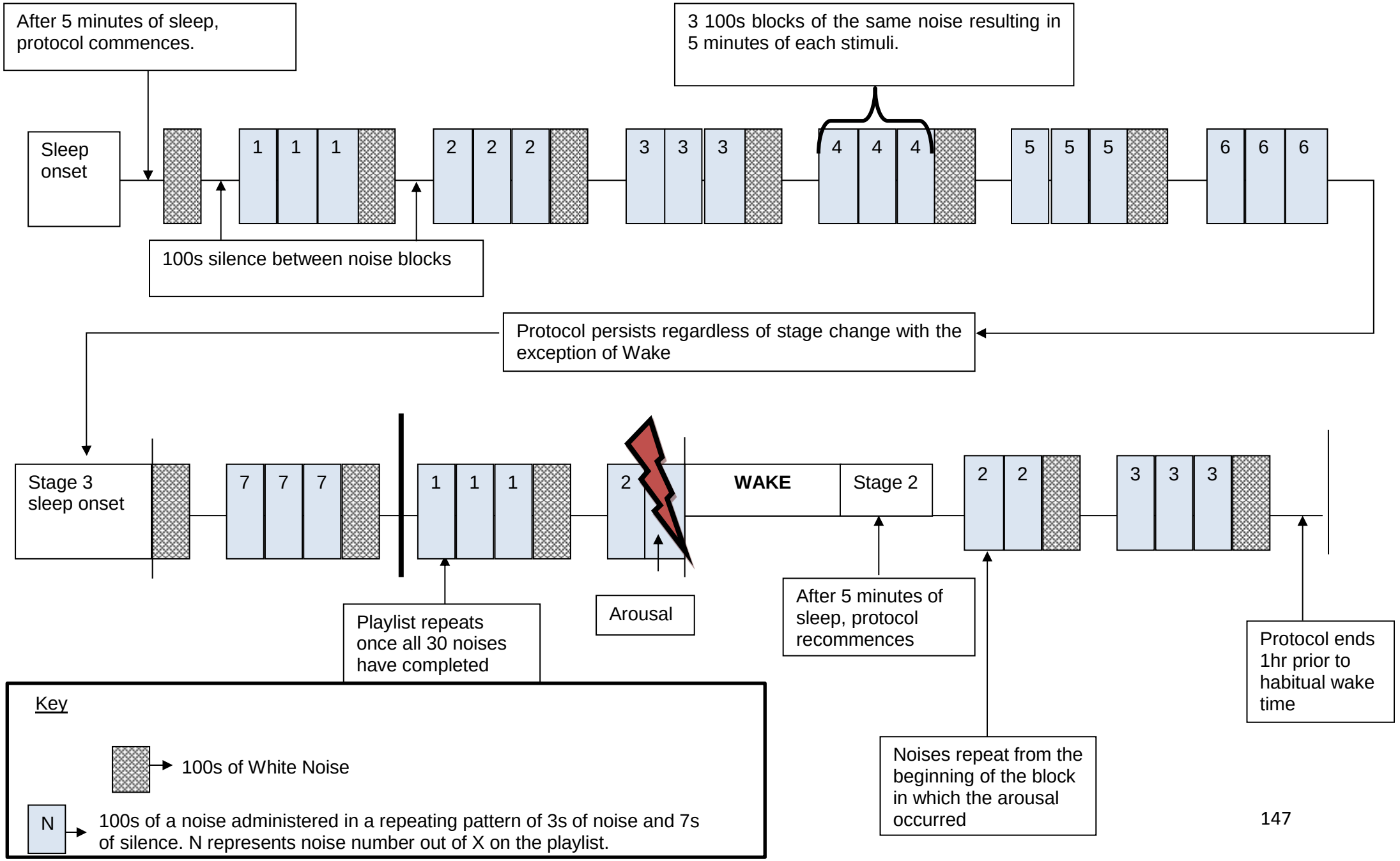
This protocol was initiated 5 minutes after sleep onset, judged as the onset of stage 2 sleep (one epoch of stage 2 indicated the start of the protocol). In the event of a natural awakening, as determined by two epochs of wake, noises were stopped and restarted again, from the beginning of the current 100s block, once sleep onset was again observed (one epoch of stage 2 sleep). See Figure 4.2 for a detailed schematic of the noise protocol.

Figure 4.1: Schematic of the study procedure.



Assessment Schedule	-14	-13	-12	-11	-10	-9	-8	-7	-6	-5	-4	-3	-2	-1	0	1	2	3
Experimental Consent																		
Eligibility Screening																		
Standard Sleep Dairy																		
Actigraphy																		
Psychometric Paper Questionnaire																		
PSG																		
Post sleep questionnaire																		
Noise administration																		
Noise recognition task																		
Noise recall task																		
Experimental Debrief																		

Figure 4.2: Schematic of the noise protocol.



4.1.4 Apparatus and Materials

4.1.4.1 Sleep Diary

A standard sleep diary, as first administered in Chapter 3, was used for two weeks prior to the study to ascertain baseline sleep measures and habitual wake and bed times. Variables used for this study were measures of sleep continuity including: Time in Bed (TIB), Total Sleep Time (TST), Sleep onset Latency (SOL), Number of Awakenings (NWAK), Wake After Sleep Onset (WASO), and Sleep Efficiency (SE). In addition, participants also completed a series of Likert scales pertaining to sleep quality. The sleep diary was also used in addition to the screening questionnaire to assess the presence of insomnia using the clinical cut-offs suggested by Lichstein and colleagues (2003) defined as SOL>30 minutes and/or WASO>30 minutes, present for 3 or more days a week.

4.1.4.2 Actigraphy

Wrist based actigraphy was used during the two week baseline period prior to study induction, as first used for the baseline period of the study in Chapter 3 (Cambridge Neurotechnology, sampling rate of 1-minute epochs). Baseline reports of measures of sleep continuity (TST, SOL, NWAK, WASO, and SE) were gathered, which were then used to corroborate reports from the sleep diary. In addition, actigraphy was also used during the three experimental nights to ensure compliance to the protocol (no napping) whilst away from the sleep laboratory. However, reports from actigraphy were not used in the determination of a sleep disorder due to the wide discrepancy often observed between actigraphy and PSG (Sadeh, 2011b).

4.1.4.3 Psychometric Questionnaires

Prior to the adaptation night, participants were required to complete a questionnaire booklet consisting of several well-validated measures to assess stress, arousal and psychopathology. These measures are indicated in the 3P model of insomnia (Spielman, 1986) as being possible vulnerability factors in the development of insomnia. Accordingly,

they may be associated with sleep disturbance measures in the present study, and as such may be indicators of susceptibility to noise based sleep disturbances through an inability to allow habituation to the sleep environment.

4.1.4.3.1 Pre-Sleep Arousal Scale (PSAS)

As first used in Chapter 3, section 3.1.3.3.1.

4.1.4.3.2 Hospital Anxiety and Depression Scale (HADS)

As first used in Chapter 3, section 3.1.3.3.2.

4.1.4.3.3 Ford Insomnia Response to Stress Test (FIRST)

As first used in Chapter 3, section 3.1.3.3.3.

4.1.4.3.4 Perceived Stress Scale (PSS)

As first used in Chapter 3, section 3.1.3.3.4.

4.1.4.3.5 Perceived Stress Reactivity Scale (PSRS)

As first used in Chapter 3, section 3.1.3.3.5.

4.1.4.4 Polysomnography

Polysomnography (PSG) was recorded on all three nights of the study. The montage for all nights included ten scalp electrode sites (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1 and O2) and two EOG's (EOG_L and EOG_R), all referenced to mastoid M1 (M2 in the case of signal loss on M1) following the international 10-20 system for electrode placement. In addition, EMG electrodes were placed on the chin to the American Academy of Sleep Medicine (AASM) guidelines (bipolar submentalis muscle referenced to the mentalis muscle) and a two lead ECG placed bilaterally on the chest (one electrode positioned below midpoint of the clavicle on the right side and one positioned on the left second to last intercostal space). On the adaptation night, electrodes were also placed on the left and right anterior tibialis muscle to screen for restless leg syndrome or periodic leg movement disorder (nocturnal myoclonus).

Electrode application was completed using GRASS 30” Gold Disk Electrodes (F-E5GH), fixated with Weaver company 10-20 paste conductive paste and electrodes were monitored to ensure all impedance was kept below 5Ω. Acquisition of the data was via the S-Med wireless PSG+ system and digitised and viewed in real time to a DELL (19”x11”) monitor, as well as recorded to an internal flash card. Real time analysis was completed using the DOMINO automatic sleep scoring system (v1.3 to AASM guidelines) to remove the variability between scorers when initiating the protocol.

Using the inbuilt marker system on DOMINO, the time at which the noises were administered was marked directly onto the PSG recording. Additional to the polysomnography recording, all participants were monitored throughout the night using night vision enabled CCTV to ensure safety of individuals throughout the night.

4.1.4.5 Post Sleep Questionnaire

Participants were administered a brief questionnaire each morning during the three night protocol immediately upon awakening to document subjective views of sleep continuity and quality. The questionnaire consisted of four items relating to sleep continuity (TST, SOL, NWAK, and WASO) and three 100mm visual analogue scales (VAS) pertaining to sleep quality (overall quality, sleep length and disturbances). The post sleep questionnaire was completed whilst participants were still in bed.

4.1.4.6 Noise Stimuli

Thirty nocturnal noises standardised for duration (3 seconds) and peak frequency (1500 Hz) were obtained from a UK based audio noise company, fonicLAB. The noises were both environmental and mechanical in nature determined by the research team to be of UK ecological validity, see appendix E. The administration order of the noises was determined by asking a convenience sample of 10 volunteers to rate the noises in order of frequency they believed would be heard during the night: with the most frequently reported occurring at the beginning of the playlist.

Noises were administered through the SoundMagic headphones that had adaptable sized tips to allow participants to find the tightest fit in the ear to ensure maximal noise isolation from the environment. These were held within the ear using surgical tape and were marketed at reducing environmental noise to below 20decibels. The headphones had a frequency range of 20Hz to 22 kHz, an impedance of 10Ω and a sensitivity of 100decibels/1mw 1 kHz.

4.1.4.7 Noise Recall Task

On the final morning, once the post sleep questionnaire had been completed, participants were given a blank piece of paper and instructed to write down anything they remembered hearing during the night. Noises that sounded similar to each other were included in the 30 noise stimuli, such as emergency services sirens (fire engine, police car and ambulance). The number of noises that were successfully recalled were summed, and compared to the number of noises falsely recalled. In the event of recalling a generalisation such as “siren”, this was scored as a single correct recall.

4.1.4.8 Noise Recognition Task

A recognition task was used to assess whether participants were able to distinguish between noises that were presented during the night and an equal number of false noises (foils). To examine this, cards were created to represent all possible salient noises along with 30 false environmental noises. Participants were instructed to separate the pack of cards into noises they heard during the night and those they did not. The recognition task was administered once the post sleep questionnaire and recall task had been completed and while the participant was still sitting in bed. Participants were timed during the recognition task and were instructed to sort the cards as quickly as they could with the upmost accuracy. Both the number of correctly recognised noises and the number of incorrectly recognised foils were documented for the analysis.

4.2 Analytical Strategy

Results from the PSG are split into measures of sleep continuity and architecture and subjective sleep reports are split into measures of sleep continuity and sleep quality. Records were blind-scored by a registered technician from another laboratory according to R&K sleep scoring criteria (Rechtschaffen & Kales, 1968a).

All statistical analysis was performed using SPSS for windows version 19. Paired samples t-tests are used to identify significant differences in subjective and objective sleep between the baseline and experimental night to determine the effect of the noise on sleep. Following this, paired samples t-tests are conducted between subjective and objective sleep measures for each of the nights (baseline and experimental) to examine whether individuals were experiencing a significant discrepancy between subjective and objective sleep reports, indicative of possible sleep state misperception. Bivariate Pearson correlation matrices are used to investigate associations between psychometric questionnaires, sleep parameters, and recollection/recognition of the noises played during the night.

Data was visually examined for normality and missing data points. Due to preforming correlational analysis, missing data points were left absent resulting in pair-wise deletion. This decision was made upon suggestions that, although removing the affected data point reduces power, it does not pose risk of an inflated error rate, which has been highlighted to occur with substitution methods in correlation analysis (Schafer & Graham, 2002).

4.3 Results

4.3.1 Baseline Characteristics of participants

Whereas only the weekdays from the two-week baseline sleep diary was used to ascertain habitual sleep and wake times, confirmation of the participants good sleeper status was drawn over the whole two week baseline period. On average, all participants reported good sleep in the two weeks prior to study start, Table 4.1.

All questionnaires administered to the participants on the adaptation night were initially examined for internal consistency and to ensure that there were no extreme outliers, Table 4.2. All scales showed acceptable internal consistency within this set of participants.

Table 4.1: Mean results over the ten weekdays from the two-week baseline sleep diary. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, TIB = Total Sleep Time, and SE = Sleep Efficiency

Sleep continuity	Mean	SD
SOL (minutes)	20.25	12.55
NWAK	0.85	0.77
WASO (minutes)	10.72	15.54
TST (minutes)	452.13	39.13
TIB (minutes)	519.62	58.84
SE	88.05	4.77

Table 4.2: Mean scores and the internal consistency of the psychometric questionnaires. PSAS= Pre-Sleep Arousal Scale (each subscale scored on a scale of 8 to 40), HADS= Hospital Anxiety and Depression Scale (each subscale scored on a scale of 0 to 21) FIRST= Ford Insomnia Response to Stress Test (scored on a scale of 9 to 36), PSS= Perceived Stress Scale (scored on a scale of 0 to 70), and PSRS= Perceived Stress Reactivity Scale (scored on a scale of 0 to 46).

Psychometric questionnaires	Mean	SD	Chronbach's α
PSAS Somatic Subscale	18.61	4.31	0.717
PSAS Cognitive Subscale	10.22	3.14	0.865
HADS Anxiety scale	5.33	2.52	0.631
HADS Depression scale	2.00	2.30	0.742
FIRST	17.76	3.36	0.568
PSS	27.65	4.69	0.533
PSRS total	19.15	5.91	0.784

4.3.2 The Impact of Nocturnal Noise on Sleep

4.3.2.1 Impact of Noise on Subjective Sleep Continuity and Quality

Paired t-tests were conducted for each measure of subjective sleep between the baseline and experimental night. Participants reported a significantly longer subjective WASO, from a baseline report of 7 minutes increasing to 24 minutes on the experimental night ($t(15)=2.15$ $p<0.05$), Table 4.3. Participants also reported significantly more awakenings on the experimental night than on the baseline night ($t(16)=2.55$ $p<0.05$). Conversely, there were no significant differences between any other measures of subjective sleep continuity (SOL, TST, and SE).

Table 4.3: Paired t-tests between subjective measures of sleep continuity from the post sleep questionnaires between the baseline and experimental nights. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency

	Night Condition					
Subjective measures of sleep continuity	**Baseline**		**Experimental**			
	Mean	**SD**	**Mean**	**SD**	**t**	**p**
SOL (min)	19.71	7.8	22.94	19.77	0.64	0.534
NWAK	1.29	1.05	2.29	1.57	2.55	**0.022**
WASO (min)	7.25	8.47	23.63	30.71	2.15	**0.049**
TST	07:17	00:35	06:58	00:50	1.74	0.100
SE	94.34	4.13	90.84	11.57	1.43	0.173

Participants reported significant alterations in perceived sleep quality after the experimental night with significantly reported shorter sleep, $t(15)=2.60$ $p<0.05$, and significantly more disturbed sleep, $t(15)=5.09$ $p<0.01$,(Table 4.4). There was also a trend towards significance in perceived sleep quality, with participants reporting poorer sleep quality on the experimental night, $t(15)=2.04$ $p=0.059$.

Table 4.4: T-tests of visual analogue scales relating to mood and sleep quality from the post sleep questionnaire. [1] Sleep quality "My sleep was 0 (worse) to 100 (better) than my usual sleep pattern" [2] Sleep length "My sleep was 0 (shorter) to 100 (longer) than my usual sleep pattern" [3] Sleep fragmentation "My sleep was 0 (more disturbed) to 100 (less disturbed) than my usual sleep pattern"

	Night Condition					
Subjective measures of sleep quality	**Baseline**		**Experimental**			
	Mean	**SD**	**Mean**	**SD**	**t**	**p**
Sleep Quality[1]	52.50	14.96	37.63	21.98	2.043	0.059
Sleep Length[2]	48.38	14.34	35.81	18.62	2.595	**0.020**
Sleep Fragmentation[3]	53.19	13.57	30.63	19.26	5.094	**0.000**

4.3.2.2 The Impact of Noise on Objective Sleep Continuity (PSG)

Paired t-tests were conducted for each measure of sleep continuity (SOL, NWAK, WASO, TST, and SE) from the PSG between the baseline and experimental night to assess the effect of the overall influence of noise on sleep, Table 4.5. The only significant alteration in sleep continuity was an increase in WASO on the experimental night, $t(17)=3.10$ $p<0.05$ and a trend in TST, reducing from 425 minutes on the baseline night to 405 minutes on the experimental night, $t(17)=2.07$ $p=0.054$.

Table 4.5: Paired t-test results for objective sleep continuity measures from PSG between the baseline night and the experimental night. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency

	Night Condition					
	Baseline		Experimental			
PSG measures of sleep continuity	Mean	SD	Mean	SD	t	p
SOL (min)	17.87	14.40	12.91	9.98	1.79	0.090
NWAK	5.56	5.00	8.11	4.56	1.84	0.080
WASO (min)	22.37	13.08	40.37	26.30	3.10	**0.007**
TST (min)	426	32	406	54	2.07	0.054
SE	91.39	3.78	87.22	8.55	1.92	0.070

4.3.2.3 The Impact of Noise on Objective Sleep Architecture (PSG)

Paired t-tests were conducted on measures of sleep architecture between the baseline and experimental nights to examine whether there were changes in sleep structure due to the noise protocol. There was a significant difference between baseline night and experimental night in the percentage of wake over the sleep period $t(17)=3.86$ $p<0.05$,

Table 4.6, with more wake occurring on the experimental night. There was also a significant difference in the percentage of stage 1 sleep over the total sleep period from 4.37% at baseline to 6.05% on the experimental night ($t(17)=2.45$ $p<0.05$). There was no difference in the percentages of stage 2, 3 and 4 between baseline and experimental nights.

Participants experienced a longer latency to REM onset on the experimental night (120 minutes) compared to the baseline night (89 minutes) ($t(17)=2.32$ $p<0.05$). There was a trend towards an increase in the latency to stage 4 from 63 minutes on the baseline night to 123 minutes on the experimental night ($t(17)=1.90$ $p=0.075$).

Table 4.6: Paired t-test results for measures of sleep architecture from the PSG on the baseline and experimental nights

	Night Condition					
	Baseline		Experimental			
PSG measures of sleep architecture	Mean	SD	Mean	SD	t	p
Stage changes	222.11	49.08	219.89	35.60	0.20	0.843
% Wake	4.07	2.22	8.43	5.27	3.86	**0.001**
% Stage 1	4.37	1.95	6.05	3.03	2.45	**0.026**
% Stage 2	56.12	5.98	57.50	5.77	1.13	0.274
% Stage 3	12.64	3.97	12.00	3.75	0.66	0.519
% Stage 4	4.52	3.96	4.79	4.43	0.45	0.658
% REM	22.35	6.14	19.65	7.91	1.80	0.090
Latency to Stage 2	21.29	14.09	16.53	10.68	1.52	0.140
Latency to Stage 3	34.01	14.43	40.38	33.15	0.81	0.430
Latency to Stage 4	63.22	80.26	122.77	152.95	1.90	0.075
Latency to REM	88.80	33.45	120.22	61.92	2.32	**0.033**

4.3.3 The Impact of Noise during Sleep on the Discrepancy between Subjective and Objective Sleep

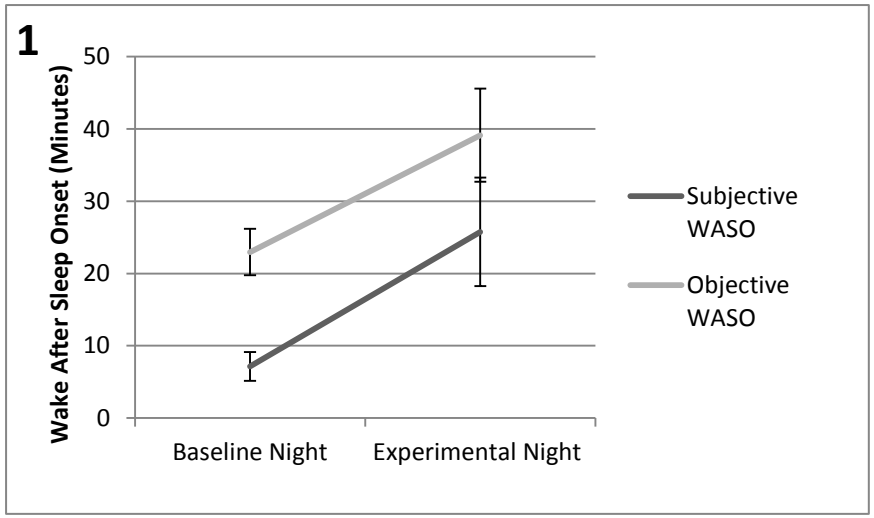
T-tests were conducted between the baseline and experimental night to examine whether there were discrepancies between subjective and objective measures of sleep continuity and whether this discrepancy differed between baseline and experimental nights

On both nights, participants experienced a discrepancy between their subjective and objective sleep, however the direction of the discrepancy differed between nights with participants reporting better sleep than what was recorded objectively by PSG on the

baseline night; and worse subjective sleep than the objectively recorded PSG sleep on the experimental night. On both nights participants reported significantly fewer awakenings (NWAK) than was observed, for the baseline ($t(17)=3.33$ $p<0.01$) and experimental night ($t(18)=5.55$ $p<0.01$. There was no significant discrepancy between subjective and objective TST on either the baseline or experimental night.

The directional differences in the discrepancies between subjective and objective sleep reports occurred within measures of SOL, WASO and SE. On the baseline night, WASO was reported significantly shorter, 7 minutes, (indicative of better sleep) than what was observed objectively, 22 minutes, $t(17)=4.974$ $p<0.01$; and SE was reported significantly better subjectively, 94%, than what was reported through PSG, 91%,($t(17)=2.30$ $p<0.05$). However, during the experimental night, both WASO and SE no longer showed a significant difference between subjective and objective sleep and the significant discrepancy was now shown within SOL, with the subjectively reported length at 24 minutes and the PSG reported length at 13 minutes, $t(17)=2.62$ $p<0.05$.

