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The dark side of artificial light: Examining the perception and intensity of light at night in the sleeping environment and its association with sleep, circadian rhythmicity, attention bias and psychological health.

Michael T. Cleary-Gaffney, B.A. (Hons), M.Sc.

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Head of Department: Dr. Michael Cooke
Research Supervisor: Prof. Andrew Coogan

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Summary of Thesis

Due to technological advances and affordability exposure to Light-at-night (LAN) is now ubiquitous both inside our private dwellings and our external environment. While LAN provides us with the agency to extend our biological day into the evening this may lead to adverse consequences for circadian rhythmicity and the timing of sleep. Additionally, LAN exposure may lead to adverse health consequences either indirectly via circadian disruption or directly impacting on health. Technological advances in both the accessibility and quality of lighting have outpaced our understanding of the impacts of LAN on sleep and health. The association between home-setting LAN with sleep and psychological health is unclear. This study specifically examines LAN exposure in the sleeping environment and its association with sleep and psychological health. Using both cross-sectional and ecological study designs this research seeks to examine what are the perceived sources and intensity of LAN which individuals are exposed to in their sleeping environment. What is the association between these sources with sleep timing, circadian misalignment and psychological health.

Our research indicates that LAN is perceived from a variety of sources in the sleeping environment. Those that perceive LAN are more likely to report that these sources are disruptive to their sleep. We report for the first time that the subjective perception of external LAN is associated with both poor sleep quality and psychological health. Due to limitations of satellite image data we employed a novel approach to objectively measure external light pollution individualized to the dwelling of the participant. We report no association between the subjective perception of LAN and objective measurements. This research examined whether the association between the subjective perception of LAN and poor sleep quality despite equally comparable levels of outdoor LAN to those that do not perceive it was due to an attention bias towards sleep related information. Our results report no significant effect of the perception of LAN in the sleeping environment with attention bias towards sleep-related word stimuli or towards images depicting LAN sleep environments. Finally, this study examined whether objectively measured LAN intensity measured from the window and bedside was associated with alterations to sleep timing, sleep quality and rest-activity patterns derived from actigraphy. Our results report that window LAN intensity is associated with delayed timing of L5 and sleep onset. However, bedside LAN is not associated with any alterations to our

outcome variables. Our research also highlights that increased LAN intensity is not associated variance in sleep timing, quality, daily mood or daily subjective sleepiness.

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Chapter 1:
Literature Review

1.1 Circadian rhythms

The circadian clock generates self-sustaining, cell autonomous oscillations with an endogenous periodicity of approximatively 24-hour (Reppert & Weaver, 2002). These rhythms allows organisms to adapt and synchronise their physiology to the daily cycle of sunlight and darkness (Gallego & Virshup, 2007; Redlin, 2001). These cell-autonomous clocks are ubiquitous throughout the body with clock genes found in almost all tissues within the body. It is argued that this periodicity of oscillations have occurred in response to the daily solar cycle of light and darkness. This internalisation of the solar 24-hour rhythm has been proposed to be beneficial to the organism in order to predict daily recurring events (Albrecht, 2012). In order to synchronise each of the individual oscillating clocks so that physiology and behaviour are regulated, the circadian system must continuously adapt to the ever-changing environmental parameters to synchronise the external environment and the body's internal oscillators. This is achieved primarily with photic information pertaining to the external environment being integrated and appropriate signals are passed to the various tissues which are involved in regulating physiology and behaviour. Given that each individual cell contains circadian clocks these individual oscillators need to be synchronised to each other in order to generate circadian oscillations which are coherent, robust, and importantly, in phase with itself and the external environment (Albrecht, 2012).

1.2 The Central Oscillator: The SCN

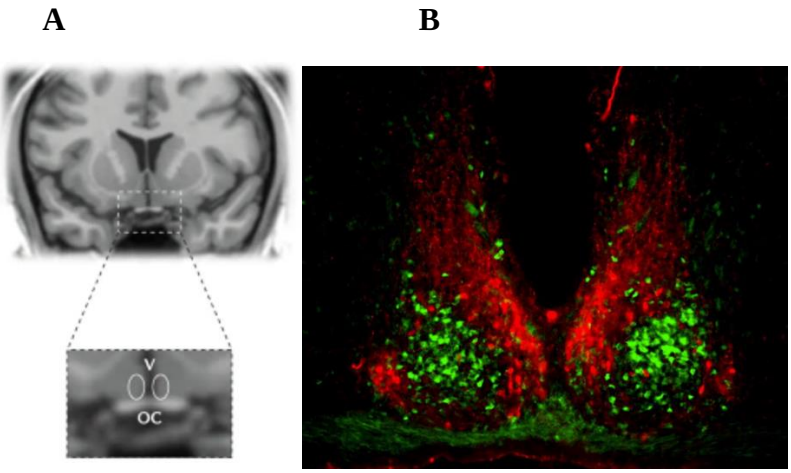
In mammals, the circadian system is a hierarchical network with the suprachiasmatic nucleus (SCN) serving as the master pacemaker. The SCN synchronises and entrains subordinate peripheral clocks distributed throughout the body with each other and to the 24-h day (Albrecht, 2012; Hastings et al., 2003). Early evidence suggesting the SCN was the master clock within the circadian system in mammals comes from lesion and transplant studies. In rodents, complete lesions to the SCN results in abolishment of circadian rhythmicity at both a behavioural (Stephan & Zucker, 1972) and endocrine level (Moore & Eichler, 1972). SCN grafts from wild-type mice restored circadian rhythmicity in genetically arrhythmic mice (Sujino et al., 2003). Partial restoration of circadian rhythms of rest-activity rhythms occurs when animals who have had their SCN lesioned have an implantation of

foetal SCN tissue (Lehman et al., 1987). These studies indicate that the SCN is involved in circadian rhythmicity. In animals who have either genetically short or long circadian periods, the period of the rhythm is determined by the genotype of the SCN donor and not that of the SCN lesioned host indicating that the recipient animal expresses the period and the phase of entrainment of the mutant donor (Ralph et al., 1990). When the SCN is isolated *in vivo* or *in vitro* it possesses an ability to generate circadian rhythms in spontaneous neuronal firing (Green & Gillette, 1982; Inouye & Kawamura, 1979) which still occur after 3 weeks when hosted in culture (Bos & Mirmiran, 1990). This indicates that SCN neurons display an ability to generate stable, self-sustained oscillations which have an intrinsic ability to generate circadian rhythms in electrical activity. Loss of the SCN results in peripheral circadian clocks to become desynchronised (Yoo et al., 2004) supporting the view that the SCN is the site of the master clock.

The master central pacemaker is located at the SCN of the hypothalamus (see Figure 1.1). The SCN is a bilaterally paired nucleus which is made up of tightly compacted, small diameter neurons which are located just lateral to the third ventricle above the optic chiasm in the mammalian brain (Van den Pol, 1980). The SCN is comprised of approximately 20,000 neurons each of which contain a cell autonomous circadian oscillator. Each of these SCN neurons can generate independent circadian oscillations of neuronal firing and clock gene expression indicating an ability to be autonomous circadian oscillators. Individual neurons show different circadian periods when isolated *in vitro* (Bos et al., 1990). However, through coupling the SCN functions as a network in which the population of SCN cells are coupled together and oscillate in a coherent manner (Herzog, 2000). This leads to a system which has a consistent period, is robust, can compensate for certain genetic defects and produce a precise, coherent output signal to other peripheral oscillators (Welsh et al., 2010).

Figure 1.1

Image (A) provides a coronal MRI image of the human brain. The highlighted area of the box denotes the suprachiasmatic nucleus. Image (B) displays a coronal section of the mouse SCN. The ventral core region is denoted in green, and the dorsal shell region is denoted in red.



Note. Image A taken from Hastings et al. (2018). Image B taken from Welsh et al. (2010).

The SCN is divided into two regions which include the ventral (core) which abuts the optic chiasm and the dorsal (shell) region which partially surrounds and receives information from the core (Figure 1.1). The core has multiple projections to the shell however, the projections from the shell to the core are sparse (Leak et al., 1999). The core region serves to receive and organise external input by receiving information from the three major pathways which include the retinohypothalamic tract (RHT), the geniculohypthalamic tract (GHT) from the intergeniculate leaflet (IGL) of the thalamus and projections from the raphe nuclei (Morin & Allen, 2006). By integrating the external information, the core neurons communicate this information to the rest of the SCN. The sensory processing cells of the core of the SCN display low amplitude rhythms in clock gene expression. Neurons within the shell display robust circadian oscillations of the clock genes (Colwell, 2011). The core also imposes the period of the rhythm with the shell when isolated, resulting in a shorter period, however, when the shell is attached to the core a longer period is expressed (Noguchi et al., 2004). The core region is critical for the maintaining coupling within the SCN with selective lesions to the core resulting in abolishment of circadian rhythms of locomotor activity, body temperature, heart rate, melatonin, and cortisol (Welsh et al., 2010). This indicates that the core is critical for coupling

of the SCN neurons and without it no coherent output signal would be generated from the SCN. The neurons in the subdivisions of the SCN are distinguished by their neurochemical content with the core regions expressing vasoactive intestinal peptide (VIP) and gastrin-releasing peptide while the shell regions express arginine vasopressin and met-enkephalin. In most SCN neurons, expression of the neurotransmitter GABA is found. Most core projections terminate on shell neurons indicating that the shell is responsible for the outgoing circadian signal by synchronising extra-SCN targets and peripheral oscillators to the master clock (Rosenwasser et al., 2009). There appears to be an interplay between SCN subdivisions being responsible for the output of circadian information from the SCN (Antle & Silver, 2005). Output information which is either neuronal or humeral signals from the SCN's subdivisions are projected to other hypothalamic regions which include the subparaventricular zone (sPVZ) and the dorsal medial hypothalamus (DMH; Colwell, 2011). From there, signaling is passed throughout the nervous and endocrine system allowing for numerous pathways by which the SCN can pass temporal information to the brain and body (Dibner et al., 2010). However, it is important to note that the SCN intrinsic anatomy and neuropeptides vary across species and are more complex than simply characterizing into core and shell regions.

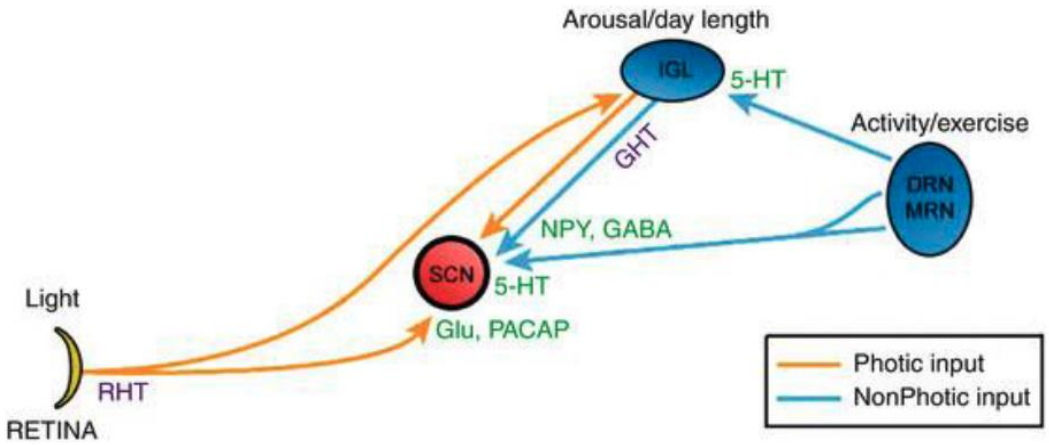
1.2.1 Afferent input to the SCN: Information from the environment

The way in which external time-cues are integrated within the circadian system involves afferent projections to the SCN core. The SCN receives projections from three input pathways which include the RHT which mediates photic information, the GHT and serotonergic input from both the dorsal raphe nucleus (DRN) and median raphe nucleus (MRN). Both the GHT and the DRN pass non-photic information to the SCN (Dibner et al., 2010). Ablation to the serotonergic pathways results in diminished entrainment response to a number of non-photic cues (Challer et al., 1997). Retinal projections directly target the SCN transducing the photic information from the optic nerve via the RHT. However, the RHT also projects photic information indirectly to the SCN via photic information passing directly to the IGL which can in turn pass information to the GHT and subsequently the SCN (Figure 1.2). This indicates that the SCN receives photic information from two different pathways. Pickard (1989) argues that the IGL only plays a modulatory

role in the regulation of photic entrainment and rhythm generation. Both the IGL and GHT act as secondary routes for passing photic signaling to the SCN with circadian responses only being attenuated following lesions to these areas (Johnson et al., 1989; Pickard et al., 1987). In addition, the IGL receives non-photic signaling from the DRN and passes this to the SCN (Figure 1.2; Dibner et al., 2010). Entrainment signals pertaining non-photic zeitgebers such as novel wheel locomotion or benzodiazepine treatment have also been found to be impacted by lesions to the IGL (Wickland & Turek, 1994). This indicates that the IGL plays a significant role in the integration of both photic and non-photic signals to entrain the SCN.

Figure 1.2

Schematic image illustrating the main pathways to the SCN.



Note. The orange line denotes the photic pathways to which light information is passed directly and indirectly (via the IGL to the SCN). The blue arrows represent how nonphotic input is directed to the SCN. Image taken from Dibner et al. (2010).

1.2.2 Output efferent pathways from the SCN

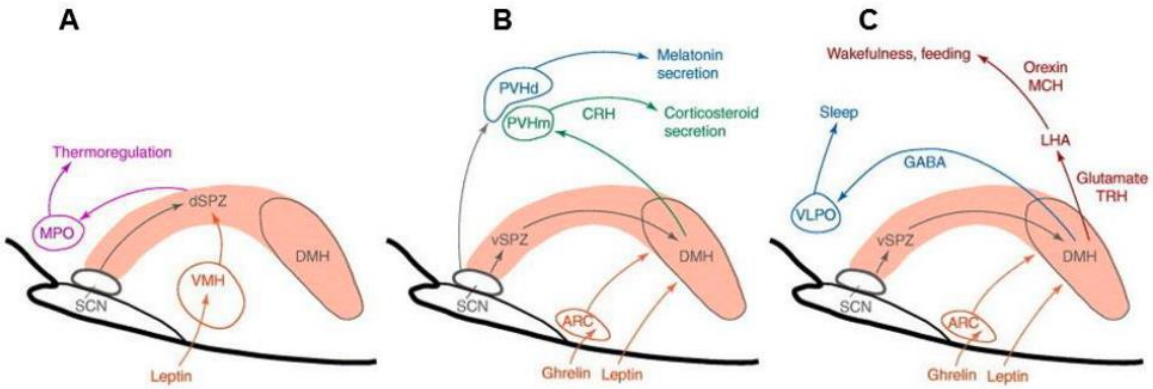
The SCN has a wide number of efferents which terminate in a range of brain areas (Dibner et al., 2010). In the hypothalamus, the SCN efferents densely terminate at the ventral subparaventricular zone (vSPZ), the dorsal subparaventricular zone (dSPZ) on the dorsomedial nucleus of the hypothalamus (DMH), rostrally in the preoptic area, the bed nucleus of the stria terminalis and the lateral septum. SCN

neurons project to the dSPZ where information is relayed to the medial preoptic region to control the circadian rhythm of body temperature which is a key phase marker of the circadian system. Lesions to the dSPZ results in attenuation or abolishment of the body temperature circadian rhythms however, other circadian outputs remain intact (Lu et al., 2001). The SCN projects information to the DMH via the vSPZ which relays this information to the paraventricular nucleus (PVHm). The PVHm is critical in the rhythmic secretion of corticosteroids from the pituitary. Disruption to these circuits has a negative impact on endocrine signaling which is essential for the coupling between the SCN and the peripheral clocks (Saper et al., 2005). The SCN projects to the dorsal parvicellular paraventricular nucleus (PVHd) which provides a signal pathway to the intermediolateral column of the upper thoracic spinal cord and the preganglionic neurons. The role of this pathway is to control the secretion of pineal melatonin (Teclemariam et al., 1999).

Non-photic entrainment of peripheral oscillators can be mediated through the rhythms of wakefulness and appetite. For example, projections from the SCN to the DMH via the vSPZ are involved in sleeping and feeding patterns while projections from the DMH to the ventrolateral preoptic nucleus (VLPO) promote the rhythm of sleep (Figure 1.3). Lesions to the dSPZ and DMH results in significant attenuation of rhythms of the sleep/wake cycle, feeding, locomotor activity (Chou et al., 2003).

Figure 1.3

Circadian signaling via efferent projections from SCN to hypothalamus.



Note. (A) Circadian rhythms in core body temperature are controlled via SCN signaling to the medial preoptic area (MPO) of the hypothalamus via projections to the dorsal sub paraventricular zone (dSPZ). (B) Hormonal factors under circadian control such as melatonin and cortisol are controlled via SCN efferents to the dorsal paraventricular nucleus (PVHd) and dorsomedial nucleus of the hypothalamus (DMH) respectively. PVHd communication runs to the superior cervical ganglion in the upper spinal cord and innervates the pineal gland which secretes melatonin. Cortisol secretion is under pituitary control the circadian rhythm of which is gated via corticotrophin releasing hormone (CRH) neurons in the PVHm. (C) Sleeping and feeding patterns are influenced by SCNvSPZ-DMH projections to the ventrolateral preoptic nucleus (VLPO) and the lateral hypothalamus (LHA). Neurotransmitters involved in these circuits are γ-aminobutyric acid (GABA), thyrotropinreleasing hormone (TRH), glutamate, melanin concentrating hormone (MCH), and orexin. Figure adapted from Saper et al. (2005).

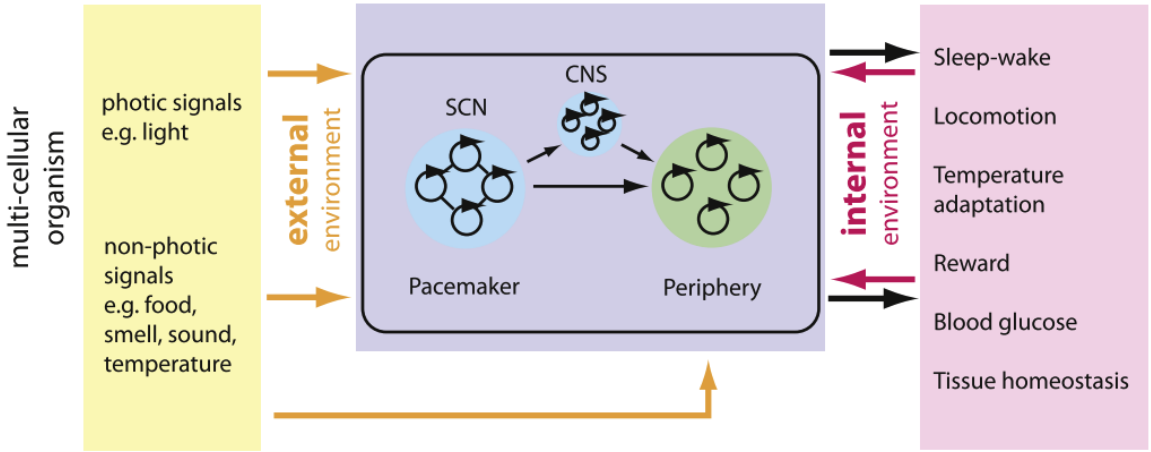
1.2.3 Output from the SCN: Passing signaling information throughout the body

For the entire organism to be synchronised in a coherent fashion this requires signal output from the SCN to be transmitted to oscillators in periphery tissues. The SCN projects to several brain areas which themselves display daily oscillations which direct behavioural, autonomic, and neuroendocrine circadian rhythms (Hastings et al., 2018). The local clocks within the various brain areas then pass signaling cues which are mediated by autonomic neural pathways and hormones to entrain the local molecular clocks of peripheral tissues (i.e. heart and liver), with these local clocks resulting in direct local programs of circadian gene expression which is involved in the regulation of rhythms which are critical for human health (Hastings et al., 2018). Thus, non-SCN tissues are primarily entrained via neural and

endocrine signals which originate from the SCN (Figure 1.4; Dibner et al., 2010). Peripheral clocks can also be entrained through the modulation of body temperature, exercise and feeding behaviour (Patke et al., 2020; Welsh et al., 2010). It is important to note that signals from the internal environment such as the rhythms of rest and activity can become inputs to other peripheral oscillators which in turn influence the master clock (Figure 1.4; Albrecht, 2012). This suggests a complex interconnectivity between the circadian system and the biological outputs to which it controls. Independent to the SCN, local signaling pathways can also influence other peripheral oscillators (Mohawk et al., 2012).

Figure 1.4

Schematic outlining the subdivision of the circadian system.



Note. Yellow box denotes the input to the clock which are the photic and nonphotic zeitgebers which entrain the circadian clock. The lilac box denotes the clock mechanism which includes the SCN passing signaling information the peripheral clocks via direct and indirect pathways. The purple box denotes the clock output. Clock output can pass signaling information back to the peripheral clocks. Image taken from Albrecht (2012).

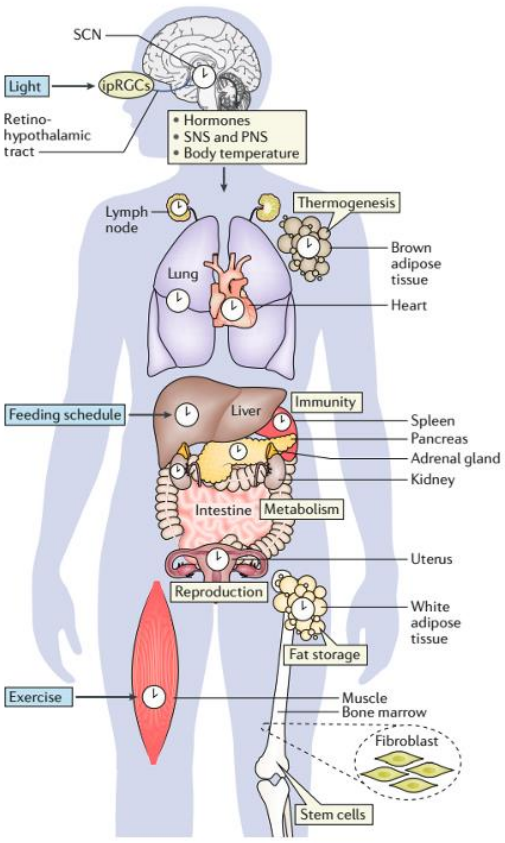
1.2.4 Peripheral oscillators throughout the body

Peripheral clocks are also self-sustained and cell-autonomous and are located in tissues throughout the body (see Figure 1.5). Oscillations within the peripheral tissues continue to persist in SCN lesioned animals (Mohawk et al., 2012). Despite expressing their own oscillation, peripheral clocks are maintained in a stable phase-relationship with each other for clock information to be optimal for the organism. This is achieved by the SCN transmitting temporal information via behavioural,

electrical, neuroendocrine, and autonomic signals to downstream peripheral clocks which entrain and impact the molecular mechanisms of cellular clocks in target tissues allowing for synchrony with the external environment to occur (Hastings et al., 2018). Asynchrony between peripheral clocks can occur when the SCN is lesioned (Yoo et al., 2004). This can lead to disturbances in communication between the various peripheral clocks resulting in desynchronisation of the circadian system leading to the development in a host of issues on health (Albrecht, 2012). Whilst peripheral clocks require entrainment from the SCN, the peripheral clocks in certain organs are critical for their organ's function and output (Patke et al., 2020). For example, even when the SCN is left intact, disruption to the circadian molecular feedback loop within the liver leads to arrhythmicity indicating that circadian oscillations of hepatic function are dependent upon an intact liver clock (Kormann et al., 2007).

Figure 1.5

Schematic image illustrating the location of the peripheral clocks throughout each of tissues and organs within the body.



Note. Image taken from Logan and McClung (2019).

While at a molecular level, there are similarities in both the SCN and peripheral clocks (Ko & Takahashi, 2006; Yagita et al., 2002) and both the peripheral clock and the SCN have the ability to generate autonomous circadian rhythms, the SCN differs for a number of reasons. Firstly, the SCN receives photic information directly from the external environment which allows for synchronisation to the solar day/light cycle which in turn synchronises other peripheral clocks (Morin & Allen, 2006). Secondly, when even in constant darkness the SCN neurons remain synchronised to each other due to topographically organised coupling mechanisms (Aton & Herzog, 2005). This is due to the individual neurons within the SCN being coupled together in order to produce a coherent circadian oscillation at the tissue level. As a result of this internal coupling the output signals from the SCN to peripheral clocks is coherent in the absence of external zeitgebers resulting in predictable periodicity to the circadian rhythm under constant conditions. This is in contrast to the peripheral tissues where independent oscillations of dissociated cells occurs. Thirdly, the SCN is able to generate a pronounced circadian rhythm of neuronal firing frequency allowing them through a number of direct and indirect pathways to synchronise other cells throughout the body (Gachon et al., 2004).

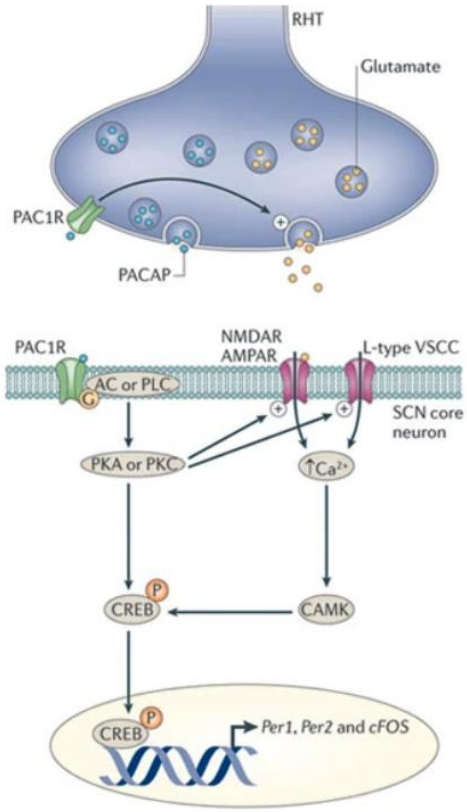
1.2.5 Light Input and Intercellular Mechanisms in the SCN

The anatomical structure which mediates light entering the body to entrain circadian rhythms is the retino-hypothalamic tract (RHT). The RHT projects from the retina to the ventral hypothalamus where the SCN is located (Moore & Lenn 1972). Ablation of the RHT around the hypothalamus results in abolishment of behavioural and hormonal rhythms in rats (Moore, 1972; Stephan & Zucker, 1972). This pathway, allows for the perception of light and darkness allowing for entrainment of the circadian system to occur. Photic information is received through the retina where it is encoded within the melanopsin expressing ipRGCs. These cells then generate action potentials to send the photic information directly to the SCN via the RHT. The monosynaptic RHT fibres end in a small subset of cells within the core of the SCN cells which express the neuropeptide VIP. VIP containing neurons process light information received from the RHT where the information is transferred to the dorsal SCN (Antle et al., 2009). From there photic information is

transmitted to neighbouring cells within the SCN. When light exposure occurs during the night this results in the release of the neurotransmitters glutamate (Glu) and pituitary adenylate cyclase-activating protein (PACAP) at the terminal synapses of the RHT where the signal is passed to the SCN. Glu has an excitatory effect on SCN neurons with its application to the SCN causing a phase shift in the SCN's electrical activity which mimics light-induced phase shifts in behaviour (Ding et al., 1994) and alters the level of PER1 and PER2 (Moriya et al., 2000). This stimulation of the RHT results in an increase in the firing rate and directly affects SCN electrical discharge (Figure 1.6). It also leads to an increase of Ca^{2+} in SCN neurons which is mediated through the activation of glutamate receptors (NMDA and AMPA) and voltage-sensitive calcium currents (Colwell, 2011). A microinjection of NMDA in a region of the SCN leads to phase-shifting effects which would be observed if light exposure occurred (Mintz et al., 1999). The PACAP type 1 receptor mediates the effects of PACAP on the SCN neurons. At a presynaptic level PACAP is involved in both aiding in the generation of Glu onto SCN neurons and postsynaptically increasing the magnitude of the NMDA and AMPA currents within the SCN which mediate the increase of firing rate in the SCN neurons. Increases in Ca^{2+} levels lead to activation of several signaling pathways which converge to alter the transcriptional and translational regulators of the molecular circadian clock. This release leads to the activation of several signaling pathways which commence chromatin remodeling, kinase activation and the induction of immediate early genes and clock genes. Specifically, increases in Ca^{2+} leads to alterations in cyclic AMP-response element (CRE)-binding protein (CREB). The phosphorylated CREB, a transcription factor, is translocated into the nucleus where it binds to CREs in promotor regions of c-FOS, PER1 and PER2. This results in chromatin remodeling resulting in the upregulation of PER1 transcription over the course of the next number of hours (Albrecht et al., 1997). This leads to a phase shifting response in the circadian clock. The light activation of the PER genes only occurs at night, and it is argued to play a significant role in the phase resetting of the clock (Hirota & Fukada, 2004) given that the SCN only responds to light during the subjective night (Takahashi et al., 1984). Glu in time is cleared from the synaptic cleft by glutamate transporters on astrocytes (Albrecht, 2012).

Figure 1.6

Schematic representation of the impact of light exposure on the intercellular signaling mechanisms in the SCN which regulate the molecular clockwork in SCN neurons.



Note. Photic information is passed to the SCN via the RHT. This results in the generation of an action potential resulting in glutamate release, which leads to kinase activation and expression of early immediate genes, resulting in a phase shift of the circadian clock. Image taken from Colwell (2011).

In traditional light/dark cycles SCN neurons display high levels of firing activity during the day and low levels during the night (Inouye & Kawamura, 1979). Within the SCN a small number of SCN neurons are light responsive through becoming active in response to light through exhibiting light-induced changes in electrical activity (Meijer et al., 1986; Michel & Meijer, 2019). When exposure to light occurs at night the firing rate of SCN neurons is significantly increased with larger increases occurring during the night than would typically occur during the day (Meijer et al., 1998). It has been argued that during the biological day neural activity is already high so stimulation of the RHT does not lead to a higher frequency of neural activity, however, during the biological night SCN neurons are electrically silent meaning that exposure to light leads to heightened RHT stimulation and in turn

greater electrical activity with the SCN responding to the change in frequency of action potentials with a phase shift (Colwell, 2011).

The intensity of light can increase the rate of neuronal firing in an intensity-dependent manner (Meijer et al., 1986) with SCN neurons responding in a sustained manner to light with their electrical discharge altered for the full duration of the light response (Michel & Meijer, 2019). The sustained response allows the SCN to perceive the length of the day or the duration of the light exposure. Longer durations of light exposure can lead to greater magnitudes in phase shifting responses compared to the shifts that occur with shorter light pulses (Meijer et al., 1992). As discussed above the increased generation of electrical activity within the SCN as a result of exposure to light-at-night (LAN) leads to the activation of proteins which reset the circadian pacemaker's core autoregulatory transcription-translation loop (Meijer & Schwartz, 2003). Specifically, LAN leads to the transcription of PER1 and PER2 with the intensity of the induction being mediated by the intensity of the light (Shigeyoshi et al., 1997). However, the timing of PER transcription impacts on the molecular clock with increases in transcription of PER genes in the early night delaying the molecular clock by postponing the normal decline of PER expression whereas exposure to LAN late into the biological night leads to a faster increase in PER (Colwell, 2011).

1.3 The Molecular Circadian Clock

Cellular circadian timekeeping in the SCN and in other tissues around the body pivots around self-sustaining cell-autonomous transcriptional translational feedback loops (TTFLs). These TTFLs are comprised of a set of core clock components which oscillate to a near 24-hour period (Figure 1.7). This is brought about through the positive and negative regulation of genes and their protein products (Ko & Takahashi, 2006). Core clock components are genes whose protein products are essential for the generation and regulation of circadian rhythms within individual cells throughout the mammal (Takahashi, 2004). The core clock genes include CLOCK and BMAL1, which encode activators and PER1, PER2, CRY1 and CRY2 which encode repressors. The TTFLs begin at circadian dawn where the positive regulators of the loop which drive the circadian cycle heterodimerize to either circadian locomotor output cycles protein kaput (CLOCK) or neuronal PAS

domain-containing protein 2 (NPAS2) with brain and muscle ARNT-like 1 (BMAL1) in the nucleus. CLOCK and BMAL1 are basic helix-loop-helix (bHLH)-PER-ARNT-SIM (PAS) transcription factors (Gekakis et al., 1998). During the daytime the CLOCK-BMAL1 complex attaches to enhancer boxes (E-boxes) which regulate and drive the daytime expression of transcription of clock-controlled genes including the repressor proteins period (PER) and cryptochrome (CRY). During this cycle, PER and CRY build up in the cytoplasm and at the end of the circadian day translocate to the nucleus. This results in negative feedback with rhythmic suppression of both CLOCK-BMAL1 effectively inhibiting the continued production of PER and CRY and in effect, closes the negative-feedback loop. Over the course of the circadian night there is a reduction in mRNA levels in both PER and CRY and the existing PER-CRY complexes are degraded and once these levels sufficiently drop, CLOCK-BMAL1 transcription can resume a new cycle of transcription. This degradation allows the cycle to be cyclical and reinitiate approximately 24-hours after the previous transcriptional initiation (Hastings et al., 2018; Patke et al., 2020; Takahashi, 2017; Takahashi et al., 2008; Reppert & Weaver, 2002). The outputs from both PER and CRY are critical to the maintenance and functioning of the circadian clock. Disruption to either PER1 or PER2 along with CRY genes induces behavioural arrhythmicity in double knockout mice when placed in constant light conditions (for review see Reppert & Weaver, 2002).

In addition to the primary feedback loop, there is a second negative feedback loop. The auxiliary loop includes the nuclear retinoic acid receptor-related orphan receptors (Rorα and RORβ) and REV-ERBs (REV-ERBα and REV-ERBβ), which are transcriptionally regulated by CLOCK-BMAL1 heterodimers. Along with the CLOCK-BMAL1 interaction activating the PER and CRY target genes, the CLOCK-BMAL1 heterodimer activate the nuclear receptors REV-ERBα and REV-ERBβ. REV-ERBα transcription is activated by the BMAL1/CLOCK heterodimer and transrepressed by CRY/PER, resulting in circadian oscillations of REV-ERBα. REVERBα represses the transcription of BMAL1 however, REV-ERBβ expression can also repress *BMAL1* transcription (Guillaumond et al., 2005). RORα competes with REV-ERBα for binding of their shared DNA binding elements, the retinoic acid-related orphan receptor (ROR), in the *BMAL1* promoter leading to *BMAL1* expression being repressed by REVERBα and activated by RORα. Thus, the rhythmic expression of REV-ERBα and REV-ERBβ results in the suppression of

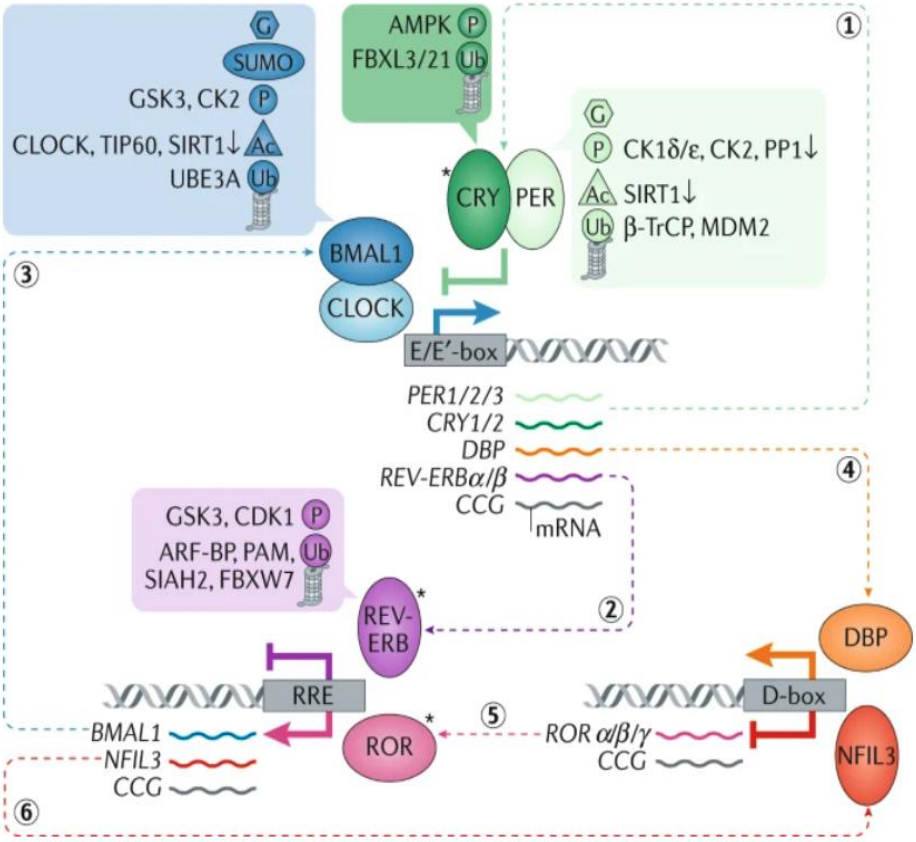
BMAL1 and CLOCK while levels of PER and CRY rise (Preitner et al., 2002). In effect the circadian oscillation of BMAL1 is positively and negatively regulated by the RORs and the REV-ERBs respectively which ensues a feedback loop which interconnects the positive and negative limbs of the core circadian clock. Although, it has been argued that this second loop is not essential but merely adds robustness to the molecular clock (Takahashi et al., 2008), it has been found that mutations in the genes of these nuclear receptors alter the amplitude and period of activity rhythms (Cho et al., 2012).

A third CLOCK-BMAL1-driven transcriptional loop involves the proline and acidic amino acid rich basic leucine zipper (PAR-bZip) factors D-box binding protein (DBP), thyrotroph embryonic factor (TEF) and hepatic leukaemia factor (HLF). Each of these proteins attach to areas containing D-boxes with the repressor nuclear factor, interleukin-3 regulated (NFIL3) which is driven by REV-ERB-ROR loop (Gachon et al., 2004). In effect, CLOCK-BMAL1 drive the transcription of several clock-controlled genes which ensue circadian rhythmic cellular-molecular processes which lead to complex circadian behaviour. Collectively, each of these three interlocking transcriptional feedback loops result in the generation of cycles of transcription with several peak gene expressions throughout the day. These transcriptions are dependent upon on the cis-elements in the promoters and enhancers of specific target genes (Ueda et al., 2005). Along with the activators and repressors, post translational modifications and degradation of circadian clock proteins are critical in determining circadian periodicity. Casein kinase 1 delta (CDNK1D) and casein kinase 1 epsilon (CSNK1E) act as the key kinases for PER and CRY phosphorylation. Mutations to either of these post-translational factors can lead to shorter circadian periods (Ko & Takahashi, 2006) and have been found to be possibly associated with familial advanced sleep phase syndrome in humans (Ex et al., 2005) The core clock gene Cry1 is a key regulator of circadian length between the amount of Cry1 and the length of the period (Oshima et al., 2015). Specifically, Patke et al. (2017) report that a mutation (CRY1 Δ11) to Cry1 was associated with lengthening of the period in individuals with delayed sleep phase syndrome (DSPS). This mutation lengthened the period of molecular oscillations by approximately 24-h. Under optimal conditions the generation of a molecular clock to a period of near to 24-hours is governed by post-translational modifications, interlocked feedback

loops of activators and repressors which impact on the stability and nuclear translocation of the core clock proteins (Ko & Takahashi, 2006).

Figure 1.7

Illustration of the mammalian circadian clock which is comprised of transcriptional-translational feedback network.



Note. The circadian clock mechanism involves transcription-translation feedback loops which are comprised of a set of core clock genes and oscillate with a near 24-hour cycle. The circadian clock is comprised of a primary negative feedback loop. As can be seen in in step 1 the positive loop is driven by the heterodimerization of CLOCK and BMAL1 in the nucleus. This heterodimerisation binds to enhancer e-boxes in gene promoters which regulate the of clock-controlled genes which encode both the PER and CRY proteins. This results in activation of Per1, Per2, Cry1, and Cry2 genes, whose proteins interact and repress their own transcription. Both PER and CRY accumulate in the cytoplasm and after time dimerizing resulting in inhibition of their own transcription by interacting with the CLOCK-BMAL1 complex. This leads to the closing of the negative-feedback loop. After time, the PER-CRY respressor complex is degraded and the CLOCK-BMAL1 complex can then activate a new cycle of transcription. In step 2 a secondary autoregulatory feedback loop is comprised of Reverbα which is a direct target of the CLOCK-BMAL1 transcription activator complex by repressing the transcription of BMAL1 and competes with ROR to bind ROR response elements in the BMAL1 promotor (step 3). Along with the transcriptional activators and repressors, post translational modifications and degradation of phosphorylation of circadian clock proteins are essential for mediating the period of the circadian rhythm. The respective kinases for PER and CRY are CSNK1D and CSNKIE. Image taken from Patke et al. (2020).