This change in discrepancy for SOL, NWAK, WASO and SE across the baseline and experimental nights can be shown graphically using line graphs (See Figure 4.3). Both WASO and SE appear to exhibit a parallel change for both the subjective and objective measures, with an increase in both the subjective sleep report from baseline to experimental night occurring with a similar increase in the objective sleep report from the baseline to experimental night. However, as Figure 4.3 below shows, this is not the case for SOL as the graph indicates divergence, with an increase in the subjective report but occurring with a decrease in the objective report from baseline to experimental night.



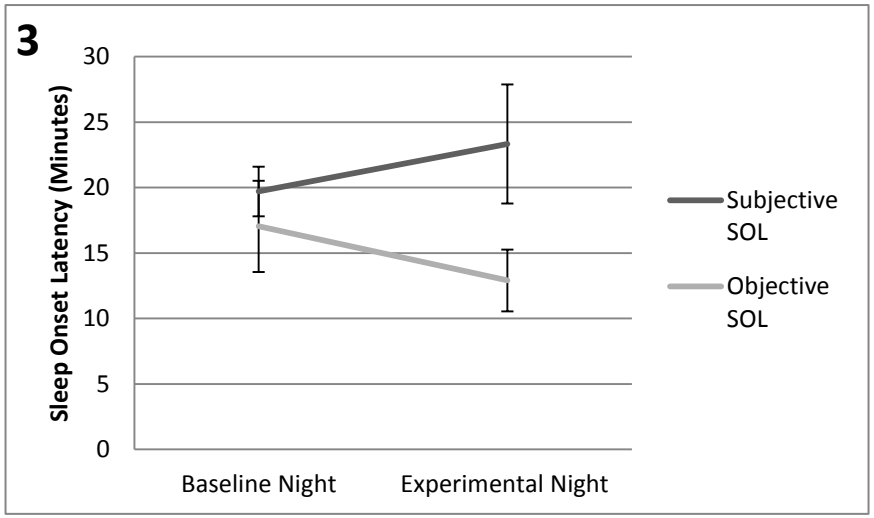
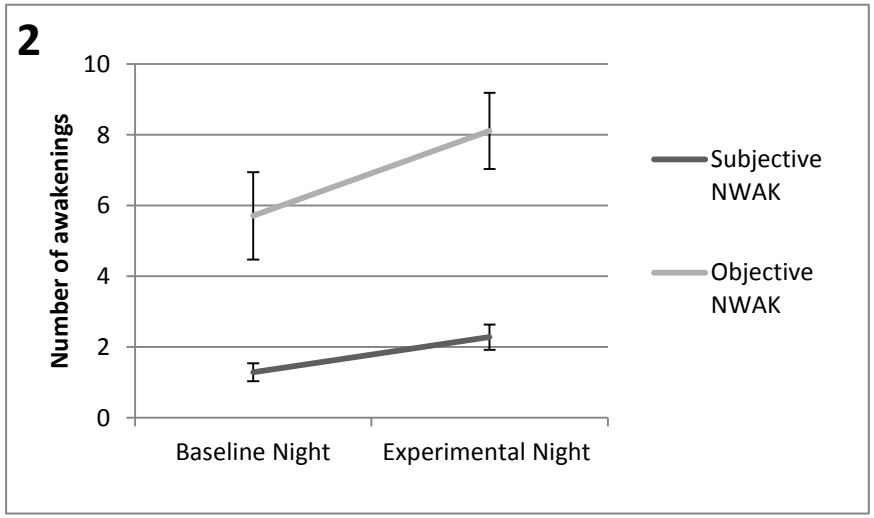
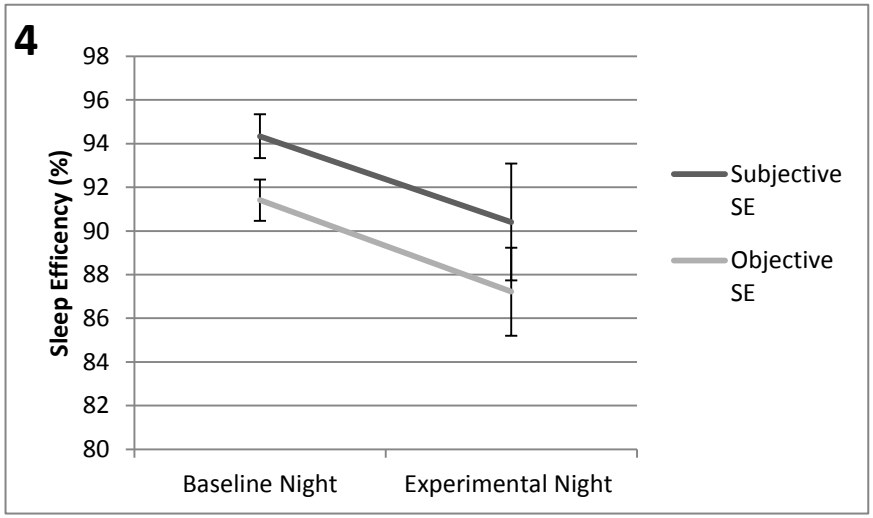


Figure 4.3 A graphical representation of the change in subjective and objective measures of sleep continuity. 1- The change in WASO 2- The change in SOL 3- The change in NWAK 4- The change in SE





4.3.3.1 Discrepancy score calculation

Discrepancy values were calculated using the method first employed in Chapter 3 whereby subjective misperception was calculated as a percentage of the observed sleep: (Subjective value/ Objective value)*100 (Edinger & Fins, 1995). As this method employs a percentage value, the directions of the discrepancy become apparent in the score; <100 is a greater objective value, 100 is no difference and >100 is a higher subjective value.

Paired t-tests were conducted on the calculated discrepancy scores between the baseline and experimental night, to examine whether the noise at night affected the direction of the discrepancy experienced. There were no significant differences between baseline and experimental nights for the discrepancy between subjective and objective sleep.

Table 4.7: Paired t-tests for the subjective-objective discrepancy scores between the baseline and experimental night. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency

	Night Condition					
	Baseline		Experimental			
Discrepancy	**Mean**	**SD**	**Mean**	**SD**	**t**	**p**
SOL	178.91	175.31	264.00	181.71	-1.674	0.113
NWAK	49.09	57.21	41.14	47.38	0.619	0.545
WASO	34.80	35.84	63.00	69.33	-1.95	0.071
TST	102.90	6.02	105.00	17.56	-0.54	0.600
SE	103.37	5.98	105.22	17.67	-0.47	0.646

4.3.4 Relationships between Measures of Stress, Arousal, and Psychopathology and the Discrepancy between Subjective and Objective Seep

Firstly, to examine whether vulnerability factors influences the relationship between subjective-objective sleep discrepancy, Pearson's correlations were computed for the measures of sleep vulnerability and the baseline discrepancy between subjective and objective sleep, see Table 4.8. There were no significant relationships identified.

Secondly, to examine whether vulnerability factors influences a change in the subjective-objective sleep discrepancy when noise is present, a partial Pearson's correlation was computed for the measures of sleep vulnerability and the experimental night discrepancy between subjective and objective sleep whilst controlling for the discrepancy experienced at baseline, see Table 4.9. A significant negative relationship between the total score on the perceived stress reactivity scale and the discrepancy in SOL ($r(5)=-0.814$ $p<0.05$) suggested that as the scores on the PSRS increased, subjective SOL was reported as increasingly shorter than the objective SOL on the experimental night. On the other hand, there were significant positive relationships between scores on the anxiety scale of the HADs and the FIRST with the discrepancy in NWAK ($r(5)=-0.765$ $p<0.05$ and $r(5)=-0.762$ $p<0.05$ respectively). This suggests that as the scores on the anxiety scale of the HADs and the FIRST increased, subjective NWAK was reported as increasingly longer than the objective NWAK on the experimental night.

Table 4.8: Correlation table of psychometric questionnaires and the subjective-objective discrepancy for the baseline night. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency. PSAS= Pre-Sleep Arousal Scale, FIRST= Ford Insomnia Response to Stress Test, PSS= Perceived Stress Scale, and PSRS= Perceived Stress Reactivity Scale.

Measures of Stress, Arousal, and Psychopathology	The discrepancy between subjective and objective parameters for the baseline night				
	SOL	NWAK	WASO	TST	SE
PSAS Somatic Subscale	-0.348	0.052	0.317	-0.185	-0.152
PSAS Cognitive Subscale	0.028	-0.201	0.187	-0.006	0.012
HADS Anxiety scale	0.195	-0.244	-0.033	-0.275	-0.244
HADS Depression scale	-0.019	-0.143	0.095	-0.095	-0.092
FIRST	0.187	-0.004	-0.056	0.000	0.010
PSS	-0.299	-0.331	-0.124	0.385	0.376
PSRS total	-0.293	0.252	-0.004	-0.252	-0.238

Table 4.9: Partial correlation of psychometric questionnaires and the subjective-objective discrepancy for the experimental night whilst controlling for baseline the discrepancy. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency. PSAS= Pre-Sleep Arousal Scale, FIRST= Ford Insomnia Response to Stress Test, PSS= Perceived Stress Scale, and PSRS= Perceived Stress Reactivity Scale. *p<0.05

Measures of Stress, Arousal, and Psychopathology	The discrepancy between subjective and objective parameters for the experimental night				
	SOL	**NWAK**	**WASO**	**TST**	**SE**
PSAS Somatic Subscale	-0.090	-0.107	-0.030	0.264	0.270
PSAS Cognitive Subscale	-0.391	0.340	0.053	-0.065	-0.061
HADS Anxiety scale	-0.482	**0.765***	0.303	-0.122	-0.123
HADS Depression scale	0.458	-0.031	-0.225	0.090	0.082
FIRST	-0.461	**0.762***	-0.077	0.087	0.082
PSS	0.036	-0.149	0.062	-0.264	-0.269
PSRS total	**-0.814***	0.547	-0.165	-0.015	-0.018

4.3.5 Relationships between Recall and Recognition of Noise and Sleep

Participants explicitly recalled or recognised very few of the noises presented to them during the night. On average, participants freely recalled only 6% of the noise presented to them during the night. For the recognition task, participants recognised a significantly higher percentage of words that were presented during the night (23.70%) then foils (16.30%), $t(17)=3.756$, $p<0.01$.

4.3.5.1 Relationships between Memory and the Discrepancy between Subjective and Objective Seep

As the Neurocognitive Model suggests, memory formation may be related to the misperception of sleep. Therefore relationships were examined between the percentage of recalled noises and the percentage of correctly or incorrectly recognised

noises/foils after the experimental night and the discrepancy between subjective and objective sleep on the experimental night whilst controlling for baseline discrepancies (Table 4.10). There were no significant associations with the degree of sleep misperception on the experimental night and measures from the recall and recognition tasks.

Table 4.10: Relationship between the subjective-objective discrepancy on the experimental night and the results from the noise recall and recognition task. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency.

	The discrepancy between subjective and objective parameters for the experimental night				
Recall/Recognition tasks	**SOL**	**NWAK**	**WASO**	**TST**	**SE**
Percentage of correctly recalled noises	0.171	-0.127	-0.010	0.264	0.258
Percentage recognition correct	-0.488	0.050	-0.130	0.003	0.001
Percentage recognition incorrect	-0.372	-0.199	-0.107	-0.218	-0.218

4.4 Discussion

This chapter aimed to use a range of ecological nocturnal noises, played intermittently (3s sound, 7s silence) to raise cortical arousal levels in good sleepers and test the Neurocognitive Model in terms of sensory processing, as determined through memory of the noise, examining, as an outcome, the potential generation of a disparity between the subjective report of sleep and what is observed through PSG.

The study within Chapter 4, aimed to determine the point where cortical arousal is said to occur, by examining NREM and REM at a greater depth based on the preliminary findings from the study in Chapter 3. Furthermore, this aimed to determine whether the ability to recall or recognise, when prompted, auditory stimuli during sleep is associated

with a misperception between subjective and objective sleep measures, as suggested by the Neurocognitive Model. Finally, this chapter examined whether individuals have specific insomnia vulnerability factors (direct or indirect measures of arousal) that prevent habituation and thus determine the extent of the disruption to sleep following a night of noise.

In the present study, there were minimal disruptions to objective sleep despite disruptions to subjective sleep: consistent with sleep state misperception. Furthermore, both sleep continuity and architecture remained intact on the experimental night with the exception of changes in wake and stage 1 sleep (endogenously of a higher EEG frequency). Moreover, there was a significant change in the direction of the discrepancy between subjective and objective sleep, moving from positive perception (i.e. WASO) of sleep on the night with no noise to a negative perception (SOL) of sleep on the night with noise. Diagnostic manuals classify insomnia based on subjective sleep and the perception of the effect of perceived poor sleep on daily functioning; therefore, any detriments to the perception of sleep quality or quantity due to the presence of noise in a non-habituated sleep environment could indicate a potential precipitating factor in the development of insomnia.

4.4.1 Overall Impact of Noise on Sleep

The first hypothesis of this chapter was to re-examine the effect of noise on sleep, a stimulus that literature suggests may alter objective sleep by increasing the lighter NREM sleep stages whilst decreasing REM sleep (Eberhardt et al., 1987). Muzet (2007) suggests that noise may be implicitly processed by an individual during sleep, and could account for the alterations seen in subjective sleep. Unlike Chapter 3, as the study was conducted in the laboratory, the noises were thought to represent a non-habituated stimulus as they were novel to the participants whilst be administered in an unfamiliar environment. Therefore, due to this inability for the individual to habituate to bedroom environment and thus increase the susceptibility for noise to increase cortical

arousal, it is unsurprising that there was a significant rise in the subjective NWAK and WASO from the baseline to experimental night, suggesting that noise during the night effects the perception of sleep maintenance.

Moreover, noise during sleep resulted in significant changes to objective sleep, with WASO increasing from 22 minutes to 40 minutes and, albeit non-significant, trend towards reduction of total sleep time. Although this does indicate an alteration to objective sleep continuity, the fact that only WASO was significant, one could state that this protocol had a minimal impact on objective sleep continuity, which may be due to the protection afforded through good sleeping practices (i.e. sleep plasticity) in the good sleeping participants. Interestingly, measures of sleep architecture remained stable in the presence of noise; however some individuals did report a total loss of both S4 and REM on the experimental night. Therefore, although not a significant difference between the nights, there may be a habituation component representative of individual differences in the response of sleep to noise, and thus suggesting that an individuals' response to noise may increase an individuals' predisposition to developing insomnia.

The results presented here are in partial agreement with previous literature investigating the effect of noise on sleep, suggesting that noise during the night affects sleep through frequent arousals and fragmentation (Babisch et al., 2009; Basner et al., 2008; Basner & Samel, 2005) and hence the use of noise as a model of transient insomnia (Dijk et al., 2012). This study supported the notion that individuals subjective sleep cannot habituate to intermittent sounds at a low level, as suggested by Townsend and colleagues (1973). Moreover, in the present study there were minimal architecture changes (an increase in wake and S1), which supports studies suggesting that low level noise creates an increase in lighter stages of sleep. This confirms the first hypothesis of this chapter that low level noise may be sufficient to disturb both objective and subjective sleep when administered in a non-habituated bedroom environment.

4.4.2 Discrepancy between subjective and objective sleep due to noise

The second hypothesis within this present study relates to the misperception of sleep through a discrepancy between the subjectively reported sleep and what is observed objectively through PSG during noise administration. Therefore this present study proposed that through the raising of cortical arousal by the low level noise, the auditory stimuli might be processed to memory by the sleeping participant even if it does not elicit an objective disruption; consequently resulting in an altered perception of sleep.

Interestingly, a significant subjective-objective discrepancy was indicated within SOL on the experimental night despite the protocol ensuring that noise was not administered until five minutes post first epoch of stage 2 sleep. This occurred despite objective adaptation of SOL to the sleep laboratory setting, with an improvement (shorter duration) from baseline to experimental. Townsend et al. (1973) did indicate that misperception in SOL can occur with low level intermittent noise administered during sleep, indicative of a direct effect of noise, however, unlike the present study, Townsend administered noise during the onset period. Once a good sleeping individual has fallen asleep it is suggested that, if woken 5 minutes after the onset of stage two sleep, most good sleeping individuals will be able to correctly report a perception of being asleep (Yang, Han, Yang, Su, & Lane, 2010). However, this would suggest that if a participant awoke shortly after the protocol initiated (during stage 2 sleep), then the perception of duration of sleep onset should not be altered, but rather the perception of WASO and NWAK should differ. Therefore, it may not be an awakening to the noise that created the disparity in the reports of SOL but rather the presence of micro-arousals as a direct result of the noise, as an increase in the number of 10-15s micro-arousals during sleep has been shown to result in an increase in subjective SOL (Smith & Trinder, 2000). However, micro-arousal analysis has not been conducted within this present study so no inference can be made to this effect.

Moreover, it could be suggested that as this group of participants were good sleepers, the protocol may have resulted in a form of sleep state misperception through the raising of cortical arousal by noise, at least concerning SOL on the experimental night. It has been suggested that a ratio change of 1.5 in subjective sleep comparative to objective sleep could be deemed sleep misperception clinically relevant to insomnia (Borkovec et al., 1979; Dorsey & Bootzin, 1997). This is in accordance with the subjective-objective difference in SOL on the experimental night where the subjectively reported SOL was 1.8 times larger than the objectively reported SOL. Indeed, when viewed graphically whereas for WASO, NWAK and SE the change in both subjective and objective sleep results in two parallel lines, the graphical representation of SOL was divergent.

However, although discrepancies between the subjective and objective existed in the individual t-tests, when the discrepancy was calculated and the differences in discrepancy between the night conditions examined, the significance disappeared. This could be due to a directional change in the misperception, from a positive subjective misperception to negative subjective misperception. Certainly, although this discrepancy is apparent on both nights, the measures that it occurs in are different between the baseline and experimental night. This is suggestive of the noise protocol reversing a positive discrepancy (perceived higher quality of sleep) experienced on the baseline night, to within the same range as the objective measure on the experimental night (WASO and SE).

Although the disparity between subjective and objective sleep was not significant across all measures, the effect of noise (be that directly or through increased cortical arousal) was still sufficient to move good sleepers from perceiving their sleep was of a higher quality than observed on the baseline to the same or lower quality on the experimental night. Therefore, the hypothesis that noise may indeed create a disparity between subjective and objective sleep is to be accepted within this present study and

thus supports the overall aim that noise may indeed be used to test the propositions of the Neurocognitive Model.

4.4.3 Associations between measures of stress, arousal, and psychopathology and sleep during the noise protocol

The Neurocognitive Model proposes that vulnerability factors (defined by Spielmans' (1986) 3Ps: Predisposing, Precipitating, and Perpetuating factors) are key in the development of cortical arousal during sleep and thus the instigation of neurocognitive factors (impaired sensory gating and an increase in memory processing of external stimuli) that lead to sleep state misperception (Perlis, Giles, Mendelson, et al., 1997). Within this present study, it was hypothesised that individuals that exhibited greater scores on questionnaires suggested to be vulnerability factors to noise based sleep intrusions, through increased arousal and sensory processing, would experience a greater disparity between subjective and objective sleep measures on the experimental night comparative to baseline due an inability to habituate to the noise.

On the baseline night, whereby there was no noise, there were no significant relationships between vulnerability factors and the discrepancy in subjective and objective sleep. However, on the experimental night and when baseline discrepancy was accounted for, significant associations were identified between measures of stress reactivity (PSRS) and the discrepancy in SOL, suggesting that higher perceived stress reactivity associates with a subjective over estimation of SOL comparative to what is seen objectively. Furthermore, scores on the HADs anxiety scale and the FIRST showed significant negative relationships with the misperception of NWAK on the experimental night. Higher scores on these scales associated with a greater subjective report of awakenings than what was observed through PSG.

Predisposing factors in the development of insomnia include a high reactivity to experience stress, or sleep disturbances phenotypical of insomnia as a response to stress. Moreover, stress and anxiety (psychiatric illness) are included within the

Neurocognitive Model as factors that could lead to cortical arousal. In accordance with the Neurocognitive Model, it could be that individuals who experience these vulnerability factors experience increased cortical arousal resulting in the disparity between subjective and objective sleep measures, due to increased sensory processing. Some of the present results are thus in agreement with these propositions of the Neurocognitive Model, partially confirming the fourth hypothesis of this present study.

4.4.4 Associations between recall and recognition of noise during the night and sleep

Evidence suggests people with insomnia may exhibit higher frequency EEG activity during sleep (Perlis, Merica, et al., 2001; Perlis, Smith, Andrews, et al., 2001) and this increase in spectral power of the EEG is suggested to alter perception of sleep through an impairment of the normal amnesia that should occur to external environmental stimuli during sleep onset (Perlis, Smith, Orff, et al., 2001). The final hypothesis of this present study suggested that the greater the memory (be that immediate recall or recognition) of the noises presented during the night, a greater disparity will occur between subjective and objective sleep. The Neurocognitive Model proposes that when an individual is appraising the previous night's sleep perception will be skewed due to the ability to recall environmental stimuli (noise) that had occurred during the night, which may alter the subjective report of continuity. Therefore, if an individual is able to recall noises that were played during the sleep, this is indicative of a sensory processing during the night, and thus, if the propositions of the Neurocognitive Model is correct, this will associate with a greater disparity between the subjective report of sleep (worse) and what is observed objectively via PSG.

However, within this study, participants recalled and recognised very few of the noises presented during sleep, which could be representative of a floor effect, with the task of recalling up to 30 individual noises too difficult. Alternatively, this may indicate that an

acute period of heightened cortical arousal via noise in a non-habituated environment is not sufficient to result in direct encoding of environmental stimuli potentially due to the innate ability of the good sleepers sleep plasticity to respond to the environmental perturbations.

Therefore, although participants significantly recognised a higher percentage of presented noises than falsely identified foils, the numbers are small and thus inferences from this study need to be viewed with caution. Due to this, it is unsurprising that this present study provided no evidence towards the memory formation component of the Neurocognitive Model as an explanation for the occurrence of sleep state misperception. As in the final hypothesis of this chapter, it was expected that as participants experience greater misperception comparative to baseline, so too would they recall or recognise more of the noises administered during the night. However, the misperception of sleep on the experimental night, once the discrepancy at baseline had been accounted for, showed no associations with either the recall or recognition of noises during the night. Therefore, in this instance, the fourth hypothesis must be rejected. Again, this is unsurprising considering this sample were all good sleeping individuals. However, this does suggest that, there may be two distinct processes surrounding the effect of cortical arousal: one resulting in sleep misperception and one involving the memory processing of environmental stimuli.

4.4.5 Limitations of this study and future considerations

The key limitation of this study is that it only focussed on analysis of sleep continuity and sleep architecture. In the first instance, it would be preferential to use quantitative EEG analysis on the data to investigate the role of high frequency EEG in the misperception of sleep in the face of noise, as this would allow quantification of cortical arousal. Although vulnerability factors pertaining to cortical arousal were assessed, the Neurocognitive Model specifies that it is the presence of high frequency EEG that

results in an increase in sensory processing of stimuli occurring during sleep onset and sleep.

Secondly, future research should examine sleep microstructure, especially EEG elements that occur within the sleep onset period or the transition to sleep after an awakening during the night, specifically sleep spindles and K complexes. Although it has been shown that there are no differences in the number of spindles or K complexes between individuals with psychophysiological insomnia and good sleepers (Bastien, St-Jean, Turcotte, Morin, Lavallee, & Carrier, 2009; Bastien, St-Jean, Turcotte, Morin, Lavallee, Carrier, et al., 2009), it has been suggested that when evoked by a stimulus, rather than a spontaneous sleep event, spindles and/or K complexes may have a role in the protection of sleep from arousals. For example, K complexes are suggested to have a protective mechanism, shielding and ensuring sleep continuation in the face of a potential awakening stimulus (Forget, Morin, & Bastien, 2011; Hairston et al., 2010; Halasz, 2005; Jahnke et al., 2012). This analysis could have the potential to explain why there were no significant associations between memory recall/recognition of the noises and the discrepancy between subjective and objective sleep, as the evoking of these mechanisms could be considered to be responding as a product sleep plasticity afforded by the good sleepers. Therefore, in light of the current lack of findings regarding memory processing of the auditory stimuli, one would expect the PSG to show increased numbers of evoked K complexes as the good sleepers were gating to the stimuli presented during the night. Sleep spindles have also been shown to act as a sleep stability mechanism, ensuring sleep continues in the face of a noisy sleep environment (Dang-Vu et al., 2010). Again, in light of the current findings, one may expect to see a higher number of spindles during the night occurring proximally to when a noise stimulus is presented during sleep, thus acting similar to the K complex by protecting the continuity of sleep.

As Fichten and Libman's (1991) model of insomnia in the elderly suggests, sleep fragmentation may be causal in the misperception of sleep. However, within this present study, although there was a disparity between subjective and objective sleep measures (SOL in particular) following the experimental night, no significant difference was found regarding the number of sleep stage changes or awakenings from the baseline night to the experimental night. Although the Cyclic Alternating Pattern (CAP) has been a known element of sleep, its use as a marker of sleep instability is reported by some to be a currently underutilized sleep measure within current literature (Parrino, Ferri, Bruni, & Terzano, 2012). Parrino and colleagues (2012) proposed that CAPs could be used as markers of sleep instability as they constitute minute changes in the pattern of the EEG independent of the transitions between sleep stages. Furthermore, the cyclic alternating pattern has been shown to correlate with subjective perception of sleep and thus may be far more sensitive as a measure of sleep instability than conventional stage changes, especially with regards to sleep state misperception in insomnia (Parrino et al., 2012; Parrino, Milioli, De Paolis, Grassi, & Terzano, 2009). So again, it would be expected that within this current study, although there was minimal disturbance to subjective and objective sleep individual, there was a misperception of sleep and therefore micro-structure analysis of this study may indicate a higher number of CAPs, particularly in stages N1 as Parrino et al. (2009) suggest and which was the stage that significantly increased on the experimental night.

However, the novelty of this present study determined that, in the first instance, basic measures of sleep analysis should be conducted which has, indeed, promoted various channels for future direction of study.

4.4.6 Conclusions

It is apparent from the results that a non-habituated environment may remove protection of sleep from noise perturbations in good sleepers resulting in increased cortical arousal. Furthermore, increased cortical arousal may be sufficient to result in

the alterations to sleep perception and memory processing as proposed in the Neurocognitive Model.

Along with disruptions to subjective and objective sleep continuity, noise results in alterations to NREM sleep, supporting the findings from Chapter 3. Specifically, there were alterations in sleep architecture, through an increase in wake and stage 1. This indicates that the whole NREM period may not be susceptible to cortical arousal, but rather limited to wake to sleep transition points including the sleep onset period. Beta EEG activity has been shown to be present prior to stage 2 sleep onset in those with insomnia that experience difficulties initiating sleep (Freedman, 1986; Perlis, Merica, et al., 2001). In good sleepers high frequency EEG occurs mainly in stage 1 and REM sleep albeit at lower relative power than found in those with insomnia (Perlis, Kehr, et al., 2001). The Neurocognitive Model suggests that it is at the wake to sleep transitions during NREM sleep as well as the initial sleep onset period where cortical arousal is at its highest and it is during this period that cognitive alterations occur. Increased beta activity (as a marker of cortical arousal) is said to persist into stage 2 sleep (Spiegelhalder et al., 2012) and that the rate of change between beta and delta EEG activity during wake to sleep transitions is lower in those with insomnia suggesting a prolonged arousal state (Merica & Gaillard, 1992). Therefore, individuals with insomnia may process noise during the night specifically during wake and stage 1 where higher frequency EEG activity is present.

The increase in stage 1 sleep and wake as a response to the presence of the noise may explain the ability to recognise some of the noises presented during the night (albeit minimal), but that levels of cortical arousal were not sufficient to result in an altered perception of sleep across all measures of sleep continuity. Therefore, one could infer that the acute nature of the arousal may have only marginally altered sleep and it was not sufficient to disrupt the good sleepers inherent sleep protection mechanisms (impair sleep plasticity). From a clinical perspective, this study indicated

that vulnerable good sleepers may be able to experience a disparity between subjective and objective sleep if sleeping in a noisy, unfamiliar sleep environment (i.e. doctors on call). As Ustinov (2010) reported, self-reported detriments to sleep associate with self-reported detriments to daytime function following sleep. Therefore, this may indicate a potential vulnerability to developing insomnia, as under Harveys' (2002) cognitive model, a perceived detriment to sleep and daytime functioning can result in a change in behaviour which in turn impairs sleep. Furthermore, the ability to recognise stimuli that has occurred during sleep may result in an attentional bias to said auditory stimuli during the day, which may result in increased sleep effort for the following night of which, as in Espies (2002) psychobiological model), may result in a failure of the plasticity surrounding sleep (ability to habituate) and thus an objective disruption to sleep occurs on subsequent nights. Therefore, when presented with a patient who is experiencing severe subjective sleep consistent with that described in the ICSD-2, a treating physician may wish to investigate the bedroom environment of the patient, addressing the issue of noise and cortical arousal directly, along with pharmaceutical and psychological treatments for insomnia.

The effect of cortical arousal is to be explored in the following chapter whereby natural awakenings and the subsequent sleep onset periods during NREM are targeted with auditory stimuli. Furthermore, examination of both explicit and implicit memory will be examined between good sleepers and those with insomnia in a non-habituated environment (sleep laboratory). Furthermore, it could be that although explicit memory is not occurring, participants may be implicitly remembering a disturbance to sleep although not able to explicitly recall the disturbance, and this creates the disparity seen within this study between the subjective and objective sleep onset, a point which could not be examined within this chapter. This is suggested to be the case by Perlis and colleagues (1997) in individuals with fibromyalgia, as although they could not explicitly recall words administered to them during the night, they did still experience alterations

to both subjective and objective sleep. The following chapter will draw on the results from both this Chapter and Chapter 3, to empirically test the Neurocognitive Model.

Chapter 5: Determining Explicit and Implicit Memory Processing in Insomnia

5.0 Introduction

The Neurocognitive Model of insomnia states that once the insomnia threshold has been met (Spielman, 1986), perpetuating factors, such as an extension of time in bed, act to maintain the insomnia disorder, resulting in persistence and chronicity of the sleep problem. One mechanism by which this may occur is through an increase in arousal prior to sleep, as lying in bed awake and ruminating about the previous or next day, can result in conditioned arousal to the bedroom environment. This conditioned arousal involves both cognitive and somatic processes, which interact creating increased cortical arousal. It is this increased cortical arousal, as identified through the presence of high frequency EEG, that alters the perception of sleep through three processes of neurocognitive alteration (short term memory formation, long term memory formation, and sensory gating impairments) (Perlis, Giles, Mendelson, et al., 1997).

It is suggested that individuals employ more attentional resources to stimuli which are perceived as threats to the sleep process (Wicklow & Espie, 2000), therefore sleep in a noisy environment may actually result in an increase in cortical arousal. Moreover, individuals with insomnia may have difficulty in maintaining sleep in the face of environmental perturbations due to insufficiencies in the sensory gating mechanism (as determined through low amplitude or reduced numbers of K complexes - a marker of stage 2 sleep, which occur spontaneously, and can be evoked by noise) (Hairston et al., 2010; Jahnke et al., 2012; Milner et al., 2009). As auditory information is processed by the brain, memories for environmental stimuli may occur during wake to sleep transitions consequently resulting in difficulties perceiving sleep “as sleep”.

This study is designed to examine whether short and long-term memory formation occur during sleep in those with insomnia compared to normal sleepers as a product of the presence of heightened cortical arousal through a non-habituated environment. In the context of the present research, short-term memory formation refers to the correct perception of being awake following a brief nocturnal awakening (2 epochs), whereas long term memory refers to the ability to recall or recognise stimuli that were presented during sleep upon awakening the following morning. It has been suggested that individuals with insomnia perceive sleep onset later than good sleepers, requiring 15 minutes of consolidated stage two sleep prior to a nocturnal awakening for an accurate perception of sleep state (Hauri & Olmstead, 1983). Furthermore, when a disparity is formed between subjective and objective sleep, in good sleepers, this is usually accompanied by reports of sensations and awareness of the external environment (Weigand et al., 2007). This raises the question of how does one know when they are asleep, and could perception of sleep be dependent on an absence of a memory of sensory stimuli or a period of amnesia.

Due to the potential sensory gating impairment through increased cortical arousal, encoding of environmental stimuli occurring during the night may arise and thus a transfer of the stimuli from short to long term memory. The Neurocognitive Model proposes that people with insomnia are able to recall these memories upon awakening which negatively biases the perception of the previous night's sleep continuity. Evidence indicates that, when presented with auditory stimuli (word nouns) during wake to sleep transitions (i.e. sleep onset and at three forced NREM awakenings), those with insomnia correctly recall more word stimuli than good sleepers (Perlis, Smith, Orff, et al., 2001), suggestive of increased memory formation during the sleep onset period when there is endogenously higher cortical arousal resulting in a failure of amnesia to stimuli at sleep onset. As introduced within Chapter 4, the ability to recall stimuli presented during sleep onset periods after a brief period of sleep has been

shown to potentially associate with cortical arousal, as inferred through the presence of increased beta EEG activity during the sleep period following the stimuli presentation (Wyatt et al., 1997). However, these all require the ability to explicitly recall or recognise the information presented during the night when tested the following morning. Specifically, the memory tasks within the study in Chapter 4 and as performed by Perlis et al. (2001), required participants to be able to retrieve the memory of the auditory stimuli that was presented during sleep/sleep onset and then write this information down without prompting. Secondly, both those studies tested explicit recognition, which is being able to identify the auditory stimuli once cued, be that through auditory cues (as in Perlis et al. (2001)) or visual cues (card sorting task within Chapter 4). These tasks measure explicit memory through episodic memory, that is the ability to recall or recognise stimuli in relation to time ("during my last sleep period"). Being able to recall and or recognise ,following cues, auditory stimuli that occurred during the night, even if one was asleep, could alter the perception of sleep "as sleep".

However, there is another type of memory that humans are able to utilise that involves the unconscious ability to retrieve memories: implicit memory. Implicit memory cannot be brought into conscious recall or recognition, as was tested in Chapter 4, but can be acted upon by an individual: the ability to ride a bike is evidence of implicit procedural memory. It has also been shown within individuals with fibromyalgia, using the same task described by Perlis et al. (2001), that even when words were not correctly recalled explicitly there was still a disparity between subjective and objective sleep (Perlis, Giles, Bootzin, et al., 1997), as was also indicated in Chapter 4. Perlis and colleagues (1997) suggest this might represent implicit memory recognition which consequently skewed the perception of sleep. Implicit memory can be assessed by embedding the auditory stimuli in noise that is louder than the stimuli to be tested or using white noise of the same loudness (Pilotti, Bergman, Gallo, Sommers, & Roediger, 2000). This Chapter aims to expand upon previous work by examining not only explicit memory to

auditory stimuli during the night, but also implicit memory. As it is suggested that those individuals with insomnia supply more attentional resources to potential disrupters of sleep, as examined by Wicklow and Espie (2000), environmental noise is one of the most frequently reported sleep disrupters by individuals with insomnia. The study within the paper by Wicklow and Espie (2000), employed immediate documentation of disrupting stimuli as the individuals were falling asleep, however, if the individuals were then able to identify the same disrupters on awakening, this would be evidence of explicit episodic memory. From a clinical perspective, this would be easy for the treating clinician to identify as, if through the propositions of the Neurocognitive Model, the disruption to sleep (subjectively) would be accompanied with a report of disturbance by external stimuli. If implicit memory is also found to associate with the creation of a disparity between the subjective report of sleep and what occurs objectively, this would highlight the need for treating clinicians to examine the sleeping environment for external stimuli without prior prompting from the patient. Furthermore, the presence of implicit memory formation would provide a link between the processing of auditory stimuli, due to cortical arousal (high frequency EEG activity) and sleep state misperception, of which no link was found in Chapter 4 (using explicit memory).

This study aims to change the external stimuli used to examine the Neurocognitive Model from environmental noise to words (nouns), along with altering the point at which stimuli is administered from NREM and REM sleep to administration only during wake to sleep transitions. As Chapter 4 found minimal recall and recognition of the environmental sounds occurring during sleep, along with minimal disruption to slow wave sleep and REM, yet an increase in the amount of wake and stage 1, this chapter will examine the proposition of the Neurocognitive Model that it is sleep onset periods and lighter sleep (S1) where increased sensory processing is occurring resulting in memory formation. Moreover, Chapter 5 will use nouns presented to the participants which will have to be repeated back to the investigator immediately on presentation

during the sleep onset periods, thus ensuring the stimuli has been attended to. This will allow a direct investigation of memory encoding occurring during sleep onset and natural awakenings from NREM sleep as a product of increased cortical arousal comparing good sleepers and those with insomnia in a non-habituated environment. Furthermore, this chapter will seek to examine whether apparent memory to stimuli during the night relates to sleep state misperception as the Neurocognitive Model proposes. This study will not only seek to test the propositions of the Neurocognitive Model, evaluating the aspects that are said to lead to insomnia through increased cortical arousal, but to also seek to determine whether good sleepers may also be able to experience neurocognitive alterations if they are at increased cortical arousal during the sleep period.

Therefore, within this chapter, four questions were proposed with the associated hypotheses tested within this chapter:

1. Firstly, before the Neurocognitive Model can be examined with regards to cortical arousal and memory, the degree of the disparity between subjective and objective sleep measures needs to be assessed between good sleepers and those with insomnia. As the outcome disruption to sleep proposed by the Neurocognitive Model of insomnia is the generation of sleep state misperception, if good sleepers also experience sleep state misperception in the presence of environmental noise (as suggested in light of findings from Chapter 4) then the Neurocognitive Model may actually provide evidence for the potential development of insomnia through negative appraisal of sleep (a falsely perceived sleep detriment) in vulnerable good sleepers in a non-habituated environment(in light of findings from Chapter 3). However, as current literature dictates, it is hypothesised that people with insomnia will experience a greater degree of sleep state misperception comparative to good sleepers in this present study;

2. Short term memory processing is said to be the first cognitive alteration to occur as a product of heightened cortical arousal. The Neurocognitive Model states that, due to processing of external stimuli during sleep wake to sleep transitions, there will be an inability to accurately perceive sleep state if questioned during an awakening. This present study questions whether there is a difference between good sleepers and those with insomnia on sleep perception (short-term memory) following a natural awakening from NREM sleep (as determined by 60 seconds of wake). It is hypothesis that, when questioned during natural awakenings from NREM sleep, individuals with insomnia will perceive themselves to have been awake prior to the arousal more often than good sleepers;
3. The second cognitive alteration is that there will be long term memory formation to the stimuli presented during these wake to sleep transitions. Therefore, the study within this chapter will aim to examine whether, if noises (words) are administered during wake to sleep transitions in NREM, is there a difference between good sleepers and those with insomnia on measures of explicit and implicit memory recognition for the noises (long term memory)? Primarily examining three potential outcomes:

    3a. Is there a difference between good sleepers and individuals with insomnia regarding explicit memory formation of the words presented during the night: it is hypothesised that individuals with insomnia will explicitly recall a higher percentage of the words presented during the sleep period than good sleepers along with being able to explicitly recognise a higher percentage of words that were administered during the night then foils in comparison to good sleepers;

    3b. Is there a difference between good sleepers and individuals with insomnia regarding implicit memory formation of the words presented during the night: it is hypothesised that individuals with insomnia will be

able to recognise (implicitly) a higher percentage of words that were administered during the night then foils in comparison to good sleepers;

3c. Finally, as this study will employ four testing points during the sleep period, sleep onset and three awakenings from NREM sleep, are there differences in memory formation dependent on when the words were administered during the sleep period: it is hypothesised that individuals with insomnia will be able to recall and recognise a higher number of words presented at each time point comparative to good sleepers.

4. Finally, the Neurocognitive Model proposes that as a result of enhanced cortical arousal and memory formation of stimuli occurring during the sleep period, the perception of sleep is altered resulting in a misperception between subjective and objective sleep continuity. This present study seeks to test whether the ability to recall or recognise the words presented during the four trials associates with a disparity between subjective and objective sleep: it is hypothesised that the percentage of words that an individual is able to recognise, be that explicitly or implicitly, from the stimuli presented during the night will associate with the degree of sleep state misperception experienced regardless of whether they are good sleepers or individuals with insomnia, indicating a direct effect of increased cortical arousal.

5.1 Methodology

5.1.1 Design

To identify memory processing, both implicit and explicit, as a result of cortical arousal during sleep onset and wake to sleep transition points during the night, a two night in-laboratory protocol was implemented. Using a between groups design, good sleepers and people with insomnia were examined for differences in sleep measures (including a discrepancy between subjective and objective sleep), and measures of explicit and implicit memory.

5.1.2 Participants

An opportunity sample of participants was obtained from Northumbria University (UK) using word of mouth, poster, and internal email advertising. Using a combination of a screening questionnaire, as first used in Chapter 3, and clinical interview using the DSM-V criteria for insomnia disorder (American Psychiatric Association, 2013)(see Chapter 1 section 1.1.2) exclusion was made at the initial screening stage if participants, other than those with insomnia, reported any sleep disorder, had a current or prior complaint of anxiety or depression, had a current medical problem, or were taking medication deemed to interfere with sleep. Exclusions were also made if there was a history of severe head injury, hearing disorder, or if employment was in a sector that required rotating shift work. Participants were required to have adequate verbal memory recall skills and normal hearing respective to age, assessed using the California Verbal Learning Task (Delis, Kramer, Kaplan, & Ober, 1987) and an online hearing test (Digital Recordings, 2012). Additionally, good sleepers were required to have an average SOL and a WASO of no more than 15 minutes (as determined through a seven-day sleep diary) along with no daytime complaints that could be associated with insomnia.

Participants in the insomnia group were required to have insomnia as defined by the DSM-V for insomnia disorder, see Chapter 1, and to report at least one daytime consequence of insomnia, as assessed by clinical interview. Moreover, participants with insomnia were required to have a subjective SOL and/or WASO of greater than 30 minutes, and a TST of less than 6 hours occurring for at least 3 nights per week over at least 6 months (Lichstein et al., 2003). Moreover, as the Neurocognitive Model suggests that extension of time in bed is a driving factor in the development of conditioned arousal, individuals in the insomnia group were required to have a subjective SE from a baseline, one week, sleep diary of less than 85% - indicative of excessive time in bed awake. It was decided that individuals with potential idiopathic

insomnia (participants who indicated difficulties sleeping since childhood) should be excluded as this subtype of insomnia is said to be of a genetic or neurological origin and not due to a conditioned arousal response (American Academy of Sleep Medicine, 2005; Edinger et al., 2004) .

Each group (good sleeper or insomnia) consisted of five individuals from consecutive age decades: 20-29, 30-39, 40-49, 50-59, and 60-69 years of age, with one participant in each decade. Overall, there were five males and five females that took part in the study. In the good sleeper group, there were three males and two females, and two males and three females in the insomnia group. Mean age of the participants was 44 years (*SD* 14.56) with an age range of 23 to 61 years of age. There was no significant difference in age between groups ($t(8)=0.041$, $p=0.968$), the average age of the good sleepers was 44.2 years (*SD* 15.42) and 43.8 years (*SD* 15.42) for individuals with insomnia. The average duration of insomnia within the insomnia group was 7.8 years (*SD* 7.56), ranging from 2 to 20 years.

All participants in this study were regular consumers of alcohol but were required to abstain during the study period. Of the 10 participants, four stated that they currently smoked but were not allowed to do so over the study period. When questioned about recreational drug use, participants had not partaken in recreational drug use for at least 6 months (cannabis) prior to the study. Of the participants, all but two were employed in full time work. Of the two that were unemployed, both were retired but undertaking part time work. All participants had completed at least 20 years of education (educated to a UK undergraduate degree level).

5.1.2.1 Consent and Ethical Considerations

This study obtained ethical approval from the Faculty of Health and Life Sciences ethics committee at Northumbria University. Prior to partaking in the research, all participants had to indicate their informed consent for both the general study and for

the overnight video monitoring. Additionally, participants signed a disclosure form, confirming acknowledgement of the statement that whilst on the study, the Northumbria Centre for Sleep Research requested they do not drive or undertake any activities that may become dangerous if drowsy. Participant remuneration was £100 for the study on a pro rata basis over the two nights spent at the sleep laboratory.

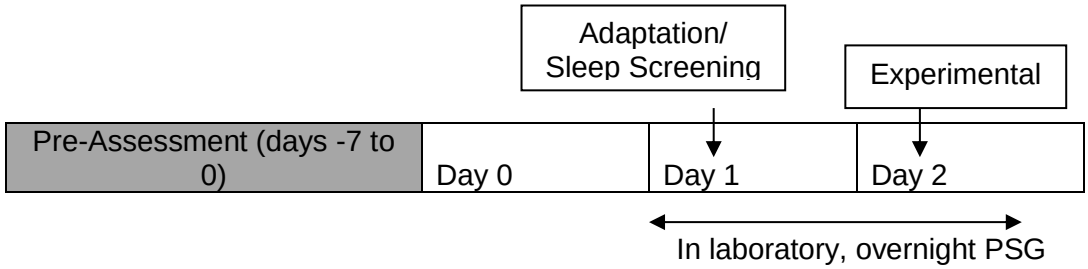
5.1.3 Procedure

Following consent and screening, the protocol consisted of one week of baseline sleep monitoring (sleep diaries and actigraphy) followed by two nights of PSG recordings (1st night screening and adaptation) within the sleep laboratory (see Figure 5.1). Unlike the study in Chapter 4, there was no baseline night (only an adaptation night) as the interest of this study was in difference between groups. Participants were advised to keep a stable sleep wake pattern across working days, ensuring that they went to bed and rose at similar times. Bedtimes and wake times for the study were calculated using the five week days of the baseline sleep diary. Weekends were not used in the determination of habitual times for the study and all experimental laboratory nights were conducted on weekdays. Following the baseline period, participants returned to the Northumbria Centre for Sleep Research for overnight PSG studies.