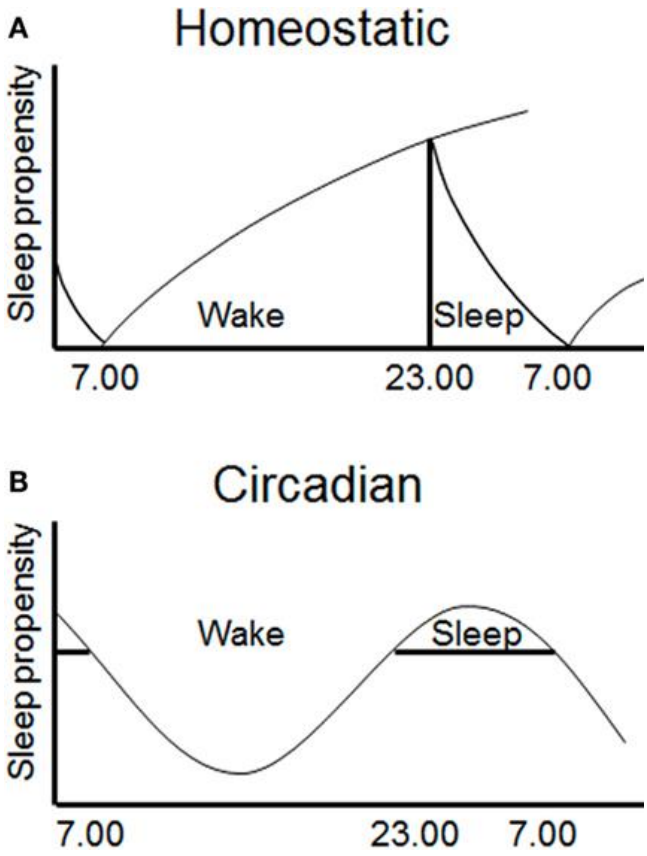
1.4 Homeostatic and Circadian Regulation of Sleep

The endogenous circadian clock modulates the timing of sleep-wake cycle, however, the cycle is also regulated by a homeostatic drive that increases with extended waking (Daan et al., 1984). This homeostatic oscillator describes the physiological need for sleep. The homeostatic sleep pressure increases during wakefulness and dissipates during sleep within a value range that oscillates with a periodicity which is typically entrained to day and night by the SCN (Borbely et al., 2016). When the sleep processes accumulation of sleep dissipates throughout the night it triggers awakening. A principal marker of this process is provided by slow wave activity (SWA) which increases as periods of wakefulness are extended and has been found to be strongly correlated with NREM sleep as measured by EEG (Vyazovskiy & Harris, 2013). The underlying mechanisms of the homeostatic process are unclear. It has been suggested that several sleep factors (e.g., adenosine in the forebrain) build up in the brain during prolonged wakefulness and dissipate during sleep which may influence the homeostatic process (Porkka-Heiskanen, 2013). Sleep and wakefulness are dependent upon the interaction between circadian and homeostatic processes (Borbely, 1982; Borbely et al., 2016). This suggests a two-process model of sleep regulation. The circadian process differs throughout the 24h day allowing for wake and sleep at alternate phases of the cycle (Fisher et al., 2013). The key regulator involved in circadian processes is the SCN as lesions to the SCN results in fragmentation of rest/activity rhythms. However, the homeostatic regulation of sleep remains intact (Mistlberger et al., 1983; Tobler et al., 1983). This indicates that two processes are regulated separately. The wake-promoting signal during the active period is argued to be driven by the SCN as SCN lesions results in an increase in total amount of sleep time (Edgar et al., 1993). This interaction between homeostatic mechanism and the circadian mechanism allows for the consolidation of the sleep-wake cycle and the transition from sleep and wakefulness (Dirk & Czeisler, 1994). During the day period the circadian propensity for wakefulness increases resulting in a suppression of the wake dependent increase in sleep propensity. Conversely, during the night the circadian propensity for sleep becomes heightened resulting in continuation of sleep despite the sleep dependent dissipation of sleep pressure. The SCN influences circadian processes through direct and indirect projections to the hypothalamic and brainstem nuclei which are involved in controlling the levels of arousal and sleep (Aston-Jones et al., 2001). The ventral

subparventricular zone exerts a liaison role between the SCN and hypothalamic and brainstem nuclei as lesions to this area results in a reduction in rhythms of locomotor activity and sleep (Lu et al., 2001).

Figure 1.8.

Schematic overview of the two-process model of sleep regulation.



Note. Sleep is regulated by two interacting processes (homeostatic & circadian). These processes interact to produce periods of wake and sleep during the day. The top panel indicates a homeostatic mechanism (Process S) which triggers a homeostatic drive for sleep the longer an individual is awake and dissipates during sleep. The bottom panel illustrates a circadian mechanism (Process C). The drive for sleep shows maximal intensity at the end of the day close to the onset of melatonin secretion. When the drive for sleep dissipates after the core body temperature minimum the propensity for wakefulness occurs. Image taken from Fisk et al. (2018).

1.5 Entrainment

As previously discussed, the internal clock is an endogenous, innate, and self-sustaining program, and as a result generates is own oscillations with its own

internal period (Czeisler et al., 2000). The intrinsic period of the circadian pacemaker deviates from 24h and is estimated to average 24.2h (Czeisler et al., 1999; Middleton et al., 1996; Wright et al., 2001). The length of the intrinsic period of the central circadian pacemaker is inferred from the intrinsic period of the phase makers (core body temperature, melatonin, cortisol rhythms) which are each correlated with each other (Dijk et al., 2012; Czeisler et al., 1999). Circadian phase is the recurring pattern in a range of psychological, behavioural, and physiological functions that repeat every 24h. As a result, the period could be estimated by measuring the time between DLMO on two successive days. When organisms are kept in temporal isolation, their circadian rhythms will begin to "free-run" with its own period. Depending on the organism and on the nature of the constant conditions, the endogenous period can become longer or shorter (Aschoff, 1979; Pittendrigh & Daan, 1976; Roenneberg & Foster, 1997). When this occurs, the temporal organisation of the phase markers under circadian control deviate from the solar 24-h cycle. Successful entrainment occurs when the central pacemaker is entrained by zeitgebers (i.e., time givers). Originally, the zeitgeber signal from the solar cycle equalized the internal and external day to match the T-cycle resulting in circadian oscillations establishing predictable phase relationships to their temporal environment (Pittendrigh & Daan, 1976). When this occurs each of the different phase markers adopts its own relationship with the clock which is essential for temporal homeostasis (Czeisler et al., 1989; Minor et al., 1991; Roenneberg et al., 1997). This means that the biological clock must be driven by zeitgebers and must establish a stable phase relationship with the external cues which is known as the phase of entrainment (Pittendrigh & Daan, 1976).

For the endogenous clock to be entrained to the solar cycle it requires resetting deviations of the endogenous clock to the solar cycle (T-cycle = 24-h). Although, the intrinsic period may deviate due to free-running conditions or due to the period being naturally shorter or longer than 24-h, this discrepancy will be relatively small and will be corrected by the solar cues of light and darkness. For the average human, whose intrinsic period is approximately 24.2-hours, the solar cycle resets the circadian clock by a daily light average of .02-h in the advance direction while individuals with a short free-running period must exhibit a phase delay (Czeisler et al., 2007; Gronfier et al., 2007).

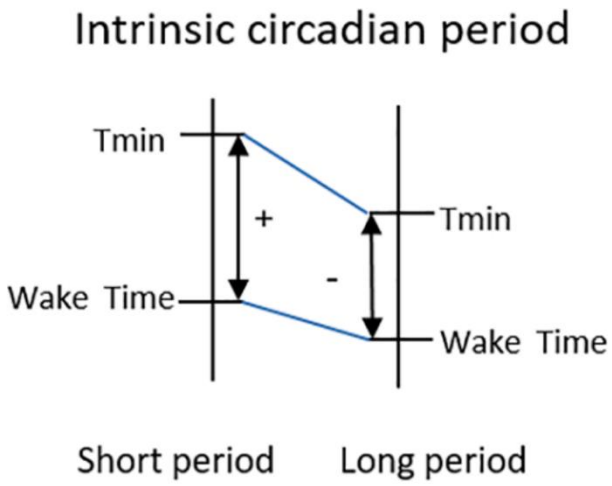
Circadian rhythms are oscillators and the phase refers to reference points in the oscillation (e.g., core body temperature minimum or DLMO). These reference points allow for the measurement of phase shifts and the change in the timing of the marker can be assessed over two cycles (Czeisler et al., 2007). This resetting of the biological clock by the solar cycle enables successful entrainment to occur whereby a stable phase relationship occurs between the biological clock and the solar cycle which is known as the phase of entrainment (Ashoff & Pohl, 1978; Pittendrigh & Daan, 1976). The phase of entrainment describes how well the clock is aligned with the light/dark cycle. When the phase of entrainment is aligned optimally the phase of an internal rhythm occurs at a similar time each day relative to environmental time (Pittendrigh & Daan, 1976). The phase angle of entrainment is the time difference between internal time expressed by the phase of the biological clock and external time expressed by the phase of the zeitgeber (Taillard et al., 2021). The relationship between sleep/darkness and the timing of DLMO is the most understood phase relationship detailing how entrainment occurs when the intrinsic period is aligned to the environmental cycle. For example, in those with stable entrainment, DLMO exceeds the fixed or relative threshold on average approximately two hours before habitual bedtime (Wright et al., 2005). The phase of entrainment is dependent upon the length of the intrinsic period, the strength of the zeitgeber, and the sensitivity of the biological clock to the zeitgeber. For example, as can be seen in Figure 1.9 an individual with a long intrinsic period will have a later circadian phase but a shorter phase angle (shorter interval between minimum body temperature and wake time.

It must be noted that in those who have stable entrainment there is variance in the rhythmicity of internal phase markers. This variance of the phase on entrainment is based upon several factors. For instance, the length of intrinsic period may differ due to the clocks having either genetically/adaptively adopted different free-running periods (Duffy et al., 2011; Dunlap et al., 2003), the period of the zeitgeber cycle and the strength and amplitude of the zeitgeber. Additionally, the zeitgeber signals may be received or transduced with different efficiency, for example, due to genetic differences in the receptor or the transduction cascade (Roenneberg, Daan & Merrow, 2003). Shorter intrinsic periods lead to earlier phases of entrainment resulting in a phenotype of getting up and going to sleep earlier (Duffy et al., 2001; Roenneberg et al., 2003). Gronfier et al. (2007) observed that shorter period was associated with earlier DLMO and habitual bedtime compared to those who have

longer period. The shorter period resulting in early wake and early sleep time may also lead to exposure to zeitgebers of with decreasing strength which will reinforce an extreme early chronotype. The same occurs for those with long intrinsic periods being exposed to weaker strength zeitgebers due to sleep and activity occurring at later phases and leading to an extreme evening type chronotype (Roenneberg, Wirz-Justice & Merrow, 2003). Exposure to the strong zeitgeber of natural light increases the amplitude of the light-dark signal. It has been found that individuals who spend longer outdoors and in stronger natural light have earlier sleep schedules (Roenneberg et al., 2003). The impact of the light-dark cycle on circadian phase is significantly influenced by the timing of light and darkness.

Figure 1.9

Illustration showcasing the phase angle of entrainment in longer or shortened intrinsic period of the circadian clock.



Note. An individual with a short intrinsic period will have an earlier circadian phase but will be entrained with a longer phase angle (longer interval between minimum body temperature and wake time). While the inverse occurs for those with long intrinsic periods. Image taken from Taillard et al. (2021).

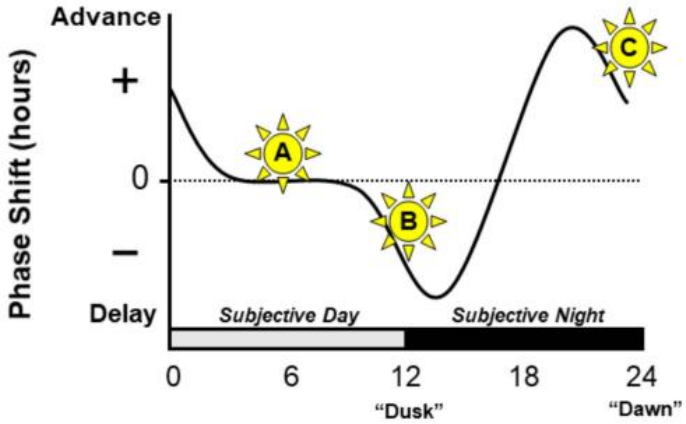
1.5.1 Phase resetting

The effect of zeitgebers on the circadian clock is biologically measured by a Phase Response Curve (PRC) which quantifies and predicts phase shifts of the circadian clock with response to a specific light stimulus over the entire circadian cycle (Czeisler et al., 1989). Both the shape and timing of the resulting PRC provides

specific information pertaining to the overall relationship between phase shift magnitude and circadian phase of the stimulus over the entire circadian cycle (Khalsa et al., 2003). In humans and in animals the speed, direction and magnitude of phase shifting responses are dependent upon the timing of exposure of light to the endogenous circadian rhythm (Czeisler et al., 1986; Czeisler et al., 1989; DeCoursey, 1960; Khalsa et al., 2003 Minors et al., 1991). It is important to note that exposure to an identical light stimulus can produce either a phase advance (shift in time to an earlier hour) or phase delay (shift in time to a later hour) depending on what time in the relative biological day exposure to a light stimulus occurs (Figure 1.10; Figure 1.11). The largest phase shifts are observed when exposure to light occurs during the subjective night. Phase delays are observed early in the subjective night before the critical phase of the endogenous phase markers (before CBT minimum) while phase advances occur late in the subjective night after the critical phase of endogenous phase markers (after the CBT minimum). Only small phase-shifting responses occur when exposure to light occurs during the subjective day (Czeisler et al., 1989). In animal studies, the subjective day region of the PRC is characterised by a region of insensitivity to light induced resetting and this has been referred to as the 'dead zone' of the PRC (Pohl, 1982). Evidence of such 'dead zones' have not been found in human studies (Jewett et al., 1997; Khalsa et al. 2003). Phase advances gradually reduce during the early subjective day, ultimately leading to gradually increasing phase delays later in the subjective day. This indicates that the human circadian pacemaker is sensitive to light induced phase resetting throughout the subjective day indicating that the entire 24-h pattern of light exposure contributes to entrainment (Jewett et al., 1997). This thesis will later discuss how mediating factors of light exposure such as the timing, intensity, wavelength, duration, and light history impact on the circadian system. However, these mediating factors do not contribute equally to the effect that light exerts eliciting a non-visual response (Houser & Esposito, 2021). The timing of exposure to a light stimulus is the most important factor in eliciting a non-visual response (e.g. phase shifting the clock) as the brain interprets time by observing the solar cycle of light and darkness (Figure 1.10; Roennenberg et al., 2006).

Figure 1.10

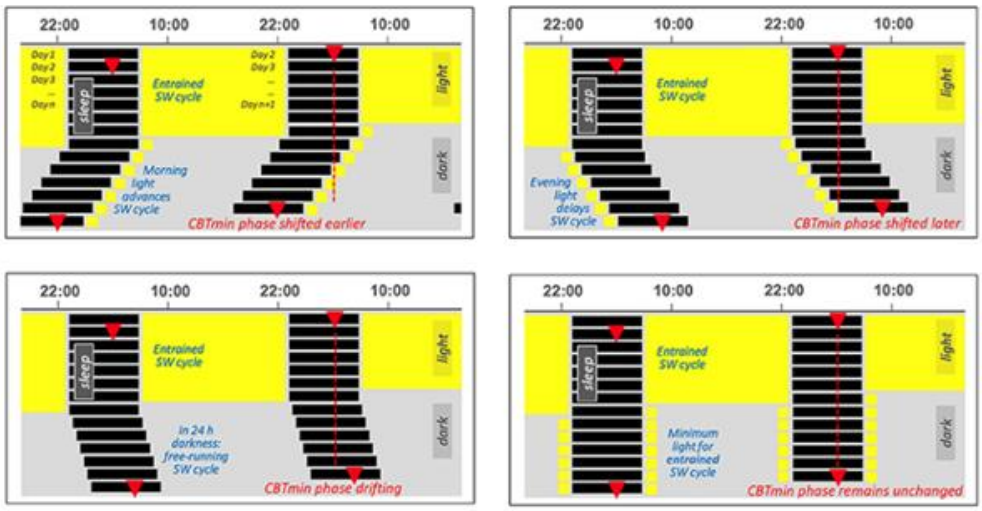
Schematic image of the phase response curve which demonstrates how exposure to light at different time points in the circadian cycle impacts on the phase of the circadian clock.



Note. Light delivered during the subjective day (denoted A) will have minimal effect on the phase of the clock. Light exposure at the during the early subjective night (denoted B) will lead to delays in the phase of the clock. Whereas light exposure at the end of the subjective night (denoted C) will lead to phase advances. Image taken from Ahston, Foster & Jagannath (2022).

Figure 1.11

Double plotted actograms showcasing patterns of the human sleep-wake cycle occurring because of different light exposures.



Note. On the top of each panel the several days occur with a 16L:8D cycle with different lighting schedules on subsequent days. (A) Light exposure occurring in the morning upon wakening resulting in phase advances of the phase markers. (B) Light exposure occurring late in the evening resulting in phase delays of circadian phase markers. (C) No light exposure resulting in a free-rhythm occurring. (D) Predictable light exposure occurring during the biological day and darkness occurring in the biological evening resulting in an entrained sleep wake cycle.

The findings of phase shifting response to light give strong evidence towards a hierarchical model in which the solar light-dark cycle ordinarily synchronises the endogenous clock which in turn, governs the internal organisation and timing of sleep. This indicates that light plays a fundamental role in entraining physiological rhythms and not sleep. This conclusion is supported by Lewy et al. (1985) where in depressed patients' exposure to light had an impact on reduction of melatonin secretion even when the timing of sleep was kept constant. Evidence of phase shifting effects due to light were essential is determining that exposure to bright light can reset the circadian clock which exerts significant influence in daily variations of physiology, behaviour, and cognitive function.

1.6 Individual differences in phase of entrainment

While everyone has an endogenous circadian rhythm, the timing of these rhythms varies across individuals whereby people synchronise differently to the same light-dark cycle either earlier or later. This results in the timing of the endogenous rhythm differing across individuals. These inter-individual differences in the phase of entrainment - also referred to as chronotype - are argued to manifest due to a combination of how individual clocks respond to light and darkness and how long the internal day they produce is (Roenneberg et al., 2003). Chronotype refers to the behavioural patterns of the endogenous circadian system that governs the active patterns in an individual's physical functions, hormone levels, body temperature, cognitive faculties, eating patterns and preferred timing sleep and wake (Roenneberg, Kuehnle et al., 2007). In the study of human sleep-wake regulation chronotype represents the preferred timing of sleep and wake (independent of environmental factors, such as work schedules) and corresponds to the timing of the circadian system (Kalmbach et al., 2017). Chronotype is distributed along a continuum ranging from early types to extreme later types (Adan et al., 2012) with epidemiological studies characterizing chronotype as near-normally distributed (Roenneberg et al., 2019). Morning types are categorized as having a preference to rising early, earlier bedtimes and have their peak performance earlier in the day. Conversely, evening types have a preference towards staying up late, sleep later into the morning and have their peak performance later in the day (Horne & Ostberg, 1976). Individuals who fall between the ends of the continuum are categorized as

intermediate types. About 40% of the adult population are categorised into one of the two extreme groups while 60% are classified as neither type (Adan et al., 2012).

Accumulating evidence provides support for the categorisation of individual chronotypes. Morning types have an earlier sleep schedule compared to late or evening types (Carrier et al., 1997; Taillard et al., 2004). Morning types display an earlier circadian temperature phase compared to evening types (Duffy et al., 1999; Gupta & Pati, 1994). The extent of the phase difference in core body temperature was 2 hours between morning and evening types (Baehr et al., 2000). Melatonin which is a predictor for propensity of sleep onset and circadian rhythm phase (Arendt, 2006; Rosenwasser, 2009), has been reported to be secreted 3 hours earlier in morning types in both salivary and plasma measurements (Gibertini et al., 1999; Mongrain et al., 2004). Additionally, phase differences in clock gene expression have been found between chronotypes (Brown et al., 2008; Novakova et al., 2003).

1.7 Assessing Chronotype

There are various ways of operationalising chronotype with several approaches employed to analyse this construct. Chronotype when operationalised as circadian phase or timing can be objectively measured based on the timing of objective markers in biological variables which exhibit circadian rhythms (e.g., melatonin concentration, core body temperature, dim light melatonin onset (DLMO)) (Arendt, 2006; Klerman et al., 2002). However, collection of these biological samples is invasive, timely, costly and limits categorization of chronotype to laboratory experiments resulting in small samples (Burgess & Eastman, 2005). To overcome this limitation several self-report questionnaires have been developed to categorise circadian preference (Horne & Ostberg, 1976; Smith et al., 1989) or typical timing (Roenneberg et al., 2003). The most used chronotype measures are the Munich Chronotype Questionnaire (MCTQ; Roenneberg, Wirz-Justice & Merrow, 2003) and the Morningness-Eveningness Questionnaire (MEQ; Horne & Ostberg, 1976). Both questionnaires conceptualise chronotype differently with the MEQ conceptualizing chronotype as a trait with individuals indicating their preferred times of the day for sleep and activity. While the MCTQ conceptualises chronotype as a biological state whereby chronotype is operationaised as the phase of entrainment

between the sleep-wake cycle and the 24-h clock. In the MCTQ individuals indicate their typical times for sleep and activity on workdays and free days.

1.7.1 Morningness-Eveningness Questionnaire (MEQ)

The Horne-Ostberg Morningness-Eveningness Questionnaire (MEQ) is a 19-item survey which examines individual differences in the time of day a person prefers to carry-out activities and classifies people as morning-type, neither type or evening type based on a score they receive on the measure. In this view chronotype is a psychological trait (Levandovski et al., 2013). This self-reported categorization of chronotype from the MEQ is correlated with circadian phase markers with studies indicating that morning types have an earlier circadian temperature phase (Baehr et al., 2000; Duffy et al., 1999) and have shorter circadian intrinsic periods (Duffy et al., 1999). The MEQ has been found to have strong internal reliability and test-retest reliability (Adan et al., 2012).

There are several criticisms of employing the MEQ to examining chronotype. Firstly, the MEQ fails to examine real behaviour given that the questions do not explicitly ask about timing of sleep, wake-up time or even differentiate behaviour of free-days (Putilov, 2000). Instead, the MEQ requires individuals to indicate a preference towards performing a hypothetical situation if they had the opportunity to do. This is problematic as preference does not necessarily translate to actual behaviour. This argument is supported by Roenneberg et al. (2019) who argue that categorizing individuals in terms of preference brings about reliability issues with actual extreme chronotypes possibly reporting a preferred desire to be more moderate in terms of their diurnal preference. This indicates that categorization arising from MEQ may not be able to accurately classify an individual's actual chronotype but instead what diurnal preference they wish they could be.

1.7.2 Munich Chronotype Questionnaire (MCTQ)

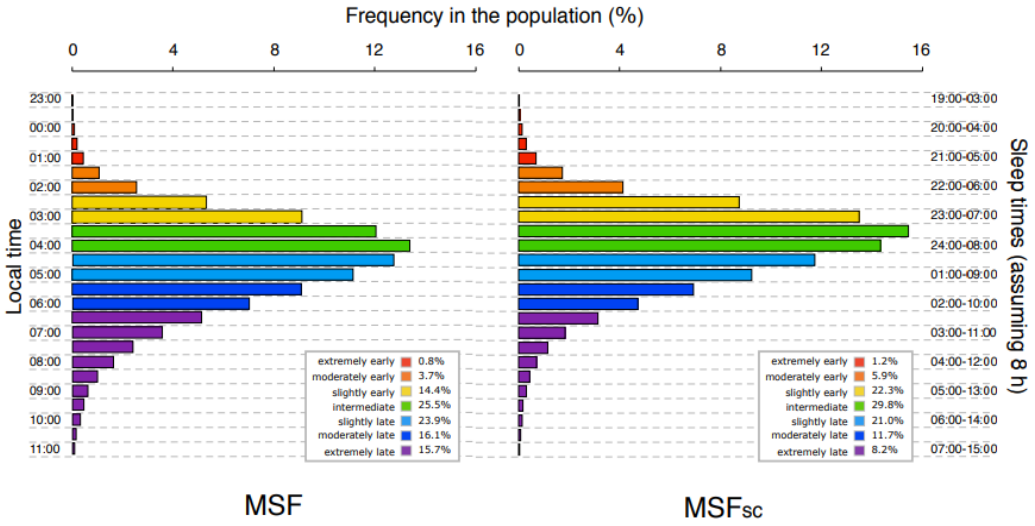
The MCTQ assesses chronotype quantitatively by approximating circadian phase by assessing sleep timing on free days (Roenneberg et al., 2003). This is achieved by finding the midpoint between sleep onset and wake-up on free-days to define chronotype (MSF). Assessing chronotype in this manner is argued to be more representative of an individual's overall circadian phase as the circadian system is

not influenced by societal demands/social clock (Roennberg et al., 2019). However, midsleep times on free days may be corrected for any sleep debt accumulated during the week to approximate chronotype. The theoretical basis for this correction is due to Roenneberg and colleagues (2007) showcasing that sleep duration differs between work- and free-days (MSF). Later chronotypes display shorter sleep duration on workdays but have longer sleep duration on free days. While extremely early types, experience shorter sleep duration on free days and longer sleep duration on workdays (Roenneberg et al., 2007). These observations suggested that sleep duration and sleep timing on free days are impacted by sleep debt which accumulates due to the sleep deprivation on workdays. This sleep debt systematically depends on chronotype with the later the MSF the larger the work-week accumulated sleep debt (Roenneberg, et al., 2003). Roenneberg and colleagues suggest that on free-days individuals compensate for their sleep debt by sleeping for longer into the day rather than going to bed earlier. It is argued that delayed offset of sleep on free days is due to recovery of sleep ensued by the sleep debt accrued over the workweek. To overcome this confounding influence of sleep-debt accumulated during the workweek, chronotype is corrected for the influence of sleep debt. The MCTQ calculates a theoretical chronotype (MSFsc) which estimates the timing of sleep as if individuals did not accrue lack of sleep on workdays. This correction from MSF to MSFsc only occurs when individuals sleep longer on work free days than on workdays. Given that most of the population accumulate sleep debt throughout the workdays, this results in the MSFsc being slightly earlier than the MSF leading to the distribution of mid-sleep (MSFsc) to become earlier and decrease the over representation of evening types which can be observed with MSF.

Scores derived from the MCTQ are continuous and are reflective of sleep behaviour rather than sleep preferences (Allebrandt & Roenneberg, 2008). The MCTQ is not categorical but instead provides continuous distributions with early or late chronotypes falling on the tail ends of a normal distribution. The distribution of MSF from data from 221,480 individuals is normally distributed with a slight overrepresentation of evening types (Figure 1.12; Roenneberg et al., 2019).

Figure 1.12

Distribution of MSF and MSFsc scores from the MCTQ database 2017.



Note. The left panel denotes MSF scores (n=221,480) and the right panel denotes MSFsc (n=185,333) scores . Bin values are based upon half-hourly bins. The y-axis denotes local time of midsleep values, and the x-axis indicates the sleep window in local time. Image taken from Roenneberg et al. (2019).

1.7.2.1 MEQ and MCTQ as a behavioural marker of circadian phase

Studies indicate that mid sleep time derived from the MCTQ provide an accurate behavioural marker for circadian phase (i.e. DLMO and cortisol; Burgess & Eastman, 2005; Kitamura et al., 2014; Simpkin et al., 2014; Terman et al., 2001) and strong associations have been found with sleep diaries and actigraphy (Roenneberg, Wirz-Justice & Merrow 2003; Roennebrg et al., 2019). However, one study has failed to report an association between the MCTQ and DLMO (Ruiz et al., 2020). Mid-sleep on free days corrected for sleep debt has been found to vary with DLMO across studies (e.g., DLMO-MSFsc: r = 0.54; Kitamura et al., 2014; DLMO-MSFsc: r = 0.68; Kantermann et al., 2015; DLMO-MSFsc: r = 0.35; Reiter et al., 2020; DLMO-MSFsc: r = 0.32; Reiter et al., 2021). However, Ruiz et al. (2020) provide evidence of no association between the MCTQ and DLMO. A high level of correlation between the MEQ and the MCTQ has been observed (Nguyen et al., 2019; Zavada et al., 2005); however, the strength of this association decreases when using the MSFsc (Zazada et al., 2005). The indicates the MEQ does not consider the confounding effect of work-related sleep deprivation. There have been mixed

findings as to which measure is a stronger predictor of DLMO with Kitamura et al. (2014) and Reiter et al. (2021) showcasing that MSFsc was a stronger predictor of DLMO compared to the MEQ. While Kantermann et al. (2015) observing the opposite. However, one study reported that the MEQ was a significant predictor of DLMO with no association found between the MCTQ and DLMO (Ruiz et al., 2020).

1.7.3 Sleep diaries and Actigraphy

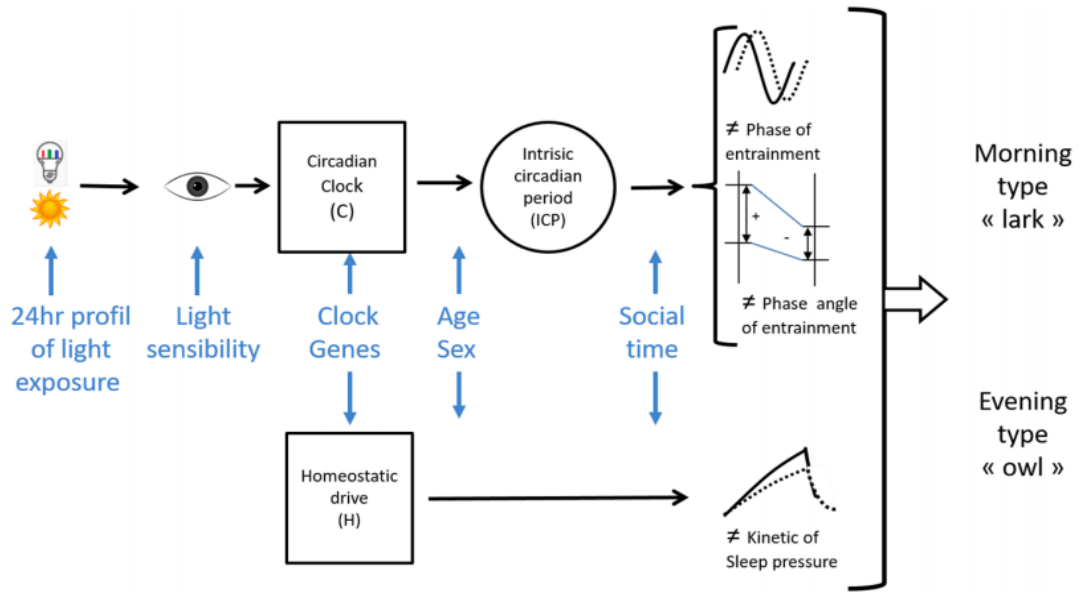
Sleep timing can also be assessed with daily sleep diaries and with actiwatches. Actiwatches provide day-to-day objective measurement of rest and activity. This daily quantification of rest/activity can be used to estimate sleep-wake schedules based on the observation that there is greater motoric activity during periods of wakefulness compared to periods of sleep. From the data generated objective measures of midsleep time on work and free days can be calculated using the same formula as the MCTQ. However, to reliably determine differences in timing between workday and free days collection of data must occur over a two-week period. This is to allow for an adequate number of free-days and workdays to sample from to depict an accurate representation of an individual's rest-activity rhythms (Roenneberg et al., 2015). Both sleep diaries and activity measurements have reported similar reliability in estimating DLMO (Reiter et al., 2020). Additionally, actigraphy and sleep diaries have shown a strongly correlated midsleep (Kantermann et al., 2007; Muehnle, 2006).

1.8 Factors Influencing Chronotype

As can be seen from Figure 1.13 individual differences in chronotype are due to a complex myriad of contributing factors which elicit differences in phase of entrainment/diurnal preference. Chronotype is a current state brought about by a dynamic system which is constantly adapting to the various internal and external conditions. This section will outline factors which contribute to chronotype.

Figure 1.13

Illustration of the complexity on how the phenotype of chronotype is derived.



Note. Showcase of the factors influencing homeostatic and circadian regulation of the timing of sleep leading in differences in the intrinsic period, circadian phase of entrainment, and sleep pressure. This leads to differences in the expression of various chronotypes. Image taken from Tailard et al. (2021).

1.8.1 Sex and Age

The epidemiology of chronotype is influenced through demographic factors which can impact chronotype across the lifespan (Adan et al., 2012; Fischer et al., 2017). Chronotype becomes progressively later during childhood and adolescence reaching a peak in 'lateness' between 18-19 years in females and males. This peak marks a sharp turn from increasingly later to increasingly earlier with advancing age, showing similar chronotypes in seniors and children (Fischer et al., 2017). Sex differences are observed with men being on average more evening orientated compared to women. However, Randler and colleagues (2019) in their meta-analysis observed that as individuals age, sex differences towards chronotype diminish with young woman who were previously morning orientated compared to men becoming more evening orientated and the opposite effects being observed in men. Possible arguments put forward for diminishing effects of sex on chronotype is due to the menopause and the reduction in testosterone in males (Randler et al., 2012; Roenneberg et al., 2004). Age is a contributor to the phenotype of chronotype with

young children typically being morning types, with a progression to evening types in adolescence (Russo et al., 2007; Tonetti et al., 2008) and after adolescence morningness scores tend to increase with age (Kim et al., 2010; Merikanto et al., 2012). This progressive change in chronotype occurs alongside alterations of endogenous circadian phase markers which is linked to earlier sleep timing (Czeisler et al., 1992; Dijk et al., 2000; Kawinska et al., 2005).

1.8.2 Genetics

Evidence that genetic factors influence the phenotype of chronotype have come from twin studies. A number of twin and family studies conducted worldwide have estimated the hereditability of chronotype to range from 14-50% (Aguiar et al., 1991; Barclay et al., 2010; Hu et al., 1998; Koskenvuo et al., 2007). These findings indicate that genetic factors explain a significant proportion of the population variability in circadian timing. Although the proportion of the genetic influence changes across the life span this occurs more predominantly during the age of 36 and 64 where the genetic effects of chronotype become weaker. Clock genes provide strong candidate genes for investigating the genetic background of chronotype as they control circadian rhythms which impacts on circadian timing and regulation of sleep. Candidate gene approach studies have found associations between circadian genes and chronotype, specifically with the CLOCK gene, each of the PER genes and the ARNTL2 genes. Studies have identified single nucleotide polymorphisms (SNP) of several clock genes (CLOCK, PER1, PER2, PER3 & Rev-Erb) which are linked to differences in chronotype. However, these studies have yielded inconsistent findings (von Schantz et al., 2017). The poor replication of studies may be because of differences in phenotyping, differences in ethnicity, sex differences, sample size and genetic differences (Adan et al., 2012). Five genome-wide association studies (GWAS) using large cohort studies (UK biobank, 23andMe & FINRISK) have identified a total of 351 separate loci which are associated with chronotype. These studies have found variants within clock genes such as CRY1, PER2 and PER3 amongst other genes (Hu et al., 2016; Jones et al., 2016; Lane et al., 2016; Maukonen et al., 2020). GWAS studies have also identified that chronotype is influenced by variants in genes which are critical in formation and functioning of RGCs (Taillard et al., 2021). This indicates individual differences in chronotype are

due to differences in detection and communication with the SCN of external light signal. Although replicable evidence has been found with some of the loci, other identified loci have not been replicated. These mixed findings suggest that multiple genes play significant roles in influencing circadian typology. Chronotype is a complex trait, involving many genes, each of which have a modest influence but through interaction result in eliciting a particular phenotype.

1.8.3 Circadian Effects

Several factors contribute to the individual differences of chronotype. The first main driver of these individual differences is due to differences in the phase of the circadian markers, the intrinsic period of the clock and the phase angle of entrainment. In terms of circadian phase, the temperature nadir and the onset of DLMO occur earlier in morning types compared to evening types (Baehr et al., 2000; Duffy et al., 1999; Duffy et al., 2001; Ruiz et al., 2020). These individual differences in the phase of the circadian markers persist to exist under constant routines suggesting that such individual differences are independent of sleep-wake timing (Dijk & Lockley, 2002). However, some morning and evening types do not differ in circadian phase despite having different sleep timing suggesting that for some individuals their diurnal preference is driven by homeostatic processes (Mongrain et al., 2005; 2006). Differences in chronotype may also arise due to variations in the length of the circadian period. Individuals with shorter periods rate themselves as morning types and those individuals with longer periods report themselves as evening types (Duffy et al., 2001; Duffy & Czeisler, 2002). A difference of 6-minutes in intrinsic period is associated with a change in the MEQ scores by 5-10 points (Duffy et al., 1999). Along with the intrinsic period being correlated with chronotype it is also correlated with circadian phase and wake time (Duffy et al., 2001**).** As stated earlier, evening type's intrinsic period is longer than 24-h and in order to be entrained requires the clock to be phase advanced. In contrast, individuals with a shorter intrinsic period need to be phase delayed. Differences in the phase angle of entrainment have been found between chronotype groups. In morning types, a shorter period will result in an earlier and longer phase angle of entrainment leading to an earlier local time but waking at a later circadian time. The opposite is observed in evening types whose phase angle of entrainment is

shorter (Dijk & Lockley, 2002; Duffy et al., 2001). Although, evening types wake up at a later clock hour, they wake at an earlier circadian phase (i.e. slightly after their trough in CBT) where the drive for sleep from the circadian system is high. In comparison, morning types are waking up earlier but at a later circadian phase given that the trough in the CBT occurred in the middle of the habitual sleep and as a result are waking at a time when there is a reduction in sleep pressure (Duffy et al., 1999).

1.8.4 Light Profiles

Differences in the daily patterns of light exposure have been found in individuals who categorize themselves as morning or evening types. This has been observed in both younger (Goulet et al., 2007) and older adults (Staples et al., 2009) along with other studies (Emens et al., 2009; Martin et al., 2012; Ruiz et al., 2020). In each of these studies, morning types in comparison to evening types were exposed to greater levels of light intensity during the morning and lower levels of light intensity during the evening compared to evening types. The opposite pattern of daily light exposure was observed in evening types. There have been mixed findings where some studies have reported differences in total bright light exposure between chronotypes (Goulet et al., 2007) and other studies reporting no differences in overall light exposure (Martin et al., 2012; Staples et al., 2009). It must be noted that it is difficult to ascertain whether it is simply the different patterns of exposure to light which is driving a particular chronotype or if differences in sleep/wake timing is leading to different patterns of light exposure and thereby driving particular chronotypes. For example, more light during the day encourages an early chronotype. However, individuals with a physiological tendency towards early schedules are being exposed to more light during the day, further advancing their sleep timing and chronotype (Swaminathan et al., 2017). This could suggest that differences in sleep-timing are leading to differences in light exposure and could be driving a particular diurnal preference. In Staple and colleagues (2009) study, the sleep-wake timing schedule was different across the two groups of chronotype with morning types going to bed on average 1 hour 15 minutes before evening types and getting up from bed 57 minutes earlier. However, in morning types, the greater exposure to a lighter pattern of exposure to light in the morning may lead to a phase advancing effect of entraining to an earlier clock time and in effect perpetuate a

preference for morningness. Although, studies have observed differences in exposure to light between chronotypes Goulet and colleagues (2007) reported that no differences between the overall sample of morning and evening types in light exposure relative to phase. Based on these findings Goulet et al. (2007) propose that only a subset of each chronotype group have a circadian cause for their diurnal preference.

When only investigating those with either extreme early or late circadian phases, a difference in light exposure relative to phase of circadian markers are found between the chronotypes. This suggests a difference in the phase angle of entrainment to the light/dark cycle in subgroups of chronotypes. Emens et al. (2009) provided further support for this argument by showing differences in the phase angle of entrainment to the light/dark cycle between morning and evening types with extreme phases. Morning types are exposed to light later in circadian time (i.e. more light before melatonin onset i.e. 0-4-h before DLMO) while evening types are exposed to less light before DLMO and instead have more light exposure in the advance zone of the PRC (12-16-h before DLMO) (Emens et al., 2009; Goulet et al., 2007). This leads to evening types have a greater ratio of phase advancing to phase delaying by light compared to morning types who show the opposite ratio of phasing effects. Van der Maren (2018) albeit not studying chronotype found that in young adults complaining of a delayed sleep schedule were exposed to greater light 9-12 hours after DLMO, which is an interval located in the phase-advancing portion of the PRC. This may suggest phase advancing light signals may aid in maintaining a stable phase of entrainment and prevent a further delay which could lead eventually to a non-24-h sleep-wake rhythm.