For each experimental night, participants were advised not to consume alcohol, nap at any point during the overnight study period, and to refrain from caffeinated substances from 6pm onwards. All participants arrived at the unit at 9pm on both study nights and left approximately 90 minutes after habitual wake time on the adaptation night and after testing on the final morning. During the overnight study period, prior to sleep participants were free to read or watch television once the California Verbal Learning Task (CVLT) and the hearing test were complete (see sections 5.1.4.5 and 5.1.4.6). An hour prior to habitual bedtime, participants were advised to get ready for bed, following which electrodes were applied to the scalp and face with the addition of electrodes placed on the body for the first night only to allow screening for potential secondary

sleep disorders that could be causing the subjective report of insomnia. Participants wore in-ear headphones on all nights to allow adaptation to sleeping with headphones in. Once in bed, and bio-calibrations (as in section 4.1.3) had been completed, participants were requested to sleep and not to sit in bed and read. Mobile phones were left outside of the bedroom during the overnight study. The recordings were conducted in noise-attenuated rooms, with blackout blinds to ensure minimal light intrusion (room at <5 Lux). On both post study mornings a post sleep questionnaire was administered and participants continued to wear the actiwatch throughout the study to ensure adherence to the protocol concerning abstaining from daytime naps when away from the laboratory.

Figure 5.1: Schematic of the timeline of the study.



Assessment Schedule- Day	**-7**	**-6**	**-5**	**-4**	**-3**	**-2**	**-1**	**0**	**1**	**2**
Experimental Consent										
Sleep Screening questionnaire and insomnia assessment										
Standard Sleep Dairy										
Actigraphy (1 min epochs)										
Polysomnography screening montage										
CVLT										
Hearing Test										
Volume Calibration										
Polysomnography (reduced montage)										
Post sleep questionnaire										
Word stimuli administration										
Word recall task										
Word recognition task										
Word recognition (with white noise)										
Experimental Debrief										

5.1.3.1 Experimental Protocol

Using a computer program (Csleep) words were presented to the participants via in-ear bud style headphones (manufactured by Sound MAGIC), held within the ear by surgical tape, as described in Chapter 4. This was connected to a marker system allowing a connection between both the test administration computer and the PSG recording device, facilitating comparative viewing of sound presentation and the EEG. Prior to sleep, a volume bio-calibration (automated from Csleep) was conducted to ensure the words were at an appropriate level for participants to comfortably hear, recognise, and repeat the word stimuli.

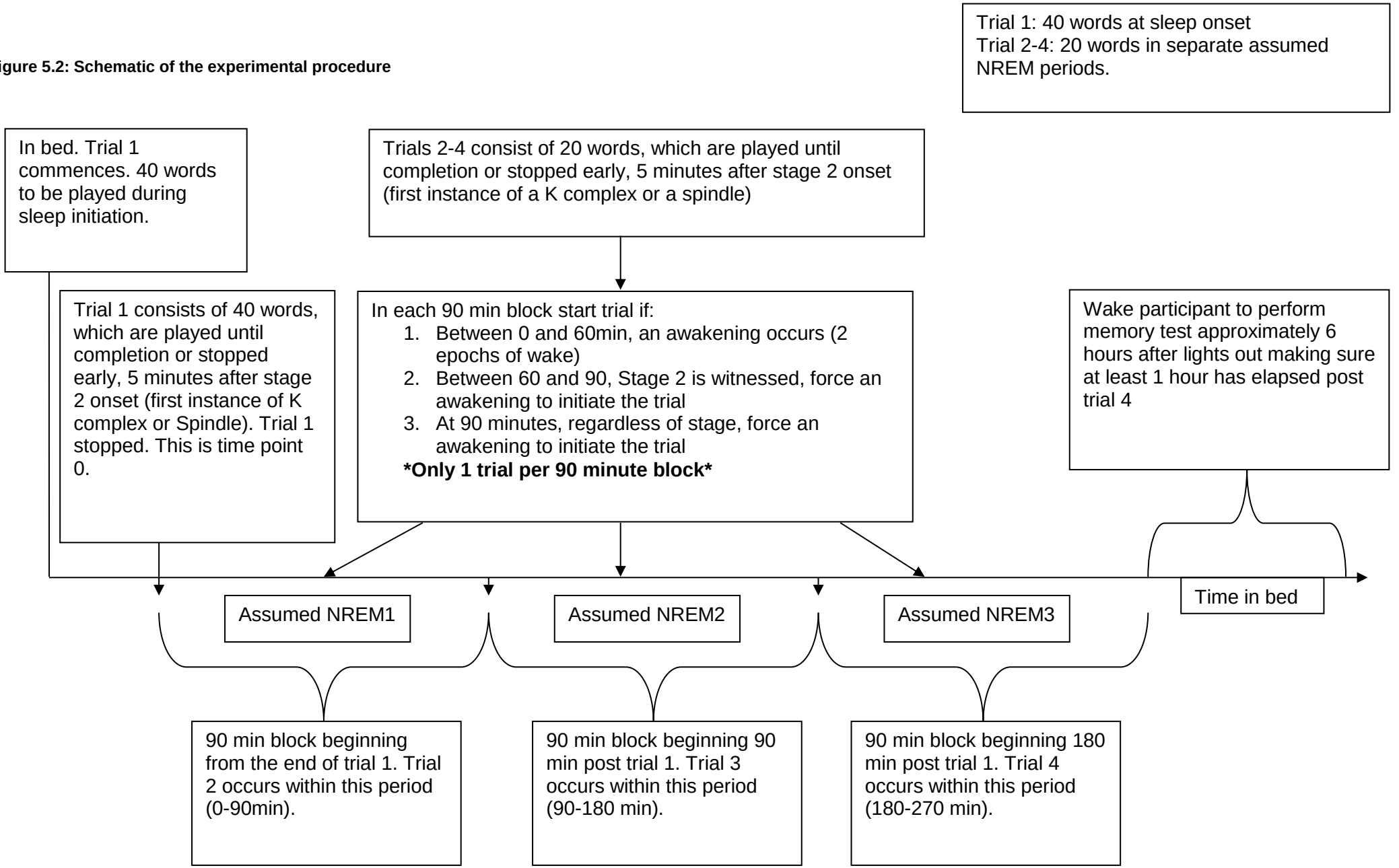
Each trial was initiated with an alarm (an auditory tone) which increased in loudness. Upon hearing, participants were instructed to sit up in bed and acknowledge that they were awake by stating, “I heard it”. Following this, the tone ceased and the participants were asked whether they were awake or asleep prior to tone administration. Upon their response, participants were then instructed to attempt to sleep. During this wake to sleep transition period, words were administered at one-minute intervals with participants instructed to say each word aloud as they heard it with their response documented within the Csleep program. Only words that had been repeated correctly during the trials were included in the analysis as these words were said to be representative of encoding, as words must have been attended to and moved through working memory as the participant attempts to repeat the word. The trial continued until either the maximum number of words was presented for that trial (40 for trial 1 and 20 for each subsequent trial) or after five minutes of N2 sleep was observed. Figure 5.2 shows a full schematic of the experimental procedure.

Trial 1 commenced at lights out. On completion of the trial (following administration of all trial words or the onset of sleep), the sleep period was then segmented into 90-

minute blocks representative of sleep cycle (NREM and REM) periods. Within each of these periods, only one trial could be initiated. The three trials subsequent to initial sleep onset were administered on the appearance of 2 epochs (60 seconds) of wake. However, if wake did not appear within the first 60 minutes of the block, then a trial was initiated from a forced awakening from stage 2 sleep using the tone present at the start of each trial. If these two conditions were not fulfilled (i.e. 2 epochs of wake or forced awakening from stage 2 sleep), after the full 90-minute trial period had elapsed initiation of the trial occurred from a forced awakening regardless of stage. Again the forced awakening was conducted using the tone present at the beginning of each trial, this increased in loudness until the participant indicated they were awake. This was to ensure that all four trials were completed within the sleep period.

To ensure comparable sleep opportunity between the good sleeper and insomnia groups, all participants were awoken 6 hours post completion of trial 1, ensuring that at least 1 hour of uninterrupted sleep followed the completion of trial 4 (the final trial). Therefore sleep opportunity for the good sleeper group was curtailed to approximately 6.5hrs. The good sleepers were aware that their sleep on the experimental night could become shorter than usual but were not made of the exact sleep duration. Upon scheduled wake time, a technician entered the room to wake the participant and administer the post sleep questionnaire. Following completion of the questionnaire, the participant was able to use the bathroom and then returned to bed. Once in a semi-recumbent position the morning testing procedure was initiated. The testing procedure involved three tasks (free recall (explicit memory), recognition (explicit memory), and white noise embedded recognition (implicit memory) that were to be completed consecutively without interruption. Instructions to participants were automated and delivered through the Csleep programme. Once testing was completed on the final morning, participants were fully debriefed and allowed to leave.

Figure 5.2: Schematic of the experimental procedure



5.1.4 Apparatus and Materials

5.1.4.1 Sleep Diary

A standard sleep diary was used for one week prior to the study to ascertain baseline sleep measures and habitual wake and bed times (see Chapter 3 section 3.1.4.2). Variables of interest from the sleep diary were those pertaining to sleep continuity (SOL, NWAK, WASO, TST, and SE).

5.1.4.2 Actigraphy

Wrist based actigraphy (Cambridge Neurotechnology) was used as per the baseline period of Chapter 3, and Chapter 4. Actigraphy acted as an objective sleep screening measure prior to induction to the study, to corroborate reported sleep and wake times from the sleep diary during the baseline period, and to ensure compliance during the day whilst not in the laboratory on the study nights.

5.1.4.3 Polysomnography

Polysomnography was recorded for the experimental nights of the study. As in Chapter 4, the montage for all nights included ten scalp electrode sites (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1 and O2) and two EOG's (EOG_L and EOG_R), all referenced to mastoid M1 (M2 in the case of signal loss on M1). In addition, EMG electrodes were placed on the chin to the American Academy of Sleep Medicine (AASM) guidelines (bipolar submentalis muscle referenced to the mentalis muscle) and a two lead ECG placed bilaterally on the chest (one electrode positioned below midpoint of the clavicle on the right side and one positioned on the left second to last intercostal space). Additionally, a trachea microphone was taped to the neck of the participant to monitor for snoring on the screening night and then as a recording device for spoken word responses during the experimental task.

Electrode application was completed using GRASS 30” Gold Disk Electrodes (F-E5GH), fixed with 10-20 conductive paste (Weaver). Monitoring of the electrodes ensured impedance was kept below 5Ω. Acquisition of the data was via the S-Med wireless PSG+ system, digitized and viewed in real time to a DELL (19”x11”) monitor, as well as saved to an internal flash card system. Recordings were blind-scored off site by a registered technician according to AASM guidelines.

Full sleep screening was completed on the adaptation night with electrodes placed on the left and right anterior tibialis muscle to screen for restless leg syndrome or periodic leg movement disorder. To screen for sleep-related breathing disorders, plethysmography was conducted through effort bands to monitor chest movement, pulse oximetry placed on the right index finger for blood oxygenation levels, and a nasal thermistor placed under the nose to measure airflow rate during the night. This was done in addition to the procedures in Chapter 4 due to the inclusion of participants with a sleep disorder (insomnia) and to ensure the participants in this group did not have a disturbance to sleep through a secondary cause, such as sleep apnoea. Additionally, on the second night, administration of the words was viewed in real-time on the EEG recording using a marker system connected to the participant via the PSG recording device. From the PSG, measures pertaining to sleep continuity were gathered (SOL, WASO, TST, and SE) along with measures of sleep architecture (%N1, %N2, %N3, and % REM).

5.1.4.4 Post Sleep Questionnaire

As first used within Chapter 4, participants were administered a brief questionnaire each morning upon awakening to document subjective sleep continuity (SOL, NWAK, WASO, TST, and SE) were used within this present study.

5.1.4.5 California Verbal Learning Task

To ensure all participants had the ability to encode and process verbal stimuli, the short form of the California Verbal Learning Task (CVLT) was administered (Delis et al., 1987). The CVLT is a psychiatry approved test that measures the ability to remember and encode a series of verbally presented words immediately, after repeated presentations, after an interference of a confounding set of words, and after a short (20 minute) delay. This study did not require any formal scoring, but ensured that participants were able to at least partially recall verbally presented material after a short delay, at the researcher's' discretion.

5.1.4.6 Hearing Test

As the experimental task consisted of word stimuli played at a comfortable hearing threshold, participants were screened to ensure adequate hearing ability. This was completed using an online hearing test and the earphones to be used during the study (Digital Recordings, 2012). The program generated a standard curve of hearing ability to different frequencies. The participants hearing ability needed to approximate to these frequencies to be included in the study.

5.1.4.7 Word Administration Program

The word administration program for this study was supplied by the University of Pennsylvania, and adhered to the guidelines as documented in Perlis et al. (2001). This program is referred to as Csleep within the present study. The Csleep program contains a range of 100 words to be presented along with a series of 100 foils. Words were matched for usage frequency (in the USA dictionary), and duration (see appendix F for complete list of words). This program also administered standardised participant instructions regarding the experiment during the night and during the word recall and recognition tasks the following morning. All words and instructions were presented by a female voice (mild American accent).

Up to 100 words could be administered during the night with a possible 40 words administered at trial 1 (sleep onset) and 20 words administered during each of the three subsequent wake to sleep transition points in NREM sleep. Presentation order was randomised between participants.

5.1.4.7.1 Word Free Recall

The first task was a free recall of the words presented during the night. Participants were instructed to document, on paper, as many words as they could remember being played to them during the four trials. At the end of the testing period, participants were instructed to read the list of recalled words aloud which were input into the Csleep program by the technician administrating the task.

5.1.4.7.2 Word Recognition

Once participants stated that they had documented all words they could remember instructions for the second task were administered. All word stimuli that were presented during the four trials along with an equal number of foils were presented in a random order determined automatically by the program. After each word was presented participants were instructed to respond, using a button box, whether they recognised the word as being presented during the night (yes or no).

5.1.4.7.3 Implicit Word Recognition (White Noise Embedded)

After the explicit word recognition task, instructions began for the implicit recognition task. This task involved the presentation of words not recognised during the previous task (both words played during the four trials and foils) embedded within 30% white noise. Participants were presented with the white noise and asked to say aloud the word that they thought was played.

This study aimed to use a novel method to examine implicit memory. Implicit memory was assessed using a modification of the widely used noise embedded method (Pilotti et al., 2000). By presenting words that were played during the night, but not explicitly

recognised, it can be inferred that when an individual is able to recognise significantly more white noise embedded words that were presented during the night than foils, implicit memory may have occurred as both the presented words and foils are equally difficult to recognise within the white noise.

5.2 Statistical Analysis

All statistical analysis was completed using SPSS for windows version 20. Univariate ANOVAs were used to identify significant differences between groups on measures of sleep, both subjective and objective. Correction for type 1 errors was accounted for using a bonferroni adjustment. Paired and independent t-tests were used when only a single dependent variable was assessed between groups. Repeated measures ANOVAs were used to examine if there were differences between and within groups on recognition of words across the four trials. Bivariate Pearson correlations were calculated to investigate relationships between sleep parameters and recall/recognition of the word stimuli for both the good sleepers and those with insomnia separately.

Words presented during the night were only included in the analysis if there was evidence that they were heard by the participant during the night, as determined by the correct repetition of the word back within the trials. This was to ensure that all words had been attended to and, due to the repetition of the word, been processed partially into memory. Only the words that were attended to (correctly repeated back) during the night were used in the analysis of the explicit memory task. Analysis of the implicit (white noise) task used the same principles for the explicit memory task but analysis was conducted on only words that were not recognised during the explicit recognition task, including foils. This is on the assumption that, although priming will have taken place during the explicit task, this will have been equal for both the words that were presented during the night and foils, therefore if an individual is able to identify more of the words that were presented during the night than foils, implicit memory may have occurred as these were words explicitly recalled or recognised.

5.2.1 Missing Data

Data was visually checked for normality and missing data points. Due to the nature of this study requiring correlational analyses, missing random data points were left absent. This decision was made upon the basis that, although removing the affected data point reduces power, it does not increase risk of an inflated error rate, which has been shown to occur with substitution methods in correlation analysis (Schafer & Graham, 2002). Pair wise deletion was applied within the t-test analyses. Missing data included one participant from the insomnia group who did not complete the white noise part of the experimental memory task due to computer failure. Additionally, one participant from the good sleep group did not complete a question on the post sleep diary pertaining to WASO on the morning following the experimental night.

5.3 Results

5.3.1 Baseline Characteristics of the Participants- Sleep Diary

Although only weeknights were used from the sleep diary to ascertain habitual sleep/wake times, the whole week of baseline sleep data was analysed to ascertain either good sleeper status or the presence of insomnia.

Due to the expected variability in sleep continuity measures between good sleepers and people with insomnia, univariate ANOVAs were used to follow up differences between groups on each of the dependent variables separately.

Before applying a bonferroni correction, data indicated that the insomnia group experienced, on average, a significantly longer SOL ($F(1,8)=7.91$, $p<0.05$, $\eta^2=0.497$), shorter TST ($F(1,8)=11.41$, $p<0.05$, $\eta^2=0.588$), and reduced SE ($F(1,8)=7.91$, $p<0.01$, $\eta^2=0.652$) than good sleepers (see Table 5.1 for means and standard deviations). Although not significant, there was a trend in WASO with the insomnia group reporting an average time of 56.66 minutes (*SD* 52.94) comparative to 4.43 minutes (*SD* 4.44) for the good sleep group ($F(1,8)=4.83$, $p=0.059$, $\eta^2=0.377$). There

was no significant difference between groups on the average NWAK over the baseline week. However, once a bonferroni adjustment was applied, reducing the significance criterion from 0.05 to 0.008 (0.05/6), only the difference in SE remained significant.

Table 5.1: Univariate ANOVAs between groups for the 7-day baseline sleep diary. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, TIB = Time in Bed, and SE = Sleep Efficiency

Measures of Subjective Sleep Continuity	Participant Group						
	Good sleepers		Insomnia				
	Mean	SD	Mean	SD	F	df	p
SOL (min)	10.37	2.67	42.60	25.49	7.91	1	0.023
NWAK	1.17	0.68	2.31	1.28	3.16	1	0.114
WASO (min)	4.43	4.44	56.66	52.94	4.83	1	0.059
TST (min)	465.60	43.35	343.40	68.17	11.41	1	0.010
TIB (min)	504.60	60.24	512.40	50.45	0.05	1	0.823
SE	92.71	3.77	68.43	13.52	14.96	1	0.005

5.3.2 Differences between Groups on Sleep Measures during the Experimental night

Of the 30 trials across all 10 participants, only 6 (20%) were initiated using a forced awakening; the remainder were initiated upon the presence of 2 epochs of wake. Three of these forced awakenings occurred within the good sleeper group and three within the insomnia group.

5.3.2.1 Differences in Subjective Sleep

A series of univariate ANOVAs indicated there were no significant differences between groups on measures of subjective sleep continuity following the experimental night (Table 5.2).

Table 5.2: Univariate ANOVAs between groups for subjective sleep quantity during the experimental night. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, TIB = Time in Bed, and SE = Sleep Efficiency

Measures of Subjective Sleep Continuity	Participant Group						
	Good sleepers		Insomnia				
	Mean	SD	Mean	SD	F	df	p
SOL (min)	57.50	61.85	16.00	9.62	2.26	1	0.176
NWAK	3.13	1.03	2.80	1.48	0.14	1	0.722
WASO (min)	57.50	62.25	27.80	21.08	1.02	1	0.345
TST (min)	296.25	140.91	378.00	40.25	1.57	1	0.250
TIB (min)	390.80	11.99	385.20	2.05	0.39	1	0.554
SE	77.78	37.82	98.11	10.17	1.37	1	0.281

5.3.2.2 Differences in Objective Sleep

A series of ANOVAs indicated that there were no significant differences between groups on measures of objective sleep continuity or architecture on the experimental night, Table 5.3 and Table 5.4. However, means across the measures suggest that, although non-significant, the good sleeper group slept worse objectively than the insomnia group on the experimental night.

Table 5.3: Univariate ANOVAs between groups for objective sleep continuity during the experimental night. SOL = Sleep Onset Latency, NWAK = Number of Awakenings, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency

Measures of Objective Sleep Continuity	Participant Group						
	Good sleepers		Insomnia				
	Mean	SD	Mean	SD	F	df	p
SOL (min)	52.82	28.45	22.12	12.39	4.89	1	0.058
WASO (min)	128.40	78.04	94.96	27.42	0.82	1	0.392
TST (min)	307.00	79.86	345.90	23.14	1.10	1	0.326
SE	63.70	18.75	74.84	6.18	1.59	1	0.243

Table 5.4: Univariate ANOVAs between groups for sleep architecture during the experimental night. N1 = NREM stage 1, N2 = NREM stage 2, and N3 = NREM stage 3 (AASM scoring criteria)

Measures of Sleep Architecture	Participant Group						
	Good sleepers		Insomnia				
	Mean	SD	Mean	SD	F	df	p
Total % Wake	36.27	18.76	25.17	6.19	1.58	1	0.245
Total % N1	3.93	1.37	4.61	0.71	0.96	1	0.356
Total % N2	38.25	11.19	42.54	11.88	0.35	1	0.573
Total % N3	8.78	4.27	12.19	9.17	0.57	1	0.473
Total % REM	12.77	6.73	15.49	6.06	0.45	1	0.520

5.3.2.3 Differences in Sleep Misperception

5.3.2.3.1 Discrepancy between Subjective and Objective Sleep within Group on the experimental night

Firstly, to examine the discrepancy between experimental night subjective sleep (through the post sleep questionnaire) and objective sleep within each group, paired t-tests were conducted for each paired sleep measure, Table 5.5. In all instances, within

both groups, subjective sleep was perceived to be better than the objective sleep. The good sleeper group had a significant difference between subjective and objective wake after sleep onset, reporting a subjective WASO of 57.5 (*SD* 62.25) minutes compared to an objective WASO of 129.75 (*SD* 90.04) minutes, ($t(3)=4.561, p<0.05$). As for the insomnia group, there was also a significant difference in WASO with the subjective report of 27.8 (*SD* 21.80) minutes compared to 94.96 (*SD* 27.42) minutes via the PSG ($t(4)=12.262, p<0.01$), and a significantly better sleep efficiency (98.11%, *SD* 10.17) compared to the PSG (74.84%, *SD* 6.18) ($t(4)=6.611, p<0.01$). In addition, the insomnia group also subjectively reported a significantly longer total sleep time at 378 minutes (*SD* 40.25) than observed through PSG at 345.9 minutes (*SD* 23.14) ($t(4)=2.972, p<0.05$).