1.9 Shifting trends in chronotype distribution

Merikanto and Partonen (2020) demonstrate using a population based cross sectional study that from 2007-2017 the preference towards morningness which was common in 2007 has seen a progressive decline from 58.7% in 2007 to 50.9% in 2017. Other studies have reported similar trends (Broms et al., 2013; Roenneberg et al., 2012). Since 2007 to 2017 a higher frequency of definite evening types has been found in each of the age groups studied but most particularly in the 35-44 age group. Merikanto and colleagues (2020) highlight that the increased frequency of

eveningness was more prominent between 2007 and 2012 compared between 2012 and 2017. Merikanto argue that this increased preference towards eveningness between 2007 and 2012 may be due to workload stress due to the recession during those years. However, it could be argued that this increase was due to the increases in availability to light emitting technologies with expose individuals to blue wavelength light. Across the decade a higher frequency of eveningness and extreme eveningness along with a delay in midsleep on both free and workdays has been found in older adults. This is surprising given that it is viewed that as individuals age there is a shift towards morningness. However, environmental reasons may explain with older adults now being more technological literate and as a result have greater exposure to light emitting devices. It has been reported that smartphone use has increased year on year amongst older adults along with more time spent on phones increasing. In older adults, this self-imposed environmental increase in exposure to blue emitting electronic devices may dictate and prolong diurnal preference towards eveningness rather than an individual's chronological age driving and influencing the chronotype (Chinoy et al., 2018; Lakerveld et al., 2016). The increase in eveningness may partly explain the increase in reporting poor quality sleep as eveningness is closely associated with many sleep problems such symptoms of insomnia and insufficient sleep (Merikanto et al., 2012).

1.9.1 Factors contributing to this trend towards eveningness

1.9.1.1 Zeitgeber Strength

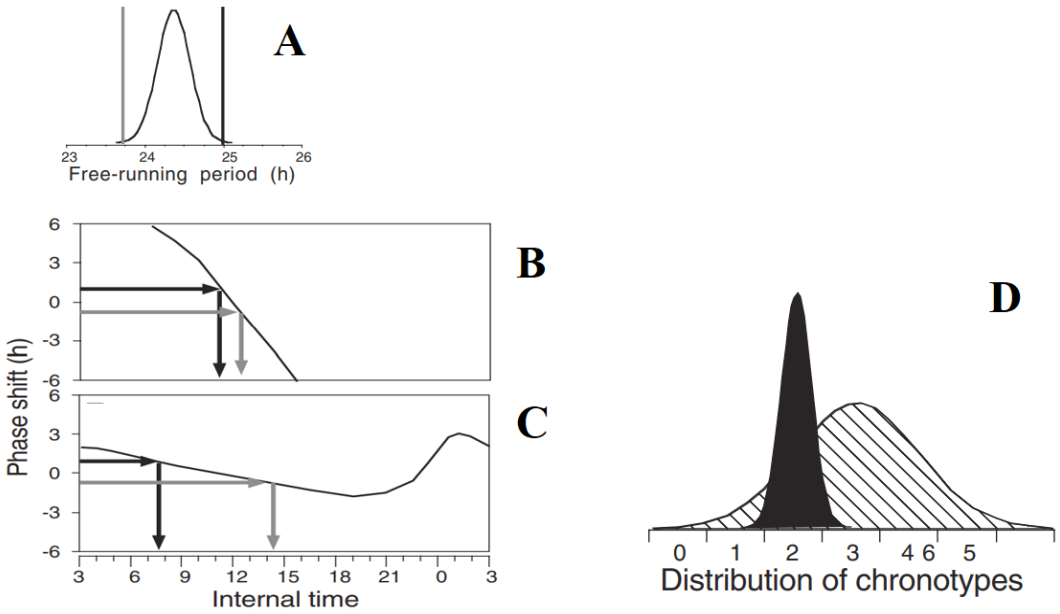
As outlined earlier when discussing entrainment, the phase of entrainment is dependent upon how much the intrinsic period deviates from the external period (Czeisler et al., 1999). For entrainment to occur depending on the length of the intrinsic circadian period exposure to light allows for either phase advance or phase delay. However, the strength of the zeitgeber is integral to phasing effects on the intrinsic period (Figure 1.14). When exposed to a strong zeitgeber, the phase of the circadian clock at which light imposes the necessary phase shifts are close to the two extremes which leads to a narrow distribution of chronotype. When the strength of the zeitgeber is weak this results in the phase of the two extremes being further apart from each other thus leading to a wide distribution of chronotype (Figure 1.14). Before the introduction of artificial light individuals were exposed to high irradiance

natural light during the day which throughout the day become progressively darker until darkness came, and light irradiance was minimal with light sources being the moon, candles, or fires. However, currently individuals spend 90% of their times indoors (Baron & Reid, 2014) with typical indoor light levels during daytime much lower than natural outdoor light and with individuals self-regulating their exposure to light after sunset (Cain et al., 2020). Empirical evidence to support this claim comes from Wright and colleagues (2013) who found that in modern house environments individuals are exposed to 4 times less natural light, exposed to significantly less natural light during the first two hours of being awake and exposed to higher levels of light from sunset till sleep. When these individuals were exposed to natural solar cycles of light/dark when camping this resulted in a reduction in individual differences in timing of the melatonin rhythm and sleep. There was also a reduction in individual differences in the timing between melatonin onset and offset with greater circadian advances in those with later chronotypes. This evidence suggests that exposure to predictable light/dark cycles which are of strong zeitgeber strength are of particular importance to later chronotypes by enabling greater circadian advances and aligning the timing of the internal clock in relation to solar cycle to exhibit an earlier chronotype. Stothard et al. (2017) showcased that exposure to a solar light cycle resulted in the phase of DLMO advancing and occurring in the middle of the biological night while it appears later in modern electrical environments. In the modern lighting environment, the clock of evening types becomes even later leading to greater inter-individual differences in sleep and circadian timing. The findings are supported by mathematical modelling which showcase that exposure to light levels experienced in home/office settings during the day is associated with a wide distribution of phase of entrainment, with the distribution narrowing as the level of light increases (Papatsimpa et al., 2021). In addition, their model found that high level daytime illuminance reduces interindividual differences in entrainment phase resulting in bringing later chronotype closer to earlier chronotypes. Interestingly, exposure to higher irradiances of light during the day was protective against developing an extreme later phase of entrainment when exposed to light during the biological night. While these findings are based on mathematical modelling they align with the distribution scores of chronotype in the real world (Roennenberg et al., 2019) and experimental field studies, showcasing that higher levels of natural light can reduce interindividual

differences in circadian timing (Wright et al., 2013; Stolhard et al., 2017). These findings indicate that light exposure which is ill-timed and of poor strength is a possible contributing factor for sleep and circadian problems such as delayed sleep phase (Sack et al., 2007) and social jetlag (Wittman et al., 2003).

Figure 1.14

Distribution of intrinsic period length, circadian system responses to light signals and the distribution of chronotype.



Note. Image (A) illustrates the distribution of the intrinsic period length. In Image A the grey line (on the left) indicates the extreme short end of the period distribution, and the black line (on the right) indicates the extreme long end. Circadian system response to light signals at different times of the endogenous cycle are illustrated with phase response curves (PRCs) that are steeper the stronger the light signal (see image B). The necessary phase shifts to entrain circadian clocks that deviate from 24 h can be achieved only if the circadian clock positions itself at a given phase angle in respect to the light signal (grey and black arrows correspond to the extreme periods indicated in A). The distribution of human periods and exposure to either strong and weak zeitgebers lead to different chronotype distributions (D; black for strong and hatched for weak zeitgeber). Individuals exposed to strong zeitgebers display a narrow distribution of midsleep timing where the opposite is observed for those exposed to weak zeitgebers. Image taken from Roenneberg et al. (2003).

Light from the solar cycle exerts a significant effect on chronotype. For example, Randler (2008) reported that in Germany, those living in the east of the country where the sun time is earlier exhibit earlier chronotypes than those living in

the west. Roenneberg and colleagues (2007) reported similar findings indicating that chronotype is driven by the sun rather than by social time. The length of the photoperiod impacts on the phase of entrainment with longer photoperiods leading to the expression of an earlier chronotype (Allebrant et al., 2014). Stothhard et al. (2017) showcased that when individuals are exposed to a natural solar cycle during winter their melatonin offset is significantly later compared to summer. This indicates a longer duration of the internal biological night after exposure to the natural winter versus natural summer light/dark cycle. However, individuals exposed to modern electrical lighting displayed no differences in the duration of melatonin duration across seasons. Exposure to a modern electrical lighting environment reduces seasonal circadian responsiveness by delaying the beginning of the biological light in both winter and summer. An individual's housing location exerts an effect on chronotype with those living in rural areas expressing an earlier chronotype and circadian phase than those living in urban areas despite both groups having access to electricity (Van Schantz et al., 2015). Although in large cities, there is a correlation between sun time and the sleep/wake cycle this association is weak and the chronotype is later. It is argued that those living in larger urban spaces are exposed to light signals which are of reduced zeitgeber strength. These weak zeitgebers are associated with later chronotypes particularly in those whose free-running period is longer that 24-hours (Roenneberg et al., 2004; 2016). Exposure to natural daylight during the day results in an increase in the amplitude of the light-dark signals. Studies have indicated that those who are exposed to natural light report a preference towards morningness and earlier sleep times compared to those that have a preference towards eveningness (Harada et al., 2002; Roenneberg et al., 2003). Furthermore, chronotype advances by more than one hour when people spend two hours outside per day.

1.9.1.2 Exposure to LAN

Artificial light-at-night (LAN) can exert an impact on the chronotype. As has been showcased by Wright et al. (2013) and Stothard et al. (2017) modern electrical environments can lead to later sleep timings along with delaying phase markers. Further evidence of the impacts of artificial light has been provided by comparative studies carried out in communities who share the same cultural background but

where part of the community have access to electric lighting and the other part does not yet have access to electricity (Nag & Pradham, 2012; Moreno et al., 2015). These studies have been fundamental in controlling for cultural effects while allowing for whether access to electricity impacts on chronotype. Moreno et al. (2015) examined differences in chronotype in rubber tappers in the Amazon Forest, who reside in areas with access to electricity and areas with no access to electricity. Amongst the community with access to electricity a later chronotype was observed along with a delay in DLMO, compared to the group without access to electricity. This later chronotype existed even with the electrified group only being exposed to relatively dim electric light exposure in the evening (approximately 30 lux inside the house with electric light). It is also important to note that these differences in chronotype are perpetuated through exposure to LAN at low levels which is not blue light emitting given that Rubber tappers do not have the income or resources to afford blue light emitting devices (i.e. smartphones, laptops or computers; Moreno et al., 2015). These studies provide evidence that light at night exposure is a contributor to later chronotype. Further evidence of LAN impacting on chronotype comes from Vollmer et al. (2012). This study demonstrated that adolescents residing in areas with higher levels of outdoor LAN displayed a higher preference towards eveningness. This association exists when controlling for other factors which would impact on chronotype (such as sex, puberty status, and exposure to light emitting devices). However, increased lighting may be proxy of increased possibility of social life which may drive a later chronotype.

Other studies have suggested that chronotype can be influenced by individuals self-selected light patterns (Swaminathan et al., 2017). Papatsimpa et al. (2021) mathematical model of variability in sleep timing and circadian phase observed that as the irradiance level of LAN increases so too does the interindividual variation in the phase of entrainment. In this research the circadian phase of both earlier chronotypes and late chronotype become later, and as a result the whole population shifts more towards eveningness. Additionally, circadian phase distribution continuously widens when nighttime light is higher or similar to that of the daytime. This suggest that an individual's physiological tendency towards later schedules may be exacerbated due to exposure to light later in the day leading to phase delays to the circadian clock and the delays in the timing of sleep. Swaminathan et al. (2017) further argue that individuals with a preference towards

eveningness are exposed to greater level of LAN emitting devices in the evening, which results in phase shifting effects on the clock and reinforces the behaviour by a positive feedback loop. In Swaminathan and colleagues (2017) model access to light at night may result in double the variation in sleep timing compared to living under natural light conditions. The impact of self-selected light exposure on chronotype was showcased in two studies which demonstrated changes in chronotype by being removed from modern electric environments to environments where light/dark cycles were aligned to the solar system (Stothard et al., 2017; Wright et al., 2013). In both these studies it was found that in the electrical environment individuals were exposed to light before sleep and displayed delayed sleep timing. As was observed in Wright and colleagues (2013) removing access to electric lighting and residing in an area with natural darkness led to phase advances among extreme evening types. New mathematical models which consider individual's physiology, their habitual self-selected patterns of light exposure and whether it's a workday or free day have been found to predict chronotype (Stone et al., 2020; Swaminathan et al., 2017).

1.9.1.3 Individual differences in how light exposure impacts chronotype

The effects that light exposure exerts on phase-shifting responses on sleep-wake timing displays significant interindividual variation (Santhi et al., 2012). For entrainment of evening types to occur the sleep-wake schedule is required to be delayed relative to the environmental light/dark cycle while the opposite is required for morning types. As discussed earlier individual differences in internal-external phase relationships is because of polymorphisms in core clock genes (Ebisawa et al., 2001; Toh et al., 2001). However, other studies have indicated that individual differences occur because of polymorphisms in genes of the phototransduction circuitry (Lee et al., 2014; Roecklein et al., 2012). Other studies have provided evidence of chronotype-dependent differential responses to light (Aoki et al., 2001; Higuchi et al., 2005). Higuchi et al. (2005) report that habitual sleep timing is associated with the magnitude of the effect of light exposure on melatonin levels with those who display less melatonin suppression in response to light having earlier habitual bedtimes. Aoki et al. (2001) reported that heightened melatonin suppression in response to light was associated with later habitual sleep timings. Van Der Meijden and colleagues (2016) provide evidence to suggest an association between

habitual sleep timing and interindividual variation in the functionality of the intrinsic melanopsin-based phototransduction circuitry. They found that with individuals with a stronger blue light responsiveness as measured by post-illumination pupil responses sleeping displayed a later phase of the 24-h cycle as measures by actigraphy and other self-report sleep timing measures. These findings are suggestive that individual differences in sleep timing may be a result of variation in the melanopsin gene. Individual differences in sleep timing have also been suggested to occur due to changes in other circadian parameters.

1.10 Chronotype and General, Mental & Cognitive Health

Circadian typology and chronotype are associated with predictors of poorer psychological and physical wellbeing (Fabbian et al., 2016). Eveningness has been repeatedly associated with poorer levels of sleep quality, sleep efficiency and higher reporting of sleep complaints (Barclay et al., 2010; Merikanto et al., 2011; Taillard et al., 2001). Evening types typically report having a shorter sleep duration due to waking up for societal commitments leading to a build-up of sleep deprivation (Roenneberg et al., 2007). Eveningness is associated with poorer health promoting behaviours such unhealthy dietary habits (Kenerva et al., 2012), higher smoking and alcohol consumption (Patterson et al., 2017; Robinson et al., 2013) and lower levels of exercise (Wennman et al., 2015). This consistent patterned interaction of evening types having poorer lifestyle choices coupled with sleep and circadian disruption leads to eveningness being routinely implicated as a risk factor for metabolic and cardiovascular illnesses (Arora et al., 2015; Knutson & von Schantz, 2018; Koopman et al., 2017; Melinska et al., 2012). After controlling for sleep duration and quality of sleep, evening types have been shown to have increased risk of arterial hypertension which is a risk factor for cardiac events (Merikanto et al., 2013). A preference towards eveningness is associated with a small increased risk of all-cause mortality; the risk of all-cause mortality was highest in older definite evening types (Broms et al., 2013; Knutson & von Schantz, 2018).

An association between eveningness is found with several neuropsychiatric conditions and/or poorer psychological wellbeing. Knutson and von Schantz (2018) in their prospective study consisting of 433,268 individuals reported that definite evening types have greater risk of having psychological disorders and neurological disorders compared to definite morning types. Fabbian and colleagues (2016)

systematic review found that eveningness is linked with anger, and anxiety disorders. Multiple lines of research have indicated associations between eveningness and depression with links being observed across all age groups (Chiu et al., 2017; Taillard et al., 2001). In Au and Reece's (2017) meta-analysis of 36 studies they found that eveningness was associated with more severe symptoms of depression, however, small effect sizes were found. These associations have been reported to be independent of sleep timing, sleep duration and sleep debt (Abe et al., 2011; Katamura et al., 2010). No causative effects of preference for eveningness contributing to depression has been established. However, genetic mutations of the circadian clock genes may alter the intrinsic period or phase angle of entrainment (Bunney & Bunney, 2000). These genetic changes result in circadian disruption which may lead to disruption to the rhythmic activity of the neurotransmitter systems involved in mood regulation (McClung et al., 2000). This may in turn lead to a preference towards eveningness and the onset of a depressive phenotype. It is important to note however, that having insomnia (Monteleone & Maj, 2008), circadian abnormalities (Bunney & Bunney, 2000) or abnormal clock gene expression may also lead to depressive symptoms (Wulff et al., 2010).

Scholastic achievement and cognitive performance is significantly impacted by a preference towards eveningness. Morning types typically perform better academically and have better attention in school (Besoluk et al., 2011; Tonetti, Natale, & Randler, 2015; Medeiros et al., 2010). However, it is important to acknowledge that the generalizability of findings about scholastic achievement in evening types may be comprised given that both school- and examination timing occur in the morning which is an optimal time of day for assessment for performance in morning types and a negative time for assessment in evening types (May & Hasher, 1998; Schmidt et al., 2007). Morning types may have heightened cognitive and academic performance due to match between their chronotype and the occurrence of scholastic activities predominantly occurring in the morning. Aligning an individual's circadian preference to the timing of cognitive testing has been found to be advantageous to cognitive ability (Cohen-Zion & Shiloh, 2018).

1.11 Mismatch between the biological and social clock

Although sleep/wake cycles are driven by the biological clock they are also influenced by our social clocks (e.g. working hours, leisure time etc.). The impact of our social clock on sleep timing is evidenced by 80% of population relying on alarm clocks to wake them up from sleep on workdays, resulting in sleep loss/curtailment (Roenneberg et al., 2012). The influence that social clocks exert on the timing of human sleep becomes visible when comparing sleep/wake activity across work- and free days with regards to the duration of sleep and the timing of sleep. For many, the duration of sleep on free days is on average typically longer compared to workdays (Foster et al., 2013; Roennenberg et al., 2012). The sleep timings and duration are argued to reflect the natural phase of entrainment or the circadian wake propensity system thereby acting as a proxy of internal time. The timing and duration of sleep on workdays is argued to be reflective of external social time due to its constrained nature (Wittmann et al., 2006). The weekly mismatch in midsleep timing between the internal endogenous circadian phase and external time is referred to as social jetlag (SJL). SJL is conceptualised to be a proxy of circadian misalignment. The term SJL draws parallels between the weekly pattern of misalignment and the misalignment accrued by rapid travel across time zones. The degree of SJL an individual experiences is calculated quantitatively by finding the difference of midsleep on free and workdays. Thus, providing an understanding of the difference between social and biological time. Social jetlag is primarily calculated using the MCTQ (Roenneberg, 2003). However, it can also be calculated by using sleep logs or actigraphy provided that information is collected on which days are workdays and free days and study duration is a minimum 7 days (Vetter et al., 2020).

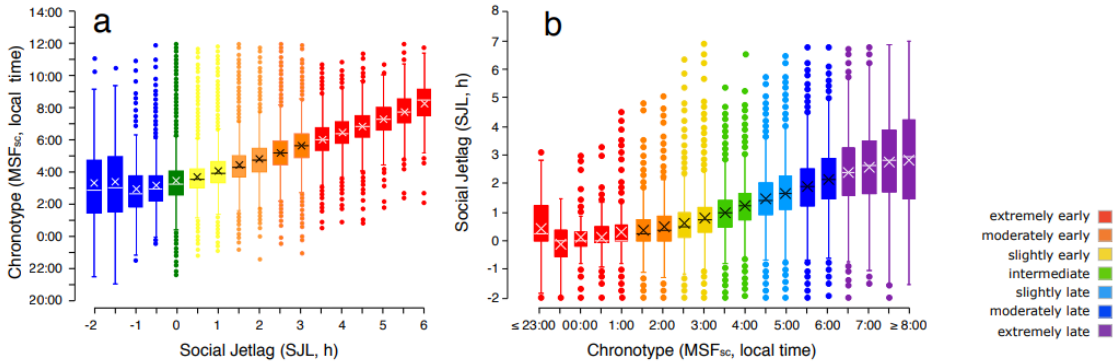
Differences in sleep timing between work and free days are observed across the lifespan with young adolescents displaying SJL due to school timings which ensues into adult life due to work. Reductions in the levels of SJL experienced decreases in retirement (Foster et al., 2013). Retired individuals do however still display SJL, albeit, to a lesser degree (Garefelt et al., 2021). It appears that SJL is a chronic phenomenon with findings indicating that the discrepancy between circadian and social clock is highly prevalent in society with around 44% displaying SJL of 1-h or more and 15% displaying greater that 2-h of SJL (Koopman et al., 2017; Roenneberg et al., 2012; 2019). As children progress from being morning types to

predominantly evening types in adolescents this leads to SJL being more acute in adolescents given societal demands of school timings (Roenneberg et al., 2012).

SJL is typically greater in evening types (see Figure 1.15) due to this group having to continuously adjust their temporal habits to social demands to a greater extent than morning and intermediate types (Merikanto & Partonen, 2020; Wittman et al., 2006). This is due to evening types sleep duration on workdays being curtailed on both ends of sleep. This occurs as evening types have a preference towards later sleep, resulting in these individuals falling asleep later and having the offset of sleep occurring prematurely due to social demands (i.e. work schedules). This results in a higher build-up of sleep debt over the course of the workweek, which is compensated for on free-days by a longer duration of sleep and later sleep offset, due to the absence of social demands (Roenneberg et al., 2004; 2007). For evening types this heightened shifting of sleep and activity times between workdays and free-days leads to MSF being much later the MSW, leading to higher levels of SJL. Even after correcting for the accumulation of sleep-debt the later the chronotype the more pronounced the SJL experienced (Wittman et al., 2006). Increased SJL in evening types may also be because of delayed circadian phase markers as late chronotypes have their circadian sleep window occurring later after the offset of melatonin secretion (Taillard et al., 2021). However, it is important to note that the association between SJL and MSFsc is not linear. Due to social norms, morning types on free-days are delayed in their timing of sleep onset as a result of staying up late into the night without sleeping later the next morning due to their normal circadian wake-up time. This leads to intermediate types having the smallest levels of SJL as a results of sleep timing being similar across work- and free- days (Wittman et al., 2006).

Figure 1.15

Indication of the association between chronotype (MSFsc) scores and social jetlag (SJL).



Note. The left panel (a) illustrates that as the timing of MSFsc (chronotype) becomes later (denoted on the x axir), this on average is associated with an increase in the amount of SJL experienced (denoted on the y axis). The right panel indicates that same however, the axis are swapped. That is, the earlier the MSFsc the lower the level of SJL experienced. The grouping of data used in this graph is derived from the midsleep on free-days corrected for sleep debt based on a sample of 185,333 participants who included their data in the Munich ChronoType Questionnaire database. The colour coding of the chronotype category as indicated by the colour legend is arbitory. Image taken from Roenneberg et al. (2019).

1.11.1 Lighting contributing to Social Jetlag

It is argued higher levels of SJL is in part brought about by widening of the chronotype distribution because of exposure to light zeitgebers which are of attenuated strength and ill-timed (Roenneberg et al., 2003). In many cases individuals during the day are not exposed to full natural daylight but instead dim low level lighting (Wright et al., 2013). In the evenings, individuals are exposed to ill-timed artificial light which enables the artificial lengthening of the day leading to delaying effects (Stothard et al., 2017). This may lead to evening types becoming more evening orientated. This has important implications for SJL as these weak and ill-timed zeitgebers leads to clocks becoming phase delayed while social schedules remain relatively unchanged. This leads to people accumulating sleep debt over the workweek which leads to even higher levels of SJL (Pilz et al., 2018). Evidence of light impacting on the timing of sleep has been found when comparing cultures with and without access to electricity (Pilz et al., 2018) and when comparing within groups the timing of sleep being earlier when there is no access to electricity compared to modern environment living (Wright et al., 2013; Stothard et al., 2017).

When individuals were resided in their modern electric environments their sleep start and sleep end is delayed by over one hour on free days compared to workdays. However, when exposed to the natural light/dark cycle sleep start and end remain similar across the week (Stothard et al., 2017). This suggests that our self-selected lighting environment may contribute to increasing levels of SJL. Additionally, the potential that lighting habits at night are driving greater eveningness leads to higher increases in levels of SJL due to sleep timing becoming incompatible with traditional work times. On workdays, individuals utilizing or being exposed to LAN or non-photic zeitgebers ensues phase delays leading to an individual failing to fall asleep after the circadian sleep window opens, and at the opposite end of sleep, work demands are leading to activity before the end of the circadian sleep window (Roennenberg et al., 2012).

The influence of exposure to light influencing larger levels of SJL comes from naturalistic studies where those living in the western border of a time zone have high levels of SJL compared to those who reside in the east (Roenneberg et al., 2007). Wright et al. (2013) observed that exposure to the natural light dark cycle for those with later chronotypes resulted in larger circadian advances. That is, the clock of the evening types appear to become even later than those of earlier chronotypes when exposed to modern environmental settings. These effects of modern lighting environments may contribute to sleep and circadian problems such as delayed sleep phase (Sack et al., 2007), and SJL (Roenneberg et al., 2012). When individuals are removed from their modern living environments and are aligned to the sun clock this results in those who previously had high levels of SJL expressing a reduction in phase delaying of sleep and circadian parameters (Crowley et al., 2010; Stothard et al., 2017). This has a positive impact on SJL levels as a decrease SJL occurs. This indicates that SJL is in part influenced by exposure to light. Interestingly, in Merikanto and Partonen's (2020) population based cross-sectional study it was found that between 2012 and 2017 SJL decreased in definite evening types, more evening types, and more morning types. This reduction in SJL is observed even in the context of LAN emitting devices becoming being more accessible and blue light exposure lighting being more frequently available. Strategies to limit decrease LAN exposure through the use of light blocking glasses and increased morning light exposure have not resulted in reducing SJL, however, has led to earlier onset of the circadian phase markers and sleep onset (Zerbini et al., 2020). Mathematical

modelling studies have reported that when individuals can self-select their lighting this results in inducing SJL (Skeldon et al., 2017). The study also reported that reducing the intensity of light resulted in reductions in the amount of SJL an individual experiences.

1.11.2 Social Jetlag & Health

Higher levels of SJL have been linked to poorer health promoting behaviours such as smoking (Wittman et al., 2006), poor dietary habits (Almoosawi et al., 2018) and less physical activity (Rutters et al., 2014). SJL has been found to be associated with increased BMI, fat mass, and probability of being categorised as obese (Roenneberg et al., 2012; Parsons et al., 2015; Zhang et al., 2018). The association between SJL and obesity is estimated to equate to an increase of 30% in the chance of being categorised as overweight/obese for each hour of SJL (Roenneberg et al., 2012). Additionally, this association is observed even after controlling for sleep deprivation. Higher levels of SJL have been associated with the biomarkers of cardiometabolic health (Wong et al., 2015), increased heart rate (Rutters et al., 2014), increased risk of having type 2 diabetes (Koopman et al., 2017). However, other studies have reported inconsistent findings with regards to SJL and parameters of weight (Johnsen et al., 2013; Pulugrudov et al., 2016). Possible reasoning for the inconsistencies in findings is due to latitude with studies in mid latitudes finding associations but higher latitudes findings no associations with metabolic health (Beuavalet et al., 2017).

Although associations between both circadian and sleep disturbances and depression have been found several studies have shown associations between high levels of SJL and higher levels of depressive symptoms (Borisenkov et al., 2015; Levandovski et al., 2011; Polugrudov et al., 2016). However, other studies have reported no association between levels of SJL and levels of depression (de Souza & Hidalgo, 2015; Sheaves et al., 2016). It is argued that the differences in findings may be because of the different ages of the sample from each study. Studies which found no associations were based on student populations ranging from 12-21years. At this stage of development depressive symptoms may be influenced to a greater degree by other variables which are separate to circadian misalignment, such as puberty and hormones (Beauvalet et al., 2017). This suggests that the association between SJL

and depression may only become apparent during adult life. Alternatively, the effects of SJL may be cumulative with the detrimental effects only becoming apparent in adulthood or it may be that younger individuals are protected against the effects of SJL compared to older adults (Henderson et al., 2019). No associations have been observed between SJL and minor psychiatric symptoms (Schmitt et al., 2013) and anxiety (Sheaves et al., 2016). Several studies have also found associations between high levels of SJL and poor academic performance (Diaz-Morales & Escribano, 2015; Haraszti et al., 2014; Lin & Yi, 2015) and productivity at work (Yong et al., 2016). Although, associations between SJL and health variables have been found there have been inconsistent findings. Several systematic reviews allude to the fact that this is due to methodological differences in approaches to assessing SJL, the computation of SJL, the cut off points for determining SJL and other psychosocial variables such as the age profile of participants, the latitude and the areas in which they live in (Beavalet et al., 2017; de Souza Tavares et al., 2020; Henderson et al., 2019; Roenneberg et al., 2019).

1.12 Circadian Photoreception

The passing of light information through the retina can elicit two human responses which can be categorized as either visual or non-visual. A visual response is an eye-brain response which enables conscious vision. Non-visual responses, also referred to as non-image forming (NIF) responses are independent of vision and refer to the impact that light exposure exerts on circadian, neuroendocrine or neurobehavioural responses (Houser & Esposito, 2021). More specifically these non-visual responses to light refer to phase resetting of the circadian clock, altering sleep architecture, constriction of the pupil, suppression of melatonin, increase of heart rate, increase of core body temperature, stimulation of cortisol production and serves as a neurophysiological stimulant (increasing subjective and objective cognitive performance in the domains of alertness, reaction time and reducing lapses in attention; Lucas et al., 2013). These non-visual responses are dependent upon the timing (discussed earlier in section 1.5), intensity, duration, temporal pattern, and spectral content of the light stimulus (Prayag et al., 2019).

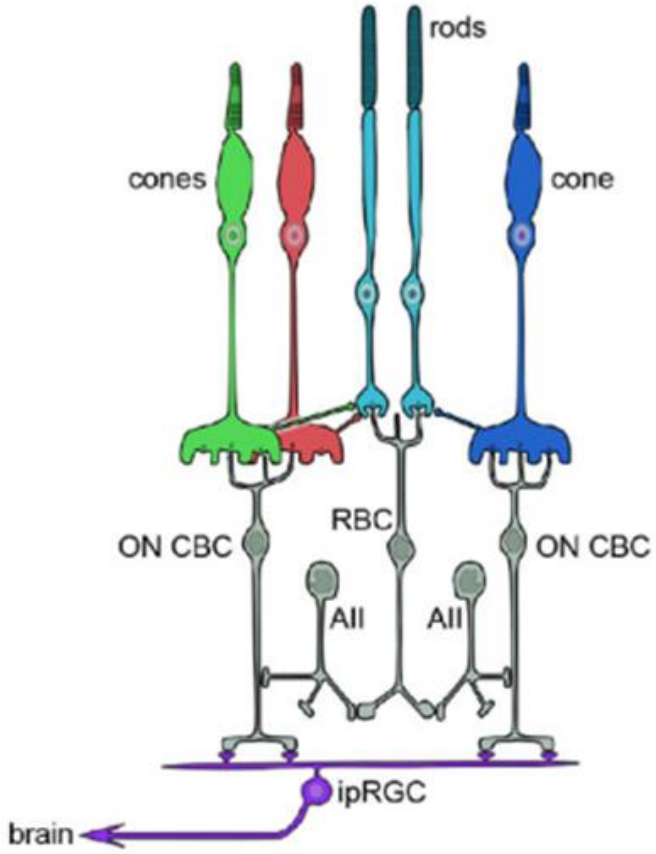
1.12.1 Classical Photoreceptors and Opsins

It was originally believed that the rods and cones were the only photosensitive cells in the retina. The identification of a third photosensitive photoreceptor came from studies where individuals who were blind due to outer degeneration of the rods and cones but who had an intact ganglion layer displayed comparable levels of melatonin reduction in response to light as sighted individuals (Czeisler et al., 1995; Hull et al., 2018). Accumulating evidence found that some blind individuals who had intact and functioning optic nerve and RHT signaling maintained a sleep/wake cycle which was aligned with the environmental light/dark cycle (Czeisler et al., 1995; Lockley et al., 1997) and in response to light displayed circadian phase shifts and PLR response to light (Lockely et al., 2005; Gooley et al., 2012; Klerman et al., 2002; Zaidi et al., 2007) while those that had damage to the optic nerve displayed no NIF response to light. Additionally, these NIF responses were occurring at action spectra which were different from those of the traditional photoreceptors (Hull et al., 2013; Zaidi et al., 2007). Vandewalle et al. (2013) reported that with individuals who are blind exposure to blue light for less than a minute resulted in activation of the prefrontal and thalamic regions of the brain which are involved in alertness and cognitive regulation. Further evidence of a third photoreceptor came from animal studies where animals who lacked rod and cones photoreceptors displayed reduction of melatonin levels with response to light, expressed an ability to photoentrain and maintained a PLR indicating that photosensitivity continues to survive with the loss of rods and cones (Hattar et al., 2003; Freedman et al., 1999; Lucas et al., 1999; Panda et al., 2003; Yoshimura & Ebihara, 1996). Only upon bilateral enucleation of the *cd/cd rd* mice were all circadian light response abolished (Freedman et al., 2009). In healthy animals, the rods and cones were unable to fully explain the different behavioural responses to light based on wavelength, intensity, and duration (Brainard et al., 2001; Lucas et al., 2001; Takahashi et al., 1984). This evidence, from both animals and humans, demonstrating light response behaviours when the rods and cones are lost implies the role of an additional photoreceptor. Given the evidence that circadian responses were kept even when removal of the whole outer and most of the inner nuclear layer of the retina suggested that the circadian photoreceptors were in the ganglion cell layer (Figure 1.16).

Berson et al. (2002) were the first to provide evidence for a third photoreceptor. In the rat they injected fluorescent microspheres into the SCN resulting in illuminance of axons down along the RHT to the retrogradely labelled a subtype of cells called the RGCs. In *rd/rd cl* mice, the retina was injected with the Ca^{2+} -sensitive FURA-2AM dye. Fluorescent imaging found light induced Ca^{2+} changes in ∼3% of neurons within the layer of the RGCs (Sekaran et al., 2003). These subtypes of RGCs displayed light-evoked depolarisation which was maintained when the rest of the retinal intercellular was blocked. The labelled RGCs displayed intrinsic light responses when dissected and isolated for the surrounding retinal tissue (Berson et al., 2002) and respond to light on their own when separated physically or blocked by drugs from receiving input from other neurons (Berson et al., 2002; Hattar et al., 2002; Lucas et al., 2003). Ablation to the ipRGCs results in the abolishment of non-image forming responses in response to light indicating that that these specific cells are essential to photoentrainment (Panda et al., 2002; Ruby et al., 2002). The photopigment of this third photoreceptor is melanopsin (*Opn4*) and is found in a subpopulation of RGCs located in the ganglion layer of the retina (Provencio et al., 2000; 2002). These unique melanopsin expressing ipRGCs are light responsive and are found in 1-3% of total ganglion cell population. The light response from the ipRGCs are associated with non-image-forming responses to light (Berson et al., 2002; Hattar et al., 2003). Melanopsin is the principle photic contributor to all non-photic responses (Cajochen et al., 2005; Gooley et al., 2010; Rahman et al., 2014). When non-photosensitive cells are transfected with the human or mouse melanopsin genes, this results in the cells becoming light sensitive (Mclyan et al., 2005, Panda et al., 2005; Qiu et al., 2005). These melanopsin-transfected cells when placed into human kidney cells have a peak spectral sensitivity of 479nm (Qui et al., 2005) and when transfected into the Xenopus oocytes the peak sensitivity is 480nm (Panda et al., 2005). This provided evidence of a third class of photoreceptors with electrophysiological and spectral characteristics which were aligned to the behavioural measures of circadian photoentrainment (Hattar et al., 2002).

Figure 1.16

Illustration of the retinal circuity in humans.



Note. While non-visual responses originate in the retina they occur due to the ipRGCs. ipRGCs are connected to the rod and cone photoreceptors via the conventional retinal circuitry. In the above image the ipRGCs are connected to the cones via on cone bipolar cells (ON CBCs) and amacrine cells (ALL). Rod bipolar cells (RBCs) connect the rods to the ipRGCs. This leads to the ipRGCs responses being mediated by melanopsin ipRGCs along with extrinsic signal which are derived from the rods and each of the spectrally distinct cone photoreceptors (as indicated by the colours red, green and blue).

1.12.2 Interaction between photoreceptors to enable non-image forming behaviour

Despite melanopsin phototransduction being only involved in moderate to high irradiance, the ipRGCs and their downstream responses have been found to be responsive at low levels of illumination (Wong, 2012). It was initially argued that exposure to light at levels of 2500 lux were required to suppress levels of melatonin (Lewy et al., 1980). However, it has been later noted in several studies that levels of light exposure as low as 1 lux has an ability to suppress melatonin secretion (Glickman et al., 2002; Zeitzer et al., 2000). This sensitivity to light indicates a

critical feature of the photoreceptive system in that the ipRGCs have two functions. First, the ipRGCs receive extrinsic mediated signals from the rods and cones (Brown, 2016). Secondly the ipRGCs are influenced by the intrinsic melanopsin photoreception due to their expression of melanopsin, which allows these cells to respond to light even when isolated from the rest of the retina (Lucas et al., 2012). The integrated signals from each of the photoreceptors are conveyed via the melanopsin expressing ipRGCs to non-image-forming brain centres (Nowozin et al., 2017). Non-visual responses can occur through the combination of the five retinal opsins (melanopsin, rhodopsin, S- M- L- cone opsins). Given the influences of both extrinsic and intrinsic signaling the unique contribution of the photoreceptors in eliciting non-visual responses is quite complex and unclear. Evidence indicates that the contribution of the photoreceptors is dependent upon the intensity of the light exposure and its duration. For instance, in dark-adapted conditions cones will dominate non-image forming responses, while under light adaption the influence of cones on non-image forming behaviour will be reduced (Gooley et al., 2012; Hommes & Gimenez, 2015). The rods are also critical for circadian photoentrainment at a wide range of light intensities but particularly at low light intensities (Altimus et al., 2010; Aranda & Schmidt, 2020). This is of critical importance given that that the ipRGCs by themselves cannot drive non-visual responses at low light intensities (Do et al., 2019). Additionally, when short-wavelength light is attenuated, long wavelength light can exert an alerting effect (Van de Werken et al., 2013). This indicates that both the rods and cones are critical for the exertion of non-image forming behaviours in certain conditions.

1.12.3 The requirement of the ipRGCs for non-image visual function

The pupillary light reflex (PLR) is the contraction of the iris muscle to limit the amount of light entering the eye. Pupillary constriction increases with irradiance up to a point where saturation occurs. This constriction is sustained for as long as the stimulus is present. Research from both animal and human studies have provided evidence that the three main photoreceptors are involved in PLR (Spitschan & Woelders, 2018). The contribution of each photoreceptor is dependent upon the when and how such as the intensity of the light stimulus, the spectral content, and changes over time in constant conditions. For instance, at low light levels which are

below the melanopsin threshold, the PLR response is dependent upon the spectral sensitivity of the rods and cones (Altimus et al., 2010). When deletion of the melanopsin protein occurs a PLR still occurs however, the constriction in abnormally transient with the response decaying over minutes despite the continued exposure to light (Keenan et al., 2016). Additionally, when the melanopsin protein has been deleted the PLR is strong at low light intensities as a result of the contribution of the rods and cones but is weaker at higher light intensities (Lall et al., 2010; Lucas et al., 2001; Lucas et al., 2003). This indicates that intrinsic melanopsin phototransduction is critical for PLR when light exposure is at high light intensities which are over a long period of time (Butler & Silver, 2011; Keenan et al., 2016; Jain et al., 2016). Deletion of the rods and cones while the melanopsin expressing cells remain intact reduces the speed and sensitivity of pupil constriction (Keenan et al., 2016; Lucas et al., 2003). Ablation specifically to the ipRGCs eliminates PLR even when the rods, cones and other subtypes of the RGCs remain intact and functional (Hatori et al., 2008). This research indicates that the ipRGCs are involved in PLR indirectly and directly. Indirectly by the rods and cones sending signals to the ipRGCs to cause a sensitive and quick PLR and directly the ipRGCs are essential for holding the PLR steady at higher irradiances (Do et al., 2019; Wang et al., 2017).

The ipRGCs play a critical role in the induction and regulation of sleep. Evidence of this comes from nocturnal animals where under normal circumstances when light is presented early into the subjective night this leads to the continuation of sleep. However, circadian photoentrainment of sleep and the continuation of sleep by light during the inactive phase becomes attenuated when the melanopsin gene is delated indicating that the rod/cone inputs can compensate for the loss of melanopsin to photoentrain sleep rhythms (Altimus et al., 2008; Lupi et al., 2008). However, ablation to the ipRGC results in continued maintenance of sleep by light becoming absent (Altimus et al., 2008; LeGates, Fernandez & Hattar, 2014; Tsai et al., 2009).