Table 5.5: Paired t-test within groups between measures of subjective (post sleep questionnaire) and objective sleep continuity on the experimental night. SOL = Sleep Onset Latency, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency

	Good Sleepers						
Sleep Continuity	**Subjective Mean**	**SD**	**Objective Mean**	**SD**	**t**	**df**	**p**
SOL (minutes)	55.00	53.85	52.82	28.45	0.168	4	0.875
WASO (minutes)	57.50	62.25	129.75	90.04	-4.561	**3**	**0.020**
TST (minutes)	321.00	134.00	307.00	79.86	0.495	4	0.646
SE	82.52	34.43	63.70	18.75	2.198	4	0.093
	Insomnia						
Sleep Continuity	**Subjective Mean**	**SD**	**Objective Mean**	**SD**	**t**	**df**	**P**
SOL (minutes)	16.00	9.62	22.12	12.39	-0.678	4	0.535
WASO (minutes)	27.80	21.08	94.96	27.42	-12.262	4	**0.000**
TST (minutes)	378.00	40.25	345.90	23.14	2.972	4	**0.041**
SE	98.11	10.17	74.84	6.18	6.611	4	**0.003**

5.3.2.3.2 Difference in Discrepancy between Groups on the experimental night

Using the method employed in Chapter 4, the discrepancy of the subjective sleep (from the post-sleep questionnaire) is calculated as a percentage of objective sleep (on the experimental night) using the formula: (subjective value/objective value)*100 (Edinger & Fins, 1995). A value greater than 100 suggests a higher subjective than objective value; 100 suggests no difference; and less than 100 suggests a higher objective than subjective value.

Again, using a series of ANOVAs, there were no significant differences between groups on the subjective-objective discrepancy (Table 5.6).

Table 5.6: Univariate ANOVAs between groups for subjective-objective discrepancy calculated as: (subjective/objective)*100) for each participant in each group using the raw data which then determined the mean to be used. SOL = Sleep Onset Latency, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency

Subjective-Objective Discrepancy (%)	Participant Group						
	Good sleepers		Insomnia				
	Mean	SD	Mean	SD	F	df	p
SOL	89.74	45.53	635.66	1321.83	0.66	1	0.442
WASO	38.46	17.10	26.95	14.28	1.22	1	0.306
TST	93.93	27.35	109.14	6.69	1.39	1	0.277
SE	116.96	35.08	131.23	10.26	0.77	1	0.409

5.3.3 Differences between Groups on Sleep Perception Prior to Task

Of the three trials in distinct NREM periods, participants were asked to report their perception of the sleep state prior to each of the trials commencing. Data ranged from participants correctly identifying sleep state prior to 33% of the three trials to 100% of the three trials. The mean percentage across all participants was 80% (*SD* 23.31). There were no differences between those with insomnia and good sleepers on the percentage of correctly identified sleep state across the three trials, (Table 5.7)

Table 5.7: Independent t-test between people with insomnia and good sleepers on the percent of trials with correct sleep state perception.

	Participant Group						
	Good sleepers		Insomnia				
Sleep Perception	**Mean**	**SD**	**Mean**	**SD**	**t**	**df**	**p**
Percent of trials (3) with correct sleep state perception	73.33	27.89	86.67	18.26	-0.894	8	0.397

Using only trials that preceded a natural awakening, again, there was no difference between groups for the percent of trials with correct sleep perception (t(8)=0.45,p=0.663). Good sleepers correctly perceived sleep state prior to awakening on 83.33% (*SD* 23.57) of the trials compared to 73.33% (*SD* 43.46) for the insomnia group.

5.3.4 Differences between Groups on Measures of Explicit and Implicit Memory Formation

5.3.4.1 Free Recall

Both groups of participants exhibited minimal ability to freely recall stimuli presented during the four trials. On average participants managed to report between 0% and 6.44% of the words presented to them, with a mean of 2.32% (*SD* 1.88). All but one (90%) of the participants only recalled stimuli that were presented during the first trial of the night (sleep onset). There was no difference in the average percentage of words freely recalled over the trials between the two groups, Table 5.8.

Table 5.8: Independent t-test between people with insomnia and good sleepers on the percentage of correctly freely recalled words across all four trials

	Participant Group						
	Good sleepers		Insomnia				
Free Recall	**Mean**	**SD**	**Mean**	**SD**	**t**	**df**	**p**
Correctly recalled words across all trials (percentage)	1.54	1.32	3.11	2.16	-1.388	8	0.202

5.3.4.2 Recognition Task (Explicit Memory)

5.3.4.2.1 Differences Within and Between Groups over the Four Trials

A mixed, repeated measures ANOVA was conducted to examine the effect of group (insomnia or good sleeper) on the percentage of correctly recognised words for each of the four trials. There was no significant effect of group on the percentage of words recognised ($F(1,8)=0.476$, $p=0.510$, $\eta^2=0.056$) and no main effect of the trial on percentage of words recognised ($F(3,24)=3.082$, $p=0.063$, $\eta^2=0.257$). There was also no significant interaction between group and trial on the percentage of words recognised ($F(3,24)=0.691$, $p=0.566$, $\eta^2=0.080$). Means across the four trials by group are shown in Table 5.9.

Table 5.9: Means percentage of correctly recognised words spilt by trial and group

	Percentage words correctly recognised							
	Trial 1		Trial 2		Trial 3		Trial 4	
Participant Group	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Good Sleepers	61.34	25.93	45.95	24.79	35.46	17.17	35.50	33.37
Insomnia	59.80	30.61	52.49	23.67	44.32	14.68	55.17	14.15

A repeated measures ANOVA, conducted on just the trials occurring during the sleep period (trials 2-4), indicated no significant effect of group on the percentage of words recognised ($F(1,8)=0.888$, $p=0.374$, $\eta^2=0.100$). There was also no main effect of the trial on percentage of words recognised ($F(2,16)=1.269$, $p=0.308$, $\eta^2=0.137$), and no significant interaction between group and trial on the percentage of words recognised ($F(2,16)=0.709$, $p=0.507$, $\eta^2=0.081$).

5.3.4.2.2 Evidence of Overall Explicit Memory between Groups

As there was no difference between groups across the four trials, the data was collapsed into a percentage of correctly recognised stimuli across all four trials. This was compared to words incorrectly identified from the foils (words recognised by participants but not presented during the night) as a measure of chance/false positives, and thus examining true explicit recognition. Table 5.10 shows that there was no significant difference between the percentages of words recognised that were presented during the night (true positives) and incorrectly recognised foils (false positives) for the good sleep group, indicating that words from the four trials were not recognised above chance. However, there was a significant difference in the insomnia group ($t(4)=3.926$, $p<0.05$), indicating that the insomnia group identified, on average, a significantly higher percentage of words that were presented during the night than incorrectly identified words from the foils.

Table 5.10: Paired t-tests between the average percentage of correctly identified words across the four trials and the number of recognised foils for both the good sleep and insomnia group. *Foils relate to words not presented during the night that were recognised by the participant (false positives)

	Percentage words recognised						
	Presented words		Foils*				
Participant Group	**Mean**	**SD**	**Mean**	**SD**	**t**	**df**	**p**
Good Sleepers	44.57	21.96	23.43	17.13	2.604	4	0.600
Insomnia	52.94	15.99	36.59	18.86	3.926	4	**0.017**

5.3.4.3 White Noise Task (Implicit Memory)

5.3.4.3.1 Differences within and between groups over the four trials

A mixed, repeated measures ANOVA was conducted to examine the effect of group (insomnia or good sleeper) on the percentage of correctly implicitly recognised words (white noise embedded) on each of the four trials. There was no significant effect of group on the percentage of words recognised ($F(1,7)=1.110$, $p=0.327$, $\eta^2=0.137$) and no main effect of the trial on percentage of words recognised ($F(3,21)=0.025$, $p=0.994$, $\eta^2=0.004$). There was also no significant interaction between group and trial on the percentage of words recognised ($F(3,21)=2.113$, $p=0.129$, $\eta^2=0.232$). Means across the four trials by group are shown in Table 5.11.

Table 5.11: Means percentage of correctly recognised words spilt by trial and group

	Percentage words correctly recognised							
	Trial 1		Trial 2		Trial 3		Trial 4	
Participant Group	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Good Sleepers	65.29	24.21	57.33	38.60	56.57	25.69	75.24	34.07
Insomnia	74.46	17.98	83.33	26.45	88.10	15.79	67.71	5.24

When a repeated measures ANOVA was conducted on just the trials occurring during the sleep period (trials 2-4), again no significant effect of group on the percentage of words recognised ($F(1,7)=1.227$, $p=0.305$, $\eta^2=0.149$). There was also no main effect of the trial on percentage of words recognised ($F(2,14)=0.022$, $p=0.978$, $\eta^2=0.003$) and no significant interaction between group and trial on the percentage of words recognised ($F(2,14)=2.476$, $p=0.1250$, $\eta^2=0.261$).

5.3.4.3.2 Evidence of Overall Implicit Memory between Groups

As there was no difference between groups across the four trials, the data was collapsed into the percentage of correctly implicitly recognised stimuli t across all four trials which compared to words incorrectly identified from the foils (words recognised but not presented during the night). Table 5.12 shows that there was no significant difference between the percentages of true positives to false positives for the good sleep group, indicating that encoded words from the four trials were not implicitly recognised above chance. However, there was a significant difference in the insomnia group ($t(3)=5.599$, $p<0.05$) indicating that, on average, those with insomnia implicitly recognised significantly higher percentage of words that were presented during the night than incorrectly identified words from the foils.

Table 5.12: Paired t-tests between the average percentage of correctly identified words across the four trials from within white noise and the number of recognised foils for both the good sleep and insomnia group. *Foils relate to words not presented during the night that were recognised by the participant (false positives)

	Percentage words implicitly recognised						
	Presented words		Foils*				
Participant Group	**Mean**	**SD**	**Mean**	**SD**	**t**	**df**	**p**
Good Sleepers	63.61	26.00	61.62	8.05	0.237	4	0.824
Insomnia	78.65	12.45	59.44	8.82	5.599	3	**0.011**

5.3.5 Associations between Explicit and Implicit Memory and Measures of Sleep State Misperception

Using a Pearson's bivariate correlation, associations were computed between the subjective-objective discrepancy for measures of sleep continuity and true and false positives on both the recognition and white noise task across all participants, Table 5.13. Only a single negative relationship was identified between the percentage of foils incorrectly recognised during the explicit recognition task and the discrepancy in WASO ($r(7)=-0.684$, $p<0.05$). Indicating that as the subjective report of WASO

increased above that of the objective WASO, the fewer foils were incorrectly recognised during the explicit memory task.

Table 5.13: Bivariate Pearsons correlation showing relationships between the subjective-objective discrepancy and responses on the memory tasks. SOL = Sleep Onset Latency, WASO = Wake After Sleep Onset, TST = Total Sleep Time, and SE = Sleep Efficiency. * p<0.05

	Subjective-Objective Discrepancy (%)			
Memory tasks	**SOL**	**WASO**	**TST**	**SE**
Number of freely recalled words	-0.179	0.040	0.159	0.078
Percentage of words correctly recognised (explicit)	0.022	-0.522	0.106	-0.024
Percentage of words (foils) incorrectly recognised (explicit)	-0.260	**-0.684***	0.092	-0.076
Percentage of words correctly recognised (implicit)	0.168	0.111	-0.221	-0.310
Percentage of words (foils) incorrectly recognised (implicit)	0.329	0.148	-0.119	-0.106

5.4 Discussion

This study posed four distinct hypotheses, firstly that those with insomnia would have poorer sleep, both subjectively and objectively, and a greater degree of sleep state misperception than good sleepers following the experimental protocol. The remaining three hypotheses relate directly to the neurocognitive alterations within the Neurocognitive Model concerning sleep perception (short-term memory), long-term memory formation (explicit and implicit memory) and relationships between these factors and sleep state misperception. It was anticipated that individuals with insomnia would be able to correctly identify wake during natural awakenings to a greater extent than good sleepers as this would be due to higher levels of cortical arousal.

Furthermore, in accordance with the Neurocognitive Model, it was hypothesised that individuals with insomnia would experience greater recall and a greater recognition of words presented during the trials than good sleepers due to the proposed association between increased cortical arousal and the impairment in sleep amnesia. Accordingly, it was also expected that a greater degree of memory processing would positively correlate with sleep state misperception, as recollection of stimuli occurring during sleep transition points would negatively skew perception of sleep continuity.

However, the findings indicate that the protocol may have improved sleep in the insomnia group yet disrupted sleep within the good sleep group. Additionally, the occurrence of sleep state misperception due to the non-habituated environment and the protocol appeared to be overall positive, as both groups perceived sleep to be better than what was recorded objectively. Furthermore, there were no differences between groups in the ability to correctly perceive sleep state prior to the trials during NREM sleep and no difference in the number of words freely recalled following sleep. Concerning explicit and implicit memory, there were no differences between groups on the percentage of correctly recalled words per trial and overall, for both the explicit and implicit recognition tasks. However, when the percentage of correctly recognised words (for both tasks) was compared to the percentage of incorrectly recognised words from the foils, only the insomnia group showed a significant difference: specifically, more words that were presented during the night were recognised than foils. However, contrary to expectation, neither explicit nor implicit memory was related to the degree of sleep state misperception observed. These results are discussed in relation to previous research below.

5.4.1 Differences between Good Sleepers and Those with Insomnia on Measures of Sleep

During baseline sleep assessments there was a marked difference in subjective sleep between the two groups. With the exception of WASO and NWAK, the insomnia group reported poorer sleep continuity (SOL, TST, and SE). This group difference, however, was not evident following the experimental night. During the experimental procedure, both subjective and objective sleep was disturbed in the good sleeper group yet those in the insomnia group had an improvement in sleep. Asking participants to repeat the words back to researcher immediately upon presentation during the sleep onset periods may have acted as a distraction from the pre-sleep cognitions that would, in the home environment, impair sleep onset in the insomnia group. Certainly excessive pre-sleep arousal and environmental monitoring has been shown to be evident in individuals with insomnia at a greater extent than those who have good sleep (Harvey, 2000). It has also been shown that, whereas asking an individual with insomnia to employ a general distraction technique before bed does not aid sleep, asking the individual to employ a specific task, such as imagery control, reduces sleep onset (Harvey & Payne, 2002). Therefore, asking the participants not to attend to the words but to repeat them if they heard them, could have aided in the shortening of SOL in the insomnia group acting as distraction technique from sleep inhibiting cognitive processes and thus lowering cortical arousal.

Conversely, the task was a distraction from the good sleeping process for the good sleeper group and therefore could explain the worsening of sleep continuity. Certainly, evidence suggests that increasing cognitive activity prior to sleep in good sleepers can lengthen sleep onset latency (Ansfield et al., 1996; Gross & Borkovec, 1982). Moreover, within the good sleeper group, the knowledge that awakenings were to occur during the night and that sleep was to possibly be shorter than usual (sleep opportunity was curtailed to approximately 6.5hrs for both groups to allow equal potential encoding time), may have resulted in increased arousal and thus impaired

sleep. Wuyts and colleagues (2012) evidenced this effect of anticipation of task/sleep disturbance within an experiment simulating a night on-call. Good sleepers were told that sleep would be disturbed periodically by a pager alarm of which they needed to attend to, although no alarms actually occurred. Wuyts and colleagues (2012) indicated that, despite sleep not being objectively disturbed by the protocol, participants within the study experienced disruptions to both subjective and objective sleep, experiencing an increase in objective SOL, an increase in both subjective and objective WASO, a lower subjective and objective SE, as well as a significant increase in beta EEG activity.

This present study also found a disparity between the groups when measures of sleep state misperception were assessed. The good sleeper group perceived WASO as shorter than what was observed through PSG. Moreover, the insomnia group reported positive sleep state misperception in measures of WASO, TST and SE. When the discrepancy was calculated by the percentage difference between subjective and objective values, there was no significant difference between groups.

Therefore, the first hypothesis which stated that, comparative to good sleepers, those with insomnia would experience poorer subjective and objective sleep along with a greater degree of sleep state misperception, was only partially confirmed by this study. The differences were apparent at baseline, but during and after the experimental night, these differences reversed. An alternative explanation may directly relate to the protocol: as with the separate measures of subjective and objective sleep, the task may have increased arousal within the good sleepers to a level sufficient to experience disturbance to sleep, as the Neurocognitive Model suggests. Furthermore, individuals with insomnia may have experienced a reduction in sleep related attention, as attention is now focussed on the presentation and repeating of words, thus reducing cognitive effort for trying to attain sleep. This may have reduced the levels of cortical arousal and, thus resulted in improvements to both subjective and objective sleep. Although

rejection of the hypotheses must occur in light of current findings, this study does re-affirm the importance of conditioned and cortical arousal in the perception of sleep in insomnia and good sleepers thus supporting the Neurocognitive Model.

5.4.2 Differences between Good Sleepers and Those with Insomnia on Sleep Perception

The Neurocognitive Model suggests that an alteration in short term memory processing occurs in response to cortical arousal during the night in insomnia. This short term processing results in an inability to recognise sleep state, in this case a false perception of wake as opposed to sleep upon awakening during the night. It is suggested, that when forcibly awoken from sleep, people with insomnia tend to report wake more frequently than good sleepers and require 15 minutes of consolidated N2 for an accurate perception of sleep onset (Borkovec et al., 1981; Hauri & Olmstead, 1983; Mercer et al., 2002) .

However, this study employed questioning of sleep perception following a natural awakening (80% of trials) from NREM sleep (1 minute of wake). Participants were aware that there would be trials during the night but were not aware whether these would be initiated from sleep or during wake periods. Across the three trials, there was no significant difference in correct identification of sleep state between the good sleepers and those with insomnia, even if only trials occurring after a natural awakening were used. Although this may seem contrary to previous research, studies suggest that both people with insomnia and good sleepers are able to correctly perceive awakenings following a consolidated block of sleep and it is only when there is a period of consecutive awakenings, prior to the awakening in question, that people with insomnia misperceive sleep state (Coates et al., 1983; Knab & Engel, 1988).

Therefore, the second hypothesis in light of current findings must be rejected. However, what is indicated through this research is that it may not be an appraisal of

awakenings that is resulting in sleep misperception as the Neurocognitive Model suggests. Therefore, if it is not short term memory, then it may be the encoding of stimuli during these awakenings that result in a skewed perception of sleep. This supplies a novel finding to current literature, as both good sleepers and those with insomnia are able to correctly perceive their sleep/wake state when questioned during a natural awakening, suggesting revisions are required of the propositions of the Neurocognitive Model.

5.4.3 Differences between Groups on Explicit Memory of Words

The Neurocognitive Model suggests that long-term memory encoding of stimuli at sleep onset and sleep transition points occurs in individuals with insomnia and, in turn, drives sleep state misperception. This proposition is novel, and only one other study has shown that when presented with word blocks (nouns), at sleep onset and at three wake sleep transition points following forced awakenings from stage 2 sleep, those with insomnia tended to show greater explicit memory for words presented across all trials comparative to good sleepers (Perlis, Smith, Orff, et al., 2001).

Unlike the study by Perlis and colleagues (2001), in the present study there was no difference between those with insomnia and the good sleepers for the number of words freely recalled from the words presented during the night, the lack of significance may be representative of a floor effect. Participants were presented with up to 100 single words during the night with no repetition and thus the difficulty of the task may have been too high for both groups. Alternatively, of the words recalled, all but one of the participants only managed to freely recall words presented in the first trial (sleep onset) suggesting that cortical arousal does not result in direct encoding of environmental stimuli during the night.

Additionally, there were no differences either within or between each of the groups for the percentage of correctly recognised words across all four trials, suggesting that the

ability to form memories through increased cortical arousal is a component of all sleep onset periods during NREM sleep. However, when examining the percentage of correctly recognised true words and the percentage of falsely recognised words from the foils, only the insomnia group showed a significant difference. This lack of difference between the groups on the percentage of correctly recognised words during each of the four trials could be due to the non-habituated environment raising cortical arousal in the good sleeper group to a level that is comparable to the endogenous levels assumed to be present in those with insomnia. As thus, the ability to explicitly recognise stimuli presented during these sleep onset periods is the same.

However, only the insomnia group showed a significant discrimination between words presented during the night and foils - confirming the hypothesis. This suggests that memory processing may indeed be occurring in individuals with insomnia at a greater level than good sleepers, and processing can occur even if direct encoding (the ability to freely recall words) has not. Moreover, it may provide evidence towards a trait vulnerability for processing auditory stimuli to recognition memory during sleep onset as, despite an improvement of sleep and thus assumed reduction of cortical arousal, explicit recognition of the auditory stimuli was still present.

5.4.4 Differences between Groups on Implicit Memory of Words

Another proposition of the Neurocognitive Model is that individuals with insomnia would exhibit higher implicit recognition memory for the words presented during sleep onset periods. Loosely defined, implicit memory is the unconscious memory formation of stimuli without the ability to remember the nature of the stimuli. If cortical arousal is impairing the ability to protect sleep from sensory intrusion as the Neurocognitive Model proposes, and that this results in a disparity between subjective and objective sleep, then stimuli may be processed by the brain even if the individual is unable to explicitly recall said stimuli. To date, no literature has assessed this in insomnia however, in individuals with fibromyalgia, it was shown that even if words presented

during the night could not be recalled, there was still a negative disparity between subjective and objective sleep after the experimental protocol of which the authors suggested to be indicative of possible implicit memory formation (Perlis, Giles, Bootzin, et al., 1997).

As with the results on explicit memory, there were no differences within or between groups on the percentage of words implicitly recalled across trials. Again, the lack of difference between the groups on the percentage of correctly recognised words indicates that those with insomnia and good sleepers may be under the same amount of cortical arousal and thus the ability to implicitly recognise stimuli presented during these sleep onset periods is the same, as the Neurocognitive Model would suggest. However, as with explicit recognition, the insomnia group indicated a significant difference between the percentage of correctly implicitly recognised true words and the percentage of falsely implicitly recognised words from the foils. This result is a novel contribution to the literature suggesting that there may also be evidence for implicit memory processing occurring in insomnia compared to good sleepers, thus supporting the hypothesis.

Along with the findings for explicit memory, this suggests that environmental noise can be processed during sleep and potentially recognised as a disturber to sleep upon awakening and during the day. If this results in a negative perception of sleep, then treatment for insomnia needs to address the role of noise and methods to control increased cortical arousal in vulnerable individuals, be that good sleepers in a non-habituated environment or those with a trait vulnerability (insomnia). Even if an individual who is experiencing sleep disturbances and unable to document to the treating professional noise which is disturbing sleep, processing (implicitly) may still be occurring and thus needs to be addressed when creating a treatment plan for an individual with insomnia, as is suggested in a study by Ellis et al. (2002).

5.4.5 Associations between Memory of Words and Sleep State Misperception

The Neurocognitive Model suggests that, due to the neurocognitive factors (cortical arousal and cognitive alterations), sleep state misperception occurs as the amnesia of sleep onset is impaired through the recall and recognition of environmental stimuli. To determine this, the present study assessed correlations between recall and recognition (explicit and implicit) memory of the words and the discrepancy between subjective and objective sleep measures.