It was originally argued that melanopsin was the most important photopigment for entraining the SCN to the external light-dark cycle. However, accumulating evidence has found that melanopsin is not fully critical for photic entrainment and that the other classic photoreceptors play a role in photic entrainment (Panda et al., 2003 Ruby et al., 2002; van Diepen et al., 2013). Melanopsin knockout mice continue to entrain to a light/dark cycle (Panda et al., 2003), display continued circadian rhythmicity (Guler et al., 2007), show a sustained

light response (van Diepen et al., 2012), weakened phase shifting response which are 50% of those in wild type mice (Ruby et al., 2002) and increased period lengthening during constant light exposure (Panda et al., 2002), attenuation of full pupil constriction (Lucas et al., 2003), attenuation of photoregulation to bright light (Altimus et al., 2010), abolishment of the intrinsic photosensitivity of ipRGC to light (Lucas et al., 2003; Hattar et al., 2003; Panda et al., 2003). This indicates that when melanopsin protein is absent the rods and cones can partially compensate for the loss of ipRGCs sensitivity however, the photoregulation of the circadian clock exhibits lower sensitivity (Do, 2019). Inversely, animals which have complete loss of rods and cones are still able to photoentrain due to the structural integrity of ipRGCs being sustained, albeit this photoregulation exhibits at lower sensitivity (Altimus et al., 2010; Hattar et al., 2003; Panda et al., 202 & Ruby et al., 2002). This suggests that non-image-forming responses is dependent upon both intrinsic melanopsin photoreception and extrinsic signaling from the rods and cones (Lucas et al., 2003; Panda et al., 2002). When melanopsin knockout mice are crossed with mice with non-functional rods and cones this results in a complete loss of responses towards light (Hattar et al., 2003). The results from these studies indicate that the rods, cones, and the ipRGCs are accountable in the execution of non-image forming responses to light exposure. However, selective ablation of the ipRGCs results in the complete loss of circadian photoentrainment, masking, phase-shifting when exposed to light while conscious vision is intact (Guler et al., 2007; Goz et al., 2008; Mrosovsky & Hattar, 2003), indicating that the ipRGCs are the principal conduit for light to drive photoentrainment.

1.12.4 Subtypes of projections of the RGCs

Studies have found that the SCN receives direct environmental photic input form a specialised subset of ipRGCs (Berson et al., 2002; Gooley et al., 2001; Hattar et al., 2002; Provencio et al., 2000) which mediate photoentrainment (Goz et al., 2008; Guler et al., 2008; Hatori et al., 2008). The ipRGCs have a widespread influence by innervating many brain areas to influence physiology, behaviour, perception, and mood but more specifically, areas of the brain that elicit non-image-forming responses. It has been reported that the melanopsin expressing ipRGCs project to non-image-forming centres of the brain such as the ventromedial region of

the hypothalamic SCN to photoentrain circadian rhythms and the olivary pretectal nucleus (OPN) to control the pupillary light reflex (Berson et al., 2002; Gooley et al., 2001; Hattar et al., 2002; Provencio et al., 2000). The ipRGCs also project to other non-image-forming centres of the brain such as vSPZ, VLPO, PTA and the IGL subdivision of the LGN (See Schmidt et al., 2011). The influences of the ipRGCs extend beyond where they target directly. LeGates et al. (2012) provide evidence that the ipRGCs are involved in the regulation of the melatonin synthesis in the pineal gland and synaptic plasticity in the hippocampus. Areas of the brain which are involved in conscious vision receive few afferent projections from the ipRGCs which express melanopsin. Gooley et al. (2003) propose that the areas in which the ipRGCs project to function as a retinal irradiance system which acts independently to image forming perception and leads driving or contributing to the non-image-forming responses.

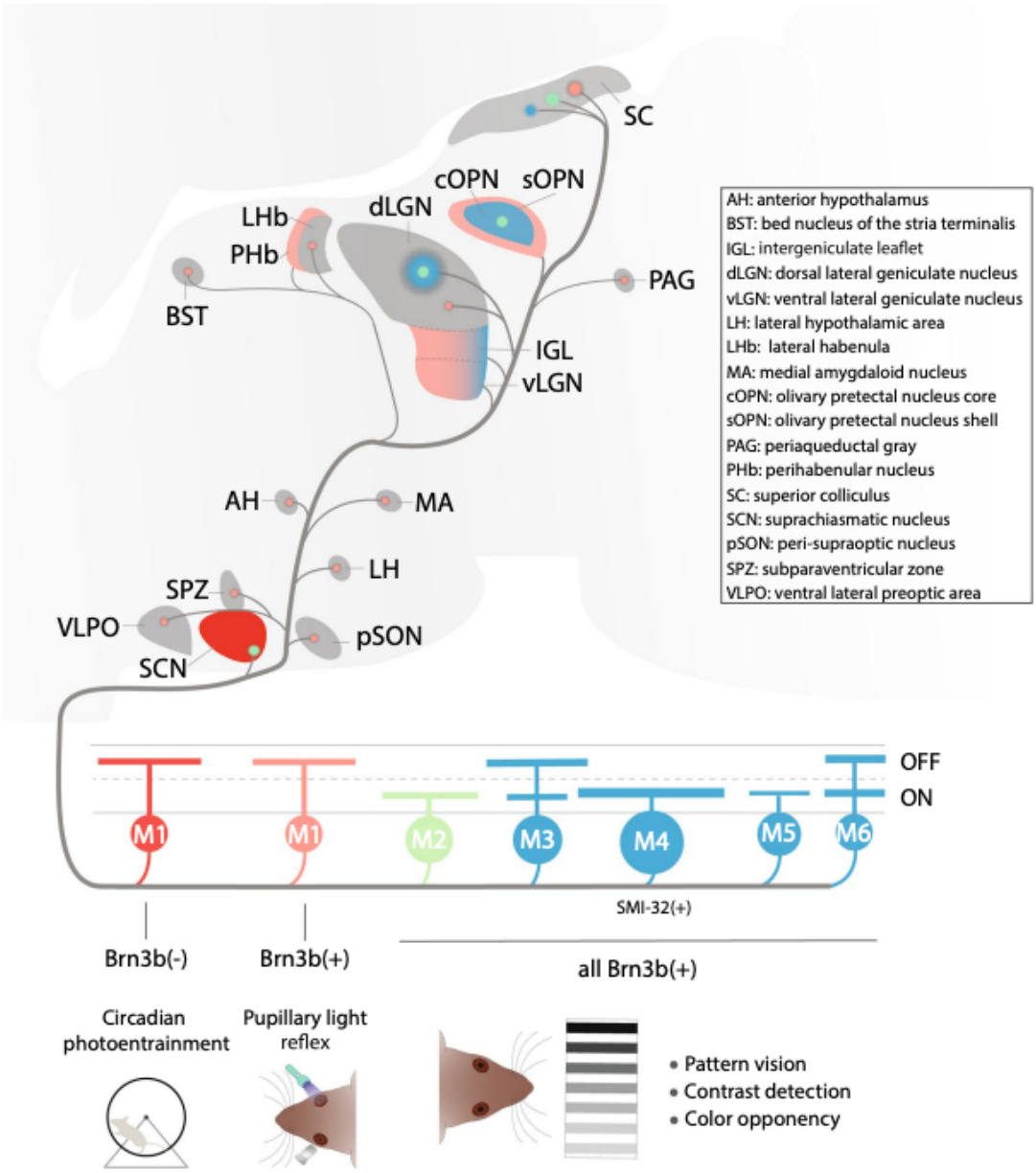
The RGCs consist of 6 subtypes (M1 through to M6) which have been identified in the mammalian species and in the human retina (Hannibal et al., 2017; Mure et al., 2019). These subtypes uniquely contribute to non-image and image forming behaviours and have potentially distinct functional roles (Schmidt et al., 2011). Each subtype exhibits differences in their morphological characteristics with differences in dendritic stratification along with differences in soma and dendritic arber size (For detailed review see both Aranda & Schmidt, 2020; Provencio et al., 2002; Schmidt et al., 2011). The M1 subtypes express high levels of melanopsin have larger and more sensitive intrinsic light-evoked responses and are seen to be central to non-visual responses (Do et al., 2019; Guler et al., 2008). The M1 combines light information from each of the photoreceptors to the non-visual response (Lax et al., 2019). The M2-M6 subtypes express lower amounts of melanopsin and exhibit reduced intrinsic photosensitivity (Mure, 2021; Quattrochi et al., 2019). However, despite differing levels of melanopsin each subtype is argued to perform specific light-regulated functions at specific levels of light intensity in order to elicit non-image forming behaviours (Do et al., 2019). The differences in light response between the various subtypes is due to differential ratio of melanopsin versus the contribution of the other photoreceptors and from the morphological differences between each subtype (Aranda & Schmidt, 2020; Quattrochi et al., 2019). Additionally, the magnitude, latency and duration of the light response varies across each of the subtypes in an intensity dependent manner. For instance, the non-

visual responses which are mediated by the M1 cells have lower threshold, higher amplitude, and faster responses than other the ipRGCs subtypes (Zhao et al., 2014).

The M1 cells are central to non-image forming behaviours and predominantly project to non-image forming centres such as the SCN and IGL (to drive circadian photoentrainment), the OPN (to drive PLR), the ventrolateral preoptic area (sleep promoting neurons), the lateral hypothalamic (wake-promoting neurons), the peri-habenular region and indirectly (via the IGL) the lateral habenula (see Figure 1.16; Aranda & Schmidt, 2020; Fernandez et al., 2018; Hatter et al., 2002; Huang et al., 2019; Schmidt et al., 2011). Circadian photoregulation is absent when all ipRGCs are ablated but persists when only a subset of M1 cells survive (Chen et al., 2011; Guler et al., 2008). M1 cells are subdivided into two types with those containing transcription factor Brn3b(+) cells which project to extra-SCN brain sites (midbrain, thalamus) and the Brn3b(-)cells which project to the SCN (Fernandez et al., 2018). The Brn3b(+) cells pass light information to the IGL impacting cognition, thermoregulation and sleep independent of the SCN (Fernandez et al., 2018; Rupp et al., 2019). M4 and M6 project to image forming areas of the brain which include the dorsal lateral geniculate nucleus, the superior colliculus, and the core of the olivary pretectal nucleus (Figure 1.16; Sanes & Masland, 2015). M2 cells project to the areas of the brain associated with visual behaviours along with areas involved in non-visual responses such as SCN albeit minimally. It is unclear the degree in which M2 cells may modulate non-visual responses (Aranda & Schmidt, 2020). M1 and non-M1 cells also project to the preoptic and lateral hypothalamic areas such as the VLPO and the ventral subparaventricular zone, which control sleep induction and general activity levels. In addition, ipRGCs innervate some limbic regions such as the lateral habenula and the medial amygdala, highlighting the possible direct role of light in the regulation of mood and cognitive functions (LeGates et al., 2014).

Figure 1.16

Illustration of the main brain targets of the ipRGCs.



Note. M1 cells are the main subtype of ipRGCs which elicit NIF. The ipRGCs projects directly to the SCN to enable photoentrainment but this subtype also innervate the thalamus, hypothalamus, and midbrain. M1 ipRGCs are subdivided into Brn3B- (direct to the SCN) and Brn3b+ (direct to the midbrain and thalamus) which target different brain areas. M2 cells project to both image forming (SC and dLGN) and non-imaging forming (SCN and OPN) visual areas. M4-M6 cells project to brain nuclei involved in image-forming vision such as the dLGN and the SC. Image taken from Arana and Schmidt (2020).

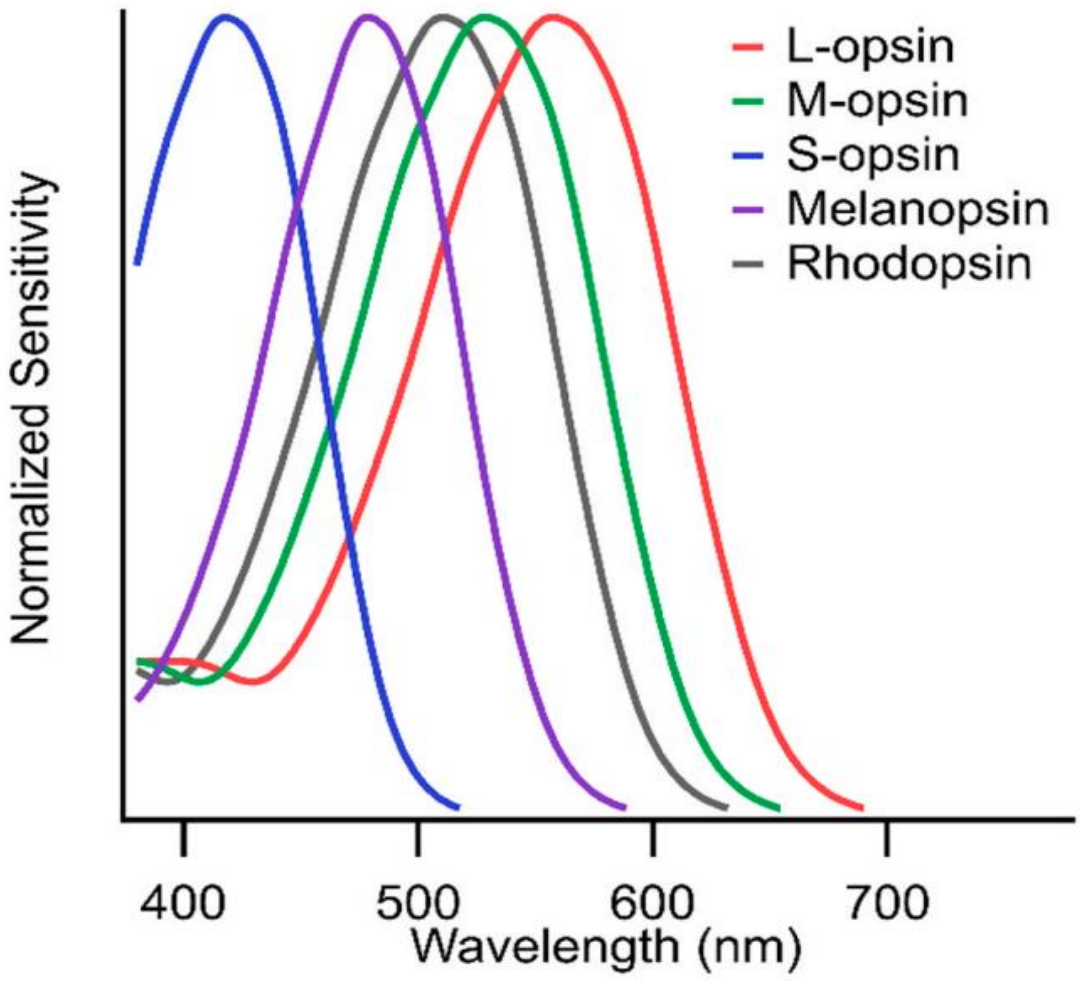
1.12.5 Action Spectra

As stated earlier, NIF responses occur through the combination of the ipRGCs intrinsic response to light along with extrinsic input from the rods and cones (Dacey et al., 2005). These responses are generated through the combined influence from each of the photoreceptors which express different spectral sensitivities to light detection (Lucas et al., 2014). What drives both intrinsic and extrinsic signaling from the photoreceptors to produce NIF responses to light is rather complex. This is due to each of the photoreceptors expressing different spectral sensitivities to light detection and it is unclear how the photopigment signal are combined by photoreceptors and processed in the brain to elicit an NIF response. Rod opsin is the photopigment of the rod receptors and expresses peak sensitivity at 500 nm across all mammalian species. Humans have three types of cones which are S cones, M cones and L cones. Each of the cones express different opsins and are maximally sensitive to different values. Cyanolabe is the photopigment of the S cones and maximally sensitive to shorter wavelengths at -420 nm, Chorolabe, the photopigment of the M cones is maximally sensitive to longer wavelengths -535mn and Erythrolabe, the photopigment of the L cones is maximally sensitive to longer wavelengths at 565nm (Stockman & Sharpe, 2000). Melanopsin is the photopigment of the ipRGCs and is maximally sensitive to short wavelength light ranging from 447-484 nm (Bailes & Lucas, 2013; Brainard et al., 2001; Panda et al., 2005; Thapan et al., 2001). The peak sensitivity is a relative sensitivity which means that each of the photoreceptors will respond to shorter and longer wavelength stimulation but to a lesser extent than when stimulated at peak spectral sensitivity (Vetter et al., 2021). This overlap of spectral sensitivity towards the different wavelengths of light indicates that each of the photoreceptors exert an influence on NIF (Figure 1.17; Michel & Meijer, 2019). However, Spitschan et al. (2019) showcase through using silent substitution methods suggests that the spectral sensitivity of non-visual responses is primarily attributed to melanopsin-containing ipRGCs rather than short wavelength cones. The effects of one particular spectrum of light eliciting a non-visual response is made more complex as it now understood that all the rods and cones photoreceptors contribute to the firing rate of the ipRGCs and this influence is dependent upon the type and duration of light exposure (Lall et al., 2010; Lucas et al., 2014). This is showcased in

pupillary responses where there is no single spectral sensitivity function which can account for this response (Gooley et al., 2012).

Figure 1.17

Spectral sensitivity of the 5 photoreceptors involved in non-visual photoreception in of the human eye.



Note. The peak sensitivity is a relative sensitivity, however, each type overlaps in their spectral response. This means that each of the photoreceptors will respond to shorter and longer wavelength stimulation but to a lesser extent than when stimulated at peak spectral sensitivity. Image taken from Patterson et al. (2021).

1.12.6 Quantifying light for non-image forming responses

Before the discovery of the third photoreceptor, photopic lux was the primary measure of human vision (CIE, 1926). Photic lux describes the luminous sensation of a light sources under conditions where conscious vision can occur. As a result,

photopic lux assumes that the light being measures has the sample spectral distribution as the visual three-cone photopic system (Brainard et al., 1986). However, photopic lux does not consider the biological potency of light on NIF responses. This is evidenced by research indicating that photopic lux poorly predicts subjective sleepiness (Hommes & Gimenez, 2015). As outlined above, NIF responses are caused by both intrinsic and extrinsic signaling from the traditional photoreceptors and intrinsic signaling from the melanopsin expressing ipRGCs. This indicates that all of the photoreceptors contribute to NIF. Given that each of these photoreceptors are maximally sensitive at different spectral sensitivities methods of quantifying the biological potency of light have been created. Lucas et al. (2014) conceptualised Equivalent Melanopic Lux (EML) which is expressed in units of melanopic lux (m-lux). This quantification incorporates the spectral power distribution of light exposure within the context of the five photopigment-specific sensitivity function. The International Commission on Illumination prosed a revision and advancement of EML and conceptualized Melanopic Equivalent Daylight Illuminance (melanopic EDI; CIE, 2018). Melanopic EDI is an international standard which defines spectral sensitivity functions, quantities, and metrics to describe optical radiation for its ability to stimulate each of the five photoreceptors that via the ipRGCs can contribute to NIF responses to light in humans (Schlangen & Price, 2021). Several studies have supported this framework of quantifying light for non-visual responses with melanopic illuminance strongly predicting melatonin suppression, circadian phase resetting and PLR (Brown et al., 2020; Nowozin et al., 2017; Prayag et al., 2019; Santhi et al., 2012; Spitschan, 2019). Ordinal ranking of short wavelength light which is blue wavelength enriched or blue intermediate leads to greater melatonin suppression and lower subjective sleepiness compared to conditions of blue-depleted light exposure. These effects occurred even though the lights with lower melanopsin-derived efficiency had higher photopic illuminance (Santhi et al., 2012). Other alpha-opic illuminances (photopic illuminance of the light stimulus, photopic lux and other photoreceptor-weighted illuminances) provided weaker predictions of melatonin suppression (Nowozin et al., 2017; Prayag et al., 2019). However, melanopic illuminance is not without its limitations in that it has been found to not accurately predict the NIF of alerting responses (Nowozin et al., 2017).

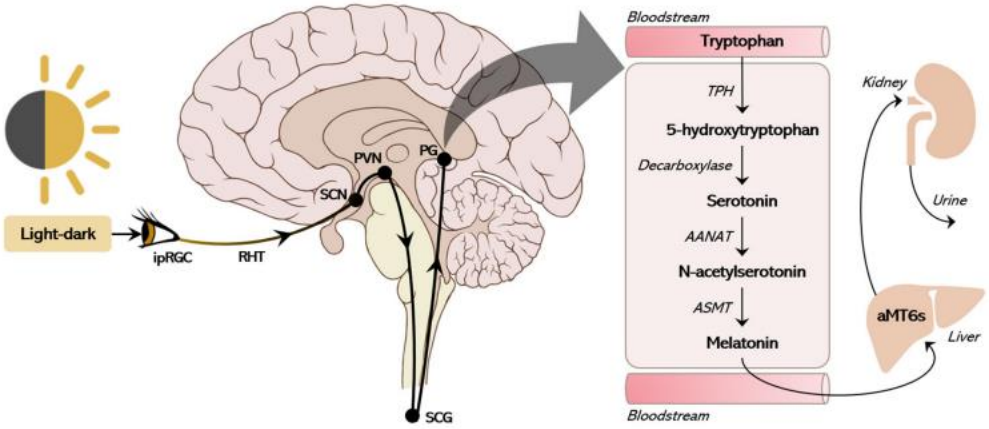
A significant criticism of both EML and melanopic EDI is that it is based on photopigment signals; however, it is unclear how photopigment signals are combined by photoreceptors and processed in the brain. It is not fully understood how EML and melanopic EDI relate to non-visual behaviors in real world-settings (Houser & Esposito, 2021). It is also argued that no single action spectrum can describe all eye mediated non-visual responses to light. Although, each of the five photoreceptors contribute to non-visual responses, the relative contribution of each individual photoreceptor type can vary depending on the specific non-visual response and upon the light exposure properties of intensity, spectrum, duration, timing, spatial pattern, prior light history, and sleep deprivation state of the individual (Schlangen & Price, 2021; Houser & Esposito, 2021). For example, although each of the photoreceptors contribute to PLR the particular influence on the photoreceptors is dependent upon when and how light exposure occurs. Rods and cones account for PLR responses when exposure to light occurs between one and 10 seconds however, when light exposure occurs over 100 seconds the pupil size is controlled by melanopsin with some contribution from the rods (McDougal & Gamlin, 2010). Additionally, the role of the S cone in photoentrainment has yielded mixed findings with some studies indicating its activation by light at night being involved in increases in alertness, melatonin suppression and phase shifting responses (Brainard et al., 2001; Mouland et al., 2019; Thapan et al., 2001). However, Spitschan et al. (2019) found no evidence for the S cone's contribution to acute neuroendocrine and alerting responses. Although Spitschan et al. (2019) findings suggest that the cones may not play a significant role in neuroendocrine and altering responses there is evidence that each of the photoreceptors contribute to PLR (Do et al., 2019; Spitschan et al., 2019). Given the differences in findings based on neuroendocrine, alerting responses and PLR it suggests that the circuit responsible for pupil control may recruit different ipRGCs which are separate to those involved in other non-visual responses, adapts to differences in cone input, or may have temporal integration properties downstream (Spitschan et al., 2019). These differences of findings highlight that the methods which attempt to quantify the biological potency of light on NIF behaviours may not be fully uniform for all responses.

1.13 Melatonin

Melatonin is a neurohormone which is secreted in pineal gland. The synthesis and secretion of melatonin occurs in a diurnal pattern with its peak expression occurring during the dark phase of the solar cycle (Reiter, 1991). The secretion increases soon after the onset of darkness, peaks in the middle of the night and declines gradually during the second half of the night (Figure 1.18; Figure 1.19; Skene, 2003). Exposure to light at night resets the rhythmic expression of melatonin by inhibiting its synthesis and secretion (Lewy et al., 1980; Shanahan & Czeisler, 1991). Klerman et al. (2002) showcased that when some individuals who have no visual awareness are exposed to light they display a suppression of melatonin concentration. Light induced melatonin suppression is a broadly employed indicator for photic input into the SCN to understand the ocular and circadian neural physiology for circadian regulation (Brainard et al., 1997). The onset of melatonin secretion is linked with an increase towards a propensity to sleep (Hughes & Badia, 1997; Rajaratnam et al., 2004), inhibiting the circadian drive for wakefulness (Scheer & Czeisler, 2005) and circulating melatonin acts as a cue which aids in the entrainment of peripheral clocks throughout the body leading to phase-resetting of clock genes (Hardeland et al., 2012). The administration of melatonin can increase subjective sleepiness (Cajochen et al., 1996), increase the quality of sleep (Deacon & Arendt, 1995), facilitate sleep (Cajochen et al., 1998; Wyatt et al., 2006), induce phase shift of circadian phase makers (Attenburrow et al., 1995; Deacon & Arendt; 1995; Krauchi et al., 1997), entrain the circadian clock (Lockley et al., 2000), increase the expression of the clock genes Per1 and Per2 (Kandalepas et al., 2016) and enhance along with synchronising behaviour and physiology in blind individuals (Lockley et al., 2000; Sack et al., 2000). In a study by Gandhi et al. (2015) criterion validity is given to melatonin being required for the regulation of sleep. Zebrafish deficient in melatonin due to a mutation displayed significant reduction in sleep when maintained in light/dark conditions and the circadian regulation of sleep was abolished when placed into free-running conditions (Gandhi et al., 2015). Melatonin receptors are located on circadian clock neurons in the SCN of the anterior hypothalamus (Vanecek et al., 1987).

Figure 1.18

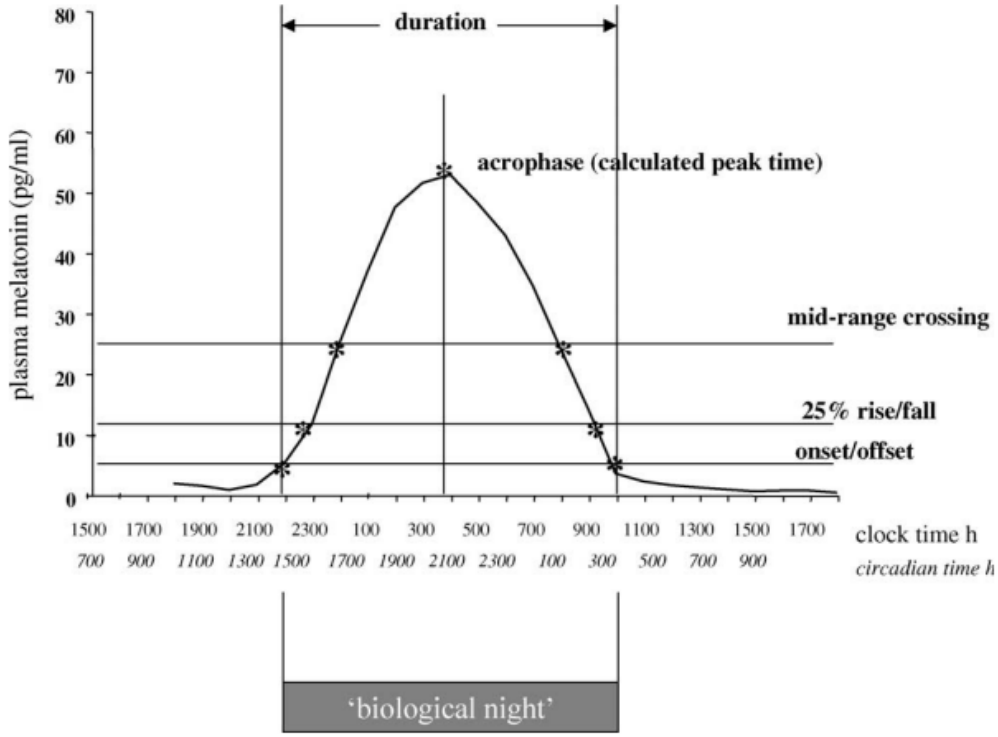
Schematic illustration of the conical pathway involved in production and synthesis of melatonin.



Note. Photic information is projected from environment into the SCN via the RHT. The SCN then projects to the paraventricular nucleus of the hypothalamus (PVN) via GABAergic neurons. When exposure to light does not occur the PVN activates the ganglion cervical nuclei (SCG, via the intermediolateral column of the medulla). This activates noradrenergic fibers that innervate the pineal gland (PG). Here in the pinealocyte the production of melatonin occurs. From there it is released immediately into the cerebrospinal fluid and bloodstream. It then passes through the liver and is converted to 6-sulfatoxymelatonin (aMT6s), which is then excreted in the urine. Image taken from Tonon et al. (2021).

Figure 1.19

Illustration of the normal profile of melatonin secretion (plasma).



Note. The features of this profile and that of salivary melatonin and urinary 6-sulphatoxymelatonin (acrophase, duration, midrange crossing, 25% rise and fall, onset and offset of secretion), used to characterise the timing of the circadian clock are indicated. Expression of melatonin is at its peaks at the dark phase of the night. Image taken from Arendt and Skene (2005).

1.13.1 Intensity of light induced melatonin suppression

It was initially thought that light exposure of at least 2500 lux was required to suppress melatonin (Lewy et al., 1980). However, later evidence suggested that there is a dose-response relationship between light irradiance and the suppression of melatonin with exposure to lower irradiance monochromatic light at levels of .03 photic lux resulting in the lowest suppression (1.83%) of melatonin but exposure to 44.66 photopic lux resulting in a 60.67% suppression in melatonin (Brainard et al., 1988). However, light to which individuals are routinely exposed to in their home settings (<100 lux) can induce suppression of melatonin secretion (Boivin et al., 1996; Cain et al., 2020; Cajochen et al., 2000; Laakso et al., 1993; Lockley et al., 2003; Philips et al., 2019; Santhi et al., 2011; Zeitzer et al., 2000) which are comparable to the effects observed at higher irradiances. For instance, Zeitzer and

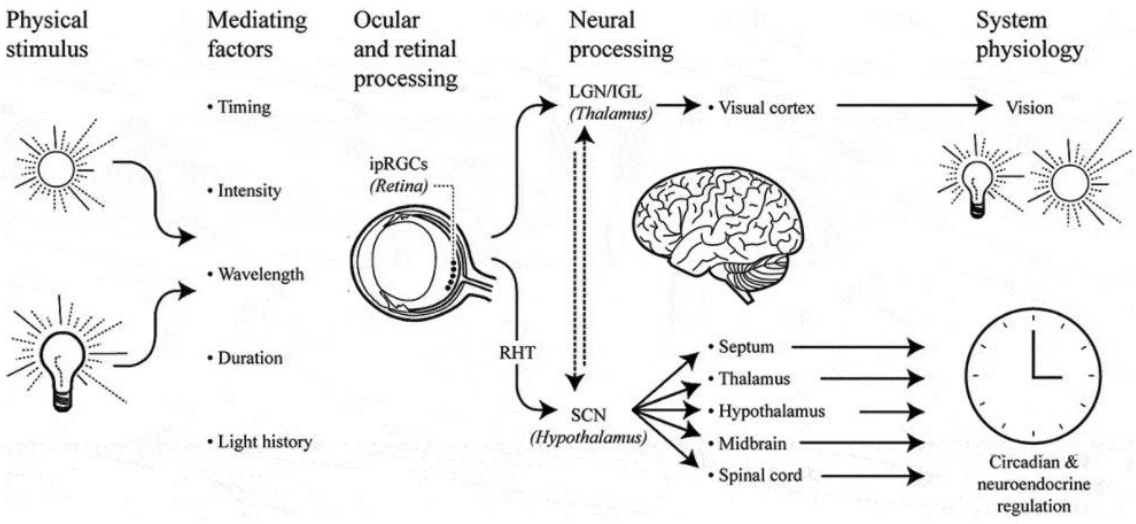
colleagues (2000) found that although exposure to LAN at levels of 9100 lux resulted in a 98% suppression in melatonin exposure to LAN at 106 lux resulted in a near equal amount of melatonin suppression (88%). Similar findings were reported by Philips et al. (2019) who showcased that the human circadian system is highly responsive to dim evening light with an average 50% reduction in melatonin suppression occurring at light levels of 30 lux. When exposure to room light continues throughout the entire night this leads to the total daily melatonin being suppressed by more than 50% with the median suppression being 73.7% (Gooley et al., 2010). These findings suggest that the circadian pacemaker is sensitive to low intensity room light. It is important to note that extreme low levels of LAN still exerts an influence on melatonin suppression (Philips et al., 2019; Zeitzer et al., 2000). For instance, in Zeitzer and colleagues (2000) study exposure to light at 3 lux resulted in a 11% reduction in the suppression of melatonin concentration and shortened the melatonin secretion and whilst these effects are small, there are nonetheless a change. Recurrent nightly exposure to light at levels of 50 lux throughout the biological night leads to melatonin secretion gradually decreasing both during the night-time and over the course of the 24-h day. However, the magnitude of the decrease in melatonin production either during the night or the 24-h period was not associated with the magnitude of the circadian phase shift (Dumont & Paquet, 2014). Across adults however, there is variance in acute responses to light. In healthy adults Philips et al. (2019) provided evidence that within groups there is a 50-fold interindividual difference in the sensitivity of the human circadian system to evening light between individuals. Their individual analysis found that the most sensitive individuals expressed greater than 50 % suppression when exposed to light as low as 10 lux. The least sensitive individual did not display suppression of melatonin until exposed to light at 400 lux (Philips et al., 2019).

As outlined above, LAN exposure has been found to suppress melatonin to low levels. However, Burgess and colleagues (2001) found that administration of melatonin reversed suppression of endogenous melatonin and significantly increased melatonin levels above normal night-time endogenous melatonin levels. The enhanced melatonin secretion observed in the pharmacological melatonin group occurred at the same time as the rise in endogenous melatonin (Burgess et al., 2001). While the onset to sleep latency was increased through exposure to bright light the administration of melatonin paired with bright light resulted in a decrease in onset to

sleep latency and a shorter onset in both stage 1 and stage 2 sleep (Burgess et al., 2001; Cajochen et al., 1998). However, in these studies, exposure to light was at levels of between 3000-5000 lux. Other studies have showcased that the administration of melatonin does not combat the negative effects of light exposure on sleep. For instance, Cajochen et al. (1998) found that exposure to light at night and administration of melatonin on the previous night leads to a subsequent delay in subjective sleepiness and onset of sleep the following day regardless of whether melatonin was administered. This suggests that LAN exposure may lead to not only impacts on sleep in the immediate night but also the following day and this is independent of suppression of melatonin (Figure 1.20).

Figure 1.20

The multiple considerations which have to be considered when investigating the effects of light exposure on non-visual responses.



Note. Light information is passed directly to the SCN and to other brain regions which are involved in non-visual responses. Image taken from Vetter et al. (2021)

1.13.2 LAN, Lux & Spectral Wavelength

As outlined earlier, the circadian system is most sensitive to short wavelength light with a peak spectral sensitivity of 446-483nm (Brainard et al., 2001; Brainard et al., 2003; Cajochen et al., 2011; Lockley et al., 2003; Thapan et al., 2001). These

studies have indicated that the mean percentage of melatonin suppression is greatest when exposed to blue wavelength light. In addition, this wavelength exerts a potent influence on phase shifting the circadian timing system (Brainard et al., 2003). Along with short wavelength light acutely suppressing melatonin these effects are more sustained compared to long wavelength stimuli (Gooley et al., 2010). It has been demonstrated that exposure to low intensity monochromatic light in the short wavelength range (460 nm) induced twice the amount of melatonin suppression than when exposed to 555-nm monochromatic light of equal photon density (Brainard et al., 2001; Cajochen et al., 2004; Lockley et al., 2003). These studies have been essential in suggesting that photopic lux which is the predominant standard unit of measurement is inappropriate when quantifying the photic drive which is required to reset the circadian pacemaker or elicit a non-image forming response. This is further evidenced by Santhi and colleagues (2012) who showcased that the magnitude of melatonin suppression is predicated by the rank order of melanopsin sensitivity and not the rank order of photopic illuminance. For instance, a light source which has greater level of illuminance but a lower melanopic activity will result in less melatonin suppression. These findings indicates that a reduction of the activation of the melanopsin system can lead to a reduced suppression of melatonin.

Along with blue wavelength light having acute effects on melatonin suppression, exposure lengthens the latency of sleep latency and reduces initial EEG delta activity which is a marker of slow-wave sleep (Cajochen et al., 1992; Munch et al., 2006). Evening exposure to monochromatic light at 464 nm can significantly reduce slow wave activity during non-rapid eye movement sleep in the first cycle however, this is compensated by an intra-night rebound of slow wave activity in the last non-rapid eye movement sleep episode (Munch et al., 2006).

The light level which was required for 50% melatonin suppression ranges from 3.1 to 181 melanopic lux (Philips et al., 2018). Cain and colleagues (2020) measured light exposure in individuals home environments and found that average melanopic lux (mlux) to which individuals are exposed to 3-h before bed ranged from 3.9-77.4 mlux with the average exposure being 17.9 mlux. This level of illuminance is high enough to elicit melatonin suppression in those which display the higher level of spectral sensitivity to light (Philips et al., 2019). In Cain et al. (2020) study they predicted that 48% of homes would cause at least 50% melatonin suppression, 73% of homes would causes at least 20% melatonin suppression and

15% of homes would causes at least 80% melatonin suppression. Their study also highlighted the prevalence of interindividual responses with regards to acute suppression of melatonin due to habitual home environment light exposure. For the most sensitive individuals, 100% of home light was predicted to cause at least 50% of melatonin suppression whereas for the least sensitive individuals 0% of homes were predicted to cause at least 50% melatonin suppression. The average home led to a wide range of predicted individual responses from 0 to 87% melatonin suppression. Cain et al. (2020) reported that home lighting persisted at biologically impactful levels throughout the evening. At 8pm the median melanopic lux was 13.4 (2.5-67.8) and at 10pm the median melanopic lux was 11 (2.4-49.6). These findings indicate that home lighting creates an extended twilight for the circadian system weakening the distinction between day and night. Cain et al. (2020) report that increased melanopic lux in the 3-h before bedtime was associated with increased wakefulness for that individual in the 90 min after bedtime. This association was maintained after controlling for age, sex, average bedtime, chronotype, sleep quality and insomniac symptoms. These results are consistent with the findings that exposure to blue-enriched light before bedtime results in reduced NREM sleep SWA which is classic hallmark of sleep pressure specifically in the first cycle (Chellappa et al., 2013). However, in a later study Chellappa et al. (2017) reported that males have increased NREM SWA compared to females. Santhi and colleague's (2011) field study which monitored light exposure in the home setting found that the levels of artificial light that individuals are exposed to in the home setting was sufficient to suppress melatonin, delay the timing of sleep. Bauer et al. (2018) highlight in their review that a number of human studies which utilise younger participants with a variety of digital devices or LEDs as the light source, varying protocols for time and intensity of exposure. Bauer and colleagues argue that although the studies are small the findings are consistent with bright light of blue wavelength origin inhibiting melatonin secretion (For review see Bauer et al., 2018).

Electric devices such a smartphones, e-readers, laptops, and smart televisions to which individuals are habitually exposed to in their home environments emit LED lighting typically is comprised of light from the blue wavelength spectrum. Cain et al. (2020) reported that over a 6/7 week period on 76% of nights there was one clearly predominant type of light source. On the nights with the predominant light source the 50 were fluorescent lights, 44 were LED and 30 were incandescent. LED

and fluorescent lighting provided similar types of melanopic illuminance which was significantly higher than the incandescent lighting. Cajochen et al. (2011) compared the impact of a LED-backlit computer screen in comparison to a LED-free computer screen over the period of 5 hours and examined differences in melatonin suppression, subjective sleepiness, cognitive performance and the EEG during wakefulness. LED-backlit computer has significantly greater melatonin suppression which rose also significantly later compared to the LED-free computer, behaviourally there was a decrease in subjective sleepiness along with an increase in alertness as measured objectively using a cognitive task. Chang et al. (2015) found that use of an e-reader for a period of 4-hours resulted in a significant suppression of melatonin secretion, a 1.5-h delay in circadian phase along with a decrease in the level of subjective sleepiness and theta activity which has been associated with a decrease in sleepiness. Wood and colleagues (2013) observed that that melatonin levels were significantly suppressed after a two-hour exposure but not after a one-hour exposure to iPads. However, Gronli et al. (2016) found that the use of an iPad for 30 minutes at full brightness decreased sleepiness and delayed slow-wave brain activity during sleep by about 30 minutes.