However, within this study there were no significant associations between both explicit and implicit memory formation of presented words during the night and the difference between subjective and objective sleep. Therefore, these results do not support the final hypothesis of this study that increased long-term memory formation of words presented during the night are associated with a greater degree of sleep-state misperception. In light of current findings, this may suggest that memory formation as a result of cortical arousal does not drive the inability to perceive sleep as sleep, at least not for stimuli occurring during wake to sleep transitions points from NREM. However, as a baseline level of sleep misperception is not known for each of the participants on a night where there is no noise, a change in sleep misperception due to the change in cortical arousal due to the protocol cannot be elucidated. So although current findings suggests a failure of this proposition of the Neurocognitive Model within this study, the limitations of this study show that further research is warranted.

5.4.6 Limitations and Future Considerations

The most prominent limitation of this study is sample size. Although both groups were matched for age and gender, there were only five participants in each group, which therefore may not be representative of the general population. Due to small sample size, there is also limited power within the statistics, which in turn limit the findings.

Another notable limitation could be within the insomnia group themselves. Participants were recruited if they fulfilled the DSM-V criteria for an insomnia disorder, that they didn't have idiopathic (childhood onset) insomnia, and that they report at least 30 minutes SOL and WASO with a TST of less than 6 hours. However, the participants in the insomnia group were not classified into whether they experienced psychophysiological insomnia or sleep state misperception insomnia, as all participants were included based on a subjective sleep diary and semi-structured interview. Psychophysiological insomnia is also known as learned insomnia and, according to the ICSD-2 (American Academy of Sleep Medicine, 2005), is thought to be due to a stressful event resulting in conditioned arousal. As the insomnia group showed improved sleep, compared to the good sleep group, the experimental protocol is suggested to have acted as a distraction technique for pre-sleep arousal processes and actually aided sleep, thus possibly skewing the results concerning associations between memory and sleep misperception. Future research may wish to singularly focus on sleep-state misperception or examine individuals in an ambulatory setting in their own home, thereby ensuring the presence of conditioned bedroom arousal.

Within this study, there was no baseline night within the laboratory to determine the individuals' baseline perception of sleep and thus this study could not examine the change in the perception of sleep continuity following the word administration. Future research may wish to consider the limitations found within this study and attempt to replicate the results using a bigger sample with the inclusion of an additional baseline night to allow, similar to Chapter 4, examination of the individuals baseline ability to correctly perceive sleep prior to manipulating sleep through changes in cortical arousal. Using this method of a baseline night to determine how far the perception of sleep has changed from a night of no environmental stimuli would allow findings to include positive misperception as well as the most documented negative misperception of sleep. As Edinger and Fins (1995) suggest, misperception of sleep may occur on a continuum within insomnia and individuals may experience either a positive or negative

perception. Testing various subtypes of insomnia, along with good sleepers, and examining the change in perception of sleep would allow determination of whether the constructs of the Neurocognitive Model apply only to sleep misperception in insomnia or whether the model is a general model of the impact of cortical arousal on sleep.

The Neurocognitive Model suggests that cortical arousal could be shown by the presence of high frequency EEG. Indeed, individuals with insomnia have been shown to experience high beta EEG activity prior to stage 2 sleep onset (Freedman, 1986; Perlis, Merica, et al., 2001). This high frequency EEG activity has been shown to associate with poorer subjective sleep quality (Perlis, Smith, Andrews, et al., 2001), and is increased in individuals with sleep state misperception insomnia (Krystal et al., 2002) suggesting a role of high frequency EEG in the perceptions of sleep. Therefore , similar to Chapter 4, future analysis of this study and indeed, future research, may wish to perform spectral analysis on the PSG to investigate associations between the level of high frequency EEG, particularly in the alpha and beta range, and measures of memory processing during wake to sleep transition periods in insomnia.

5.4.7 Conclusions

Due to the small participant numbers within this study, all conclusions need to be viewed tentatively, but this study does suggest that those with insomnia show an ability to both implicitly and explicitly recognise environmental stimuli that occur at sleep onset and natural wake to sleep transition points during NREM sleep. As the experimental protocol affected those with insomnia and good sleepers differently, this study indicates that nocturnal noise could be a problem for individuals with insomnia and good sleepers in non-habituated environment (i.e. if the noises occur spontaneously without prior knowledge of the participant), influencing cortical arousal. Yet further research is needed to assess whether memory processing is indeed associated with greater sleep misperception as the Neurocognitive Model suggests, which could simply be provided by repeating the above protocol with the inclusion of a baseline night of no noise. In

which case, it would be expected, that the greater the change in the perception of sleep from baseline to a night whereby auditory stimuli are administered would associate with memory of said auditory stimuli.

Interestingly, the study within this chapter identified no difference in sleep perception between good sleepers and those with insomnia when questioned during natural arousals. This provides a novel finding to current insomnia literature and suggests that the process surrounding the creation of the disparity between subjective and objective sleep may not be due to an exaggeration of the perception of wake during nocturnal awakenings. Therefore, although this study did not supply evidence towards the short-term memory formation component of the Neurocognitive Model, this same evidence provides potential support towards the encoding to long-term memory, auditory stimuli that may alter the perception of sleep upon sleep appraisal the following morning.

Furthermore, the identification of evidence pertaining to increased implicit memory formation to auditory stimuli provides significant clinical relevance that warrants future study. Although in this instance implicit memory did not associate with sleep state misperception, if upon further analysis, the presence of implicit memory associated with an increase in cortical arousal (as is presumed to be occurring in the insomnia group), this may not only provide support for the Neurocognitive Model but also for insomnia theory. Furthermore, although literature examining sleep state misperception have found that it associates with high frequency EEG (Perlis, Merica, et al., 2001) and that it is not due to insufficiencies in time estimation (Tang & Harvey, 2005), current literature has not agreed on why sleep state misperception occurs. As, unlike psychophysiological insomnia whereby the initiating factor is said to be a stressful life event, neither the ICSD-2 nor the RDC provide causative factors for sleep state misperception occurs. If implicit memory processing is occurring within insomnia, then clinicians need to be made aware that, even if the patient is unable to recall stimuli that is disturbing sleep, subjectively this disturbance may still be occurring and, as Semler

and Harvey (2005) have shown, can result in real detriments to performance the following day. So although Edinger and Krystal (2003) proposes that sleep state misperception may not be a distinct insomnia subtype could, through the Neurocognitive Model, the processing of auditory stimuli drive the development of an insomnia disorder through cortical arousal and the altered sleep perception?

The following chapter will draw conclusions from this present study and the studies within Chapters 3 and 4 to discuss the findings in relation to the Neurocognitive Model along with the application this body of work provides to insomnia theory and treatment practices.

Chapter 6: Overall Thesis Discussion – Empirically Testing the Neurocognitive Model

6.0 Thesis Aims

The body of work presented within this thesis sought to empirically test the cortical arousal component of the Neurocognitive Model of insomnia providing a novel contribution to the literature. Specifically, to determine whether heightened cortical arousal influences a disparity between subjective and objective sleep and if this relationship occurs due to memory formation for nocturnal auditory stimuli. This thesis employed a variety of auditory stimuli administered at various points across the sleep period as a method of: a) eliciting increased cortical arousal, and b) exploring whether memory formation (either implicitly or explicitly) occurs. Additionally, a parallel theme was to explore whether characteristics in the individual increased their vulnerability to cortical arousal, specifically a habituated versus a non-habituated environment.

6.1 Research Questions Proposed by this Thesis

On the assumption that the administration of auditory stimuli during sleep is a suitable method for examining the cortical arousal component of the Neurocognitive Model three central questions were posed:

1) Does habituation to nocturnal noise occur equally amongst individuals or are there specific vulnerabilities or situational factors that influence habituation?

2) Is there increased cortical arousal when non-habituated novel stimuli are presented, and are there specific vulnerabilities that facilitate or prevent habituation to the novel stimuli?

3) Is the pathway to misperception, as suggested by the Neurocognitive Model, through memory formation?

In terms of the first question, cortical arousal was examined in the home environment within a sample of good sleepers. The home environment was chosen to ensure that habituation would most likely have occurred at the time of testing ensuring individual differences could be examined without the interference of novelty of environment or noise. In terms of the second question, again self-reported good sleepers came to the sleep laboratory and were exposed to novel but ecologically valid noises to determine whether non-habituated noises influenced sleep subjectively, objectively, and/or the discrepancy between them (i.e. evidence of increased cortical arousal) and to examine whether individual differences facilitated habituation. The final question was addressed by an in-lab sleep study with good sleepers and individuals with insomnia. This study examined group differences in cortical arousal following exposure to auditory stimuli (words) during sleep onset periods to assess whether heightened cortical arousal associated with explicit or implicit memory formation and/or the occurrence of a disparity in the perception of sleep comparative to objective report of sleep upon final awakening.

6.2 Overview of Findings from Chapter Three

The study in Chapter 3 provided the initial observation of the relationship between noise and cortical arousal within participants own homes. By utilising the habituated bedroom environment, examination of individual differences in the response of sleep dependent on stage, vulnerability factors that result in disruption of sleep, and relationships between sleep state misperception and noise recall could be examined. Findings indicated, unsurprisingly, that noise during the night was likely to occur concurrent with an epoch of wake as determined through Actigraphy, supporting the studies by Öhrström et al. (1990) and Öhrström (1995) that an increase in body movements correlates with noise levels during sleep.

This study evidenced that noise had a differential effect on sleep dependent on when the noise occurred, as a mixed ANOVA indicated a significant interaction effect

between the percentage of each epoch classification (wake with noise, wake with silence, sleep with noise, and sleep with silence) and the sampling period. When epoch classifications were grouped into hits (sleep with silence, wake with noise) and misses (sleep with noise, wake with silence), the first 90 minute sampling period (NREM) had a significantly higher percentage of epochs classified as hits and a significantly lower percentage of epochs classified as misses compared to the last 90 minute (REM) sampling period. This indicates that habituation of the bedroom environment may negate the effect of noise on the final 90 minutes of sleep, providing support for the Neurocognitive model that it may be the initial sleep onset period and NREM sleep that is most vulnerable to noise and thus the influences of cortical arousal.

When examining individual differences between participants on measures identified as vulnerability factors for experiencing cortical arousal, the somatic subscale of the Pre-Sleep Arousal Scale (PSAS) (Nicassio et al., 1985) showed a significant positive relationship with the percentage of epochs classified as hits (wake when noise was present, sleep when there was silence silent) in the last 90 minute sampling period. Additionally, there was a significant negative relationship between the percentage of epochs classified as misses (wake when silent, sleep in the presence of noise) in the last 90 minute sampling period and the somatic subscale of the PSAS. Together these findings indicate that higher levels of somatic arousal may be a vulnerability factor for nocturnal disturbances by auditory environmental stimuli, irrespective of habituation to the bedroom environment. Furthermore, scores on the Ford Insomnia Response to Stress Test (FIRST) (Drake et al., 2004) showed a similar significant positive relationship with the percentage of epochs classified as hits and a significant negative relationship with the percentage of epochs classified as misses in the last 90 minute sampling period. This suggests that a vulnerability for stress related sleep loss is positively associated with awakenings and negatively associated with sleep in the presence of auditory stimuli. As these scales showed significant associations during

the last 90 minute sampling period, and as this period could be assumed to consist of predominately lighter EEG frequency stages sleep (including REM, N1 and N2), this may indicate a period which may be endogenously alterations to sleep through noise induced cortical arousal.

The Neurocognitive Model states, processing of external stimuli during NREM sleep can result in an altered perception of sleep. On average participants managed to recall 0.92 (*SD* 0.92) noises as having disturbed sleep although as we only assessed noise presence rather than type we cannot confirm if these recalled sounds occurred and, if they did, the specific stage they occurred in. Additionally, the study in Chapter 3 demonstrated minimal relationships with the presence of noise and the subjective report of sleep. A single significant relationship was identified with subjective TST whereby a significant negative correlation occurred with the percentage of epochs classified as hits (wake with noise and sleep with silence) and thus also a significant positive correlation with misses in the first 90 minute sampling period, after controlling for the number of noises recalled. This suggests that, during initial sleep onset period (assumed to be predominately NREM sleep), good sleepers who were able to sleep in the presence (more misses) of noise recalled fewer noises in the morning and had a better perception of their total sleep time.

When the discrepancy between subjective and objective sleep was examined, on average across the three nights, the self-reporting good sleeping participants experienced minimal differences between the subjective report of sleep and what was observed objectively via actigraphy. The single measure that significantly differed was WASO, with participants reporting a positive misperception of sleep (subjective report of 7.45 minutes comparative to 49.06 minutes from actigraphy). Therefore, it was unsurprising that there were no significant relationships found for either of the sampling periods with the discrepancy in sleep indicating that, the number of noise reported as

having disturbed sleep (memory encoding) was not associated with a subjective/objective sleep discrepancy over the night.

6.2.1 How the findings of Chapter Three Informed the Thesis

Overall, it would appear that there may be an endogenous vulnerability period to cortical arousal due to the presence of auditory stimuli when sleep is thought to contain a predominance of lighter sleep stages (i.e. the sleep onset period determined as the first 90 minute sampling period). This examination of vulnerability to cortical arousal across the first and last 90 (NREM/REM) minute sampling periods in a habituated sleep environment is a novel contribution to the literature and the finding that the period believed to contain predominately NREM sleep (first 90 minute sampling period) may be vulnerable provides support for the Neurocognitive Model.

The second novel contribution to the literature was through the examination of vulnerability to cortical arousal in good sleepers. As Spielman's 3P model of insomnia (1986) proposes some individuals may be predisposed to developing insomnia and Espies (2002) Psychobiological model proposes that good sleepers have good plasticity and can therefore respond adequately (habituate) to sleep interfering factors. Certainly the findings support this concept with regards to the protection (habituation) of sleep continuity (misses) in the face of environmental perturbations (noise). Furthermore, as there was no ability to recall noise, and no discrepancy between subjective and objective sleep measures, habituation to the bedroom environment appears to attenuate the impact of cortical arousal through noise during the NREM and REM period at least in terms of sleep misperception and memory formation of environmental stimuli. However, this study did not asses the type or loudness of the nocturnal noises. The Neurocognitive Model proposes that it is the ability to recall or recognise environmental stimuli occurring during sleep or sleep onset that results in a negative appraisal of sleep continuity upon awakening and thus sleep state misperception. As such, the type of noise stimuli would need to be documented so as

recognition tasks can be conducted for auditory to stimuli to examine relationships with the misperception of sleep.

The study in Chapter 3 sugessted that, although the first 90 minutes of sleep (NREM predominately) is vulnerable to disturbance by cortical arousal through noise, habituation to the bedroom may afford protection to the good sleepers against the neurocognitive alterations posited in the Neurocognitive Model to lead to sleep state misperception. Therefore, informing the thesis that, to examine the effects of raised cortical arousal through noise, a non-habituated environment was required. Consequently, the study in Chapter 4 examined noise in a laboratory setting where the nature and loudness of the auditory stimulus could be controlled. By providing a non-habituated environment, it was thought this would raise the potential for the noise to increase cortical arousal affording the ability to test the propositions of the Neurocognitive Model using good sleepers. Moreover, the auditory stimuli consisted of a combination of ecologically valid noises administered in intermittent short 3s pulses, of which has been previously demonstrated to increase the lighter sleep stages at the expense of deep sleep (Eberhardt et al., 1987) along with altering the perception of SOL and inhibiting habituation effects (i.e. results in both subjective and an objective detriment to sleep) (Townsend et al., 1973).

6.3 Overview of Findings from Chapter Four

The study within Chapter 4 aimed to determine the point where cortical arousal is said to occur, by examining NREM and REM at a greater depth based on the preliminary findings from the study in Chapter 3. Furthermore, this aimed to determine whether the ability to recognize, when prompted, auditory stimuli during sleep is associated with a misperception between subjective and objective sleep measures, as suggested by the Neurocognitive Model. Along with utilising a non-habituated environment, participants were unaware that noise was to be played to them whilst they slept, thus avoiding any alterations in cognitive load and arousal through anticipation of the task. As Wuyts et

al., (2012) indicated with their simulated on call study, telling a good sleeper that noise is to disturb their sleep during the night may result in sleep disrutpion despite no noise being administered.

In line with the propositions of the Neurocognitive Model and the previous study in Chapter 3, NREM appeared to be the most vulnerable period for cortical arousal, evidenced through alterations in sleep architecture: an increase in wake and N1 from the baseline to the experimental night. This suggests that the whole NREM period may not be susceptible to alterations through the effects of increased cortical arousal via noise per se but rather wake to sleep (N1) transitions, expanding on the findings from the study in Chapter 3.

Moreover, this study indicated that continuous noise played at or below the guidelines set by WHO (2009) for nocturnal environmental noise (40 decibels) may be sufficient to disturb both subjective (larger NWAK and WASO) and objective sleep (larger WASO) in good sleepers. However, most importantly, noise resulted in a discrepancy between subjective and objective SOL occurred following a night of noise - a discrepancy that did not exist on the baseline night. This is surprising, as sounds were not administered on the experimental night until five minutes post onset of stage 2 sleep.This could indicate that, in terms of the Neurocognitive Model, just the perception of sleep onset that is altered through increased cortical arousal in good sleepers as opposed to a global misperception across all sleep continuity measures. This, non-habituated, effect of noise resulting in subjective and objective detriments to sleep along with a discrepancy in SOL supports the seminal work of Townsend et al., (1973).

Similar to the study in Chapter 3, participants in this study freely recalled (without prompting) very few of the noises that were administered during sleep. This indicates that good sleepers, despite the novel auditory stimuli and a non-habituated environment, may not be able to directly encode auditory stimuli during sleep despite

an increase in cortical arousal. However, participants did manage to recognise (via a card-sorting task) significantly more of the noises that were presented during sleep than foils the following morning. This suggests that increased cortical arousal may result in memory formation but one which can only be retrieved after prompting.

Furthermore, similar to the study in Chapter 3, there were no associations between explicit memory (percentage of correctly recalled items or percentage of noises correctly recognised) and thedisparity between subjective and objective sleep. This suggests that noise may have had a direct effect on the discrepancy of sleep as evidenced by Townsend et al., (1973), or through implicit memory of the auditory stimuli altering the perception of sleep “as sleep”.

6.3.1 How the Findings of Chapter Four Informed the Thesis

The studies in Chapter 3 and 4 highlighted that noise is a suitable candidate to test the Neurocognitive Model by not only providing environmental stimuli that has the potential to be explicitly recalled during the night but also as a means of increasing cortical arousal. This informed Chapter 5 by raising the question as to what extent this process occurs in those with already heightened cortical arousal, such as individuals with insomnia, as the Neurocognitive Model proposes.

Overall, the study in Chapter 4 suggested that it might be the perception of sleep onset that is most sensitive to noise, indicating that even if the sleep onset period is not disturbed, noise during the night may alter the perception of being able to fall asleep easily in good sleepers. As significant sleep misperception is usually attributed to insomnia, and that it has been suggested the perception of a poor night of sleep can promote an actual deficit in sleep (Espie, 2002; Harvey, 2002), this finding is of importance when examining the development of insomnia. As defined by Semler and Harvey (2005), a perceived decrement in subjective sleep precedes a perceived decrement to daytime functioning. In vulnerable individuals, this negative appraisal of

sleep may results in the employment of safety behaviours to try to counteract the perceived sleep loss. As there is no real deficit (through objective means) these compensatory safety behaviours will not result in an improvement in sleep, as the disturbance is perceptual, and actually create the objective sleep disturbance via increased sleep effort and associated arousal.

Although there was no evidence of direct encoding of the auditory stimuli, following prompting via a card-sorting task, there was recognition of noise stimuli that was administered during the night. Chapter 4 only examined explicit recognition of noise, therefore the altered perception of sleep suggests, as one possibility of many, implicit memory may be in the mechanism underlying the perception of sleep. Within individuals with fibromyalgia (a population with high frequency EEG intrusion in sleep), similar to the study in Chapter 4, the explicit memory of words administered during sleep did not associate with the discrepancy created in sleep, therefore the authors proposed that implicit recognition of the auditory stimuli was the factor that led to the misperception (Perlis, Giles, Bootzin, et al., 1997).

Both the alterations in sleep architecture (the increase in wake and stage 1) and the created disparity between subjective and objective sleep onset latency provided direction to Chapter 5, by determining the point at which stimuli were to be administered (wake to sleep transition points) to assess encoding of stimuli during the sleep period. Furthermore, it evidenced that a non-habituated bedroom environment utilising novel auditory noise may be sufficient to increase cortical arousal in good sleepers as determined through alterations in subjective and objective sleep. Therefore, the study in Chapter 5 questioned whether there are differences between individuals already experiencing heightened cortical arousal (insomnia) and good sleepers when presented with a non-habituated bedroom environment (proposed in this thesis to result in an acute period of heightened cortical arousal), aiming to

examine the propositions of the Neurocognitive model surrounding the effect of cortical arousal on sleep.

6.4 Overview of Findings from Chapter Five

Primarily, this study aimed to examine whether there are differences between individuals with insomnia and good sleepers which would give an indication of whether higher levels of cortical arousal results in both alterations to memory (through recognition of auditory stimuli upon awakening) and perception of wakefulness during sleep periods. Furthermore, would this result in a subjective/objective sleep discrepancy upon awakening, in line with propositions of the Neurocognitive Model of insomnia.

The study in Chapter 5 aimed to examine whether encoding of information during sleep onset periods, as a direct result of heightened cortical arousal, is apparent in individuals with insomnia in comparison to good sleepers. As heightened cortical arousal was expected to be present in those with insomnia regardless of the effect of noise and environment, then increased sleep state misperception would be specific to the insomnia group following the administration of noises. However, unexpectedly, the individuals with insomnia showed, albeit non-significantly, better objective and subjective sleep on the experimental night compared to the good sleeper group. Furthermore, the insomnia group experienced more measures whereby there was a significant difference between the subjective and objective measure of sleep continuity (WASO, TST, and SE) compared to good sleepers (WASO) on the experimental night. Moreover, these were all a positive misperceptions of sleep, with both the people with insomnia and good sleepers perceiving themselves to have slept better than what was observed via PSG on the night where sleep was disturbed by the word noise task.

The study in Chapter 5 found that there was no difference in the perception of wake during sleep periods (short term memory) between those with insomnia and good

sleepers. Furthermore, in agreement with the study in Chapter 4, there was no evidence of direct encoding of the words presented during the night as again very few words were freely recalled by both groups with no significant difference in the number of words freely recalled.