When spectral characteristics to light are fine-tuned to reduce blue wavelength light exposure this can attenuate non-visual responses to light in humans. For instance, a number of studies have demonstrated that filtering out short wavelength light from high intensity (> 1000 lux) blue wavelength light can attenuate the suppression of melatonin (Rahmen et al., 2011; Sasseville et al., 2006; Souman et al., 2018). Rahmen et al. (2017) compared fluorescent light and blue depleted LED light at a level of lux which is the intensity of light that individuals are typically exposed to in their bedroom to examine differences in levels of melatonin suppression, alertness, and sleep. Their results found that blue-depleted LED lighting attenuated melatonin's suppression response to light and decreased levels of alertness as measured by EEG and behavioural reaction time performance on a cognitive task however no differences were observed between the two groups on sleep related outcomes as measured by polysomnography. Kayumov et al. (2005) demonstrated that the use of goggles which eliminated wavelengths of light less than 530 nm resulted in a preservation of melatonin levels which was similar when individuals were exposed to light as low as 5 lux compared to the suppression observed at 800 lux. Similar findings were reported by Zerbini et al. (2018) who

found that the use of blue-light blocking glasses in the evening led to an advance in melatonin onset and sleep onset on weekdays. However, it is important to note that these effects did not continue after one week. Strategies have been employed by the manufactures of the light emitting devices to reduce the impacts their devices have on eliciting non-image forming responses. However, these strategies such as Apple's night mode may not be having the strongest effect. For instance, Nagare et al. (2019) examined whether enabling night mode on Apple devices attenuated the suppression of melatonin. Although, typical use of the device without night mode resulted in the largest percentage of melatonin suppression the difference between the night mode with low and high correlated colour temperature still displayed reduction in melatonin suppression albeit in an attenuated manner. This indicates that adjusting the spectral composition of the device without adjusting its brightness is insufficient in avoiding melatonin suppression and the circadian timing system.

There is conflicting evidence of whether the intensity of light or the wavelength of light is more important in having acute circadian effects. Souman et al. (2018) provided strong evidence that it is not illumination level or the correlated colour temperature that causes the acute suppression of melatonin but instead its exposure to light which is of specific spectral range to elicit greater suppression of melatonin. These results are further supported by Brown et al. (2020) who showed that a melanopic EDI below 4lux results in minimal responses (<25%) of maximum melatonin suppression) and melanopic EDI above 300lx strongly suppresses salivary melatonin (>75% of the maximum) (Brown et al., 2020). However, Brainard and colleagues (2008) displayed that at high light intensities acute suppression of melatonin may be induced by longer wavelengths of 630 and 700 nm and short wavelength of 420 nm (Brainard et al., 2008). This suggests that the magnitude of melatonin suppression at all wavelengths is proportional to light intensity. Prayag and colleagues (2019) provide evidence that it is not the spectral wavelength of light that is critical for eliciting a non-visual response but instead the duration of light exposure. In their study Prayag et al. (2019) demonstrated that exposure to blue-enriched (230mlux) or red-enriched (90mlux) light for a period of 50 minutes does not lead to differences in eliciting of a non-visual response. Prayag et al. (2019) argue that the reasoning for no differences in non-visual behaviours across the lighting conditions was due to all non-visual responses reaching a saturation response when exposed to light for a sufficient duration. This indicates that after

passing a certain threshold of brightness a particular non-visual response can reach saturation leading to the actual spectral composition above this brightness less relevant for the response-size (Cajochen et al., 2019). These findings highlight those non-visual responses can be saturated by relatively low light levels regardless of the spectral wavelength of light. This suggests that the effects of light on the circadian system is dependent upon the type of non-visual response being investigated which is mediated by the type of exposure, duration, prior photic history, and the experimental context.

Although, home setting exposure to devices which emit blue wavelength have been found to suppress melatonin in young and older adults (Gabel et al., 2017) it has been found that younger aged children have significantly greater melatonin suppression when exposed to high or low colour lighting compared to older adults (Lee et al., 2018). This may be suggestive that as individuals age there is less absorbance to short wavelength light due to loss in crystalline lens transmittance and pupillary miosis (Barker et al., 1991; Winn et al., 1994). These physiological differences to the eye may result in differences in melatonin sensitivity towards light as age increases. Significantly to note from Lee and colleagues (2018) study is that in older adults no within groups differences are observed between high and low colour lighting and melatonin suppression. This may be due to a reduction in the amount of melatonin secretion as individuals age (Crowley et al., 2012).

1.13.3 Light Duration

Suppression of melatonin has been observed for when light exposure only occurred for a small period of time (i.e. 15 minutes; Gronfier et al., 2004; Hilaire et al., 2007). Gronfier et al. (2004) demonstrated that exposure to intermittent light is also effective at resetting the human circadian system. The phase-resetting effects of 5-h of continuous bright white light (-10,000lux) are comparable to a 5-h intermittent exposure of six cycles of 15 mins of bright light (-10,000lux). This indicates that a single sequence of intermittent bright-light pulses has a greater resetting capacity on a per minute basis than does continuous light exposure. Chang and colleagues (2012) have provided evidence that that magnitude of melatonin suppression increased as the duration of exposure to light increased. However, the relationship between duration and intensity is non-linear for phase shifting responses with shorter pulses

of light leading to larger physiological effects per minute of exposure. Specifically, exposure to short wavelength light for .2-h resulted in one hour phase delay with a 4-h exposure leading to 2.6-h phase delay. These findings suggest that shorter duration of light exposure is 5 times more efficient in phase shifting of the clock than longer durations of light exposure. Prayag et al. (2020) demonstrate that the duration of light exposure required to elicit a non-visual response differs across responses with low light levels PLR and EEG density being modulated within one minute of light exposure while heart rate and thermoregulation can be modulated within 2 and 5 minutes respectively. These findings highlight that low intensity and short duration light exposure can elicit a non-visual response. This suggest that even brief exposure to technology before bed can lead to negative consequences for sleep.

1.14 Light and Disruption to Sleep

Light when ill-timed influences the circadian clock through the acute suppression of melatonin and the shifting of the circadian phase (Blume et al., 2019). However, it important to note that melatonin suppression cannot be used as a proxy of circadian phase shifting. Studies have reported no association between light induced melatonin suppression and light induced phase shifting of the circadian clock with phase shifts occurring after exposure to light despite negligible suppression of melatonin suggesting that acute responses or phase shifting responses do not arise from a unitary pathway (Rahmen et al., 2018; Zeitzer et al., 2011). Additionally higher levels of light intensity are required for melatonin suppression than is required to induce phase shifts. This indicates that phase shifts can be induced by exposure to light in the absence of an accompanying decline in melatonin suggesting that there is no direct relationship between each of these non-visual responses. Although both melatonin suppression and phase shifting responses to light occur the same portion of spectral sensitivity (Brainard et al., 2001; Wright & Lack, 2001) they may not be necessarily mediated by the same neural pathways and are not necessarily proxies for one another. For example, the non-visual response of PLR can be modulated by classical photoreceptors. Evidence has suggested that initial pupillary constriction is mediated by rods and cones in the outer retinal whereas the ipRGC in the inner retina mediates a sustained pupil constriction after the offset of light stimulus known as post-illumination pupil response (Park et al.,

2011). Secondly, although light exposure may elicit a non-visual behavioural response the speed at which this non-visual response occurs is different across responses and therefore a direct relationship from one non-visual response cannot be inferred onto another response. For instance, at low light intensities of 90 mlux pupillary response and EEG power densities can occur at one minute of light exposure while changes to heart rate and thermoregulation can be modulated at 2 and 5 minutes respectively (Prayag et al., 2019). Finally, Prayag and colleagues (2020) showcase those non-visual responses do not exhibit the same sensitivity curves and instead display their own unique sensitivity to light and a specific range in response to light amplitude which is constrained by light intensity and the duration of exposure to light. This indicates that each non-visual response differs in terms of sensitivities which is dependent upon the intensity and duration of light (Chang et al., 2012; Zeitzer et al., 2000; Prayag et al., 2019). The findings from these studies suggest that melatonin suppression cannot be used as a proxy for circadian phase resetting. For example, in a number of participants exposure to light for a long period of time resulted in robust phase shifts however, this occurred with negligible suppression of melatonin. This is of critical importance to understand in that if light exposure is below the threshold of irradiance required for melatonin suppression other systems may, in turn, undergo light effects, such as homeostatic rather than circadian mechanisms (Lockley & Gooley, 2006).

1.14.1 Effect of Irradiance on Phase Shifting

Bright light was more strongly associated with objective sleep compared to moderate and dim light. Specifically, bright evening light was associated with a delay of the sleep period evidenced by polysomnography longer sleep onset latency (Burgess et al., 2001; Campbell & Dawson, 1992; Cooke et al., 1992; Dijk & Borbely, 1991, Komoda et al., 2000) and delayed sleep onset (Burgess et al., 2001; Cajochen et al., 1998; Campbell & Dawson, 1992). However, light exposure which is at the irradiance level to which individuals are routinely exposed to in their home settings has been observed to lead in phase shifting effects (Gooley et al., 2011; Philips et al., 2018; Zeitzer et al., 2000). For example, Zeitzer and colleagues (2000) observed that exposure to light at levels of 106 photopic lux resulted in more than half (1.8h) of the phase shifting response that occurred when individuals were

exposed to 9100 lux of light (3.2h). Cajochen et al. (2000) report that exposure to light of 106 lux which is typical home setting light can lead to increases in subjective alertness, a reduction in subjective sleepiness, reduction in slow eye movements, a reduction in of EEG activity in the theta-alpha frequencies. Burgess and Molina (2014) carried out a field study with a within groups design where individuals were exposed to levels of LAN of 65 lux and then 3 lux. Their results found that when individuals were exposed to light at levels of 65 lux their DLMO was on average 1.03-h later and this also coincided with a later timing of sleep. In addition to this, exposure to light even at levels as low a 3lux leads to phase shifting effects. Again Zeitzer et al. (2000) demonstrated that 3lux resulted in a phase shift of DLMO by .07h. Although this effect of light on the circadian parameters was small it nonetheless is a change. Similar observations have observed that light for a period of 4-h before habitual bedtime at levels of 10, 30 and 50 lux can lead to DLMO phase shifting responses of 22, 77 and 109 minutes respectively (Philips et al., 2018). Each of these studies provide strong evidence that the human circadian pacemaker is sensitive to light. Duffy and Wright (2005) report that humans have the capacity to keep stable entrainment to a 24-h cycle even when ambient light levels are around 1.5lux. This suggests that low light environments can induce small phase shifts in the circadian system. In animal studies, exposure to LAN as low as 1 lux has been found to impact on the common circadian rhythm parameters such as expanding the intrinsic period (Butler et al., 2012; Evans et al., 2007), increasing of the active phase as waveform of the cycle (Coomans et al., 2015) and amplitude (Wallbeek et al., 2021). Additionally, entrainment still occurred during exposure to dim light of .0005 lux for 12-h during the light phase followed by 12-h of darkness (Butler & Silver, 2011).

1.14.2 Effect of Spectral Sensitivity on Phase-Shifting Responses

The human circadian system is highly sensitive to blue- enriched short wavelength light (LED lighting). Short wavelength light is commonly used in rooms including lamps, night lights, desk lamps, ceiling fixtures and accent lights amongst all mobile devices, TVs fitness trackers, laptops, and e-readers (Anderson, 2013; Bauer et al., 2018). Cain and colleagues (2020) in their observational study noted that 35% of participants are exposed to LED lighting before sleep and 40% are

exposed to fluorescent light. Both of these light sources have a high melanopic lux level (Cain et al., 2020). Most personal light emitting technologies such as computers, televisions, electronic tablets, and smartphones which are routinely used in close proximity to the eyes before bedtime contain blue wavelength light. A large self-report study found that 90% of individuals use a technological light-emitting device <1-h before bed. Before bedtime, 60% report to watching television, 72% of adolescents and 67% of young adults admitted to using smartphones versus 36% of middle aged and 16% of older adults (Gradisar et al., 2013).

Lockley et al. (2003) showcased that exposure to monochromatic light which is blue wavelength elicited more a 1.31h greater phase delay in DLMO compared to longer wavelength light. This occurred despite an equal photon density between the two light sources (Lockley et al., 2003; Warman et al., 2003; Wright et al., 2004). The strong effect of short wavelength light was repeated by Warman and colleagues (2003) who reported that exposure to short wavelength light after habitual wake time led to similar phase advancing effects on melatonin acrophase and offset as exposure to white light which contained 185-fold more photons. Bauer and colleagues (2018) in their review highlight several studies carried out on healthy participants which found that exposure to LED from devices which are typically used in the biological evening was associated with delayed onset to sleep, reduced total sleep time, and increases in daytime sleep. Experimental studies have reported that exposure to short wavelength light results in lower levels of subjective sleepiness in comparison to blue-depleted light or longer wavelength light (Lockley et al., 2005; Santhi et al., 2012). These effects in using these light emitting devices can be seen the following day with using an iPad in the 4 hours before bedtime delaying DLMO by 1.5h the following day (Chang et al., 2015). Studies using objective measures of sleep have reported that evening exposure to blue-enriched light leads to a greater delay in sleep onset and latency to persistent sleep after lights were turned off along with delayed latency to slow-wave sleep and REM sleep latency in comparison to blue depleted light (Santhi et al., 2012) or complete darkness (Munch et al., 2011). Additional research demonstrating the effect of short-wavelength light on sleep architecture comes from Chellappa and colleagues (2013) who reported that exposure to 40lux short wavelength light before sleep weakens frontal NREM sleep slow wave activity which is a classical marker of sleep pressure compared with exposure to longer

wavelength light. Exposure to short wavelength light stimulates enhanced expression of the clock gene PER2 compared to green light (Cajochen et al., 2006).

Although, light exposure at a particular time can phase delay the timing the sleep, it is proposed that this could also be mediated through light having an alerting effect and delaying the homeostatic drive for sleep (Altimus et al., 2008; Cajochen et al., 1992; Chellappa et al., 2013; Lupi et al., 2008; Tsai et al., 2009). As outlined earlier the timing of sleep is regulated by both homeostatic and circadian processes. Put briefly, the homeostatic drive for sleep increases throughout the evening and dissipates during sleep. However, exposure to light particularly short wavelength light has been found to increase alertness and arousal (Cajochen et al., 2000; Cajochen et al., 2005; Lockley et al., 2005; Lockley et al., 2006) with may reduce the drive for sleep at night which leads to delaying the onset of sleep. This is argued to negatively impact homeostatic sleep regulation. How blue enriched light leads to these differences in homeostatic sleep pressure is possibility mediated through light exposure increasing alertness (Chellappa et al., 2011). Support for this argument comes from the above-mentioned studies showcasing that exposure to short-wavelength light increases alertness (Chapella et al., 2017) while also reducing NREM slow wave activity which is an index of sleep pressure (Cajochen et al., 2010; Chellappa et al., 2013).

In rats filtering out of wavelengths shorter than 500nm have been found to attenuate phase delay shifts in locomotor activities by 40-50% compared to unfiltered light exposure (Gladanac et al. 2019). The reduction in phase-delay shifts correspond were associated to regionally specific attenuation in molecular markers of SCN activation in response to light exposure which include c-FOS, Per1 and Per2. However, when light at levels were of low irradiances there were no differences on phase shifting effects between filtered and non-filtered light. This indicates that filtering short wavelength light under low irradiance conditions does not reduce the activation of visual photoreceptors, which can continue to signal to the pacemaker and contribute to phase resetting. This is of particular importance to suggest that strategies of filtering wavelength of light on electronic devices which are commonly used during the night may not be effective in attenuating phase-shifting effects of light if the intensity of the light is low.

1.14.3 Photic History

The previous photic history – the intensity of daytime light exposure during the biological day – can impact on acute and phase shifting responses. Evidence from animal studies have reported that the circadian resetting response to a light stimulus can be reduced by a preceding non-saturating light stimulus (Nelson & Takahashi, 1999). In humans, Hebert et al. (2002) demonstrated that melatonin suppression was greatest in those who were exposed to dim light (<200 lux) during the day and exposed to 3-h light exposure (500-lux) during the night compared to those who were exposed to bright light during the day (5000-7000-lux). Two other studies reported that exposure to very dim light history sensitizes the circadian system to eliciting heightened phase-shifting and acute responses to light (Chang et al., 2011; Smith et al., 2004). In particular, Chang and colleagues (2011) report that a prior history of light exposure at levels of 1lux leads to a 68% greater suppression in melatonin compared to a light history 90lux. Additionally, the same study reported that a light history of 1lux elicited 38-minute (62% greater efficiency) phase delay compared to a photic history of 90lux. These findings are supported by Rangtell et al. (2016) who found that exposure to bright light during the day led to no differences in suppression of melatonin or differences in the subjective and objective sleep parameters when comparing reading a novel and using a tablet computer for two hours. Separately, Chang and colleagues (2013) using the same crossover design as the previous study found that a prior photic history to dim light resulted in alerting responses being greater and lasting longer. The impact of prior photic history is not restrictive to non-image forming behaviours with previous light history also impacting the sensitivity of the intrinsically photosensitive retinal ganglion cells (ipRGCs) which controls circadian photoreception. These cells display light and dark adaptation by becoming desensitized by exposure to brief light flashes and becoming re-sensitized by period spent in darkness (Wong et al., 2005).

While a prior light history to low level light during the day increased levels of LAN in the biological night can have adverse impacts on circadian responses and sleep, a prior light history of bright light during the day has been found to be associated with positive impacts on sleep and circadian rhythms. Cajochen and colleagues (2019) provided evidence that photic light history modulates homeostatic regulation of human sleep. In a forced wakefulness protocol exposure to strong

bright light during the day has been found to be associated with less sleepiness and a higher homeostatic sleep pressure response compared to being exposed to dim light (Cajochen et al., 2019). This indicates that environmental factors during wakefulness may impact on human sleep architecture. Further studies in animals have indicated that exposure to brighter light during the day is associated with increases in rhythm robustness (as indexed by locomotor activity), increases in SCN electrical activity (as indexed by increases in the spontaneous firing rate) and increases in circadian amplitude (as indexed by increases in membrane depolarization in the SCN). Most notably, the positive impacts of a bright photic light history was maintained once the light stimulus was removed and placed into free-running conditions (Bano-Otalora et al., 2021). In a study comprising of 400,000 adults increased time spent outdoors was associated with lower frequency on insomnia symptoms, less frequent tiredness (Burns et al., 2021). These findings are consistent with Wams et al. (2017) who report that being exposed to higher light intensities during the day lead to higher SWS accumulation. The plausible argument for improved outcomes to sleep due to a photic history to bright light during the day could be explained by increases in the rhythm amplitude (Ancoli et al., 2003) or improving the alignment between sleep onset and the circadian signal (Dijk & Czeisler, 1994).

1.15 Circadian Disruption – What does it mean?

As highlighted above light exposure when ill-timed can result in both acute and phase shifting responses in the circadian system. The chronic exposure to LAN may lead to chronic disturbance of the circadian system which is referred to as circadian disruption (Vetter, 2020). Circadian disruption is operationalized as a disturbance of biological timing which can occur at different organizational levels and/or between different organizational levels (Qian & Scheer, 2016). This disruption can result in disturbances in molecular rhythms of individual cells to misalignment of behavioral cycles to environmental changes. However, within the literature other terms are interchangeably used to refer to circadian disruption such as circadian misalignment, circadian desynchrony, circadian desynchronization. However, circadian disruption should be conceptualized as an umbrella term which each of the aforementioned terms referring to disturbance at different levels within the system or between the system and the environment. Table 1.1 outlines how each

of these terms are operationalized. It is important to recognize however, that the metrics often employed to quantify the different levels of circadian disruption are often proxies of the variable as sleep timing as indicated by self-report measures or actigraphy is not pure circadian output and do not provide information on endogenous clock function or facilitate understanding of organismal or molecular interplay of clock-regulated process (Broussard et al., 2017; Vetter, 2020).

Table 1.1

Overview of definitions used to define circadian disruption.

Circadian Disruption		
(i)	**Circadian desynchrony/ Circadian desynchronization**	Interchangeable terms which refer to differing periods between two (or more) rhythms (e.g. relationship between temperature and sleep/wake rhythms)
(ii)	**Circadian Misalignment**	Abnormal phase angle between two or more rhythms (central and peripheral). Rhythms may be both internal or internal and external (central and sleep/wake)
(iii)	**Chronodisruption**	Synonymously used to refer to circadian disruption. However, also used as a metric to quantify the overlap between sleep timing on a biological night and work hours.
(iv)	**Circadian Dysfunction**	Resulting from damage to the SCN or adverse genetic variations.

1.16 Effect of Lighting Schedules on the circadian clock

Exposure to light may induce misalignment between environmental or behaviour cycles and physiology or exacerbate disruption to the circadian clock at a behavioural and molecular level. However, the effects are different dependent on the type of light exposure.

1.16.1 Constant Light

In both mice and rat studies constant light exposure can lead to circadian arrhythmicity, disruption of the sleep-wake cycle, circadian rhythm behaviour and decreased locomotor activity and rhythm splitting (For review see Coomans et al., 2013; Fonken et al., 2010; LeGates et al., 2014; Stephenson et al., 2012). In nocturnal animals constant light results in decreased time spent awake (Stephenson et al., 2012). At a molecular level long term constant light leads higher levels of PER2 (Munoz et al., 2005) and clock gene rhythms become desynchronised and/or altered organization of cellular oscillators within the SCN (de la Iglesia et al., 2000; Ohta et al., 2005). At an electrophysiological level, the amplitude of SCN firing is reduced with a more variable firing rate (Coomans et al., 2013). However, some animals still display rhythmicity under constant conditions which can be achieved through lengthening of the period or splitting of locomotor activity rhythms (Fonken et al., 2008; Fonken et al., 2010; Fujioka et al., 2011; Ma et al., 2007; Ohto et al., 2005).

1.16.2 Jet Lag

Transmeridional travel can lead to sudden shifts in time-zones resulting in a shift of the light/dark cycle. Animal models been used to investigate the effects of acute jetlag whereby there is a single advance or delay in the light/dark cycle and chronic jetlag were repeated shifts in the light/dark cycle occur. At a behavioral level, acute jetlag protocols have found that rodents gradually shift their activity over several days to re-entrain to the new light/dark cycle. For example, if a 6-hour advance in the light/dark cycle this results in the animal shifting their activity by approximately one hour per day resulting in it taking about 5-6 days to re-entrain. At a molecular level, the speed in which SCN clock genes adjust is more rapid

(Yamazaki et al., 2000). However, the speed of shifting across peripheral clocks differ (Kiessling et al., 2010).

1.16.3 Dim Light at Night (dLAN)

dLAN protocols aim to mimic urban/house levels of LAN exposure to study effects on sleep and circadian rhythmicity. These studies aim to investigate the impact of mankind's modern lifestyle to LAN sources such as light-emitting personal devices, light pollution from the external environment, and LED lighting. In these studies, the intensity of dLAN employed is between 5-20lux as is suggested to be comparable to the levels of light exposure experienced in urban areas (Gatson et al., 2012) and sleeping environments (Obayashi et al., 2013). Dim light has been observed to reduce locomotor activity (Bedrosian et al., 2013a; Bedrosian et al., 2013b; Fonken et al., 2010; Stenvers et al., 2016) and increase the period of the rhythm (Stenvers et al., 2016). At a molecular level the amplitude of rhythms such PER1 and PER2 in the SCN have been found to be blunted (Bedrosian et al., 2013; Stenvers et al., 2016). Expression of BMAL1, PER1, PER2, CRY1, CRY2 and Rev-Erb are all repressed in the liver by exposure to dLAN (Fonken et al., 2013). Other studies using other animals have found that although dLAN does not lead to differences in locomotor activity, there were reductions in PER1 and PER2 (Bedrosian et al., 2013). Tam et al. (2021) reported that exposure to 12h light/4h dLAN/8h dark cycle results in locomotor activity onsets, midpoints and offsets becoming delayed by up to 2-3 hours. At a molecular level clock rhythms are delayed in peripheral tissues along with modifying patterns of hypothalamic and cortical cFos signals which are a molecular correlate of recent neuronal activity. There have been inconsistent findings to the extent that dLAN impacts on sleep architecture and sleep quality. Some studies using animal models have significant effects on sleep disruption (Borniger et al., 2013) while others have reported that dLAN increases time spent in NREM in during the dim phase, gradually decreased duration of NREM sleep during the light phase and decreased the amplitude of the SWA rhythm (Stenvers et al., 2016; Panagiotou & Doeber, 2020).). Additionally, Stenvers and colleagues reported that dLAN increased sleep in the active phase and decreased sleep during the light (inactive) phase.

1.17 Light and Circadian Disruption

1.17.1 Light as an effector of circadian disruption

Exposure to light at night has both a direct effect on circadian rhythms and enables additional activities which lead to circadian disruption. As previously outlined, circadian rhythms are 24-h recurring rhythms of physiological processes which are coordinated by internal biological clocks which are regulated at the molecular level by clock genes. Without time cues these clock genes oscillate to a near 24-h period. However, both internal and external cues result in the circadian rhythm being entrained to a precise 24-h period. This leads to a precision for several cycles under circadian control such as sleep-wake cycles, body temperature, blood pressure and metabolism (Lunn et al., 2017).

When light exposure is ill-timed and not aligned to the light/dark solar cycle this can lead to direct effects on circadian rhythms through disruption of the circadian rhythmicity which may lead to downstream dysregulation of circadian rhythms in peripheral systems (Walker et al., 2020). At the circadian molecular clock level, LAN exposure leads to the disruption of the clock genes involved in the transcriptional-translation cycle by rapidly inducing the expression of Per1 or Per2 which is dependent on whether the light occurs during either the early or late night (Albrecht et al., 1997). LAN can lead to alterations to non-photic synchronising cues such as hunger, timing of food intake, and composition of food eaten which can lead to negative alterations of synchronising cues of peripheral clocks (Lunn et al., 2017). As previously discussed at an endocrine level LAN exerts a direct effect on delaying and suppressing production of melatonin which is a robust marker of circadian rhythmicity. Melatonin's suppression by an external environment stimulus can disrupt the synchronisation of peripheral clocks (Hardeland et al., 2012). Given that light exerts a significant influence on entraining circadian rhythms, exposure to LAN could dysregulate both direct and indirect signaling which leads to shorter sleep duration and quality of sleep and may be a contributing factor to various negative health outcomes (Blume et al., 2019; LeGates et al., 2014; Lunn et al., 2017; Mason et al., 2018; Touitou et al., 2017; Tahkamo et al., 2018; Walker et al., 2020).

1.17.2 Light as an enabler of circadian disruption

Light is argued to act as an enabler of circadian disruption. Electric lighting affords individuals the opportunity to carry out work and social activities during the night which have indirect effects on the timing of sleep which can lead to shorter sleep duration (Cho et al., 2015; Stothhard et al., 2017; Xiao et al., 2020) and poorer quality sleep (Cho et al., 2016; Esaki et al., 2019). Individuals are routinely exposed to artificial lighting in their homes and sleeping environment (Cain et al., 2020) and outside their living environments environment (Falchi et al., 2016; Kyba et al., 2017). Satellite images of the Earth at night have showed that outdoor lighting from street lighting, buildings and advertisements are observed in major cities and surrounding areas resulting in LAN covering around 80% of the world (Falchi et al., 2016) with this percentage increasing yearly (Kyba et al., 2017). The light intensity of an average urban street is estimated at 5-15 lux and a typical living room is between 30-300 lux (Cain et al., 2020; Gatson et al., 2013). Those residing in areas with higher levels of outdoor light as measured by satellite data report delayed bedtime and wake up time, shorter sleep duration, increased daytime sleepiness and report poorer quality and quantity of sleep, higher incidences of insomnia and increased daytime sleepiness (Koo et al., 2016; Ohayon & Milesi, 2016).

LAN exposure can elicit disruption to sleep (Cain et al., 2020; Obayashi et al., 2014). For instance, Wams et al. (2017) report that individuals with exposure to light greater than 10 lux in the evening had more nocturnal awakenings and less slow-wave sleep. Individuals who sleep with the light (40 lux) displayed more shallow sleep, increased arousals, and decreased brain oscillations during sleep (Cho et al., 2013). Munch et al. (2006) reported that exposure to short wavelength light for a period of two hours before habitual bedtime sleep resulted in decreased slow wave activity leading to shallow sleep. This suggests that the alerting effects of light impact on the architecture of sleep. However, this study also reported increased slow wave activity later in the night suggesting a compensatory mechanism.

Personal light emitting devices are now routinely used in the sleeping environment and in the hours proceeding sleep. These devices emit around 40 lux of blue wavelength light depending on the size of the screen (Wood et al., 2013). This is the spectrum and intensity of light which elicits both acute- and phase shifting-responses (Cajochen et al., 2011; Chang et al., 2015). However, these devices can

additionally lead to disruption to sleep both subjectively and objectively. Chang and colleagues (2015) reported that utilisation of personal light emitting devices such an e-reader for a four- hour period resulted in an increase in latency to sleep onset, a reduction in both subjective sleepiness and next morning alertness. These findings are further supported by Gronli et al. (2016) whom in addition to previous studies found that the use of an iPad delayed the EEG dynamics of slow wave activity by approximately 30 minutes and reduced slow wave activity after sleep onset compared to reading from a book. However, no differences were observed with regards to time spent in different sleep states and self-reported sleep onset latency

1.18 Sleep and Circadian Rhythm Disruption associated with negative health outcomes

As previously outlined the molecular circadian clock operates in almost every cell throughout the body imposing control and regulation over the physiological activity of different tissues and organs resulting in cyclic variations in gene expression and tissue function (Hastings et al., 2003; Patke et al., 2020). The circadian system is a product of complex interactions among multiple brain regions neurotransmitter systems and modulatory hormones (Wulff et al., 2010). When the circadian clock is optimally synchronized this results in physiological homeostasis however, when the clock is not synchronized this leads to circadian disruption which can disrupt physiological activity resulting in adverse health outcomes such as neurological, psychiatric, metabolic, endocrine, cardiovascular and immune function comorbidities (Logan & McClung, 2020). As a result, both sleep and circadian rhythm disruption (SCRD) are observed to be a common feature of adverse health outcomes (see Figure 1.21; Jagannath et al., 2013; Logan & McLung, 2019).

Sleep disruption and abnormalities in sleep architecture are also a risk factor for the development of adverse health outcomes (Karatsoreos, 2014; Wulff et al., 2010). A major component of mood, anxiety and psychotic disorders is disruption to the sleep-wake cycle with up to 80% of those with diagnosed depression or schizophrenia reporting sleep abnormalities (Wulff et al., 2010). Some studies have provided evidence that sleep disruption can precipitate mood and psychotic episodes in individuals who already have psychiatric disorders (Malkoff-Schwartz et al., 1998; Melo et al., 2017). The sleep architecture in those with MDD has also been

found be altered with MDD patients having a shorter latency to REM sleep (Kupfer, 1976), increased REM sleep, decreased slow-wave sleep and decreases in total sleep time and sleep efficiency (Benca et al., 1992; Kupfer & Foster, 1972; Kupfer et al., 1984; Menlewicz & Kerkhofs, 1991; Shaffery et al., 2003; Tsuno et al., 2005), with the severity of depressive symptoms positively correlated with the level of circadian misalignment induced by phase delays (Emens et al., 2009).

Association between SCRD and poor health outcomes may be mediated either through disruption of the circadian clock or its downstream components of the circadian timing system (Jagannath et al., 2013; Lunn et al., 2015). Bilateral lesions to the SCN resulted in a depressive-like phenotype as measured by the Forced Swim Test (Arushanyan & Popov, 1995; Tataroglu et al., 2004). Landgraf et al. (2016) found that genetic disruption of circadian rhythms in the SCN via SCN-specific BMAL1 knockdown increased depressive-like behaviour in mice. Mutations to other clock genes such as BMAL1 and CLOCK which are transcription factors which directly control the expression of numerous genes have been found to induce increased risk of obesity (Turek et al., 2005), substance abuse (Haslet et al., 2011), bipolar disorder (Jagannath et al., 2013) and depression (McClung, 2013). Circadian patterns of gene expression were much weaker due to shifted peak timing and disrupted phase relationships between individual circadian genes in brains of those with depression compared to controls (Li et al., 2013). Further studies have demonstrated that the molecular components of the clock can act as a pharmacological target for improving health outcomes (Hastings et al., 2003). For instance, several studies have identified that REV-ERB agonists can result in reducing anxiety, alleviating the adverse effects of diet induced obesity, and reduce neuroinflammation (For review see Patke et al., 2020). Other pharmacotherapies such as SSRIs and lithium have been observed to induce effects on the SCN (see Vadnie & McClung, 2017 for review). This indicates that the circadian molecular framework exerts an influence on physical and psychological wellbeing and disruption to this circadian system can lead to adverse health consequences.

Given the association between SCRD and adverse health outcomes research has attempted to investigate whether environmental risk factors such as LAN exposure contribute to SCRD. As stated in earlier sections exposure to LAN can adversely impact the timing, quality, and duration of sleep and lead to circadian disruption. Evidence suggesting an association between circadian disruption

mediated through chronic exposure to LAN and adverse health outcomes were originally observed in shift workers who were of increased risk of developing cancer (Blask et al., 2011; Granwisch, 2009; Haus & Smokensky, 2013), obesity, heart disease, sleep disorders, diabetes (Drake et al., 2004; Granwisch, 2009; James et al., 2017; Lunn et al., 2017; Ramin et al., 2015) and psychiatric disorders such as depression (Scott et al., 1997; Roth, 2012; Wright et al., 2012). The severity of circadian disruption is associated with the chronicity of shift work with severe disruption leading to heightened risk of developing depression (Hall et al., 2018).

Osibona et al. (2021) carried out a systematic review investigating the associations between light in the home and health. The review investigated the impact of home setting light at night has on physical health, mental health, and sleep health. Most studies in this area have been carried out in Housing environments and Health Investigation among Japanese Older People from the HEIJO-KYO cohort. With specific reference to physical health, higher levels of LAN in the home environment were associated with carotid atherosclerosis (Obayashi et al., 2015), higher risk dyslipidaemia, body mass index and abdominal obesity (Obayashi et al., 2013), higher blood pressure (Obayashi et al., 2014) and diabetes (Obayashi et al., 2014). Some of these cohort studies have specifically investigated mental health where associations were found between exposures to light at levels over 5 lux and incidence of depression (Obayashi et al., 2013). However, it is important to note that the severity of depressive symptoms was not associated with melatonin concentrations. In a longitudinal design higher levels of light exposure were associated with depression at follow-up (Obayashi et al., 2018). In these studies, higher levels of light exposure in the bedroom environment were associated with poorer sleep quality, greater prevalence of insomnia and delayed latency to sleep (Obayashi et al., 2014; Obayashi et al., 2014).

The widespread use of light-emitting devices (Chang et al., 2016; Chinoy et al., 2018) and increased exposure to light in our sleeping environments (Cain et al., 2020) may potentially lead to circadian disruption through circadian misalignment. It has been reported that 87% of non-shift workers display some form of circadian misalignment (Roenneberg & Merrow, 2016). This suggests that the health hazards from LAN exposure may not only be specific to shift workers but instead to a larger proportion of the population. The association with these health hazards and LAN exposure is plausible given that incidence of depression, cancer and obesity have

grown alongside the increased prevalence of electric lighting. Although, this does not suggest causation, research findings from the Amish population who do not have major access to electricity it is found that incidences of depression are lower (Egeland & Hostetter, 1983) and incidences of depression and cancer have paralleled the expansion of LAN (Figure 1.21; Stevens et al., 2014).

Figure 1.21

Psychophysical and physiological responses which are influenced as a result of light exposure.

Time Course	Psychophysical	Physiological
Immediate (seconds or minutes)	• Brightness perception • Visual amenity • Visual discomfort • Attention response	• Pupil size • Acute melatonin suppression • Luminance adaptation • Short-term chromatic adaptation
Delayed (hours, days, or weeks)	• Mood • Cognition • Motivation	• Circadian phase shift • Sleep quality • Long-term chromatic adaptation
Long-Term (months or years)	• Productivity • Depression	• Stress • Poor health • Seasonal affective disorder • Depression

Note. Some of the non-visual responses can occur immediately after exposure to light while others non-visual responses can occur over a delayed period of time. Over time exposure to light at night can have negative impacts on physical health and psychological well-being. Image taken from Houser & Esposito (2021).

1.18.1 LAN exposure exerting direct effects on eliciting mood

It is unclear how the association between LAN and affective disorders is mediated. Some argue that the association occurs through direct pathways where light exposure directly leads to impacts on mood and cognitive deficits independently of circadian arrhythmicity or sleep deprivation (LeGates et al., 2014). While other researchers argue that the association occurs through indirect pathways whereby light exposure leads to alterations in circadian rhythms which in turn influence sleep leading to alterations in mood (see Figure 1.22).

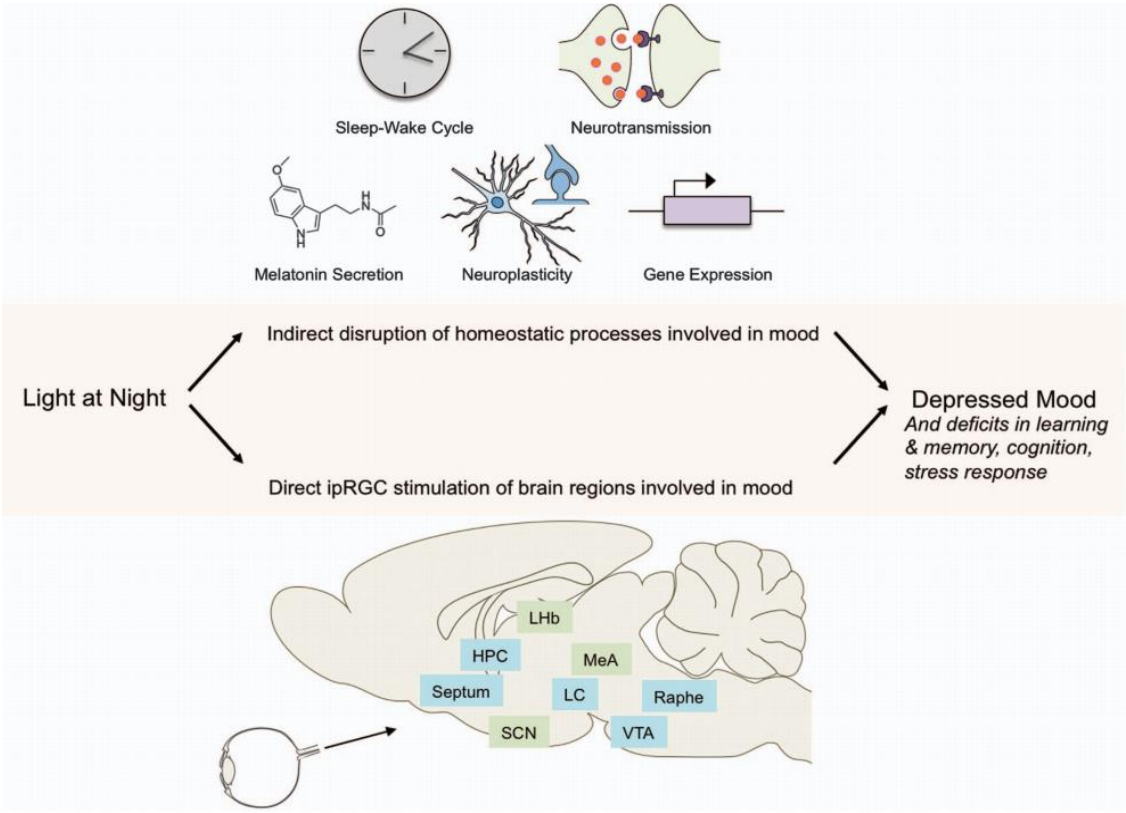
Evidence for the direct pathways has come from studies where light exposure can directly impact mood though ipRGCs projections to brain regions which are involved in emotionality (LeGates et al, 2014; LeGates et al., 2012). Fernandez et al. (2018) have showcased that the ipRGC cells also directly project to the habenula and has been associated with specifically mediating light induced alterations in mood. Further paucity has been given from imaging studies which showcase that the human

habenula is sensitive to modulations of ambient light (Kaiser et al., 2019). ipRGC signaling also passes from the amygdala to the ventral tegmental area and hippocampus which have also been shown to be areas involved in affective state (LeGates et al., 2014). These findings are suggestive that the association between LAN and mood disorders is mediated through light passing through the ipRGCs and projecting either directly or via the SCN to brain regions which are involved in mood regulation. LeGates et al. (2012) provide further evidence of direct effects of LAN on mood independent of sleep or circadian system. This study demonstrated that animals who were exposed to a cycle of light and darkness every 3.5 hours, which did not disrupt cycling clock gene expression or sleep, expressed a depressive-like phenotype (LeGates et al., 2012). Additionally, a separate study also observed that animals who had a deletion to the melanopsin gene did not express a depressive-like phenotype (Hattar et al., 2006). Predicative validity of direct pathways have been provided by light treatments which have been found to modulate emotional responses (Vandewalle et al., 2010). From the above studies presented it is clear there is complexity with light having a direct, clock independent role in influencing affective state. However, light may impact affective state via an SCN-dependent mechanism where light information transmitted by or changed the coupling of the SCN to downstream brain regions (Le Gates et al., 2014).