Despite the study in Chapter 4 showing that recognition of auditory stimuli may occur in good sleepers, in Chapter 5 only the individuals with insomnia were able to significantly distinguish between words presented during the night and words that were not (foils) for both explicit and implicit recognition. As good sleepers appear to experience amnesia to stimuli presented during sleep onset (Wyatt et al., 1994), evidence from this Chapter suggests that those with insomnia may have impairments to this property of sleep onset which is proposed to be due to cortical arousal. However, similar to the study in Chapter 4, the recognition of auditory stimuli (words) was not associated with the discrepancy between subjective and objective sleep as would have been expected according to the propositions of the Neurocognitive Model. This suggests two distinct pathways resulting from increased cortical arousal: one that results in memory formation and one that alters the perception of sleep upon awakening.

6.4.1 How the Findings of Chapter Five Informed the Thesis

The unexpected improvement of some measures of sleep continuity within the insomnia group of Chapter 5 suggests that the administration of noise during wake to sleep transition points may have reduced cortical arousal and temporarily relieved the sleep symptoms of insomnia. It is possible that the noise acted as a distraction to the normal efforts to obtain sleep. Distraction techniques, such as imagery distraction, are often employed in cognitive behavioural therapy for insomnia and are known to improve sleep (Harvey & Payne, 2002). In the context of the present study, the auditory distraction could have altered the individuals' focus on attaining sleep to completing the experimental task. Thus, the auditory stimuli may have reduced cortical arousal and subsequently improved sleep. Additionally, the task may have increased cognitive load

prior to sleep in the good sleeper group, which in turn may have resulted in an increase in cortical arousal through somatic or cognitive means (Hall et al., 2004; Wuyts, De Valck, Vandekerckhove, Pattyn, Exadaktylos, et al., 2012). Therefore, as is proposed in the Neurocognitive Model, a potential increase in cortical arousal would explain the disruption to subjective sleep and the generation of sleep state misperception within the good sleeper group, albeit the manifestation of a positive misperception.

Rather than employing forced awakenings (as Perlis et al., 2001) or continuous noise throughout the night (Chapter 4) to increase cortical arousal, the study in Chapter 5 examined the point whereby one may infer there is an endogenous higher level of cortical arousal, the transition from wake to sleep during NREM. By finding no differences between those with insomnia and the good sleepers on the percentage of words correctly freely recalled the following morning, it could be suggested that cortical arousal does not interfere with the normal amnesia of sleep onset as there was no evidence off direct encoding of the auditory information. In the seminal work of Wyatt et al., (1994), normal sleepers show a severe detriment to the ability to freely recall information prior to sleep onset when tested 10 minutes post sleep, of which one reasoning was posited to be that sleep onset interferes with the ability to consolidate memory into long term memory. However, studies have indicated that increased cortical arousal might allow individuals to recognise that auditory stimuli have occurred during sleep. Portinoff et al., (1966) suggested that the ability to recognise information presented during sleep onset is more robust than the ability to freely recall stimuli and that recognition may be associated with higher levels of arousal, as the length of sleep onset post presentation associated with a higher percentage of recognition of stimuli by good sleepers. As only the insomnia group showed an ability to distinguish between words administered during the night and foils, it is suggested that the chronicity of the presence of cortical arousal aids this recognition memory beyond that of the good

sleepers experiencing an acute period of increased cortical arousal through the protocol.

The study within Chapter 5 provided a novel contribution to the literature by providing evidence that individuals with insomnia may experience greater implicit memory formation to stimuli presented during wake to sleep transitions. Within individuals with fibromyalgia Perlis, Giles, Bootzin, et al. (1997) argued that, due to a lack of explicit memory recognition for auditory stimuli, implicit memory of auditory stimuli presented during sleep onsets resulted in an observed disparity between subjective and objective sleep. Similar to explicit recognition memory, although both good sleepers and those with insomnia were able to implicitly recognise the same percentage of words that were presented during the night, only those with insomnia showed an ability to discriminate between words that were presented and foils, indicative of implicit recognition memory. Again, this suggests that a mechanism present in individuals with insomnia beyond heightened cortical arousal may be driving the process of recognition memory of auditory environmental stimuli during sleep. It could be that, the amount of high frequency EEG intrusion differs between those with insomnia and good sleepers under an acute period of arousal (novel bedroom and noise task).Again, as Portinoff et al., (1966) hypothesized, this higher level of arousal may be resulting in the recognition memory of environmental auditory stimuli.

Contrary to the Neurocognitive Model, the study in Chapter 5 did not find any associations between the encoding of the environmental stimuli (words), as determined through the recall and recognition (explicit and implicit) task administered post sleep and sleep state misperception. Therefore, the process of direct encoding of auditory stimuli may not result in an altered perception of sleep, but rather that th perception of sleep and memory formation of environmental stimuli during sleep may be two distinct products of cortical arousal. However, as the protocol in Chapter 5 assessed sleep immediately upon awakening which was then followed by the memory task, due to the

absence of direct encoding (recall) of the auditory stimuli, it is not surprising that there was no association with sleep misperception, as prompting for recognition memory had not occurred. As thus, the subjective report of sleep needs to be tested following the recognition task this would examine if recognition of noise can alter the daytime perception of prior sleep quality. If so, this could initiate safety behaviours if sleep is then deemed to have been poor along with appraisal of daytime functioning.

6.5 Clinical and Scientific Implications of the Findings

6.5.1 Implications of Findings for the Neurocognitive Model

The Neurocognitive Model is one of the least tested of the cognitive models of insomnia in current literature. It is the only model to define the etiology of sleep state misperception as a product of heightened cortical arousal. By examining cortical arousal (using noise), this thesis provides a novel ecological testing of the propositions of the Neurocognitive Model. Most people do not reside in sleeping environments similar to the controlled environment of a sleep laboratory. In ambulatory settings, noise is present whether we would like it to be or not, (as shown within the study in Chapter 3), and thus has the potential to disrupt sleep on a nightly basis, albeit without awakening the individual. The findings from this thesis tested three aspects of the Neurocognitive Model in that a) the ability to recognise sleep state during awakenings, b) the encoding of information during the sleep period, and c) the generation of sleep state misperception, are the mechanism by which heightened cortical arousal affects sleep.

The studies within this thesis identified that nocturnal noise is able to elicit cortical arousal and that vulnerability to cortical arousal is dependent on sleep stage. It was found that NREM sleep, specifically the wake to N1 transition points of sleep onset are periods whereby an individual may be more susceptible to the neurocognitive alterations said to occur through heightened cortical arousal. Therefore, the

propositions of the Neurocognitive Model of insomnia are specific to certain points in the sleep cycle. As thus, normal sleepers who have a significant number of awakenings (fragmented sleep), and thus wake to N1 transition points, may be at higher risk of experiencing alterations in sensory processing during sleep and thus greater sleep state misperception, providing support to the propositions of the Neurocognitive Model.

The Neurocognitive Model proposes that, through precipitating factors for insomnia, conditioned arousal to the bedroom occurs resulting in increased cortical arousal. The presence of cortical arousal is the factor that results in the processing to memory of environmental stimuli, which results in a misperception of sleep. This is supported by the evidence from Chapter 5 whereby the protocol acted as a distraction technique as the individuals with insomnia where going to sleep, as supported by the work of Harvey and Payne (2002). This distraction is proposed to have alleviated cognitive arousal prior to sleep and thus negated the effects of cortical arousal, resulting in a positive misperception of sleep.

This thesis evidences that the Neurocognitive Model may equally apply to good sleepers experiencing cortical arousal through a non-habituated environment. As the study in Chapter 4 highlighted, raised cortical arousal through the administration of novel noise removed the protection afforded through the habituation to the bedroom environment at home (as in Chapter 3) and a misperception of sleep onset occurred. Therefore, it is proposed that the Neurocognitive Model has a second application, distinct from the effects of conditioned arousal specific to insomnia, for vulnerable individuals whereby a precipitating event (i.e. high period of biopsychosocial stress) coupled with a novel sleep environment that is noisy, can result in a disruption to, and a misperception of sleep. This is supported by the study in Chapter 5, where the protocol may have increased cognitive load and subsequently cortical arousal in the good sleeper group resulting in a disturbance to sleep. Which in turn, the impact of raised cognitive load prior to sleep as a mechanism for disrupting sleep is supported by Wuyts

et al., (2012). This suggests that the presence of cortical arousal is a state characteristic that can occur in good sleepers, which may result in a vulnerability to the disturbance of sleep through noise via the propositions of the Neurocognitive Model.

Increased cortical arousal is proposed to result in an impairment of the sensory gating system which results in neurocognitive alterations, the first of which is said to be an alteration in the ability to perceive sleep “as sleep” prior to NREM awakenings. Although evidence suggests that it takes individuals with insomnia longer to recognise sleep onset has occurred than good sleepers (Hauri & Olmstead, 1983) and have a diminished sleep/wake discrimination by misperceiving the period before an awakening from sleep as wake (Knab & Engel, 1988; Mercer et al., 2002), this was not supported by the findings from Chapter 5. There was no difference between the individuals with insomnia and good sleepers on the perception of sleep state prior to natural awakenings, and furthermore the perceptions of sleep state prior to awakenings were predominately correct for both groups. This implies that increased cortical arousal does not alter the immediate perception of sleep or wake state and thus suggests revision of the Neurocognitive Model.

Increased cortical arousal is said to result in the ability to process information to long term memory of environmental stimuli occurring during sleep onset periods (wake to sleep transitions). Good sleepers are evidenced to exhibit deficits in memory to stimuli presented during sleep, suggested to represent a natural amnesia to environmental stimuli during this onset period (Portnoff et al., 1966; Wyatt et al., 1994). Furthermore, Portinoff et al., (1966) suggested that that recognition may be associated with higher levels of arousal, as the length of sleep onset post presentation of auditory stimuli associates with a higher percentage of recognition, of which Wyatt et al., (1997) implicated to be measured through beta EEG power (indicative of cortical arousal). Despite this, findings from this thesis did not provide evidence of any ability to directly encode (as measured through free recall tasks) auditory stimuli presented during

NREM and REM (Chapter 4) or during wake to sleep transition points (Chapter 5) as a result of increased cortical arousal, be that through the effect of non-habituated environment or as a trait of insomnia . Again suggesting an amendment of the Neurocognitive Model is required.

However, as indicated in the study in Chapter 4, long term recognition memory can form for auditory stimuli presented during the whole sleep period in good sleepers experiencing raised cortical arousal through a non-habituated environment. Further suggesting that the Neurocognitive Model can be applied to vulnerable good sleepers and a distinct pathway needs to be added examining the effects of novel environment on arousal levels. Although, this recognition (explicit or implicit) does not occur for stimuli presented during sleep onset periods from NREM (as Chapter 5), supporting the work of Wyatt et al., (1994) whereby they evidenced an inability to encode information during sleep onset periods in good sleepers. Therefore, increased cortical arousal does not alter the amnesia associated with sleep onset in good sleepers but potentially impairs an unknown property of the total sleep period when sleep protection via habituation to the sleeping environment is lost. One explanation may involve an alteration of the plasticity of sleep (as Espie, 2000) within in the good sleepers, and that the continuous noise not only raised cortical arousal but dampened the ability of the sleep system to respond to the continual interruption by noise over the course of the night.

Contrary to this, individuals with insomnia were shown to experience both explicit and implicit recognition of stimuli presented during wake to sleep transitions during NREM sleep. Along with the seminal work by Perlis et al., (2001), this suggests that individuals with insomnia, through heightened cortical arousal, have an increase in recognition memory to auditory stimuli occurring during sleep onset periods in NREM regardless of whether they are natural onset periods or from a forced awakening. As both good sleepers and individuals with insomnia were assumed to be experiencing increased

cortical arousal through the protocol, the findings suggests two differential effects of cortical arousal dependent on whether the arousal is through an acute period of heightened stress (state characteristic in the good sleepers) or whether the cortical arousal is present as trait vulnerability (those with insomnia). This trait vulnerability to heightened cortical arousal results in a perpetual impairment of the ability to habituate noise, as despite an improvement of sleep, individuals with insomnia could still process auditory stimuli during sleep onset periods.

Despite the measure whereby a misperception of sleep occurred, and the direction of the misperception (positive in Chapter 5, negative in Chapter 4), throughout this thesis, sleep state misperception was not associated with any ability to recall or recognise auditory stimuli that was presented during the night. Therefore, there are two potential implications for the Neurocognitive Model based upon these findings. Firstly, this suggests that an increase in cortical arousal (be that from a state characteristic or trait vulnerability) may result in two distinct processes: memory recognition and misperception. Secondly, as there was no identification of direct encoding of auditory stimuli through recall, and that subjective sleep was recorded immediately upon awakening, prior to the recognition task, it is possible that memory encoding may result in a negative appraisal of sleep during the day, a point which was not examined within this thesis. The Neurocognitive Model does not include daytime evaluation of sleep within the processes leading to insomnia, however if auditory stimuli is being processed through recognition memory during the sleep period, then hearing a similar stimulus during the day may result in a re-appraisal of sleep and daytime functioning. As in Harveys' (2002) Cognitive model, a negative appraisal of sleep and functioning during the day, can result in the instigation of safety behaviours aimed at protecting sleep, which can result in an actual deficit to sleep. Therefore, the work in thesis proposes that an extension of the Neurocognitive Model is required to examine the effect of

recognition on the daytime perception of sleep quality and functioning as a product of raised cortical arousal.

6.5.2 Implication of Findings to Insomnia Theory

As Öhström and Björkman (1988) suggested, susceptibility of sleep to noise-related sleep disruption is dependent on those who can habituate to noise or not and therefore fits within Espie's (2000) framework regarding plasticity and sleep. Habitation to noise must involve a response of the sleeping brain to the noise stimuli, be that an arousal response or the evocation of the sensory gating system. From the findings presented here, it could be suggested that there are two possible pathways following noise in the bedroom. Good sleepers, with good plasticity, may be conditioned to sleep within an environment with occasional noise. As sleep plasticity is the ability of the sleep system to respond to the external environment to maintain the good sleep process, a good sleeper would respond to auditory stimuli through the instigation of sleep protective mechanisms (such as sensory gating through the K complex). Conversely, those with insomnia may be conditioned to wakefulness through impaired sleep plasticity and heightened cortical arousal. Therefore, when the sleep is disturbed through noise, the sleep system cannot respond adequately (impaired plasticity) as the sleep protection mechanisms are said to be reduced, which results in memory processing of auditory stimuli. Similarly, for those who cannot habituate, the sleep switch (Saper et al., 2001) may be impaired, resulting in sleep instability and reduced sleep plasticity. This in turn may result in implicit encoding of environmental auditory stimuli due to cortical arousal, as neuronal columns remain in a wake-like state, as suggested in the Neurobiological model (Buysse et al., 2011). Certainly, the testing of this is beyond the scope of this thesis, but if the number of localised high frequency events is found to be associated with the ability to process memory, then a link could be drawn between neurobiological alterations during sleep as a product of cortical arousal and sleep state misperception.

The work presented in this thesis suggests individuals who experience sleep state misperception are likely to have a trait vulnerability towards faulty perception of sleep through increased cortical arousal, as this could be seen as the predisposing factor that lead to the altered perception of sleep. Whereas, those with learned psychophysiological insomnia, it is the conditioning response that may result in increased cortical arousal as postulated by the Neurocognitive Model. Therefore, in individuals with psychophysiological insomnia, heightened cortical arousal may be the state characteristic that results in sleep state misperception through the memory processing of environmental noise. However, as the studies in this thesis indicated, the Neurocognitive Model may be a model applicable to individuals vulnerable to cortical arousal (i.e. through a non-habituated environment) as opposed to just simply individuals with insomnia. Therefore, identification of those individuals that may develop cortical arousal and are thus vulnerable to sleep state misperception may be beneficial for insomnia clinicians due to the high persistence and relapse rates associated with insomnia syndrome (Jansson-Frojmark & Linton, 2008; LeBlanc et al., 2009; Morin et al., 2009; Morphy et al., 2007). Individuals who are vulnerable to insomnia may experience state characteristics, such as heightened arousal through stress, which can result in a period of increased cortical arousal. If these individuals sleep in a noisy non-habituated environment, as the study in Chapter 4, this may lead to the development of sleep state misperception, for example doctors who are sleeping in the hospital on call or flight crew on long haul flights. Whereas a trait vulnerability to cortical arousal is a persistent factor, state characteristics are transient events, be that biopsychosocial stress or simply a change in sleep environment, and therefore treatment needs to address this difference between individuals experiencing sleep state misperception.

6.5.3 Implication of Findings to Insomnia Practice

In current clinical practice, insomnia treatment is contingent on a diagnosis of primary insomnia through a subjective report of difficulties initiating or maintaining sleep (American Psychiatric Association, 2013) therefore, the objective assessment of sleep is not routinely carried out. If an examination of objective sleep is not included in patient assessment then the presence of sleep state misperception may be missed. As Wicklow and Espie (2000) indicated, noise is commonly reported by people with insomnia as a disruptor to sleep onset. Further, the monitoring of environmental noise is included within Harvey's (2002) cognitive model of insomnia and Ellis et al. (2002) proposed that, at least for elderly insomniacs, perceived nocturnal noise was the biggest complaint compared to good sleepers.

Therefore, in terms of treatment, the clinician administering Cognitive Behavioural Therapy for Insomnia (CBT-I) would be able to identify a relationship between a reported noisy bedroom (explicit encoding) and the sleep disturbance and therefore able to advise the removal of noise from the bedroom (for example through the wearing of earplugs). However, in the case of implicit encoding of noise, the stimuli that are disturbing sleep are not able to be reported to the clinician by the individual with insomnia, yet may still influence the subjective detriment to sleep. As thus, the treating clinician may wish to include noise stimuli control to ensure, despite the lack of explicit reporting of noise as a factor disturbing sleep, the influence of memory to auditory stimuli on sleep perception is reduced. For individuals that experience a misperception of sleep but where objective sleep remains normal (i.e. trait vulnerability), advising the use of earplugs to reduce noise every night may help facilitate accurate perception of sleep. Alternatively, those with psychophysiological insomnia would need to wear earplugs during treatment until re-conditioning of the bedroom environment has occurred and conditioned arousal has ceased. Similarly, individuals vulnerable to increased cortical arousal through sleeping in a non-habituated environment may wish

to use earplugs until they either return to their normal sleep environment or the state characteristic that is resulting in the disruption to sleep has ceased.

To address the presence of cortical arousal directly, sleep restriction may be of benefit. Conditioned arousal to the bedroom through an extension of time spent in bed is stated within the Neurocognitive model as the primary perpetuating factor that leads to increased cortical arousal. A recent study has evidenced that restriction of time in bed, not only improves sleep efficiency, but reduces the amount of beta EEG activity occurring in N2 and N3 in individuals with insomnia by (Vallieres, Ceklic, Bastien, & Espie, 2013). Therefore, sleep restriction therapy may be used to reduce heightened cortical arousal during sleep for individuals with insomnia experiencing sleep state misperception through increased cortical arousal.

Alternatively, for individuals' vulnerable to experiencing heightened cortical arousal prior to sleep, treatment could involve the utilisation of Neurofeedback therapy. Neurofeedback involves the patient being able to observe the EEG live and learning techniques to reduce the frequency of the brain waves whilst listening to an auditory tone stimulus (operant conditioning) (Hammond, 2007). When compared to Biofeedback (the observation and reduction of EMG activity analogous to muscle relaxation), Neurofeedback has been shown to significantly increase objective total sleep time for individuals with insomnia and improve subjective reports of sleep continuity (SOL, WASO,TST, and SE) (Cortoos, Valck, Arns, Breteler, & Cluydts, 2010). Cortoos et al., (2010) not only evidenced a link between cortical arousal and the perception of sleep, but also provided a potential treatment that can be practiced by individuals who are vulnerable to cortical arousal. Unlike sleep restriction, Neurofeedback is a tool that can be utilised by an individual when they perceive that they may be at risk of heightened cortical arousal, such as when experiencing a period of high cognitive demand or having to sleep in a non-habituated noisy environment.

6.6 Limitations

Although limitations specific to each study chapter have been discussed, some general limitations applicable to the whole thesis are discussed below.

6.6.1 Sample

One of the primary limitations of this thesis is the chosen sample of participants. As the primary aim of this thesis was to test the constructs of a model of insomnia, then only having one study utilizing individuals with insomnia could be classed as a limitation. However, it could be argued that to determine the implications of cortical arousal and to understand the processes present in good sleepers that aid the normal sleep process through habitation it is important to examine sleep before it is disordered. Furthermore, as Edinger and Fins (1995) stated, at least regarding sleep estimation ability, symptoms of insomnia may exist on a continuum, from normal sleep to severely disrupted sleep. As such, the processes that underpin good sleep are disrupted at varying degrees as the intensity of disruption to the normal sleep processes increases in individuals with insomnia.

In terms of gender, the studies employed an equal ratio of males and females. As discussed in Chapter 1, women are more likely to report insomnia or dissatisfaction with sleep during their lifetime than males and are also roughly twice as likely to experience clinically defined insomnia (Johnson et al., 2006; Leger et al., 2000; Ohayon, 1997; Ohayon, 2002; Ohayon & Sagales, 2010; Ohayon & Smirne, 2002; Pallesen et al., 2001). Therefore, it could be seen as a limiting factor that there was an almost equal split between genders within the three studies when application of the findings are to be made to primary insomnia. That said, as we were examining a potential aspect (i.e. cortical arousal) in the development of insomnia it is important to note that the ratio of males to females with acute insomnia is roughly equal (Ellis et al., 2012)

However, the key limitation within the sample over each of the three studies within this thesis is the size of the sample used. This indicates that potential significant findings may have been missed due to a lack of overall power in the sample, that being said the samples here are representative of similar work in the area, such as the fibromyalgia study by Perlis et al., (1997) which had a sample population of 20.

6.6.2 Measurements of sleep

The methods employed to examine sleep within each of the studies have clear benefits and limitations within each of the studies. Each method was chosen based on the balance between cost, time, and disruption to the participant.

6.6.2.1 Polysomnography

Although not used for the ambulatory study within chapter 3, the studies in Chapter 4 and 5 were conducted within the laboratory and therefore polysomnography could be utilised as the objective sleep measure. However, both studies that employed polysomnography as the objective sleep measure only focused on measures pertaining to sleep continuity and sleep architecture and did not utilise more sensitive measures, such as sleep microstructure or quantitative analysis of the EEG.

The Neurocognitive Model suggests that it is an increase in sensory processing during the night, which leads to insomnia, of which can be measured through the K complex (a marker of stage 2 sleep) as those with insomnia experience a diminished number of evoked K's to sensory stimuli (Hairston et al., 2010; Jahnke et al., 2012). Sleep spindles are another grapheoelement of sleep, with an increase in the presence of spindles shown to protect sleep from environmental auditory stimuli (Dang-Vu et al., 2010). Additionally the cyclic alternating pattern (CAP) is a measure of sleep instability, through short duration EEG changes within a staged epoch itself, that is proposed to be more sensitive than looking at the number of arousals or stage changes (Parrino et al., 2012). Moreover, as high frequency EEG, is said to associate with a poorer

subjective, but not objective sleep quality, thus is a potential measure of sleep state misperception (Krystal et al., 2002; Perlis, Smith, Andrews, et al., 2001). Power spectral analysis of the EEG, the analysis of the frequency of the sleep, could further the analysis of data from the studies within Chapters 4 and 5 allowing for a more sensitive determination of the level of cortical arousal within sleep (to be a discussed further in section 6.7.2).