Predictive validity to the LAN inducing depressive-like phenotypes hypothesis was provided when administration of the antidepressant agomelatine was given to animals exposed to 3- and 6- weeks of constant light and resulted in prevention in the increase of the depressive-like phenotype and restored the melatonin rhythms (Tchekalarova et al., 2018; Tchekalarova et al., 2019). Both the removal of animals from dim light and the separate administration of antidepressant medication resulted in depressive-like phenotypes being reversed. Some studies have reported that independent of changes to rest-activity cycles that constant light resulted in depressive-like phenotype and decreases in spatial memory performance (Fonken et al., 2009). When an opaque tube was provided to shield from the constant light a reduction in depressive-like behaviour was observed. However, it is unclear whether the effects of constant light on depression are independent of the circadian clock as other studies have reported similar findings of no differences in rest-activity rhythms, but LAN resulted in molecular changes to circadian clock genes in the liver and SCN (Fonken et al., 2011).

Figure 1.22

Possible pathways to which light at night exposure may mediate low mood.



Note. Light exposure may impact mood by first disrupting sleep, circadian rhythms, hormone secretion neurotransmission or gene expression (indirect pathway) or it can directly affect mood without disruption to sleep or circadian rhythms but by aberrant signals transmitted from the ipRGCs in the eye directly to brain regions which are involved in mood regulation (direct pathway). Image taken from Bedrosian & Nelson (2017).

1.18.2 LAN exerting effects on mood mediated via indirect pathways

It is also argued that light impacts mood and cognitive function in an indirect manner. This is achieved by the light environment leading to alterations in circadian rhythms which in turn influence sleep and contribute to alterations in mood and cognitive function (LeGates et al., 2014). A number of indirect pathways which are under circadian control are impacted by LAN and may potentially lead to the onset and maintenance of mood disorders. LAN exposure can lead to a variety of alterations which include behavioural and brain changes have been found to be associated with mood disorders which include disruption to sleep, brain plasticity, neurotransmission, hormone secretion and gene expression (Bedrosian & Nelson, 2017).

Disturbances to sleep have been associated with being a contributing factor in the onset and maintenance of mood disturbances (Harvey, 2011) with a sizeable proportion of those with depression reporting poor quality sleep weeks before the onset of depressive symptoms (Perlis et al., 1997). Associations between alterations to a lighting schedule and disturbances to mood/depression have been found to be mediated by changes in the photoperiod (Rosenthal et al., 1984), transmeridian travel (Young, 2005) and shift work (Foster & Wulff, 2005). A number of studies have demonstrated that home setting LAN exposure is associated with disturbances to sleep by altering the timing of sleep either by delaying sleep onset, resulting in shorter duration of sleep, and leading to poorer quality sleep as indexed by more shallow sleeping, and more awakenings (Cain et al., 2020; Chang et al., 2015; Cho et al., 2013; Koo et al., 2016; Ohayon et al., 2016). However, it is important to note that causation cannot be inferred from the association between LAN disturbing sleep and leading to mood disturbances. Animal studies have reported that rats exposed to constant light developed a depressive-like phenotype along with loss of diurnal rhythms in activity and melatonin (Tapio-Osorio et al., 2013). Findings from nocturnal animal studies have observed that LAN exposure can lead to depressive-like phenotypes which occur independent of sleep disruption (Borniger et al., 2013; Fonken & Nelson, 2013). Additionally, the use of nocturnal animals allows for LAN exposure to occur during their active and awake phase and as a result LAN does not directly lead to alterations in sleep. This conceptual underpinning is important given that associations between circadian rhythm disruption and mood disturbances in humans is often attributed to disturbances to sleep. These findings of affective state and associations with LAN are found in both diurnal and nocturnal animals (Fonken et al., 2012). These findings indicate that sleep disturbances may be a contributor and not a principal mechanism involved in mood disturbances.

As previously outlined LAN exposure results in the suppression of melatonin which impacts on sleep propensity and the regulation of circadian sleep phase (Gandhi et al., 2015). This can lead to disruption to the timing the sleep and quality of sleep and as previously outlined these disturbances have been associated with the maintenance of depression (Tsuno et al., 2005). Although the suppression of melatonin by exposure to LAN has been attributed to depressive symptoms (Satyanarayanan et al., 2018) the findings are not conclusive. Studies have reported that animals who do not produce melatonin (i.e. C57bl/6) or have abnormal patterns

of melatonin production (i.e. Siberian Hamsters) display depressive-like phenotypes in response to light exposure similar to that of animals that have robust melatonin (Bedrosian et al., 2013; Cleary-Gaffney & Coogan, 2018; Walker et al., 2019). These findings albeit based on animal models would indicate that melatonin suppression due to LAN exposure is not the only mechanism which mediates the onset and maintenance affective states. However, in humans, melatonin suppression may provide a significant influence on inducing an affective state and may be a potential point of intervention (Walker et al., 2020).

Other studies have reported that LAN exposure directly alters the expression of clock genes and interacts with existing circadian gene variants which may pose as a risk factor to mood disorders. Findings from animal studies have reported that dLAN exposure can lead to blunted expression of clock gene expression in the brain (Bedrosian et al., 2013; Fonken et al., 2013; Shuboni & Yan, 2010; Walker et al., 2019). Circadian genes are associated with mood disorders with evidence coming from mutations to a number of clock genes being associated with symptoms of mood disorders (McClung et al., 2013). Genome-wide association studies have identified polymorphisms in circadian diseases which are associated with depression (Etain et al., 2011). This suggests that abnormalities in clock gene function may be a cause rather than an effect of mood disorder pathology (Bedrosian & Nelson, 2017).

Exposure to LAN can lead to alterations in the availability of neurotransmitters with some of these neurotransmitters being heavily involved in sleep and circadian timing (Wulff et al., 2010). In addition to LAN possibly disrupting the circadian system through alteration of neurotransmitter it may have indirect effects on psychiatric illness. For example, serotonin is one neurotransmitter impacted by LAN which is involved in mood regulation (Bedrosian & Nelson, 2017; Blume, et al., 2019). Predictive validity to serotonin being associated with depression is provided with most antidepressant medication having therapeutic efficacy primarily targeting abnormalities in serotonin neurotransmitter signaling (Vadnie & McClung, 2017). Additionally, serotonin neurotransmitters and their receptors display a circadian rhythm in their concentration, release, and expression (Wirz-Justice, 1987) with mutations to clock genes leading to reductions in serotonin activity (Hampp et al., 2008). These findings potentially suggest that environmental stimuli such as light which disturbs clock genes may in turn disrupt neurotransmitter transmission. This suggests that LAN may be one indirect mechanism in the

aetiology of mood disturbance via disturbance to neurotransmitter signaling (Wulff et al., 2010).

A common feature of major depression is impaired neuroplasticity with individuals displaying reduced hippocampal volume (Gueze et al., 2005; Sheline et al., 2003; Videbech & Ravnkilde, 2004), lower levels of brain-derived neurotrophic factor (BDNF; Murakami et al., 2005; Pandey et al., 2008; Sen et al., 2008), increased levels of receptors for pro-inflammatory cytokines, such as interleukin (IL) 1B and tumor necrosis factor (TNF; Bedrosian et al.,, 2012) and deficits to functional plasticity (Player et al., 2013). In a number of animal models of depression alterations in levels of neurogenesis or neuronal morphology have been observed (Pittenger & Duman, 2008). Post-mortem studies of individuals with depression report lower levels of BDNF in brain tissue (Aydemir et al., 2006)

Predictive validity has been observed in these models with administration of antidepressant treatment increasing CA1 spine density in rats (Hajszan et al., 2005; Norrholm & Ouimet, 2001), increasing levels of BDNF (Altar, 1999; Duman & Monteggia, 2006), increasing levels of neurogenesis in the hippocampus and simultaneously blocking the effects of stress (Boldrini et al., 2013; Malberg et al., 2000; Santarelli, et al., 2003; Sapolsky, 2004). In humans, administration of antidepressant medication and electroconvulsive therapy results in increasing BDNF levels in blood (Sharma et al., 2016).

Studies have highlighted that disruption to circadian rhythms can induce structural changes in the brain which may potentially impact on the functional connectivity of regions involved in mood regulation (Bedrosian & Nelson. 2017). Evidence to support this claim comes from several animal studies demonstrating that dLAN results in the increase of hippocampal cytokine expression and a reduction in BDNF expression (Bedrosian et al., 2013; Fonken et al., 2013; Walker et al., 2019) with these molecular changes occurring simultaneously with an increase in depressive-like behaviours. Other studies have reported that dLAN exposure was sufficient to reduce the density of dendritic spines on hippocampal CA1 and dentate gyrus granule neurons with an association between a reduction the density of CA1 dendritic spines and depressive–like phenotypes being observed (Bedrosian et al., 2011; Bedrosian et al., 2013; Fonken et al., 2012). Bedrosian et al. (2013) report that these alterations to neuronal plasticity may be mediated by the ipRGCs as exposure to white and blue wavelength light led to reduction in CA1 total spine density

compared with exposure either red light or darkness. Alongside this the same study reported exposure to white and blue light elicited depressive-like behaviour but this was not observed with exposure to red light or darkness. Predictive validity for an association between expression of a depressive-like phenotype impaired neuroplasticity being mediated through indirect effects of LAN was provided by Bedrosian et al. (2013) who demonstrated that removal of animals from a light/dLAN cycle to a L/D cycle resulted in increases of hippocampal BDNF expression, increases in dendritic spine density, reduction of TNF expression and reduction in depressive-like behaviours. Additionally, in the same study administration of inhibitors to cytokines resulted in a reduction of depressive-like symptom in animals exposed to LAN. In addition, Bedrosian et al., (2012) found that the administration of antidepressant medication resulted in CA1 dendritic spine density being moderately restored in animals housed under dLAN along with depressive-like symptoms being improved. This supports evidence that LAN exposure may results in depressive-like phenotypes because of impaired neuroplasticity. In some reports dLAN is also associated with an increase in neuroinflammation as indexed by an increase cytokine expression which may contribute to depressive-like behaviour (Bedrosian et al., 2013; Walker et al., 2019). Inhibition of hippocampal cytokine expression intracerebroventricular administration of a dominant negative TNF reverses depressive-like behaviour (Bedrosain et al., 2012). Studies using constant light, dLAN or jetlag paradigms have been associated with reduced production of neural progenitor cells (NPCs; Cleary-Gaffney & Coogan, 2018; Fujioka et al., 2011; Gibson et al., 2010). Reduced levels of NPCs results in a lower level of neurogenesis which as outlined earlier is associated in the pathophysiology of anxiety and depression (Benninghoff et al., 2002; Kempermann & Kronenberg, 2003; Snyder et al., 2011).

1.19 Thesis overview

The literature presented above indicates the significant effect that light exposure exerts on the circadian clock. However, many of these studies have been based upon the effects of bright light exposure inducing NIF responses either through acute or circadian responses (Chang et al., 2012; Gooley et al., 2011; Zeitzer et al., 2000). However, the intensity of this light is may not be reflective of the LAN

experienced in home settings or the low-level LAN experienced in the sleeping environment during sleep. Additionally, the intensity of home-setting LAN varies between home settings and is not fixed which occurs in experimental studies. The motivation for this thesis was set against the backdrop of several studies which showcased that those who have access to light in their homes display delayed sleep onset, delayed timing in DLMO, adverse alterations to sleep structure, duration, and quality (Beale et al., 2017; de la Iglesia et al., 2015; Moreno et al., 2015; Pilz et al., 2018; Peixoto et al., 2009; Stothard et al., 2017; Wright et al., 2013). The work was also influenced by a small number of experimental human and animal studies which showcased that low level LAN during the sleep is associated with alterations to sleep timing, sleep quality and sleep architecture (Burgess & Miolina, 2014; Cho et al., 2015; Cho et al., 2018; Stebelova et al., 2020). While these studies have been informative due to their experimental set-up in allowing to understand the mechanistic underpinnings of low level of LAN exposure on sleep and circadian rhythmicity these studies lack ecological validity. It is unclear under naturalistic conditions the extent to which LAN exposure in the home environment impacts on sleep circadian rhythmicity and psychological health. As outlined previously, a small number of studies have carried out observational studies to investigate the effects/associations of low-level LAN on sleep and health (Obayashi et al., 2014, Obayashi et al., 2013; Obayashi et al., 2018). However, these studies have been based upon an older population to which the findings cannot be generalized due to differences in photic sensitivity poorer compared to younger adults and adolescents.

The main aim of this research was to examine the perception of LAN in the sleeping environment and its association with sleep, circadian rhythmicity, attention bias and psychological health. **Chapter 2** examines individual's attitudes towards LAN being a disruptor to sleep. This study then specifically examines what are the sources of LAN exposure in the sleeping environment and how individuals perceive these sources as being disruptive to sleep. The study also assesses strategies that individual's employ to minimise the LAN exposure. **Chapter 3** examines how subjective perceptions of LAN correlate with estimates of outdoor illuminance due to public lighting at the level of individual residences, and how subjective perceptions of ALAN as well as objectively-measured household illuminance levels associate with measures of psychological distress, cognitive failures, sleep duration, quality, social jetlag and chronotype. **Chapter 4** examines whether the perception of

LAN sources in the sleeping environment is associated with sleep related attention bias using the Emotional Stroop Test. **Chapter 5** builds upon the work from chapter 4 by using the Dot Probe Task to assess whether the perception LAN in the sleeping environment is associated with increased attention bias towards images depicting LAN sleeping environments. **Chapter 6** uses an observational design to examine whether increased in bed LAN intensity measured at the window and bedside is associated with sleep and circadian rhythm disturbances as measured by actigraphy derived measures of sleep quality and circadian rhythm function. This chapter also assesses whether increased LAN intensity is associated with increased day to day variance in sleep timing, efficacy, mood, and subjective sleepiness. **Chapter 7** provides a general discussion of the overall results and conclusions of the research carried out in this thesis. Strengths, limitations, and areas for future research are discussed.

Chapter 2

Examination of the sources of LAN in the sleeping environment and individual's attitudes towards these sources of LAN being disruptive to sleep.

Abstract

Exposure to LAN in the sleeping environment may come from a variety of sources. However, studies to date have only examined the presence of either one particular LAN source or perceived room level brightness. Using a novel survey this study assesses LAN in the sleeping environment. This study for the first times provides a comprehensive understanding of the perceived sources of LAN in the sleeping environment and their perceived impact on sleep. 552 participants aged between 18-74 (*M*=37; *SD*=13) completed the survey. Using an exploratory design our study reports that overall the majority respondents perceive LAN to be disruptive to sleep and that exposure to LAN comes from a variety of sources. However, the perceived impacts of LAN adversely impacting sleep are more frequently endorsed by those that perceive LAN. Our results indicate solutions individuals can take to minimise LAN in their sleeping environment. Our results also indicate that citizens need to be informed about the impacts of LAN in the sleeping environment and strategies and behaviours they can employ to minimize their exposure.

2.1 Introduction

Before the widespread use of artificial light, individuals were exposed to solar light/dark cycles which cyclically progressed from intense (>300lux) to dim (<30lux) environmental light throughout the day period (Philips et al., 2019). The technological innovation of artificial light has led to individuals extending their exposure to light into the biological night, resulting in LAN exposure now being ubiquitous in the home environment. Gatson et al. (2013) propose that home setting LAN exposure ranges between 100-200lux. More specifically, LAN exposure in the sleeping environment has become more commonplace (Philips et al., 2019; Santhi et al., 2012; Xu et al., 2022) with an observational study reporting with older adults that pre-sleep bedroom light intensity was 27.3lux (Obayashi et al., 2014). This is the intensity of light which which has been reported to elicit suppression of melatonin (Philips et al., 2018). Objective evidence to support this comes from Cain et al. (2020) who found that, in the home-setting, individuals are routinely exposed to levels of LAN which in 73% of homes may result in at least a 20% reduction in melatonin suppression. Additionally, they reported that the spectral composition and irradiance of LAN exposure to which individuals are exposed to persists at continuous levels throughout the biological night before sleep (Cain et al., 2020).

Several experimental research studies have demonstrated the impact of LAN exposure on sleep architecture (Cho et al., 2013; 2016), sleep timing (Santhi et al., 2012; Wright et al., 2013; Stohlhard et al., 2017, alertness (Cajochen et al., 2011; Cajochen et al., 2005Chapella et al., 2018) and on circadian phase markers (Cajochen et al., 2006; Gooley et al., 2011; Zeitzer et al., 2000). Although these studies have been essential to understand the mechanistic underpinnings of how light exposure impacts on the circadian system, these studies may not provide an accurate representation of LAN exposure in home-settings. For instance, many experimental light exposure studies occur in highly controlled environments where the type of LAN stimulus and its intensity may not be representative of the type of light individuals are typically exposed to in their sleeping environments (Cain et al., 2020; Santhi et al., 2012). Secondly, light exposure is typically presented well after habitual sleep, at varying pulses or even after exposure to dim light (Gooley et al., 2010). Each of these factors may lead to findings which are not fully aligned with individuals lighting habits in their home environments. Additionally, these studies

failed to assess the sources or indeed the perception of these sources of LAN on sleep quality.

Two pieces of research have examined perception of bedroom brightness during the sleep period with a study comprising of 19,136 individuals finding that 7.4% report that their bedroom was bright (Ohayon & Millesi, 2016) while another comprising of 105,000 females found that 49% perceived to have medium levels of LAN exposure in the sleeping environment with only 21% reporting that the level of LAN exposure in their sleeping environment was low (Johns et al., 2017). Observational field studies which have objectively measured LAN have reported that in bed LAN levels are between .08-2lux (Obayashi et al., 2014; Esaki et al., 2020). However, these studies are limited by self-report measures of bedroom brightness. For instance, Johns et al (2017) examine the Likert response options to the question examining levels of room brightness, noting they were worded in a manner that was difficult to ascertain sleeping environment brightness induced by LAN. In their question, the response options were “light enough to read”; “light enough to see across the room, but not read”; “light enough to see your hand in front of you but not see across the room”; and “too dark to see your hand or you wear a mask”. The middle two responses were categorised as high LAN exposure despite this level of LAN not being meaningfully bright. This is supported by Kyba and Spitschan (2018) who argue that exposure to light as low as .1lux still allows individuals to see their hand and that at starlight levels, it is possible to see the walls and objects in a room (Hanel et al., (2017). Although, this previous research has provided an understanding of perception of room level brightness, it did not fully examine the specific sources of LAN which contribute to this perceived level of brightness. Sources of LAN in the sleep environment may arise from 4 main sources which include: (i) main lighting inside the bedroom such as bedside lighting and main bedroom lighting; (ii) light from usage of emitting devices such as tablets, smartphones and televisions; (iii) trespassing light either from inside the house into the sleeping environment or; (iv) light externally from the environment. Each of these sources in isolation and in combination may have deleterious effects on sleep quality.

2.1.1 Main Bedroom Lighting

Sleeping with a bedside light on has been found to induce shallow sleep, frequent arousals along with sustained effects on brain oscillations which are associated in sleep depth and stability (Cho et al., 2013). In large cohort studies, it has been reported that between 3.9-7% of individuals report sleeping with a light on (Hurley et al. 2014; Ohayon & Milesi, 2016). However, the wording of the question in Hurley's and colleagues (2014) research may have led to findings which underestimate the frequency of sleeping with a light source on as the question asked only addressed sleeping with a bright light on. This may have led to participants only considering the main light source in the bedroom environment and not bedside light sources. Additionally, these studies have failed to examine other sources of LAN exposure in the bedroom such as light from inside the house (i.e. light from the landing) trespassing into the sleeping environment.

2.1.2 External LAN trespassing into the sleeping environment

Exposure to LAN in urban cities has increased rapidly with rates between 5%-20% per year (Zhang et al., 2021). From 1992-2017 global satellite observable light emissions increased by at least 49% (de Miguel et al., 2021). Over 80% of the global population is now affected by nighttime light pollution (Falchi et al., 2016; Xu et al., 2021) with urban locations associated with subjectively brighter skies compared to locations with lower population densities (Coogan et al., 2020). Light pollution is so severe that over one-third of the world population are no longer able to see the Milky Way as result of artificial night sky glow (Falchi et al., 2019). The prevalence of light pollution differs across continents with 60% of Europeans and 80% of North Americans not able to see the Milky Way (Falchi et al., 2016; Kyba et al., 2017). This indicates that light pollution external to the home is now commonplace. The irradiance of a common street-light is 5 lux and a parking lot light in a shopping mall is approximately 20 lux (Ohayon et al., 2016). The accumulation of different light sources external (e.g., street lighting, commercial lighting and external domestic lighting) to the home environment has led to increased irradiances of LAN exposure and light pollution in our environment. Sources of light pollution are also present in rural areas with individuals reporting that personal external domestic lighting and neighbors lighting are important sources

of LAN in rural settings (Coogan et al., 2020). It is plausible that this external LAN may trespass into the sleeping environment (Kang et al., 2016).

Much recent research has demonstrated associations between external light pollution, poor sleep quality (Ohayon & Milesi, 2016), shortened sleep (Xiao et al., 2020; Zhong et al., 2021), later sleep onset and later wake-up time. In many of the studies, these associations persist when controlling for age, sex, population density and noise pollution (Ohayon & Milesi, 2016; Zhong et al., 2012). In addition, associations between outdoor nighttime light and multiple adverse health outcomes have been reported in the domains of obesity (Koo et al., 2016; Zhang et al., 2020), cardiovascular diseases (Sun et al., 2021), cancer (Xiao et al., 2021), mental disorders (Paksarian et al., 2020) and suicidality (Min & Min, 2017)

Although, these studies have reported associations linking external LAN and adverse health outcomes, external LAN cannot be used as a proxy for LAN exposure in the sleeping environment. These studies do not take into consideration individual level factors such as window covering practices with curtains/blinds, the presence of outdoor vegetations to shield external LAN and the location of the bedroom in relation to streetlights. This suggests that satellite data studies may not act as a reliable proxy to represent an individual's exposure to light within their sleeping environment. Evidence from Garcia-Saenz and colleagues (2018) supports this assertion, as they contend that there is no association between the subjective perception of indoor LAN and outdoor satellite measures of LAN. Two separate studies have reported no association between photometer measured LAN inside the sleeping environment and satellite image data (Huss et al., 2019; Rea et al., 2011). However, these findings are in contrast with Ohayon and colleagues (2016) who reported an association between individuals that perceived their bedroom environment to be bright and living in areas with higher levels of outdoor night light. This may suggest that part of the perception of room level brightness comes from external LAN sources. However, this study did not further examine these specific effects on sleep timing but instead controlled for perceived room level brightness when demonstrating that outdoor night light is a predictor of delayed bedtime, wakeup time and decreased sleep duration (Ohayon & Milesi, 2016). Weaker evidence to support the claim that external LAN trespassing into the sleeping environment may be a disrupter to sleep comes from Park and colleagues (2019) who examined various sources of LAN in the sleeping environment (e.g., external

LAN, internal dwelling trespassing LAN, sources of LAN in the sleeping environment). The presence of LAN from these sources was then aggregated to categorise individuals into either high LAN exposure or low LAN exposure. Although, the study found that high LAN exposure was associated with delayed sleep timing and increased disturbances, it is unclear whether the perception of external LAN specifically contributed to that. This suggests that direct assessment of perception of external LAN in the sleeping environment and its association with sleep timing/quality is limited. To date, only one study has specifically examined whether light entering the sleeping the environment effects sleep. The results from this study found that those living in rural locations are more likely to endorse this statement (Coogan et al., 2020). However, there are some methodological shortcomings as the study did not separately assess the perception of external LAN in the sleeping environment and its perceived impact on sleep, but instead addressed using one question whether external LAN in the bedroom effects sleep. Assessing this statement with one question limits the reliability of the findings. In particular, no study to date has assessed the frequency to which external LAN is perceived in the sleeping environment and how the specific perception of external LAN is perceived to impact on sleep.

2.1.3 Use of personal light emitting devices in the bedroom

Light exposure also occurs through the ubiquitous use of personal light emitting devices such as computers, television screens, e-readers, smartphones and tablets. These devices have become more accessible, lightweight and portable making them easier to use, resulting in their use being indispensable and a huge part of our daily lives, especially at bedtime. These devices exhibit both the high levels of LAN irradiance and express the specific spectral composition to which the circadian system is most sensitive to (Cajochen et al., 2011; Chang et al., 2015; Chiny, Duffy & Czeisler, 2018). Numerous studies have indicated an emergent problematic and worrying excessive use of new technologies in people of all ages (Clayton et al., 2015; Jelenchick et al., 2016; Exelmans & Van den Bulck, 2016; Oiedo-Trespalacios et al., 2019). An American poll study carried out by the National Sleep Foundation reported that nine out of 10 Americans report using technological devices in the hour before bed (Gradisar et al., 2013). This finding is supported by Jniene et al. (2019)

who observed that in a student population 97.3% use a light emitting devices at bedtime.

In the aforementioned Gradisar and colleagues' (2012) American poll, it was observed that were age group differences in the use of light emitting devices before sleep. Those under 30 were more likely to use mobile phones compared to those over 30. Conversely, 72% of adolescents (13-18) used cell phones before sleep while only 16% of older adults (46-64) used cell phones before sleep. In recent years however, the prevalence and frequency in the use of light emitting technologies has changed, resulting in the above findings being possibly no longer valid. Additionally, the same trends may not be observable in older adults now, given the increased ability and confidence in adult IT literacy which may lead to older adults more frequently using light emitting devices.

Although, the number of light-emitting devices has increased and numerous studies have showed both the prevalence of their use before sleep and the negative impacts they have on sleep, only one study to date has assessed individual's perception of sleep disturbance due to the night use of personal light emitting devices and its impacts on the quality of sleep (Jniene et al., 2019). 65.7% perceived that using personal light emitting devices before bedtime lead to sleep disturbances in quality and or quantity. However, the generalisations of these results may be over representative as the sample comprised of young medical students who may have knowledge about the disruptive impacts of LAN exposure on sleep. It is unclear whether if this question was examined in the broader general population would the association between the use of light emitting devices before sleep and poor sleep quality be found.

2.1.4 Rationale

It is plausible to propose that exposure to LAN in the sleeping environment is not due to exposure to one source but through a combination of sources. LAN exposure in the bedroom environment may concurrently occur due to light pollution trespassing into the sleep environment, traditional lighting in the bedroom being left on throughout the night and/or use of personal light emitting devices before sleep. Although, separate studies have singly assessed the perception of room level brightness and the perception of different environmental sources to LAN in the

sleeping environment, this study will be the first to concurrently assess the prevalence of LAN from multiple sources and lighting habits in the sleeping environment. This understanding of the prevalence of LAN sources in the sleeping environment is important in developing approaches to reduce LAN in the sleeping environment. The study aims to obtain findings which are more reliable in doing so. Findings from previous studies which have assessed room level brightness and the prevalence in perception of LAN exposure from various sources in the bedroom may not be fully generalisable. This may be due to previous research only examining specific ages (Jniene et al., 2019), specific populations (Davis & Stevens, 2001; Hurley et al., 2014; Johns et al., 2017; O'Leary et al., 2006), specific demographics (Johns et al., 201), out of date findings due to changes in the prevalence and use of light emitting technologies (Gradisar et al., 2013) or due to the wording of the question (Coogan et al., 2020; Hurley et al., 2014; Johns et al., 2017). Alongside assessing the sources of LAN exposure in the sleep environment, this study will extend upon Jniene and colleagues (2019) and Coogan and colleagues (2020) methodology to examine individual's attitudes about LAN. For the first time, this research will assess the perception and awareness of how each of these specific LAN sources are perceived to be disruptive to sleep. Accordingly, it is important to explore whether individuals can assess the risk that these sources of LAN have on their sleep.

The aim of this study is to assess and describe:

1. What are the perceived sources of LAN in the sleeping environment and how common are these sources in a typical sleeping environment.
2. What are individual's attitudes towards the perceived sources of LAN as being disruptive to sleep and/or sleep quality.
3. What strategies can individual's employ to minimise the perception of LAN.

2.2 Method

2.2.1 Participants

552 participated in the study of which 66.3% (n = 366) were female. The age range of participants was 18-74 (M = 37, SD = 13.52). The study was reviewed and approved by the Maynooth University Ethics Review Board. Participants were recruited from voluntary organisations, trade shows, email distribution lists, media outlets and on social media platforms. The sampling method employed was a mixture of convenience sampling and snowball sampling. Those that resided outside of the Republic of Ireland, who were under the age of 18 or those who were shift-workers were excluded from the study. Participants did not receive any renumeration for their involvement.

2.2.2 Materials

The survey instrument comprised of 30 structured questions with coded responses (for full survey see Appendix A). The survey opened with a number of questions assessing key demographic information (i.e., age, sex, home locations, type of residence) followed by questions which assessed individual's general attitudes towards light at night exposure on sleep quality before sleep and during sleep. The survey then set out to assess the sources of LAN in the sleeping environment. The four sources of LAN exposure assessed in this study were: (i) sleeping with main bedroom or bedside light on (ii) perception of external LAN from the outside environment trespassing into the bedroom (iii) perception of internal LAN from inside the house trespassing into the sleeping environment and (iv) assessment of the frequency in using light emitting technologies in the bedroom. Finally, the study assessed how each of these light sources are perceived to be disruptive to sleep.

As no established questionnaire exists which concurrently assesses the multiple sources of LAN exposure in the sleeping environment and attitudes towards LAN exposure, the researcher conducted a review of the literature which had examined light at night exposure in the sleeping environment. The researcher found that studies to date had singly assessed room level brightness after lights were turned off (Johns et al., 2017; Ohayon et al., 2016;), sleeping with a bedroom light on

(Hurley et al., 2014; Ohayon et al., 2016) and frequency of use of technological devices before bed (Gradisar et al., 2013). The researcher acknowledged the limitations of previous work where the wording of the question impacted on the reliability of the findings. This was found in Johns and colleagues (2017) study where room brightness was assessed by indicating their response to statements such as "light enough to read"; "light enough to see across the room, but not read"; "light enough to see your hand in front of you but not to see across the room" and "too dark to see your hand, or you wear a mask". It has been argued that the two middle responses cannot be meaningfully differentiated and secondly the perception of one's hand can be perceived at low light levels that would not be considered bright (Kyba & Spitschan et al., 2018). In consideration of this limitation, the researcher utilised Ohayon and colleagues' (2016) question which assessed room brightness as a dichotomous variable. However, the question asked about room level brightness once all bedroom lights were turned off. A separate question assessed room level brightness as an ordinal variable ranging from vary dark to very bright. Separately, the researcher considered that a previous study which assessed sleeping with a light on may have underestimated sleeping with a bedside light, as the question assessed whether an individual slept with a bright light (Hurley et al., 2014). The current survey examined whether individuals slept with either a main light or bedside light on. When the survey was designed, no research had specifically examined whether external environmental LAN or LAN from inside the dwelling trespassed into the sleeping environment. The researcher separately assessed the perception of each of these sources in the sleeping environment with the use of a dichotomous response.

When assessing the use of personal light emitting devices, the questions were guided by a poll conducted by the National Sleep Foundation (NSF) which examined sleep and technology use of Americans. In the NSF's study, individuals were separately asked about the presence and use of each light emitting device (e.g., TV, computer etc.) and the frequency of technology use. Unlike the NSF's study, which separately assessed the use and frequency of use of different light emitting technologies, the current study assessed use and frequency of use of light emitting technologies collectively. For example, in the current study the question was worded as "*Before sleep do you use electronic devices in bed (i.e, mobile phone, tablet, ebook, personal computer?."* The justification for collectively assessing light emitting technologies is due to most devices now emitting blue wavelength light to

which the circadian clock is most sensitive (Cajochen et al., 2011; Chang et al., 2015; Gringras et al., 2015) and to further reduce the length of the questionnaire. The NSF survey examined use of the devices in the hour before bed (but not limited in use bedroom). However, the current survey separately assessed whether individuals used light emitting devices an hour before bed and if they specifically used light emitting devices in bed before sleep. The inclusion of the latter question was to specifically understand light exposure habits in the bedroom.

When the perception of each of LAN from the various sources was assessed, a follow-up question separately asked how individuals perceive each of these sources impact on their sleep. Although, consideration was taken to ensure that neutrality was maintained in the wording of questions, so that there was no priming of respondents' responses, the researcher acknowledges complete neutrality is difficult to achieve as all expressions frame and guide interpretations to some direction. This has been also acknowledged in other surveys which have assessed perceptions of light pollution (Lyytimaki & Rinne, 2013).

In addition to the survey, participants completed a number of other questionnaires which included the Pittsburgh Sleep Quality Index, the Cognitive Failures Questionnaire and the Munich Chronotype Questionnaire. Discussion and analysis of these measures will be discussed in the following chapter.

2.2.3 Procedure

The survey was conducted both online and face-to-face between March 2017 and June 2018. In order to collect a representative sample, with a diverse age range and residing in various locations around Ireland, the researcher actively recruited participants by advertising the survey on social media (Twitter, Facebook and targeting specific Facebook groups such as community Tidy Town pages and community pages. In order to target more specific groups, the researcher contacted specific organizations such as the Irish Countrywomen Association and the Men's Shed. These organisations allowed for the dissemination of a brief overview of the study and the URL to participate in the study which was sent in an e-newsletter to their members. Individuals clicked on a URL link which sent them to the survey which was hosted on Google Forms. Participants read a brief information sheet and were then asked to confirm that they had read the information sheet and understood

what would be assessed. The survey took between 20-30 minutes to complete. Once participants completed the survey, they submitted their responses which were hosted on a password protected Google Forms account. The data was not fully anonymous at time of collection as participant's provided their Eircode which provides a geolocation of their home address. The researcher also targeted specific rural groups by attending trade fairs (Tullamore Agricultural Show). When attending these trade shows the participants completed a pen and paper version of the survey. This was identical to the surveys presented online. Individual's geolocation was used to cross-reference with objective measures of external environmental light. Discussion and analysis of objective measurements will be provided in the following chapter. Once the data had been cross-referenced all online responses were deleted from Google Forms.

2.2.4 Analysis

Associations between categorical responses were tested in Pearsons's chi square test. $P < .05$ was interpreted as indicating a statistically significant effect. All data were analysed using SPSS version 26 (IBM Corporations Armonk, NY, USA). The statistical approach employed was exploratory and not hypothesis testing.

2.3 Results

A total of 552 individuals participated in the survey. Most respondents were female (66.3%). The age range of the participants was between 18-74 years (M = 37, SD = 13.53). Age parameters were defined and categorised into 18–29-year-old (38.3%), 30–45-year-old (34.8%) and 46–75-year-old (27%). The wide range of participants for the older adults was due to small number of respondents being 65 years and over. However, the age categorisation was similar to Gradisar et al. (2013) NSF study assessing technology use in the bedroom. Within this sample, 20.80% resided in the city, an equal proportion of the sample resided in either suburbs (24.3%) or urbans towns (25.20%) with the remaining proportion living in semi-rural environments (11.8%) or rural environments (17.90%).

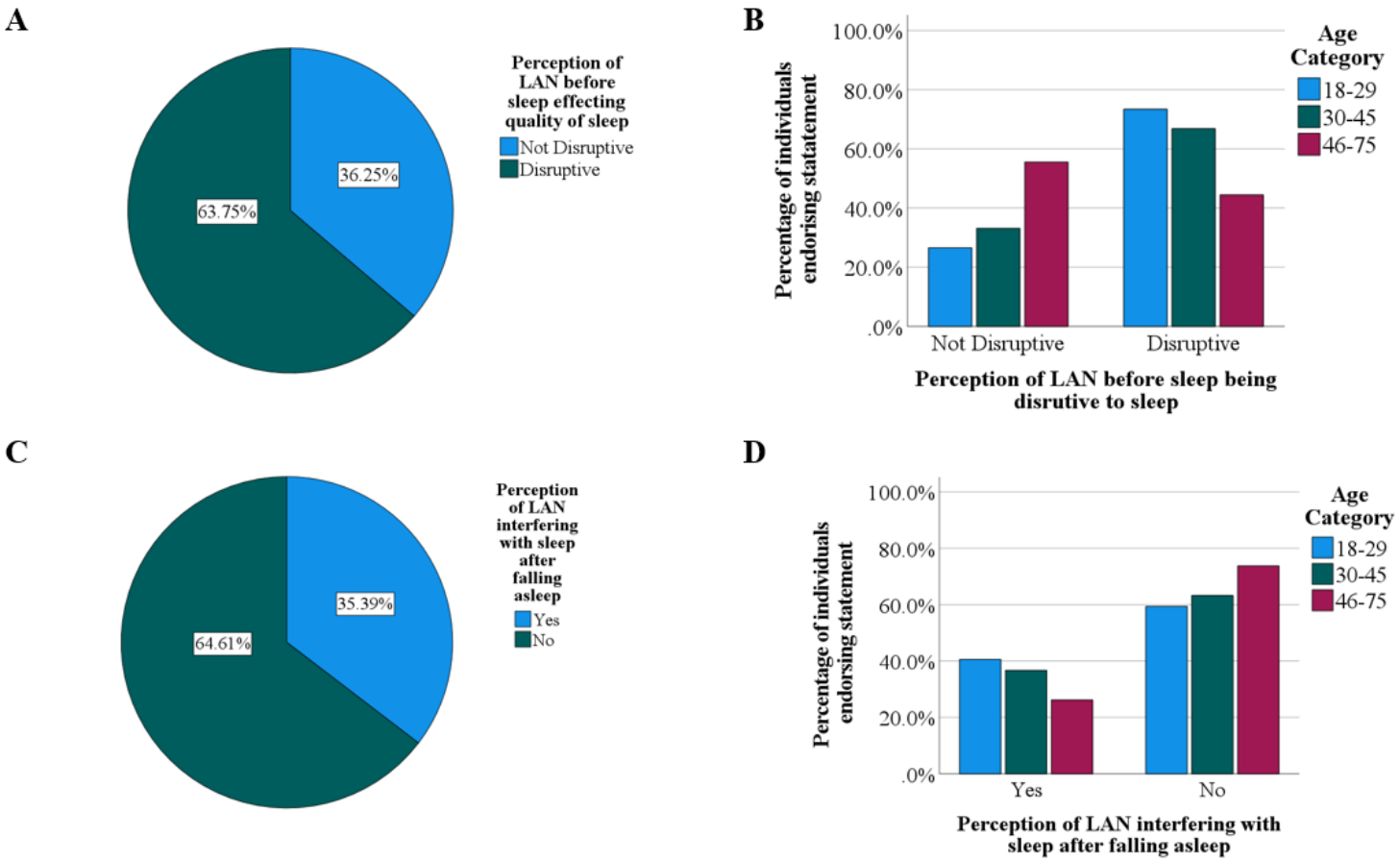
2.3.1 Attitudes towards LAN exposure

64% (n = 350) of the sample report that LAN exposure before sleep is disruptive to sleep (see Figure 2.1A). No significant sex differences were observed on the endorsement of LAN being disruptive to sleep, x^2 (1, n = 538) = .942, p = .332. However, there was a significant association between age type and the perception of LAN before sleep being disruptive to sleep quality, x^2 (2, n = 538) = 32.22, p < .001, phi = .245). As can be seen from Figure 2.1B, younger adults more frequently endorsed (73%) that LAN was disruptive to sleep quality while older adults were more likely to report that LAN was not disruptive to sleep quality (56%).

65% of respondents perceive that LAN does not interfere with the quality of their sleep after falling asleep (see Figure 2.1C). No sex differences were observed on the endorsement that LAN exposure was disruptive to sleep quality after falling asleep x^2 (2, n = 540) = .045, p = .832). A significant association was observed between age type and perception of LAN being disruptive to sleep quality after falling asleep, x^2 (2, n = 540) = 7.93, p = .019, phi = .121. As can be seen from Figure 2.1D younger adults more frequently endorse (44%) that LAN exposure is disruptive to their sleep quality after falling asleep. While for older adults are more likely endorse that LAN exposure is not disruptive to sleep after falling asleep (30.7%).

Figure 2.1

(A) Pie chart illustrating the percentage of the sample that report that LAN exposure before sleep is / is not disruptive to sleep. (B) Bar chart illustrating that percentage of individuals endorsing that LAN before sleep was disruptive to sleep by age categories. (C) Pie chart illustrating the percentage of respondents who perceive that LAN does/does not interfere with the quality of their sleep after falling asleep. (D) Bar chart illustrating the percentage frequency of the sample endorsing whether the LAN is perceived as disruptive to sleep after falling asleep across age categories.