However, the aim of this thesis was to determine where sleep might be vulnerable to disturbance through increased cortical arousal, specifically NREM versus REM sleep, along with examining sleep state misperception, which is determined through measures of sleep continuity. Therefore, as the studies within this thesis were examining the effect of cortical arousal as opposed to directly measuring it, analysis of the EEG for sleep continuity and sleep architecture was the most appropriate on the balance of cost, time, and questions that needed to be addressed over the studies in this thesis.

6.6.2.2 Actigraphy

Actigraphy was used throughout the thesis as a method of confirming compliance with study protocols whilst absent from the laboratory and to confirm sleep reports on the subjective sleep diary. However, the ambulatory study in Chapter 3 utilised Actigraphy as the sole objective sleep measurement. The sensitivity and specificity of Actigraphy has improved over recent years and reviews suggest it does have validity for use in good sleepers (Morgenthaler et al., 2007; Sadeh, 2011b; Sadeh & Acebo, 2002). Moreover, it is suggested that arousals due to environmental noise, if accompanied by a movement, can be measured using Actigraphy (Öhrström, 1995). Therefore, if there is no movement by the individual, then there could be a risk that an arousal could be determined as sleep, and thus Actigraphy may not be a sensitive enough measure, although it is one of the only possible objective sleep recording measures bar PSG.

Furthermore, as the study in Chapter 3 was examining a habituated sleep environment, by employing a minimally invasive technique to measure objective sleep ensured that the natural sleep environment was as undisturbed as possible.

6.7 Future Directions

6.7.1 Looking Beyond Sleep Continuity and Architecture in Insomnia

Although the Neurocognitive Model states that cortical arousal, as determined through high frequency EEG, is the overarching factor in the generation of sleep state misperception, it briefly alludes to sleep microstructure, the K complex, as the mechanism by which sensory gating is impaired allowing the processing of environmental stimuli. Throughout this thesis, the measures employed to analyse sleep were able to determine the overall impact of cortical arousal on sleep continuity and architecture however, by increasing the sensitivity of the analysis using power spectral analysis and perhaps the analysis of sleep microstructure, further discrimination between potential sensory gating and cortical arousal within the Neurocognitive Model could be elucidated.

6.7.1.1 The K complex

Although Bastien, St-Jean, Turcotte, Morin, Lavallee, Carrier, et al. (2009) suggest that there may be no differences between those with insomnia and good sleepers in spontaneously generated K complexes, there may be differences within the evoked K complex. Individuals with insomnia may have an inability to generate K complexes in response to external sensory stimuli during sleep (Hairston et al., 2010) occurring during stage 2 sleep in particular (Milner et al., 2009). Future research may wish to examine the generation of evoked K complexes when environmental noise occurs during sleep within good sleepers and people with insomnia. Is it possible that

individuals who can habituate to noise during the night have a stronger evoked K complex response than those who cannot habituate to noise?

Furthermore, as the perception of sleep onset was found to be altered, could diminished sensory gating during stage 2 sleep be responsible for the perception of an inability to initiate sleep in both good sleepers and those with insomnia? Future research may wish to address these questions, as well as examining the possible associations between the presence of K complexes and the ability to recall stimuli presented during sleep. The examination of associations between the K complex and memory formation to auditory stimuli would provide support for the sensory processing component of the Neurocognitive Model and the influence of heightened cortical arousal on the microstructure of sleep.

6.7.1.2 The Sleep Spindle

The spindle, similar to the K complex, is also a marker of stage 2 sleep and also thought to be a sleep protective mechanism, yet there is also no difference between those with insomnia and good sleepers occurring across the sleep period (Bastien, St-Jean, Turcotte, Morin, Lavallee, & Carrier, 2009). However, the number of spindles has been suggested to correlate with the ability to recall verbally encoded names prior to sleep (Clemens, Fabó, & Halász, 2005). Furthermore, the presence of spindles have been shown to have a protective sleep effect, with the number of spindles associating with a protection of objective sleep continuity when sleeping in a noisy non-habituated environment (hospital) (Dang-Vu et al., 2010). Although no difference between good sleepers and those with insomnia on the number of spindles, differences may lie between those who misperceive sleep and those who do not. In sleep sate misperception, sleep is not necessarily disturbed objectively, therefore, although spindles provide objective protection of sleep (as evidence by Dang-Vu et al., 2010), auditory processing may be occurring for noises occurring during natural awakenings (as Clemens et al., 2005), and thus, driving a disparity between the subjective report of

sleep and the objective sleep recording. Future research may wish to examine the role of the sleep spindle concerning memory processing, protection of objective sleep from noise, and the generation of sleep state misperception.

6.7.1.3 The Cyclic Alternating Pattern (CAP)

Although the Cyclic Alternating Pattern (CAP) is a known element of sleep, its use as a marker of sleep instability is currently under recognised in the literature (Parrino et al., 2012). Parrino and colleagues (2012) proposed that CAPs could be used as markers of sleep instability as they constitute minute changes in the pattern of the EEG and are independent of the transitions between sleep stages. Furthermore, CAPs have been shown to be induced with auditory stimuli presented during sleep in good sleepers and correlate with the perception of sleep quality (Terzano et al., 1990). Research suggests that the number of CAPs present during sleep are significantly higher in those who misperceive sleep than those who don't and that these differences in numbers of CAPs are found primarily during N1 and N2 (Parrino et al., 2009). Therefore, as the presence of the CAP may be associated with both the presence of nocturnal auditory stimuli and perception of sleep quality it would seem logical that future research address this element of sleep EEG in relation to the Neurocognitive Model of Insomnia.

6.7.2 Application to Future Studies

Firstly, as pharmacological agents can disrupt sleep microstructure and thus the sensory gating system, these agents afford the ability to directly examine the sensory processing component of the Neurocognitive Model. Benzodiazepines used to treat insomnia have been shown to increase the percentage of N2 sleep over the course of the night (Borbély, Mattmann, Loepfe, Strauch, & Lehmann, 1985). Although the characteristics of N2 sleep (spindles and K complexes) may afford sleep protection from noise perturbations, it must be noted that an increase in stage 2 sleep would occur at the expense of other sleep stages, notably REM, which may have a

detrimental effect on health. Furthermore, Benzodiazepines have also been implicated in the increase of beta EEG activity (Bastien, LeBlanc, Carrier, & Morin, 2003), which is suggested to be a biological marker of cortical arousal (Perlis, Merica, et al., 2001). By testing the ability to encode auditory information during sleep, using a population where both S2 and cortical arousal are present would allow determination of the factor(s) responsible for encoding during sleep.

Moreover, Benzodiazepines have been shown to increase the number of spindles during sleep whilst reducing the amplitude of the K complexes. (Borbély et al., 1983; Johnson, Hanson, & Bickford, 1976) This combination effect on sleep microstructure may result in sleep misperception as, although an increase in spindles may protect sleep from noise, the reduction in sensory gating by the dampened K complex in response to auditory stimuli may allow processing of sensory information that is then encoded to memory through the increase in spindles. Future research may wish to examine this in detail with regards to the Neurocognitive Model utilising a sample of people using benzodiazepines. This would demonstrate whether the K complex and sleep spindle negates the effect of cortical arousal in response to noise.

A second method is to examine the point of sleep where the effects of increased cortical arousal are said to occur by utilising individuals who have a reduction or absence of N2 sleep. As infants age, sleep architecture changes, with N2 sleep increasing at the expense of REM, occurring concurrently with a reduction in the number of awakenings (Louis, Cannard, Bastuji, & Challamel, 1997). This association with a reduction in awakenings further highlights the potential of the elements of N2 that provide sensory protection for sleep which may negate the effects of cortical arousal. Therefore, examination of the effects of noise on sleep in infants as they age could allow determination of potential associations between noise and memory formations concerning sensory gating during sleep. If novel auditory stimuli is administered during sleep, and memory is shown to be present (by using eye tracking

as a measure of stimuli recognition following awakening), inferences could be made regarding memory encoding during sleep and insufficiencies in sensory processing.

However conducting a study on infants may not be a feasible option, and therefore an alternative could be to employ a study utilising individuals with depression. Depression is said to result in an instability of NREM sleep as measured through the cyclic alternating pattern (Farina et al., 2003) which, as suggested above, may be associated with an inability to habituate to noise and thus result in sleep misperception. Furthermore, individuals with insomnia co-morbid with major depressive disorder have been shown to also experience an increase in high frequency EEG activity (in the beta and gamma range) during sleep, albeit with less intrusion into normal sleep architecture than those with primary insomnia (Perlis, Kehr, et al., 2001). By comparing individuals with untreated depression co-morbid with insomnia with good sleepers, inferences could be made regarding the role of cortical arousal, sensory gating, and sleep state misperception. Individuals with insomnia co-morbid with depression, due to higher levels of cortical arousal, would experience higher explicit and implicit recognition for words presented during the wake to sleep transitions than good sleepers, but less than individuals with primary insomnia where cortical arousal is posited to be at its highest, as Perlis, Kehr et al., (2001) suggests. Furthermore, as the study in Chapter 5 did not show associations between memory processing and sleep state misperception, if the findings from individuals with co-morbid insomnia do, then one could infer that instability of sleep is the factor relating to sleep state misperception through encoding of environmental stimuli during sleep.

6.8 Final Conclusions

This thesis provided a novel contribution to the literature by empirically testing the Neurocognitive Model of insomnia, examining the relationship between cortical arousal,

the ability to form memories of external stimuli during sleep, and the generation of sleep state misperception. The studies in this thesis highlighted that lighter stages of sleep may be endogenously susceptible to cortical arousal, and it would appear that it is the transition from wake to sleep where neurocognitive alterations are most likely to occur. Furthermore, it would seem that the memory formation component of the Neurocognitive Model and sleep state misperception are two distinct, unrelated aspects as no associations were found, across the three studies, between sleep state misperception and memory formation of auditory stimuli occurring during the night. It is proposed that the Neurocognitive Model of insomnia is in fact a model of cortical arousal applicable to good sleepers and those with insomnia. It could be that susceptibility to cortical arousal can result from either state factors or be a trait vulnerability, of which can be measured utilising the effects of auditory stimuli on sleep. Moreover, the findings highlight the importance of controlling noise at night and thus current treatment for insomnia may wish to not only re-iterate the importance of noise control within the sleep hygiene component of CBT-I but also examine individuals for the presence of increased cortical arousal and treat accordingly to protect against both the memory processing of environmental stimuli and the development of sleep state misperception. The research presented within this body of work suggests that implicit encoding and cortical arousal may be impairing the ability of the sensory gating system to appropriately respond to auditory stimuli creating a potential target for future research and treatment.

Future research may want to address the points raised in this body of work and continue to examine the role of environmental noise in sleep perception and insomnia, as it is possible that some individuals perceive themselves to be kept awake by bleats whilst they are counting sheep.

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Appendix

Appendix A: Eligibility Questionnaire first used in Chapter 3

Date:_____
Name of potential participant:_____

Please answer the following questions so that it can be determined whether or not you meet the criteria to take part in this study.

1. SLEEP DIFFICULTIES

Have you ever had a sleep problem: __Yes __No **(If no go to section 2)**

If yes, Is this an ongoing problem at the moment? __Yes __No

How long have you had the sleep problem or how long did it last? _____Years _____Months

What is the nature of your sleep problem?

Getting off to sleep / Staying Asleep / Waking too early / Feeling unrefreshed on waking / Sleeping at odd times / Excessive daytime sleepiness

2. OTHER SLEEP/WAKE DISORDERS

Have you travelled over three time zones in the last six months? __Yes __No

Have you or your spouse/bed partner ever noticed one of the following in the last month:

A. RESTLESS LEGS: Crawling or aching feelings in the legs (calf) and inability to keep legs still ; __Yes __No
B. PERIODIC LEG MOVEMENTS: Leg twitches or jerks during the night; __Yes __No
C. APNOEA: Snoring, pauses in breathing at night, short of breath, chocking at night; morning headaches, chest pain, dry mouth; __Yes __No
D. NARCOLEPSY: Sleep attacks, sleep paralysis, hyp. hall., cataplexy; __Yes __No
E. GASTRO-ESOPHAGEAL REFLUX: Sour taste in mouth, heart burn; reflux; __Yes __No
F. PARASOMNIAS: Nightmares, night terrors, sleep walking, sleep talking; __Yes __No
G. SHIFT WORK DISORDER: Rotating work patterns __Yes __No

3. NATURE OF SLEEP/WAKE DIFFICULTY

How much sleep do you think you need to function during the day ____Hours ____Minutes

How much sleep do you get on a typical weekday _____Hours ____Minutes

How much sleep do you get on a typical weekend _____Hours ____Minutes

4. GENERAL HEALTH

Do you consider yourself to be in good health? __Yes __No
Do you suffer from an ongoing illness (e.g. Asthma or Diabetes?) __Yes __No
If yes, could you specify?

_____

Are you currently on any medication? __Yes __No

-If yes, what, and at what dose_____

Have you suffered a head injury? __Yes __No
Do you have a history or current complaint of depression or anxiety ___Yes ___No

5. DEMOGRAPHICS

Address:_____

_____

_____

Post code:_____ E-mail-address:_____

Telephone: _____ Mobile phone:_____

Preferable time for future contact (check one): Morning __ Afternoon __ Evening __

Date of birth:_____ Age:_____ Gender: Male__ Female__

I agree to be contacted in the future from the Northumbria Centre for Sleep Research?__Yes__No

What would be your preferred medium to receive the questionnaires, sleep diaries and more information regarding this study? __Online (internet access __Hard copies

Now please e-mail this questionnaire back to rachel.sharman@northumbria.ac.uk so that your eligibility to take part in the study can be assessed. Once you have done this the researcher will be in touch with you shortly. Please send any questions to the same e-mail address.

Thank you for your time!

Appendix B: Demographic questionnaire first used in Chapter 3

ABOUT YOU

1) Your date of birth: -------/----------/----------

2) Your postcode: _____

3) Are you: (please tick) Male □ or Female □

If you are female, would you describe yourself as: **(please tick box)**

Pre-menopausal □

Experiencing the menopause now □

Post-menopausal □

4) Which of the following best describes your ethnic background?

Please tick one of the following groups, if not applicable please specify your ethnic origin

White	□	Pakistani	□
Black – Caribbean	□	Bangladeshi	□
Black – African	□	Chinese	□
Black – Other	□	Asian – Other	□
Indian	□	Other– please specify _____	□

5) Which of the following best describes your marital status?

Please tick one of the following.

Single	□	Widowed	□
Married	□	Divorced/Separated	□
Living as married		Other (please specify) _____	□

6) How tall are you? _____(feet) _____(inches) or _____(cm)

7) How much do you weigh?_____(stone)_____(pounds) or_____(kg)

8) Are you **currently**: (please tick as many boxes as apply

In active paid work	□	Unemployed and seeking work	□
Retired	□	Unemployed due to illness or disability	□
Doing voluntary work	□	At home doing housework	□
Full time student	□	Other (please specify) _____	□

9) Please complete for present or last paid job (for retired or unemployed)

Job title:_____

10) Is / was this job: Full time □

Part time □

11) Major activities in job (e.g. tasks, level/grade, and qualifications):

_____

_____

Employee □

12) Are / were you an:

Employer/Manager ☐
Self-employed ☐
Supervisor/Foreman ☐

13) How many people do/did you supervise? _____

14) How old were you when you left school? _____

15) Do you have any of these qualifications? (Please tick all that apply)

CSE / O levels / School Certificate / GCSE	☐	City and Guilds Certificate	☐
A levels / Highers / BTEC	☐	Recognised Trade Apprenticeship	☐
HND	☐	Clerical / Commercial Qualification	☐
First degree (BA, BSc etc)	☐	Higher Degree (MSc, PhD etc)	☐
Medical / Nursing / Teaching qualifications	☐	Membership of Professional Institute	☐
		Other (Please describe) _____	☐

ABOUT YOUR HEALTH

16) In the last 12 months, have you been diagnosed with / experienced any of the following health conditions?

(Please tick as many conditions as apply)

- ☐ Depression
- ☐ Anxiety Disorder
- ☐ Excessive Worry
- ☐ Bipolar Disorder
- ☐ Dementia
- ☐ Insomnia
- ☐ Head Injury
- ☐ Haemorrhage
- ☐ Meningitis
- ☐ Migraine
- ☐ Gastric Bleeding
- ☐ Pancreatitis
- ☐ Heartburn
- ☐ Oesophageal Reflux
- ☐ Cystitis
- ☐ Kidney Stones
- ☐ Menopause
- ☐ Hives or Rashes
- ☐ Dental Problems
- ☐ Sleep Apnoea
- ☐ Restless Legs
- ☐ Liver Disease
- ☐ Colitis
- ☐ Constipation
- ☐ Pneumonia
- ☐ Tuberculosis
- ☐ Gastric Ulcer Disease
- ☐ Cancer
- ☐ Diabetes
- ☐ Multiple Sclerosis
- ☐ Thyroid Problems
- ☐ Parkinson's
- ☐ ADHD
- ☐ Obesity
- ☐ Seizures
- ☐ Gout
- ☐ Stroke
- ☐ Arthritis
- ☐ Shingles
- ☐ Fibromyalgia
- ☐ Chest Pain
- ☐ Chronic Fatigue Syndrome
- ☐ Psoriasis
- ☐ Irregular Heart Rhythm
- ☐ Ovarian Cysts
- ☐ Congestive Heart Failure
- ☐ COPD
- ☐ Pelvic Inflammatory Disease
- ☐ Heart Attack
- ☐ Grinding Teeth
- ☐ Vision Problems
- ☐ Hearing Problems
- ☐ Tinnitus
- ☐ Speech Problems
- ☐ Blood Clots
- ☐ Blood Disorders
- ☐ Asthma
- ☐ PTSD
- ☐ Chronic Pain
- ☐ Flu
- ☐ Headaches
- ☐ Tropical Disease
- ☐ Phobia
- ☐ Obsessive Compulsive Disorder
- ☐ Other

Appendix C: Drug use questionnaire first used in Chapter 3

Drug	**When did you <u>first</u> use?**	**When did you <u>last</u> use? (Please circle one only)**				
	mm/yy	**Hours Previous**	**Days Previous**	**Weeks Previous**	**Months Previous**	**Years Previous**
Alcohol			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Tobacco			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Ecstasy (MDMA)			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Amphetamine			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Cannabis			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Cocaine			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Crack			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Heroin			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Ketamine			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
LSD (Acid\Blotters)			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Mushrooms			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Poppers			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Salvia Divindrum			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10
Other			0 1 2 3 4 5 6	1 2 3	1 2 3 4 5 6 7 8 9 10 11	1 2 3 4 5 6 7 8 9 10

Appendix D: Sleep diary example first used in Chapter 3

Day 1

Please complete today's date ___ / ___ / ____

	MEASURING THE PATTERN OF YOUR SLEEP	
1	What time did you wake this morning?	
2	At what time did you rise from bed?	
3	At what time did you go to bed last night?	
4	Lights Out: At what time did you put the lights out to go to sleep?	
5	How long did it take you to fall asleep (minutes)? (After Lights Out)	
6	How many times did you wake up during the night?	
7	How long were you awake during the night (in total)?	
8	About how long did you sleep altogether (hours/mins)?	
9	How many sleeping pills did you take to help you sleep?	

	MEASURING THE QUALITY OF YOUR SLEEP				
	not at all		**moderately**		**very**
10	How well do you feel this morning?				
	0	1	2	3	4
11	How enjoyable was your sleep last night?				
	0	1	2	3	4
12	How mentally alert were you in bed last night?				
	0	1	2	3	4
13	How physically tense were you in bed last night?				
	0	1	2	3	4

Read each statement then mark on the line at the most appropriate point to indicate
how you feel right now, at this moment

I feel calm* ——————————————————
not at all very much

I feel tense* ——————————————————
not at all very much

I am upset* ——————————————————
not at all very much

I feel relaxed* ——————————————————
not at all very much

I feel content* ——————————————————
not at all very much

I feel worried* ——————————————————
not at all very much

I feel stressed ——————————————————
not at all very much

Appendix E: Noise stimuli administered to participants in Chapter 4 in playlist order

Nocturnal Noises
Wind on the Windows
Clock ticking
House Creaking
Toilet Flushing
Phone Ringing
Rain on the Window
Tap Dripping
Cats Fighting
Car Alarm
People Arguing
Cat Meowing
Keys Rattling
Police Car Siren
Rain Falling
Music from Neighbours
Traffic
Fridge Humming
Fire Engine Siren
Dog Barking
Ambulance Siren
Fireworks
Birds Chirping
Hail Stones falling
Wind in the Trees
Washing Machine
Thunder Clap
Gravel Being Blown
TV Static
Litter Being Blown
Milk Float

Appendix F: Noise stimuli administered to participants in Chapter 5

Foils			Trial 1	Trial 2	Trial 3	Trial 4
baffle	fuel	tabby	barter	beast	bamboo	badger
baggage	garbage	tadpole	bleach	beggar	booze	cashew
bandit	gravel	tap	blush	blurb	butler	drain
barrack	geyser	tape	branch	bully	camel	greed
biscuit	goblin	tar	caress	cactus	denture	hermit
blade	graft	tattle	cave	casket	detract	hunt
blazer	graze	tingle	clown	crate	ferret	kink
bleed	grime	toad	cog	drench	fleck	sapphire
blindfold	grit	tongs	couch	drip	foe	slush
bouquet	gum	tongue	crib	flagon	freeze	squab
cabbie	gutter	topcoat	crook	frosting	graveyard	starve
cake	hag	toss	crust	glob	grove	tarnish
calf	halo	tread	daze	growl	hallo	teak
cascade	hammock	trickster	dealer	hamper	hick	teal
cedar	harpoon	trombone	devil	holly	hump	tent
clang	harpy	trunk	dolly	horror	secludes	tombstone
clap	heather	tube	dolphin	scour	smudge	tooth
claw	hemlock	tuna	dream	slur	snitch	tort
cliff	henna	twig	dryer	toupee	spud	townsman
crow	hockey	wheat	fleece	trample	trail	twist
cub	hound		flood			
dagger	sap		garlic			
daisy	sappy		glow			
deafen	sausage		halter			
detach	scallion		harass			
detain	scissors		seamstress			
doll	scuffle		siren			
donkey	skewer		skate			
dosage	skim		slay			
drank	sled		smother			
dredge	slime		sprain			
drift	slink		stalk			
duct	snail		tacky			
feather	snare		termite			
feline	snicker		tin			
flap	snob		torch			
flaw	sob		trash			
floss	spout		troop			
frown	squawk		turnip			
fruit	tab		whisker			