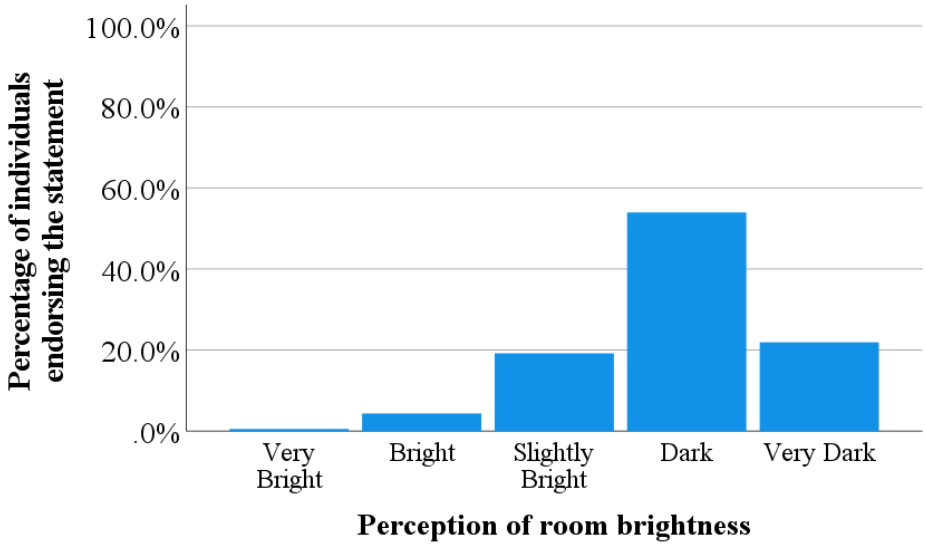
2.3.2 Sources of LAN exposure in the sleeping environment

When asked what the most prevalent light-emitting source in the sleeping environment is just under half of the respondents (47.5%) reported that their mobile phone was the most prevalent light source in the bedroom. This was followed by street lighting (20.5%), alarm clock (15.9%), computer/tablet (9.6%), television (5.3%) and car headlights (1.8%).

Most participants (84%) reported never sleeping with a main bedroom light or bedside light on. Only a small number of respondents reported to either always (1.8%) and very often (1.8%) sleep with bedroom lighting on. The majority (82%) of the sample reported that when both the bedside light and main bedroom light was turned off, that they perceived their room to be dark. However, when asked to rate the brightness of the room on a 5-point Likert scale, the frequency of those endorsing that their room was dark was reduced to 75%, while 25% endorsed that their sleeping environment ranged from either very bright to slightly bright (Figure 2.2).

Figure 2.2

Bar chart illustrating the percentage frequency of the sample indicating the level of room brightness based on a likert response.



Most participants (86.6%) report not turning the light on when they awake from sleep during the night. There was no significant association between age category and turning on a light source after awakening from sleep (x^2 (1, n = 541) = 3.80, p = .150). For those who do turn on the light during the night, the majority tend

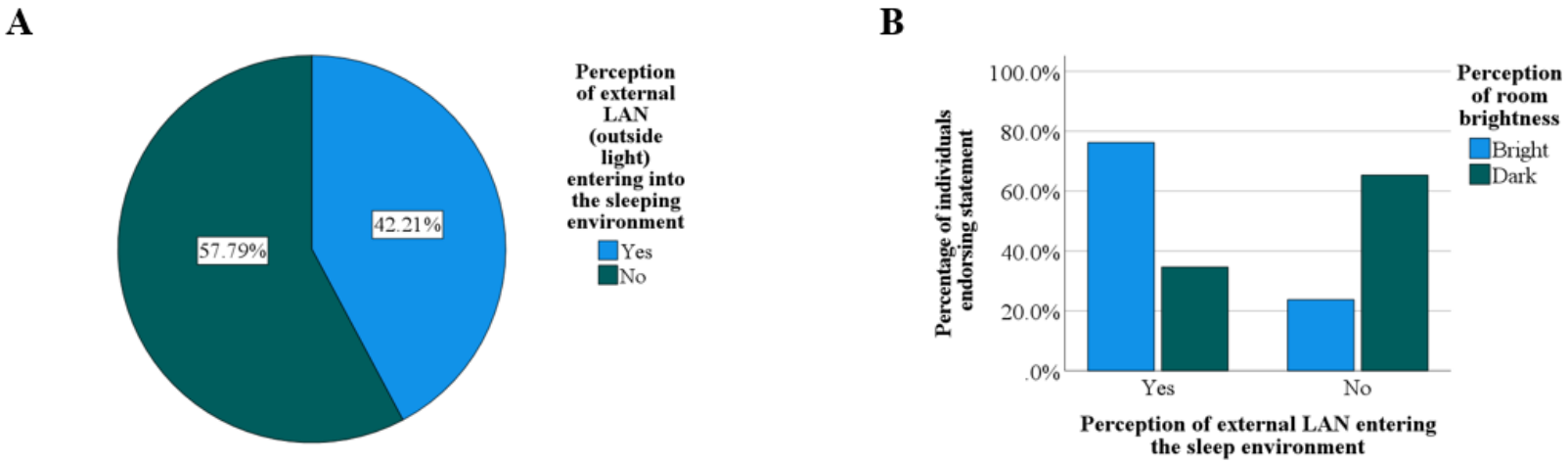
to do so for 0-5 minutes (71%), with smaller proportions turning the light on for 5-10 minutes (17%), 10-20 minutes (4%) and 20+ minutes (8%). No association was observed between turning the light on after awakening from sleep and perception of room brightness (x^2 (1, n = 551) = 2.17, p = .141).

2.3.3 Perception of External LAN trespassing into the sleeping environment

As can be seen from Figure 2.3A most respondents (57.8% n = 319) reported not perceiving external LAN from the outside environment (e.g., street lighting, car headlights) entering the sleeping environment. As can be seen from Figure 2.3B, those who perceive no external LAN passing into their sleeping environment more frequently report that their sleeping environment is dark once all bedroom lights are turned off (65%; x^2 (1, n = 551) = 58.409, p < .001, phi = .326). Concurrently, those who perceive external LAN more frequently report that their sleeping environment is bright (76.2%). To separately assess whether individuals who perceived LAN to be disruptive to sleep were more biased in their perception of external LAN and room brightness, a split file analysis was conducted. Significant associations were observed for perception of external LAN and room brightness for those that perceive LAN to be disruptive (x^2 (1, n = 350) = 27.72, p < .001, phi = .30) or not disruptive to sleep (x^2 (1, n = 552) = 27.72, p < .001, phi = .374). In both cases, those who do not perceive external LAN more frequently endorse that their sleeping environment is dark, while those who perceive external LAN more frequently report their sleeping environment to be bright.

Figure 2.3

(A) Pie chart illustrating the percentage frequency of the sample endorsing the perception of external LAN (e.g., car light, street lighting) entering the sleeping environment. (B) Bar chart illustrating the percentage frequency indicating the frequency of endorsing the perception of external LAN and the perceived room brightness after bedroom lights are turned off.

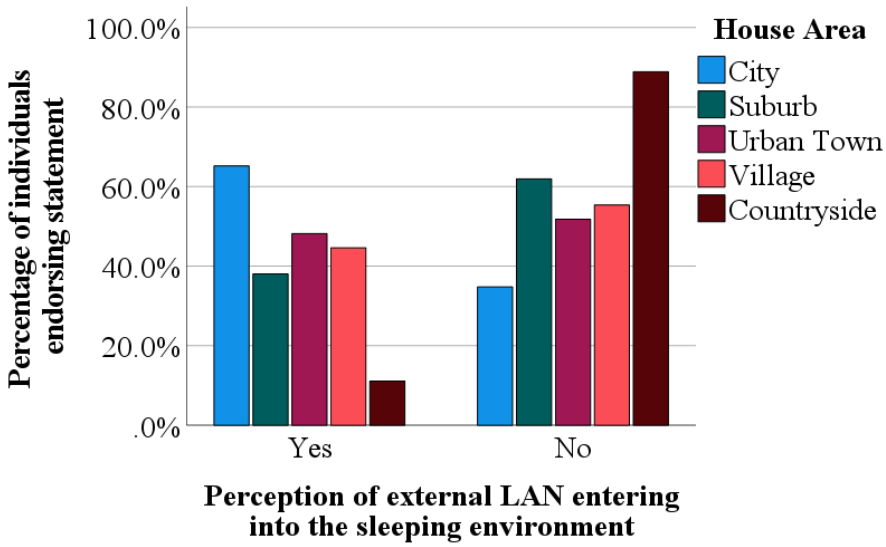


2.3.3.1 Population density and perception of External LAN

The location of the residence is associated with whether external LAN is perceived in the sleeping environment (x^2 (1, n = 552) = 67.35, p < .001, phi = .349). Those that live in city environments more frequently report perceiving external LAN in their sleeping environment, whereas those living in rural environments more frequently endorse not perceiving external LAN. More specifically as can be seen from Figure 2.4 for those who perceive external LAN, there is an ascending frequency in endorsing perception of external LAN and urban density perception of LAN. City inhabitants (65.2%) experience more external LAN followed by urban towns (48.2%), suburbs (38.1%), semi-rural environments (44.6%) and rural environments (11.1%). Those that do not perceive LAN more frequently report living in rural environments (88.9%) and those that live in cities are less likely (33.4%) to report not perceiving LAN. Surprisingly, those living in suburbs and urban towns are more likely to report not perceiving external LAN (61.9% and 51.8% respectively).

Figure 2.4

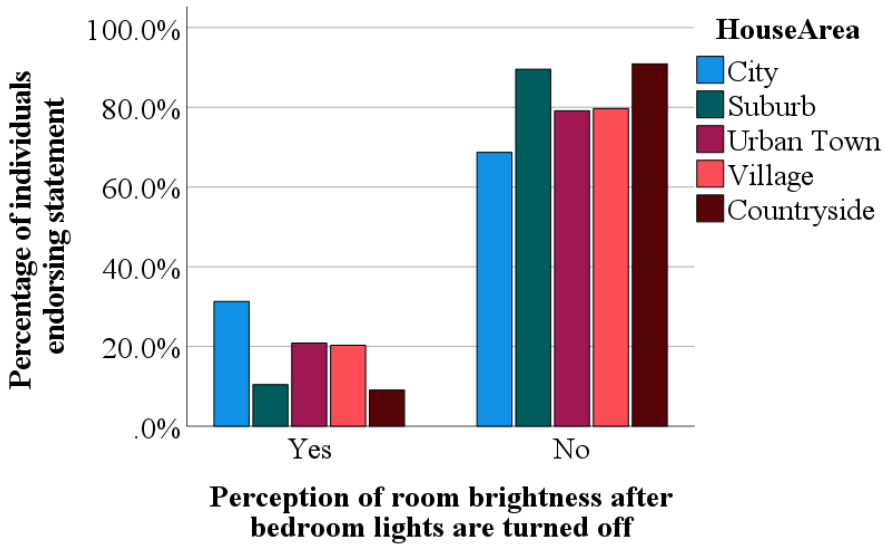
Bar chart illustrating the percentage frequency indicating the frequency of endorsing the perception of external LAN and living location.



Perceived room level brightness and living location were significantly associated (x^2 (4, n = 551) = 24.90, p < .001, phi = .21). As can be seen from Figure 2.5 those perceiving their sleeping environment to be bright more frequently resided in cities (31%), urban towns (21%), villages (20.3%) relative to suburbs (10.4%), or the countryside (8.9%). Surprisingly, those that perceive their room to be dark more frequently reside in the countryside (91%) suburbs (90%) or urban towns (79%) relative to those living in the city (68.7%) or a village (79.7%).

Figure 2.5

Bar chart illustrating the percentage frequency indicating the frequency of endorsing the perception of external LAN and living location.



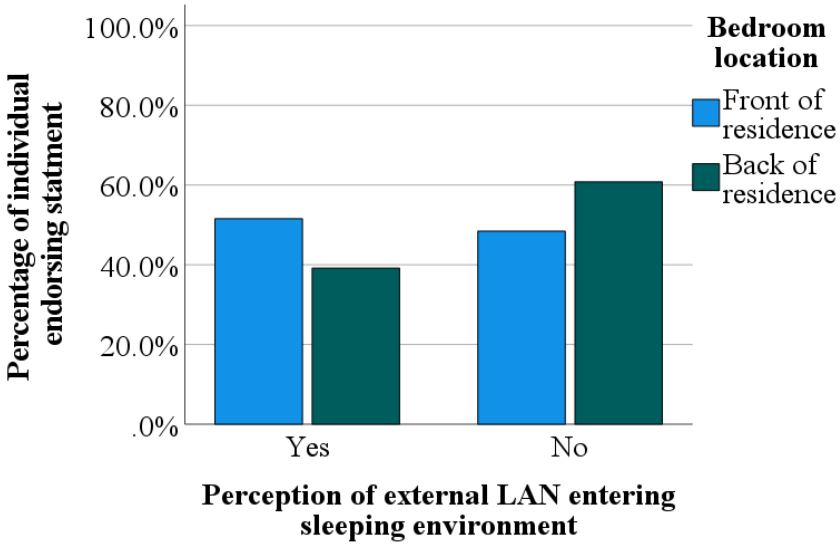
2.3.3.2 Bedroom location and external LAN perception

Out of the full sample, 27% of individuals who report perceiving LAN have their sleeping at the front of the residence while only 19% whose sleeping environment is located at the back of the residence report perceiving LAN in their sleeping environment. There was a significant association between location of the bedroom in the residence and the perception of external LAN (x^2 (1, n = 309) = 4.75, p = .029, phi = .12). As can be seen from Figure 2.6, individuals whose bedroom was at the back of their residence were more likely to report not perceiving external LAN trespassing into the sleeping environment (54%), whereas individuals whose

bedroom was at the front of the residence are more likely to report perceiving external LAN (59%). Further analyses with the file split according to house location found no association between the perception of external LAN and location of the bedroom in the residence.

Figure 2.6

Bar chart illustrating the percentage frequency indicating the frequency of endorsing the perception of external LAN and location of the sleeping environment in the residence.



2.3.3.3 Effectiveness of blinds in reducing the perception of External LAN

Most respondents (97.8%) report having blinds/curtains on their windows. However, there were various degrees to how individuals perceive the effectiveness of the blinds/curtains. Although only a small number (2.8%) reported the blinds/curtains being ineffective, 34.8% reported that their blind/curtains were moderately/somewhat effective in preventing light entering the bedroom. Most respondents (62.4%) perceive their blinds/curtains to be very effective/effective in preventing light trespassing into the bedroom.

Analysis found that those who rated their blind/curtains as either effective or very effective more frequently reported to not perceive external LAN in their sleeping environment (x^2 (1, n = 552) = 110.54, p < .001, phi = .447). As can be seen from Table 2.1 for those who perceived external light trespassing into their bedroom environment, only 12.7% of respondents report that their blinds were very effective.

In comparison, 87.3% of those that do not perceive light trespassing into their sleeping environment find that their blinds and curtains are very effective in preventing light trespassing into the sleeping environment. Combining respondents that perceive their blinds to be either effective or very effective found that from the overall sample, just 16.3% reported that their blinds were effective in blocking external LAN. Conversely, 44.7% from the overall sample reported that their blinds were either effective or very effective in blocking external LAN. Participants who perceive LAN are equally likely to report their blinds as either somewhat effective or moderately effective (Table 2.1). By combining these responses, it is found that 23.1% report that their blind/curtain to some extent are effective while only 10.8% of respondents who do not perceive external LAN endorse this belief.

Table 2.1

Illustrating the frequency of response and percentage of endorsing a statement across the whole sample.

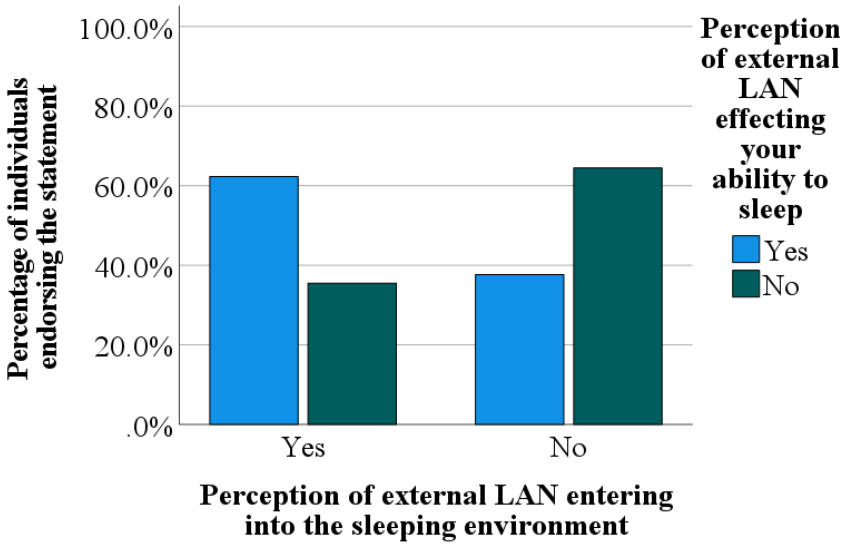
	Yes	No
Very Effective	19 (12.7%)	131 (87.3%)
Effective	71 (38%)	108 (62%)
Moderately Effective	72 (66.7%)	36 (33.3%)
Somewhat Effective	56 (70%)	24 (30%)
Ineffective	10 (66.7%)	5 (33.3%)

75% of respondents do not perceive that external LAN sources impact on their ability to fall asleep. Further examination found that there was a significant association between the perception of external LAN and the perceived impacts of external LAN being disruptive to sleep, x^2 (1, n = 309) = 30.50, p < .001, phi = .24. As can be seen from Figure 2.7, individuals that do not perceive external LAN (65%) more frequently endorse that external LAN is not disruptive to sleep. Individuals that do perceive external LAN (62%) more frequently endorse that the perception of external LAN is associated with perceiving this source of LAN as disruptive to sleep. Similarly, those that do not perceive external LAN, more frequently reported that LAN sources were not disruptive to sleep after falling asleep (62%; x^2 (1, n = 551) = 6.29, p = .012, phi = .11) compared to those that perceive LAN and more frequently

endorse that the perception does impact on disruption to sleep maintenance (49%). Finally, an association was found between the perception of external LAN and the general perception of LAN being disruptive to sleep, x^2 (1, n = 549) = 24.32, p < .001, phi = -.21. Individuals that do perceive external LAN (50%) more frequently endorse that the LAN is disruptive to sleep. Conversely, those that do not perceive LAN (71%) more frequently endorse that LAN is not disruptive to sleep.

Figure 2.7

Bar chart illustrating the percentage frequency indicating the frequency of endorsing the perception of external LAN and its perceived perception on ability to fall asleep.



2.3.4 Perception of Internal LAN trespassing into the sleeping environment

72.1% of respondents reported that they did not have any internal lighting entering the sleeping environment (see Figure 2.8). No association was found between the perception of internal LAN in the sleeping environment and rating of room brightness (x^2 (1, n = 548) = .083, p = .773). Further analysis found no association between the perception of both internal LAN and external LAN (x^2 (1, n = 549) = .044, p = .834). An association was found between the perception of internal LAN entering the bedroom and the perception that LAN exposure being disruptive to sleep after falling asleep (x^2 (1, n = 548) = 4.44, p = .035, phi = .090). As can be seen from Figure 2.9 individuals who perceive internal LAN (33%) more frequently endorse that LAN adversely impacts on sleep quality after falling asleep.

While those that do not perceive internal LAN in their sleeping environment (75%) more frequently endorse that LAN is not disruptive to sleep quality after falling asleep.

Figure 2.8

Pie chart illustrating the percentage frequency of the sample endorsing the perception of internal LAN (e.g., hall lighting/bathroom lighting) entering the sleeping environment.

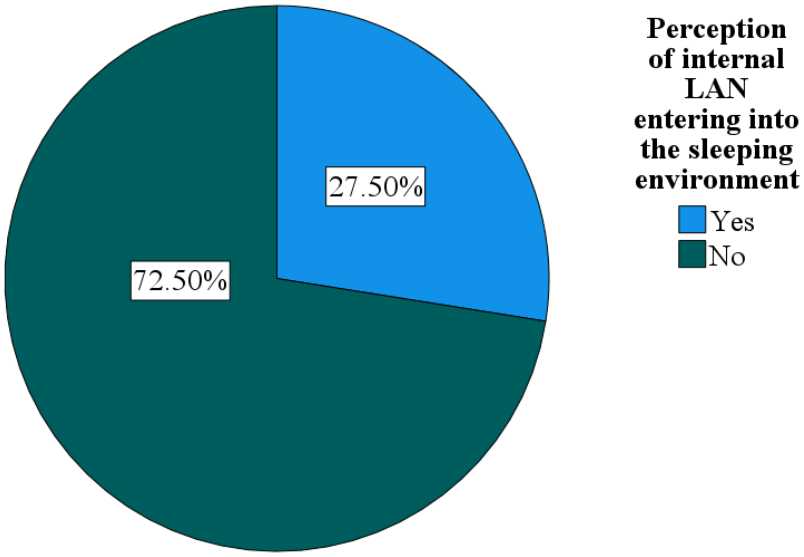
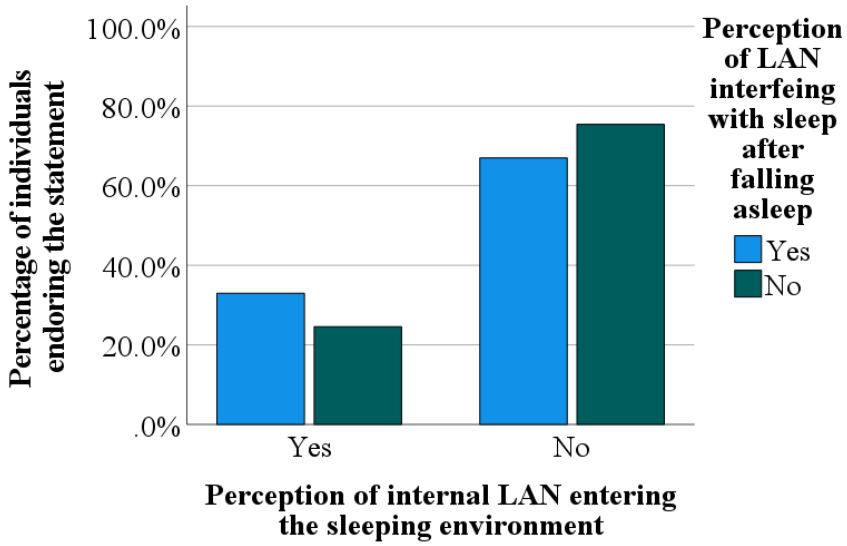


Figure 2.9

Bar chart illustrating the percentage frequency indicating the frequency of endorsing the perception of internal LAN and its perceived perception on impacting sleep after falling asleep.



2.3.4.1 Personal Light Emitting Devices

81.4% of respondents use electronic devices within an hour of attempting to sleep, while 83.8% of respondents state that they use these light-emitting personal devices in bed (see Figure 2.10). An association between age and frequency of light emitting devices in bed was found (x^2 (1, n = 538) = 52,08, p < .001, phi = .311, Figure 2.11), with younger adults more frequently endorsing (43.1%) using light devices in comparison to older adults (20.9%) and older adults more frequently reporting not using devices in bed (56.8%) compared to younger adults (14.6%). From those who reported using light emitting devices in bed 46.1% reported to always using them, 36% reported regularly using them and a small proportion (9.8%) of respondents reported to rarely using them. After using light emitting devices most respondents reported to either falling asleep within 10 minutes (32.7%), 10-20 minutes (31.8%) or 20-30 minutes (17.6%). A smaller number of respondents reported longer latencies to sleep after using personal devices (30-40 mins, 7.5%; 40-50 mins, 4.6% and greater than 60 minutes 5.9%).

Figure 2.10

Pie chart illustrating the percentage frequency of the sample endorsing using light emitting technologies in bed before sleep.

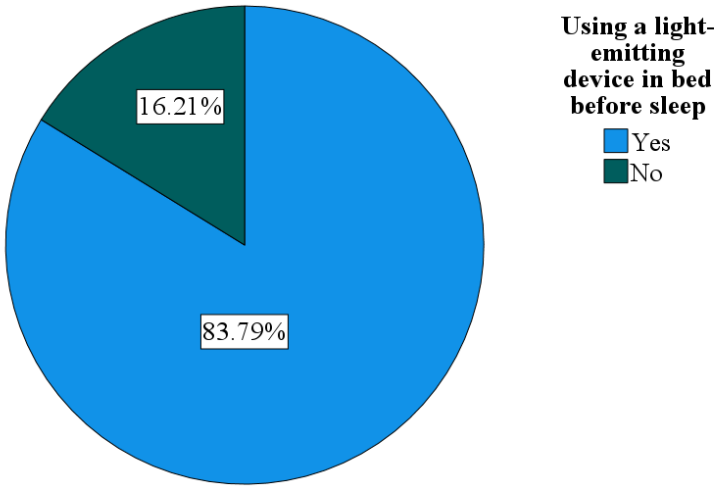
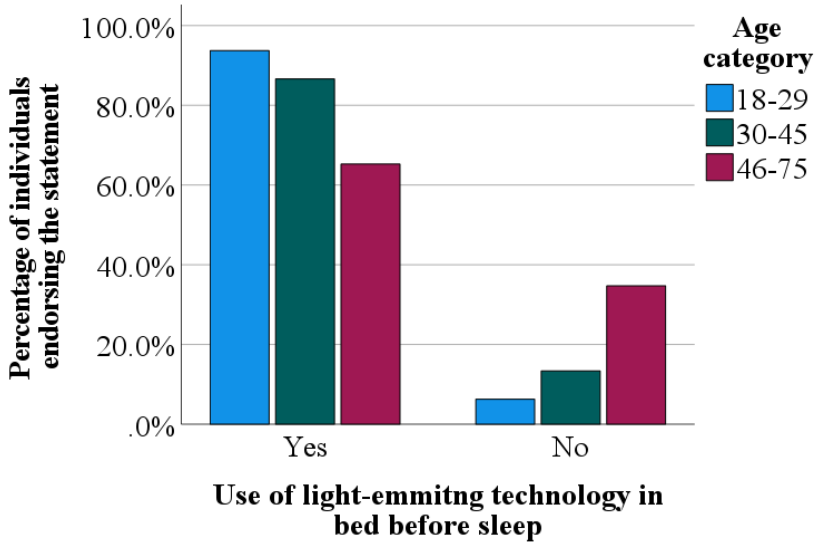


Figure 2.11

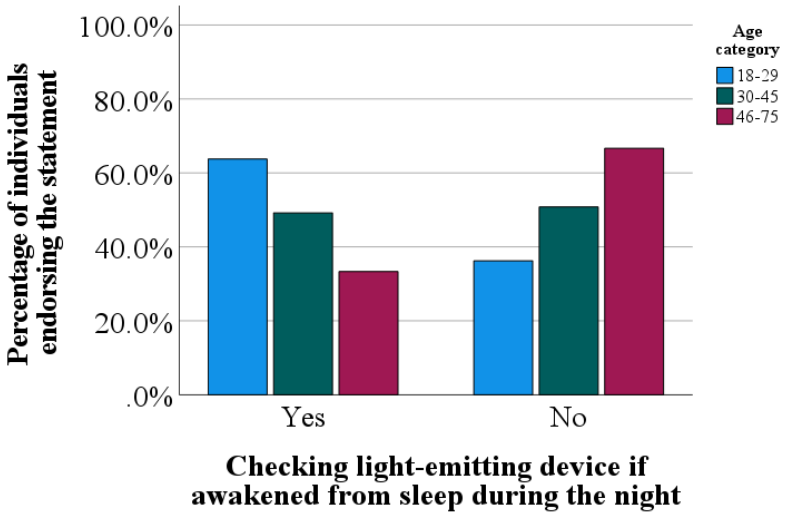
Bar chart illustrating the percentage frequency indicating the frequency of using light-emitting technologies across age groups.



50.3% of the sample report checking personal devices if they awake from sleep during the night. A significant association between age category and frequency of phone checking during the night was found (x^2 (2, n = 538) = 31.68, p < .001, phi = .243). As can be seen from Figure 2.12 younger adults were more likely to report the checking of devices (64%) compared to older adults (33%). When the frequency of checking devices was investigated, it was found that the majority either regularly (29.5%) or always (8.6%) check their personal device, with 34.1% stating that they rarely check their device and 27.5% reporting to never checking their device.

Figure 2.12

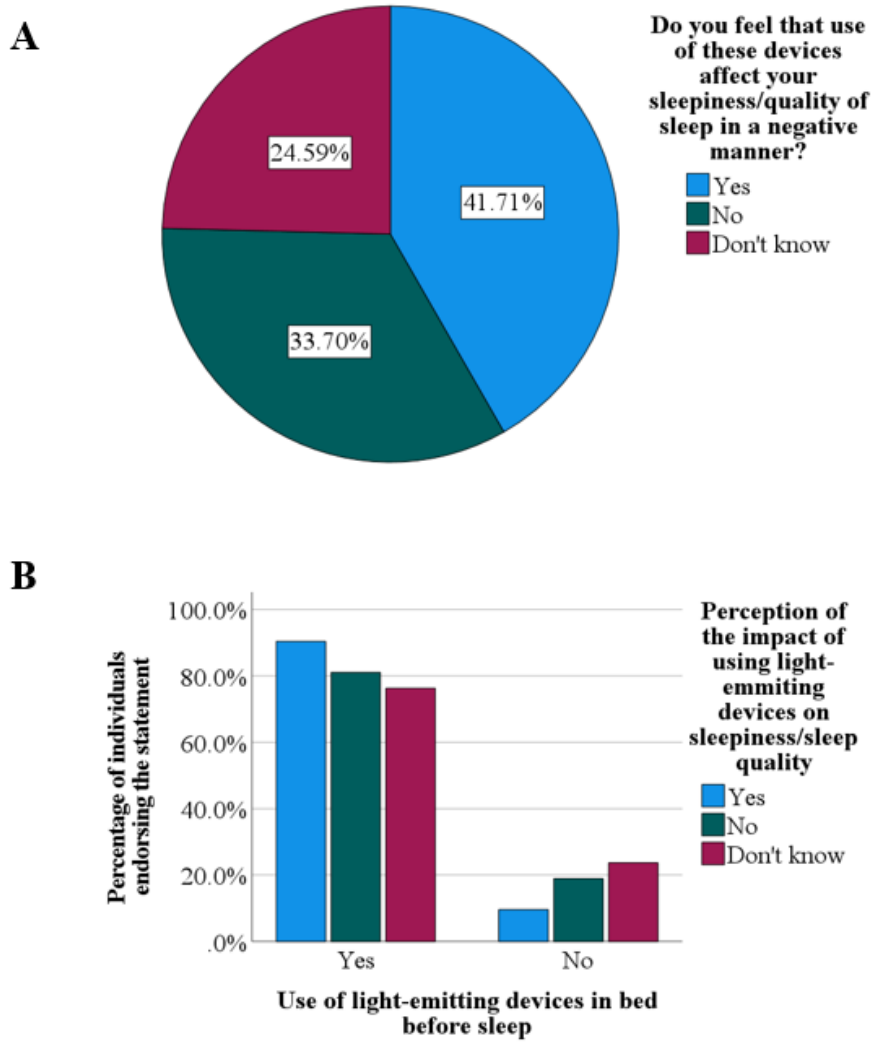
Bar chart illustrating the percentage frequency indicating the frequency of using light-emitting technologies and their perceived impacts on sleepiness/sleep quality.



41.5% of respondents believe that the use of electric devices impact on the quality of their sleep/levels of sleepiness in a negative manner (see Figure 2.13A). A significant association was found between the use of light emitting devices in bed and the perception that these devices on levels of sleepiness and sleep quality x^2 (2, n = 549) = 13.93, p < .001, phi = .16). As can be seen from Figure 2.13B, 90% of those who use devices in bed endorse that these devices negatively impact on sleepiness and sleep quality, those that do not use these devices in bed are more likely to report that these devices are not disruptive to sleepiness/sleep quality (19%) or more likely to report that they do not know if their use is disruptive to sleep (24%).

Figure 2.13

(A) Pie chart illustrating the percentage of respondents that believe that the use of electric devices impact on the quality of their sleep/levels of sleepiness in a negative manner (B) Bar chart illustrating the percentage frequency indicating the frequency of using light-emitting technologies.

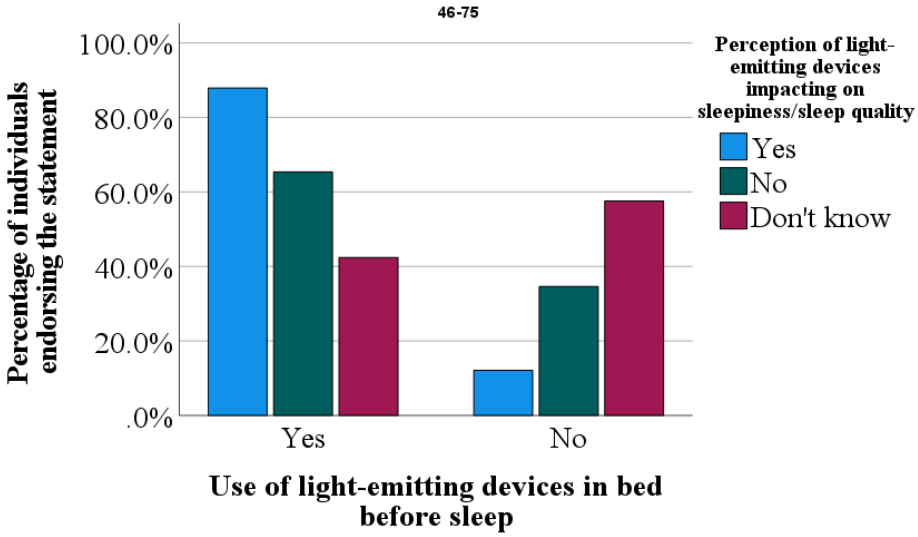


When analysis was split by age group, it was found that older adults who used devices in bed more frequently reported that these devices were disruptive to sleep quality/sleepiness 88%; x^2 (1, n = 144) = 15.04, p < .001, phi = .323, see Figure 2.14). Those that do not use devices (57.6%) were more likely to report that they do not know whether the use of devices are disruptive to sleepiness or sleep quality. There was no differences in the observed and expected counts in those that use/do not use light emitting devices. No significant association was observed between young-aged adults and middle-aged adults when examining the association of

between light emitting devices in bed and the perception of their impact on sleep quality.

Figure 2.14

Bar chart illustrating the percentage frequency indicating the frequency of using light-emitting technologies and their perceived impacts on sleepiness/sleep quality for older adults (46-75 years).



2.4 Discussion

The aim of this study was to describe individuals' light habits in their sleep environment, what are the perceived sources of LAN in the sleeping environment and what are the perceived impacts of these LAN sources on sleep. In this exploratory and descriptive study, it was observed that most individuals endorse that LAN exposure is both disruptive to sleep quality and interferes with the quality of sleep after falling asleep. However, when assessing the specific sources of LAN exposure and their perceived impacts on sleep, findings are mixed. Individuals who perceive either internal or external LAN in the sleeping environment more strongly endorse that it is disruptive to sleep. Conversely, those that do not perceive LAN either from internal or external sources more frequently endorse than LAN is not disruptive to sleep/sleep quality. Separately, those that more frequently use light emitting technologies in bed before sleep more routinely endorse their negative impacts on sleep quality/levels of sleepiness.

Our findings are consistent with other studies which report that only a small percentage sleep with a light on (Hurley et al., 2014; Ohayon & Milesi, 2016). Our finding of no association between sleeping with a light and the perception of light being disruptive after falling asleep are in contrast with experimental findings, which showcase that bedroom lights left on are associated with shallow sleep and frequent arousals (Cho et al., 2013, 2016). Our study is the first to report that just under 30% perceive LAN exposure from inside their dwelling trespassing into the sleeping environment. However, there are differences in the perceived impacts of this LAN exposure on sleep. Specifically, those who perceive internal trespassing LAN more frequently report that LAN is disruptive to sleep after falling asleep while the opposite trend was observed in those who do not perceive internal trespassing LAN. These findings may suggest that those who do not perceive LAN in the sleeping environment may have taken steps to minimise trespassing LAN from entering the bedroom and do not experience the potential adverse impacts LAN exposure has on sleep, and conversely. Alternatively, those that perceive internal trespassing LAN may be sensitive to light exposure (Chellappa et al., 2021; Philips et al., 2018). However, it must be noted that this level of LAN exposure may not be sufficient intensity levels or project in the direction in relation to the retina may not be sufficient to produce physiological effects to disrupt sleep.

42.2% of the sample report the perception of external LAN those that perceive their sleeping environment to be bright also report perceiving external LAN from the outside environment. These findings align with Ohayon and Milesi (2016), who report that sleeping in a bedroom which was perceived as bright was associated with living in areas with higher level of environmental outdoor light. Our findings along with Ohayon and Milesi (2016) suggest that part of the room brightness of the sleeping environment comes from the external environment. The relative frequency of those perceiving their sleeping environment to be bright are similar to that of Johns et al. (2017). However, our findings are substantially higher than that of Ohayon and Milesi (2016) who reported that only 7.4% of respondents perceived their room to be bright using a similar question to one posed in the current study. This is despite light pollution being reported to be higher in the USA compared to Europe where the current participants were from (Falchi et al., 2019). The possible reason for the difference in the frequencies is that rates of outdoor light pollution has increased since 2003-2013 when their study was completed (Kyba et al., 2017). Additionally, the type of lighting in the outdoor environment has changed with de Miguel and colleagues (2021) reporting that globally since 2010, there has been a phased transition from high pressure sodium lighting which are perceived as less bright to blue rich LEDs, which are perceived as brighter and more disruptive to circadian rhythms. As a result of this shift, individual's may be more aware of perception of external LAN than when older light technologies were present.

Our results indicate that those who perceive external LAN are more likely to report living in cities, while those that do not perceive LAN are more likely to living in rural areas. These findings align to some extent with an Irish Citizen Science study which found that individuals living in cities reported experiencing dark skies and that public lighting was observed to be a main source of light pollution for city and town dwellers (Coogan et al., 2020). These findings are further supported by Spitschan and colleagues (2016) who report that at night, the downwelling illumination of the sky in cities are strongly influenced by artificial illumination while in rural areas the average spectral wavelength is irregular but displays a peak near emission which is reflective of light pollution-free conditions.

The results from the current study provide practical solutions that individuals can take to minimise external LAN trespassing into the sleeping environment. Although nearly all participants report havening curtains/blinds, the efficacy of these

blinds impacts on the perception of LAN, those who rate their curtains/blinds as either very effective or effective more frequently reported not perceiving LAN while those who rated their blinds as either moderately/somewhat effective or ineffective reported a higher frequency of perceiving LAN.

Surprisingly, the current study observed that those that perceive LAN more frequently endorse that these sources are disruptive to their ability to fall asleep, with this endorsement even stronger in those that do not perceive external LAN. These findings consistent with other studies showcasing that ALAN in the sleep environment are associated with increased risk of delayed latency to sleep onset (Obayashi et al., 2014a) and insomnia (Obayashi et al., 2014) in older adults along low level LAN altering sleep architecture (Cho et al., 2016; 2018). Additionally, although individuals will have their eyes closed when sleeping, closed eyes may not fully block light or its impacts on non-image forming behaviours (Cole et al., 2002; Figueiro et al., 2012; Figueiro et al., 2014; Zeitzer et al., 2014). It is proposed that level of light which can transmit through the closed eyes while sleeping can result in phase shifts in melatonin rhythms (Zeitzer et al., 2014). For those that perceive external LAN, the level of light perceived may be of sufficient intensity at an indecent level of the retina to produce physiological effects on the circadian clock or sleep timing. Findings indicating that those who do not perceive LAN more frequently endorsing that LAN is not disruptive initiation of sleep may have good overall sleep quality and not place their attention towards sleep related environmental stimuli (Harris et al., 2015).

A significant proportion of the sample indicate utilising light emitting devices in bed before sleep. These findings align with other large-scale poll studies which report that 90% of individuals report using technological devices either in the hour before bed (Gradisar et al., 2013) and when all lights are turned off in the bedroom (Jniene et al., 2019). Our findings align with an earlier study showcasing that overall, there has not been a change in the age profile of those using light emitting technologies before bed with older adults more likely to report not using these devices in the bedroom environment (Gradisar et al., 2013). These findings are surprising given the prevalence and useability of light emitting technologies has increased in recent years, it could be expected that older adults' use would of such devices would also increase. Just under 40% of respondents indicate checking their phone if they awake from sleep during the night. This is concerning given that light

when ill-timed and when of the spectral wavelength typically found in light emitting technologies can increase alertness, decrease subjective sleepiness (Cajochen et al., 2011; Chang et al., 2015), and even when exposure occurs for short duration can induce larger physiological effects on the circadian system per minute of exposure than longer durations of light exposure (Chang et al., 2011; Prayag et al., 2020).

41.7% perceived that using electric devices impact on the quality of their sleep/level of sleepiness in a negative manner. This frequency is lower than that reported by Jniene et al. (2019) who found that 65.7% perceived night usage of blue light-emitting smart devices to be disruptive to sleep. However, it plausible that the findings from Jniene and colleague's (2019) study may not be reliable as the profile of participants were medical and pharmacy students who may through their studies previous knowledge of the impact of light on both sleep and the circadian system. Additionally, our responses to the question included a "don't know" response which may have spread out the endorsement to dichotomous response option provided in Jniene et al's (2019) study. Our findings indicate that despite being consciously aware that use of light technologies is disruptive to sleep, individuals still report using them in bed before sleep. These findings provide some support to the argument that knowing about proper sleep hygiene habits does not influence sleep quality (Brown, et al. 2002) or behaviour (Kwok et al., 2017). This suggests that although individuals perceive that their lighting habits are disruptive to sleep however, this does not translate to changing usage habits. Additionally, in the current data just under a quarter of participants report not knowing whether using light emitting technologies in bed was disruptive to their sleep. This suggests that more public health information needs to be provided to the population about the negative impacts that light emitting technologies can have on sleep timing. Individuals could also be made aware of possible strategies they could employ to modify their behaviour to reduce LAN exposure from light emitting devices. This could potentially have a positive effect by reducing the possible adverse impact of LAN exposure on sleep and health.

There are several strengths to this study. Firstly, this is the first study which attempts to concurrently assess individuals lighting habits and the sources of LAN exposure in the sleeping environment along with individually assessing how these perceived sources are perceived to impact on sleep. It is of importance to measure and understand what sources are contributing to LAN exposure in the sleeping

environment and what measures can be taken to reduce it. Additionally, at a population level this study gives an insight into what individual attitudes towards these sources have on sleep. This can provide a basis in understanding the degree to which individuals know the disruptive impacts of light exposure. Our study builds upon simply using satellite data as surrogate measure of LAN exposure, but instead to classify the frequency of perception of external LAN in the sleeping environment and co-exposures, thus providing a more detailed account of individual light exposures. Our findings showcase possible strategies individuals can take to minimize external LAN by using good quality curtain/blinds to block urban lighting. Policy makers can also ensure that shielding is placed on lighting to prevent it dissipating into the sleeping environment. Secondly, the study advances from some of the limitations of previous work which assessed room brightness and lighting habits by examining strategies individuals take to prevent LAN exposure. The study also provides a more specific understanding on the impact external LAN has on sleep. While satellite studies have provided evidence indicating an association between higher level outdoor lighting and adverse sleep outcomes these studies possibly overgeneralise light exposure at an individual level in those living in areas with high levels of outdoor light. For example, an individual living in a dark rural area with no outdoor light may have high levels of LAN exposure in their sleeping environment while an individual in high levels of outdoor LAN living in an urbanised area may use shutters or blinds to keep out external LAN and sleep in total darkness. The study also recruited participants from a large age range and was not a case referent study, which previous studies accessing light exposure have been based upon.

There are several limitations to this study. Firstly, the survey used is not a validated measure however, questions used were influenced by previous research and limitations of previous work was considered in question and response selection. The study was a cross sectional survey which may be influenced by recall and selection bias. No objective measurements of light exposure in the bedroom were collected which limits the reliability of the findings. Although, this research investigated the perception of LAN, the study did examine the characteristics of light exposure which can have impact on circadian rhythmicity, sleep and health. These include intensity, duration, light spectral composition and distance from the LAN sources. Although the labels of our response options in the questions were common

in much observational research, (Arora et al., 2013; Arora et al., 2014) they can be ambiguous as it is rather subjective what is to be understood by “occasionally”, “sometimes,” “regularly” and “often.” Perhaps response options with a more distinct indication of frequency, such as “about once a month,” “several times a month,” “about once a week” and “several times a week” may be less ambiguous. Assessment of light emitting technologies was broadly assessed however, the specific light emitting technologies and the frequency of light emitting technologies used was not investigated. Several studies have indicated that individuals use more than one light emitting source in the bedroom (Gamble et al., 2014) with increased number of light-emitting technologies used at night being a predictor of delayed sleep timing and sleep quality (Chahal et al., 2013; Gradisar et al., 2013).

The prevalence of exposure to LAN is widespread and individuals to a large extent have control over the amount they are exposed to. Our findings indicate that despite a global perception of the disruption of LAN to sleep, most individuals either voluntarily or involuntarily are exposed to various sources of LAN in the sleeping environment. While the sleeping environment should be a space to afford sleep, our lighting practices could paradoxically influence wakefulness. Given the spate of studies indicating the negative effects of light on sleep timing, sleep architecture and sleep quality and the current study findings that light sources are prevalent in the sleeping environment, public health education must be given to individuals to reduce their exposure to LAN but also the negative impacts it has on their sleep.

Chapter 3

Associations of Perception of Artificial Light-at-night with Psychological distress, Sleep Quality, and Chronotype

Abstract

Exposure to artificial light-at-night (ALAN) is increasing globally, and there are concerns around how ALAN may impact sleep, psychological and physical health. However, there is a lack of evidence in the literature on how individuals perceive ALAN relative to their sleeping environment and habits, and how such perceptions match against objectively assessed night-time illuminance at the level of the individual residence. This cross-sectional study examined how such perceptions and their associations with sleep quality, sleep timing, psychological distress and cognitive failures, as well as to illuminance levels calculated as the biologically-relevant melatonin-suppression index estimated at the level of the individual residence using a database of public street lighting. 552 adult participants completed a survey addressing perception of ALAN in sleep environment along with the Pittsburgh Sleep Quality Index, Munich Chronotype Questionnaire, Cognitive Failure Questionnaire and the Global Health Questionnaire. We report that perception of external ALAN in the sleeping environment was associated with poorer sleep quality, more cognitive failures and greater psychological distress. Minimal associations were found between the perception of external ALAN and MSI scores, and MSI scores were not associated with scores on any of the self-report measures. Internal lighting passing into the sleeping environment was associated with poorer sleep quality but not with psychological wellbeing. Habitual use of light-emitting devices was associated with poorer psychological wellbeing but not with sleep quality and sleep timing. These results may suggest heightened attentional bias towards ALAN associated with poor sleep quality and higher levels of psychological distress, and highlight the need for more granular approaches in the study of ALAN and sleep and psychological health in terms of levels individual ALAN exposure, and an interpretation that seeks to integrate biological and psychological perspectives.

Keywords: Light-at-night, sleep, psychological health, physical health.

3.1 Introduction

Exposure to artificial light-at-night (ALAN) has become part of everyday life, with individuals routinely exposed to ALAN through the use of electronic devices, indoor electric lighting and environmental light pollution (Falchi et al, 2016). Whilst ALAN brings many societal benefits such as extending the length of productive days and recreational activities (Fonken & Nelson, 2011), ALAN may also disrupt both the internal biological circadian clock and sleep by artificially extending the biological day leading to desynchrony of the circadian timing system and impaired sleep (Zeitzer et al., 2000). Such effects may contribute to a number of adverse health outcomes at a metabolic (Park et al., 2019), psychiatric (Wulff et al., 2010) and neurological level (Musiek & Holtzman, 2016), as well as potentially elevating the risk of some hormone-dependent cancers (Davis et al., 2001). Circadian rhythms are the product of endogenous oscillators which are responsible for the regulation of our physiology and behaviour with a near 24-h period (Dijk & von Schantz, 2005). The core pacemaker is located in the suprachiasmatic nucleus (SCN) of the hypothalamus and is entrained to the 24-h day, with light being the major synchronising cue (Hughes et al, 2015). The primary neural mechanism of such photic entrainment is via a pathway involving intrinsically photosensitive retinal ganglion cells which direct neural projections to the SCN allowing for non-visual synchronisation of circadian rhythms to cycles in environmental light (Hughes et al., 2015).

Through industrialisation and modernisation, the natural pattern of light and dark has been altered by the lengthening of the period of daily light exposure resulting from man-made lighting (Lunn et al, 2017). Exposure to ALAN at sufficient intensity, duration, wavelength and timing impacts on the circadian timing system (Gooley et al., 2003; Cajochen et al., 2000, 2005; Lockley et al., 2006). ALAN may also suppress the sleep-promoting hormone melatonin (Gooley et al., 2011) and alter the expression of the molecular components of the circadian clockworks in the SCN (Bedrossian et al, 2017). Through the advancement of technology, individuals may now be routinely exposed to low level ALAN in their sleeping environments through the use of computer screens, tablets and smartphones which emit short wavelength visible light to which the circadian clock is most sensitive (Hughes et al, 2015). Dim light at night (dLAN) emitted from may

increase both alertness and heart rate and a reduced propensity to sleep (Cajochen et al., 2005) and alter clock gene expression (Cajochen et al., 2016).

Bedroom lights left on during sleep is associated with more shallow sleep, frequent arousals and negative impacts on brain oscillations essential to sleep depth and stability (Cho et al., 2013). dLAN exposure during sleep results has been reported in increased number of awakenings, shallow sleep (Cho et al. 2016), poorer sleep quality and in older adults increased risk of insomnia (Obeyashi et al., 2014). Observational and longitudinal studies in older adults have found ALAN exposure in sleeping environments is associated with a greater risk of depression (Obeyashi et al., 2013; Obeyashi et al., 2018). Similar findings have also been reported from animal studies, with ALAN perturbing circadian rhythms (Stenvers et al., 2016; Panagitou et al., 2020) and associating with depressive-like behaviour (Bedrosian et al., 2014; Borniger et al., 2014; although such findings are not ubiquitous and may vary according to the animal model (Cleary-Gaffney and Coogan, 2018).

Outdoor street lighting is common in industrialised countries, with outdoor ALAN increasing annually by 5-10% (Hölker et al., 2010) resulting in around 80% of USA citizens living in areas where the natural appearance of the night sky cannot be observed, and up to 40% living in areas where night adaption of human eyes is inhibited by light (Falchi et al., 2016). The recommended mean lighting level along residential roads has surface illuminance levels of 2-15 lux, with lighting levels along busier routes or in city centres being higher (BSI, 2015). However, lighting from other sources such as private dwelling and commercial outdoor lighting may result in the maximum permitted level being exceeded in urban settings. Such light may also trespass into individual's sleeping environments, particularly in urban environments where there is closer proximity between public lighting and house windows both in horizontal and vertical alignment. A number of studies have associated outdoor ALAN with poorer sleep quality, reduced night time sleep, and delays in both bedtime and waking-up time (Koo et al., 2016; Ohayon & Milesi, 2016). Living in areas with high outdoor light may result in greater risk of depressive symptoms and suicidal behaviours in adults (Min & Min, 2018) and increased risk of mood and anxiety disorders in adolescents (Paksarian et al., 2020). In addition, individuals over 60 years old living in areas with high external ALAN had greater hypnotic medication use.

There are a number of important limitations in the current literature relating ALAN to real-world effects. Although previous studies in older adults have indicated negative associations between indoor ALAN and sleep, physical and psychological health, the results of these studies may not be generalizable to the general population as around 40% of older adults would be expected to report insomnia symptoms such as delayed sleep onset and difficulty maintaining sleep (Calem et al., 2010; Walsh et al., 2011), and as such the additional impact of ALAN on top of normal age-related changes in sleep and circadian function is not clear. Another important limitation is the level at which ALAN is assessed; ecological studies have provided evidence that high levels of outdoor LAN measured via satellite data are associated with sleep disturbances, poor sleep quality and poor psychological well-being (Paksarian et al., 2020; Xiao et al. 2020). Because of the use of low-resolution external estimates of LAN, these studies potentially under- or overestimate personal experience of ALAN in the sleeping environment. This comes from evidence which suggests there is a lack of association between external ALAN and the ALAN measured in the bedroom (Huss et al., 2019; Rea et al., 2011) and the subjective perception of external ALAN entering the bedroom and satellite measurements of ALAN (Garcia-Saenz et al., 2018). As a result it is unclear whether outdoor ALAN is impacting on sleep and general psychological well-being in the sleeping environment. A second disadvantage of using satellite image data as a measure of ALAN exposure is that provides an average light intensity of a region and may not reflect the true values, amounts, and patterns of ALAN exposure; as an example, Katz and Levin (2016) found only a low to moderate correlation between ALAN measured at ground level and satellite measured ALAN.

Given the high prevalence of ALAN and its potential negative health implications, it is important to investigate its impacts, and the perception of the same, in the general population. No studies to date have investigated how the *perception* of ALAN exposure is associated with sleep and psychological well-being. The current study aims to examine how individuals perceive ALAN exposure in their sleeping environment, how subjective perceptions of ALAN correlate with estimates of outdoor illuminance due to public lighting at the level of individual residences, and how subjective perceptions of ALAN as well as objectively-measured household illuminance levels associate with measures of psychological distress, cognitive failures, sleep duration quality and chronotype. Our *a priori* hypotheses were: 1)

That the perception of ALAN trespassing into the sleeping environment will associate with poorer sleep quality, delayed circadian timing, and higher levels of psychological distress; 2) The self-reported use of light-emitting technology at night will associate with poorer sleep quality and more psychological distress; and 3) There will be an association between objective measure of external lighting and subjective perception of external lighting.

3.2 Methods

3.2.1 Participants

Recruitment of participants was through a mixture of snowball and convenience sampling via flyers, emails, personal contacts, and tradeshows. A total of 552 of Irish residents completed the questionnaires. Data was collected between March 2017 and June 2019. Both pencil and paper and online versions of the questionnaires were used. The inclusion criteria were that participants were aged 18 years and above who were not current shift workers. All participants gave their electronic informed consent before participating in this study and were informed that all data collected would be stored anonymously and participation was voluntary and unpaid. Ethical approval was obtained from the Maynooth University Research Ethics Committee.

3.2.2 Questionnaires

Participants provided demographic information, including age, gender, location of house (city, suburb, town, semi-rural, rural), type of residence (detached house, semi-detached house, bungalow, apartment), the exact GPS of the residence (expressed as the Irish Eircode, a residence-specific geolocation identifier), and whether the respondent's bedroom located at the front or back of their premises.

3.2.2.1 Light at Night Questionnaire

We employed the questionnaire which was outlined in chapter 2. The survey asks about perceptions of ALAN, its potential sources, and its perceived impact on sleep. The nature of most response items were dichotomous (yes/no), and as such the questionnaire generated categorical data. Specifically, there were items relating to the perception of whether outside ALAN enters the bedroom at night, if outside ALAN impacts on sleep, whether there are indoor sources of ALAN that enter the bedroom at night and whether such ALAN impacts on sleep, a question rating the brightness of bedroom with no bedroom lighting, and questions pertaining to electronic device usage in the run-up to, and after, sleep onset and the perceived impacts of device usage on sleep. Furthermore, there were questions relating to noise

pollution, asking whether noise pollution at night a nuisance in the bedroom is and whether it impacts on sleep.

3.2.2.2. Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989).

The PSQI is a retrospective self-report questionnaire on sleep quality over the previous 4 weeks (see appendix B). It is a 19-item measure which is divided into 7 domains called component scores (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications and daytime dysfunction). Each component score ranges from 0 (no difficulty) to 3 (severe difficulty) and is summed to produce a global score which ranges from 0 to 21. Scores of 5 and above were categorised as poor sleep quality. In the current study the Cronbach alpha coefficient was .72

3.2.2.3 Munich Chronotype Questionnaire (MCTQ; Roenneberg et al., 2003)

The MCTQ is a self-report measure (see appendix C) which investigates sleep–wake behaviour by asking participants to indicate their sleep and wake times on both "work" and "free" days resulting in the calculation of the mid-point of sleep on each of these types of days. The average sleep duration over the course of a week was calculated using a formula which weighted the amount of self-reported sleep on "work" and "free" days (Roenneberg et al., 2012). Mid-sleep on free days (the midpoint between sleep onset and wake time), corrected for sleep debt accumulated during the week and provided a measure of chronotype as sleep timing without social constraint provides an indication of the underlying phase of circadian entrainment (MSFsc; Roenneberg et al., 2003). Social Jetlag (SJL) was measured by subtracting mid-sleep on workdays from mid-sleep on free days and presenting this as an absolute value (Wittmann et al., 2006). The numbers of work and free days were also assessed through this instrument, as was whether there was any meaningful distinction between "work" and "free" days for the participant

3.2.2.4 Cognitive Failures Questionnaire (CFQ; Broadbent et al., 1982)

The CFQ is a 25-item self-report instrument (see appendix D) which assesses the frequency of everyday slips and errors an individual has in the domains of memory, perception, and motor function. Scores for the CFQ can range from 0 to

100 with the total CFQ score being simply the sum of all the individual responses on for the 25 items. In the current study the Cronbach alpha coefficient was .91

3.2.2.5 Global Health Questionnaire (GHQ; Goldberg, 1978)

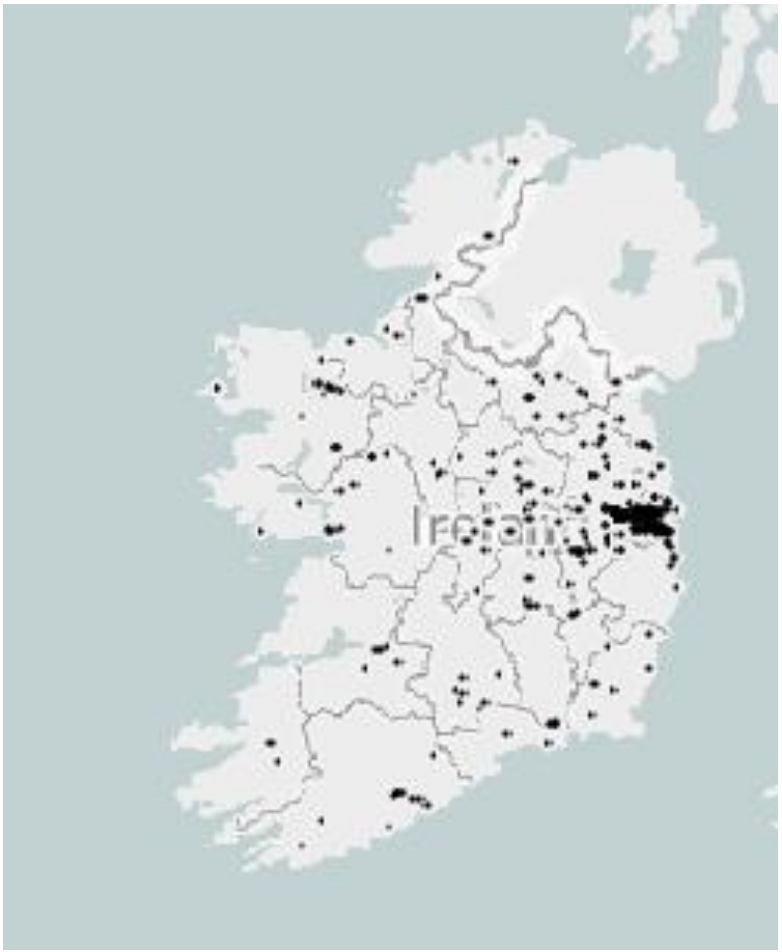
The GHQ is 28-item self-report questionnaire (see appendix E) which assesses how participants rate their general psychological health over the past weeks. The questionnaire is divided into four specific subscales which are somatic symptoms, anxiety/insomnia, social dysfunction, and severe depression. Global scores range from 0-84 with higher scores indicate greater levels of psychological distress. In the current study the Cronbach alpha coefficient was .94

3.2.3 Address Geocoding and Residence-Level Melatonin Suppression Index

Each participant provided their Eircode; unlike postcodes in other jurisdictions which define clusters of residences, an Eircode is specifically unique and assigned to each residential address. After Eircodes were collated, each address was geocoded to the corresponding geographic position using the Google Geocoding API service through Python. A randomly selected subsample was used to verify the positions by comparing our derived locations with those obtained using Google Earth's location based solely on the of the address location and, additionally, using the Eircode map positions. Figure 3.1 provides a graphical illustration of the location of each of each of the residences in Ireland.

Figure 3.1

Schematic image illustrating where each participant's residence is located in Ireland.



3.2.3.1 Melatonin Suppression Index (MSI)

The contribution of street lighting to sleep disruption was calculated using the Melatonin Suppression Index (MSI) for a range of lamp types as discussed in Aubé et al. (2013). MSI provides an estimate of the potential impact of each lamp type on humans based on its blue content and is normalised such that the spectrum of daylight - represented by the International Commission of Illumination (CIE) illuminant D65 - has a value of unity. On this scale, low pressure sodium light has a MSI of 0.017, while LED white light with a colour temperature of 4000K has a MSI of 0.465.

3.2.3.2. Application to individual addresses

The methodology in generating these values was as follows:

1. For each location, generate a list of the public lights within one km, ranked in increasing distance;
2. Calculate the expected illuminance at each residence's location by scaling the wattage by the typical lumens/watt for that lamp type, and weighted by the inverse distance between the lantern and the residence;
3. Calculate the estimated human health impact of the light by multiplying by the Melatonin Suppression Index (MSI) for each lamp type;
4. Rank the impact for each streetlight and stop including lights when the additional contribution to the cumulative total is smaller than the selected cutoff or no further lights are available within the 1 km limit.

3.2.3.3 Code output

For each location a series of metrics were produced by the software: the total lux at the nominal location; the summed light weighted in terms of both lux and MSI value; similar values for those sources closer than 30m, and the distance to the nearest public light, irrespective of spectrum or light output. Missing data were recorded for 23% (n = 127) of the sample, due to either incorrect Eircode, an address provided which was outside a jurisdiction, incomplete data and, in a small number of cases, no data being available.

3.2.4 Statistical Analyses

For statistical analysis time-based variables from the MCTQ (MSFsc, average sleep duration and SJL) were decimalised (i.e. 6:30 become 6.5, 45 min became 0.75). Data was assessed for normality via Kolmogorov-Smirnov tests, and all variables to be treated as dependent variables were found to be not normally distributed, and non-parametric inferential testing was employed for initial unadjusted analysis (Mann-Whitney U and Kruskal-Wallis tests), Chi Square tests for independence were employed to investigate associations between categorical variables. When bivariate unadjusted analysis revealed statistically significant results, associations were retested using ANCOVA adjusting for the relevant covariates of age, sex, and house location MSI. For this analysis the variables of

CFQ, GHB and MSI scores were log-transformed and age and PSQI were centred, and partial eta-squared statistics were reported as indicators of effect sizes. In order to account for missing data and to minimise removal of whole participant data, pairwise deletion was employed when carrying out inferential testing.

The study sample size was estimated using a-priori power calculations based on it being important to detect effect sizes of moderate size ($d = 0.5$) with anticipated variance in dependent variables based on standard deviations from previous studies in our group in similar populations with the psychometric instruments used; these calculations indicated a required study sample of approximately 500 would be required to detect differences of a likely-to-be-meaningful magnitude. All statistical analysis was conducted using IBM SPSS (V25, IBM Corporation) or JASP (V 0.9.1.0, https://jasp-stats.org/)

3.3 Results

3.3.1 Demographics of the study sample.

Key demographic features of the study sample are presented in Table 3.1. The mean age of respondents was 36.7 years. There were significantly more female than male respondents (66% vs 32%) and a reasonably even distribution of residence location across cities, suburbs, towns, semi-rural and rural locales. Most respondents reported living in a house of some type, with only 12% of respondents reporting living in an apartment. The mean, median and standard deviation scores for the chronometric and psychometric data is presented in Table 3.2.

Table 3.1

Key demographics of the responding sample.

Variable	N	Valid Percentage
Gender		
Male	175	31.7%
Female	366	66.3%
Prefer not to say	11	2%
Location of Residence		
City	115	20.8%
Suburb	134	24.3%
Town	139	25.2%
Semi-Rural	60	11.8%
Rural Environment	99	17.9%
Bedroom Location		
Front of Premises	152	28.7%
Back of Premises	131	24.8%
Residence Type		
Detached House	73	13.2%
Semi-detached House	94	17%
Terraced House	45	8.2%
Apartment	68	12.3%
Bungalow	29	5.3%

Table 3.2

Mean, median and standard deviations for scores on the psychometric instruments used to assess psychological health, subjective sleep quality, daily cognitive failures, sleep timing and descriptives on objective MSI data.

	Mean	95%CI	Median	SD	Range
Age (yrs)	36.82	[34.86-38.68]	35	13.01	18-71
Total CFQ	35.46	[33.45-37.41]	33	14.41	2-91
Total Score GHQ	23.31	[21.62-25.09]	20	12.99	2-64
GHQ A - Somatic Symptoms	5.75	[5.17-6.30]	5	4.07	0-19
GHQ B - Anxiety & Depression	6.70	[6.06-7.35]	6	4.74	0-21
GHQ C - Social Dysfunction	7.83	[7.44-8.24]	7	2.97	0-17
GHQ D -Severe Depression	3.02	[2.50-3.59]	1	4.10	0-18
Global PSQI	6.45	[6.00-6.88]	6	3.26	1-17
Average Sleep Duration (h)	7.50	[7.35-7.67]	7.54	1.11	3.86-10.71
MSFsc (h:m)	4.36	[4.18-4.53]	4.17	1.29	1.00-7.61
Social Jetlag (h)	1.16	[1.04-1.30]	1.00	.91	0-4.79
MSI	.05	[.04-.07]	.01	.14	.0-1.56

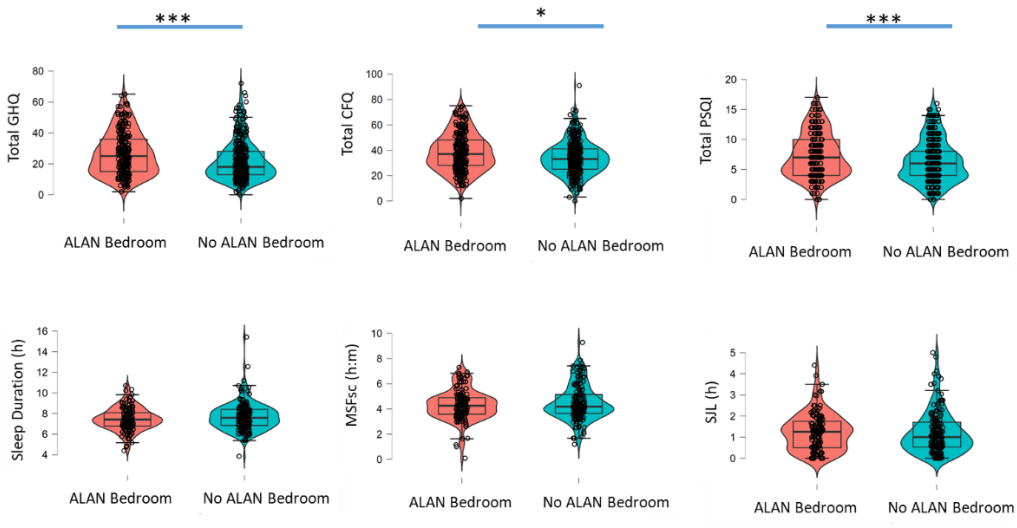
3.3.2 Perceptions of ALAN and sleep and psychological health indicators.

42.2% of respondents reported that external lighting entered into their sleeping environment during sleep. Compared to those who did not perceive external ALAN entering into the sleeping environment, respondents who perceived outdoor ALAN entering into the sleeping environment had poorer sleep quality (median PSQI total score 7, 95% CI [6; 7] vs 6, 95% CI [5; 6], p = .002, r = .11), more cognitive failures (median CFQ total 37, 95% CI [34; 40] vs 33, 95% CI [31.51;

35], p = .001, r = -.15) and higher GHQ scores (median GHQ total 25, 95% CI [22; 27] vs 18, 95% CI [17; 20], p = .001, r = -0.18, Figure 3.1). When adjusting these associations for the potential confounders of age, sex, house location and MSI at residence level, the perception of external ALAN was associated with higher GHQ score (F(1, 336) = 14.2, p < .001, partial eta squared = 0.043), higher PSQI scores (F(1, 336) = 11.23, p = .001, partial eta squared = 0.035;adjusted R^2 for the model = 0.014) and with higher CFQ score (F(1, 336) = 8.72, p = .003, partial eta squared = 0.026). Participants did not vary on average sleep duration (p = .48), MSFsc (p = .62) or SJL (p = .46) according to their perception of external ALAN entering their bedroom (Figure 3.1). When participants rated the brightness of their bedroom at night when the internal lights were switched off (on a five-point scale from very bright to very dark), after adjusting for covariates there were no effects of rating of brightness on GHQ scores (p = .221), CFQ (p = .054), PSQI (p = .061), sleep duration (p = .371), MSFsc (p = .508) and SJL (p = .119).

Figure 3.1

Box-and-violin plots of scores on the GHQ, CFQ, PSQI and sleep duration, MSFsc and SJL from the Munich Chronotype Questionnaire, split by responding on the question "Does artificial outside light (i.e. street lights, traffic lights, headlights etc.) enter the bedroom when you are sleeping?".



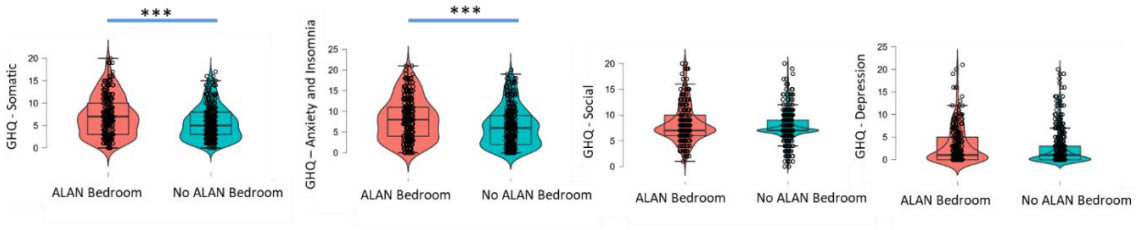
Note. *** indicates p < .01 and * indicates p < .05 by ANCOVA controlling for age, sex, house location and MSI.

When the subscales of the GHQ were examined, participants who perceived external ALAN entering their bedroom had higher scores in unadjusted analyses on

the GHQ-A Somatic Symptoms (median GHQ-A total score 7, 95% CI [6; 7] vs 5, 95% CI [4; 6], $p < .001$), GHQ-B Anxiety/Insomnia symptoms (median GHQ-B total score 8, 95% CI [7; 9] vs 6, 95% CI [5; 6.48], $p < .001$) and GHQ-D severe depression (median GHQ-D total score 1, 95% CI [1; 2] vs 1, 95% CI [0; 1], p = .002), but not GHQ-C social dysfunction (p = .121); Figure 3.2. The associations between ALAN perception and GHQ-A and GHQ-B remained after adjusting for the covariates of sex, age, residence location and MSI ($F(1, 336) = 14.8$, $p < .001$, partial eta squared = 0.043 for GHQ-A; $F(1, 336) = 13.3$, $p < .001$, partial eta squared = 0.039 for GHQ-B), but the association of ALAN perception with GHQ-D was not significant in ANCOVA ($F(1, 336) = 2.9$, $p = .089$).

Figure 3.2

Box-and-violin plots of scores on the subscales of the GHQ split by responding on the question "Does artificial outside light (i.e. street lights, traffic lights, headlights etc.) enter the bedroom when you are sleeping?".



Note. *** indicates p <.01 by ANCOVA controlling for age, sex, house location and MSI.

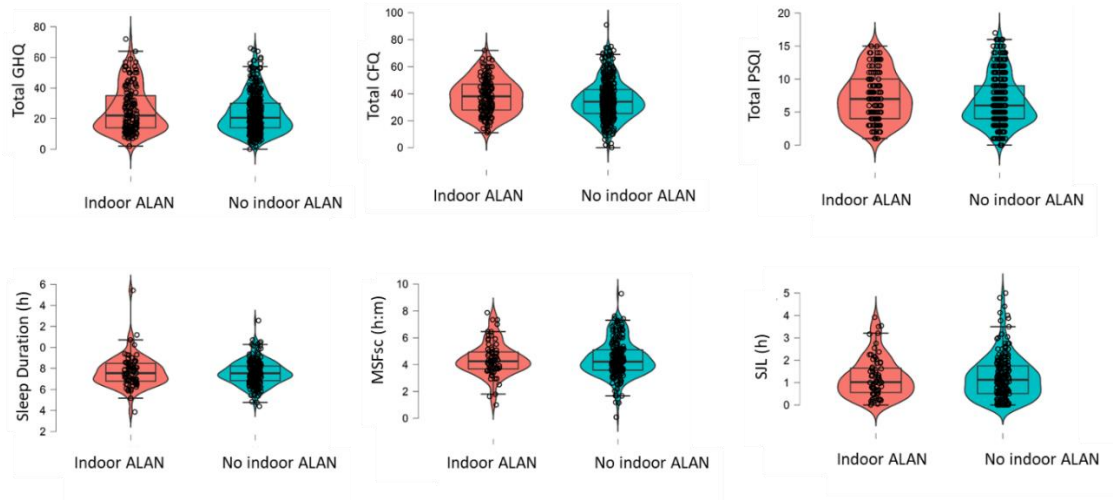
63.4% of participants perceived that exposure to external ALAN before sleep was disruptive to their sleep. Participants who reported this perception, when compared to those that did not in unadjusted analysis, had poorer sleep quality (median PSQI total 7, 95% CI [6; 7] vs 5, 95% CI [4; 6]; $p = .001$), more cognitive failures (median CFQ score 37, 95% CI [33; 40] vs 29.5 95%, CI [27; 32], $p = .001$, $r = 0.22$) and higher GHQ total scores (median total GHQ score 22, 95% CI [20; 25] vs. 17, 95% CI [14; 19], $p = .001$, $r = 0.16$). However, when controlling for age, sex, MSI and residence location, only the association between the perception of ALAN disrupting sleep and CFQ remained statistically significant ($p = .016$, partial eta squared = 0.018). Those endorsing a perception of external ALAN disrupting sleep also reported later MSFsc (median MSFsc 04:50, 95% CI [4.23; 4.67] vs 04:01, 95%

CI [3.79; 4.21]) and greater levels of social jetlag (median SJL of 1.25h 95% CI [1.00; 1.33] vs. .79, 95% CI [.58; 1.15]), although these differences were not shown to be statistically significant in ANCOVAs when age, sex, house location and MSI were controlled for. Examining the subscales of the GHQ, participants reporting perception of external ALAN disrupting sleep displayed statistically significant higher scores when controlling for age, sex, MSI and residence location on GHQ-B anxiety and insomnia (median GHQ-B total score 7, 95% CI [6; 8] vs 6, 95% CI [4; 6], p = .034, partial eta squared = 0.014), GHQ-C social dysfunction (median GHQ-C total score 7, 95% CI [7; 7] vs 7, 95% CI [7; 7], p = .011, partial eta squared = 0.020 and GHQ-D severe depression (median GHQ-D total score 1, 95% CI [1; 2] vs 0, 95% CI [0; 1], p = .035, partial eta squared = 0.013) compared to those who do not perceive ALAN exposure to be disruptive to sleep.

Regarding perception of indoor ALAN, 27.4% of respondents endorsed that indoor lighting (e.g. from the bathroom or landing) intruded into their sleeping environment (42% of these respondents also reported intrusion of external ALAN). Respondents reporting internal lighting intruding into the bedroom reported no statistically significant differences, after controlling for covariates, in GHQ score (p = .449), sleep quality (p = .073), cognitive failures (p = .069), MSFsc (p = .90), sleep duration (p = .193) or SJL (p = .298; Figure 3.3).

Figure 3.3

Box-and-violin plots of scores on the GHQ, CFQ, PSQI and sleep duration, MSFsc and SJL from the Munich Chronotype Questionnaire, split by perception of ALAN from within the residence assessed by the question "Do you usually have lights on outside your bedroom door which illuminate your bedroom (i.e. bathroom or landing).



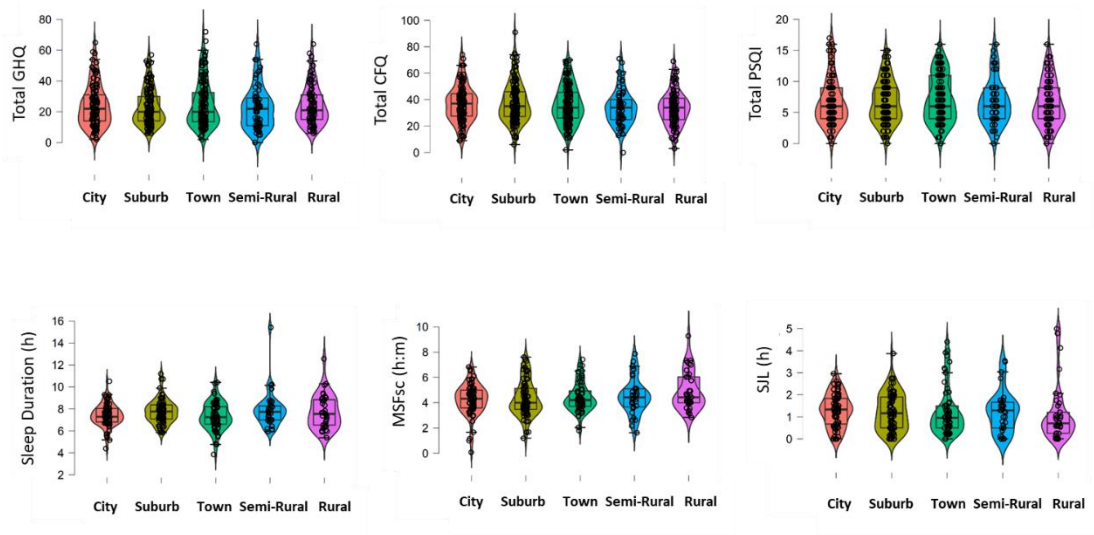
Note. There were no statistically significant differences by ANCOVA.

3.3.3 Residence location, illumination levels and sleep and psychological health indicators.

When GHQ, CFQ, PSQI, sleep duration, MSFsc and SJL were analysed against residence location type, there were no significant effects (Figure 3.4). MSI varied by location of the residence, $F(4,342) = 6.71$, $p < .001$, eta squared = 0.074, with city location associated with highest MSI and countryside with the lowest; Figure 3.5).

Figure 3.4

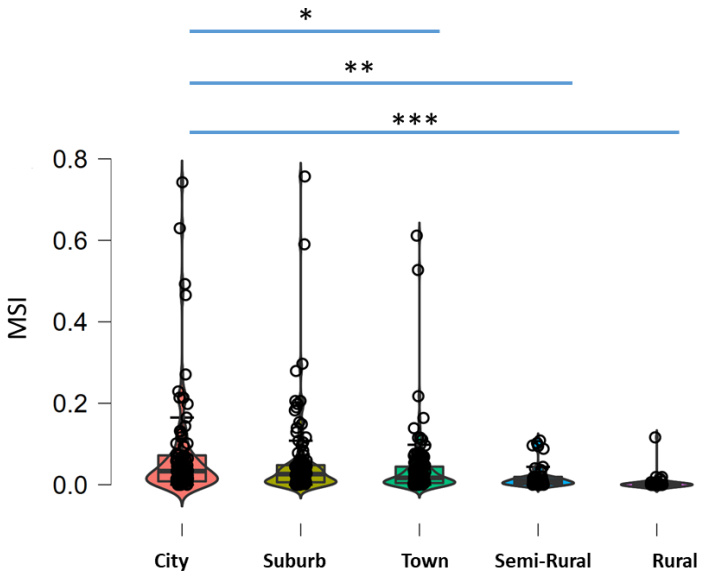
Box-and-violin plots of scores on the GHQ, CFQ, PSQI and sleep duration, MSFsc and SJL from the Munich Chronotype Questionnaire, split by the location of respondents' residences.



Note. There were no significant differences as assessed by Kruskal-Wallis tests.

Figure 3.5

Association of residence MSI with location categorisation of the residence.

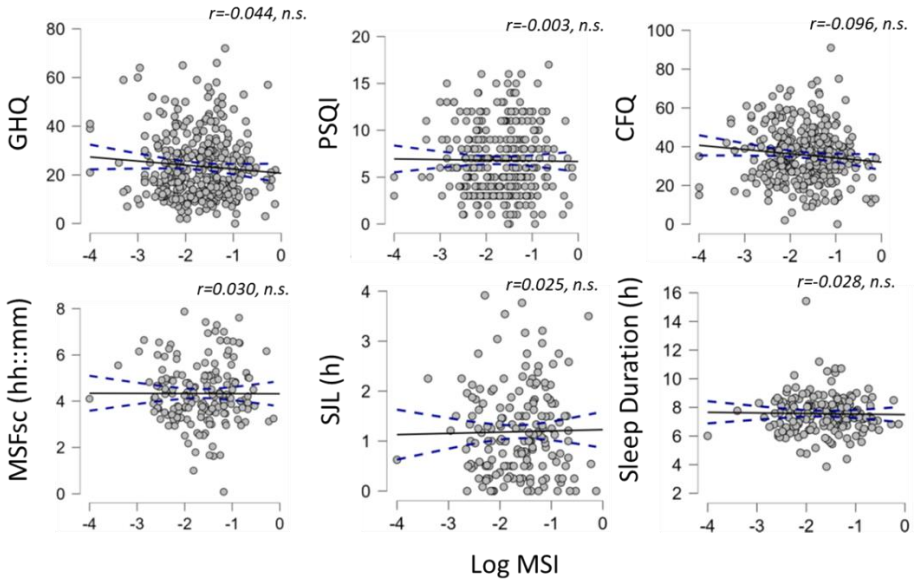


Note. *** indicates P<0.001, ** P<0.01, * P<0.05 by Kruskal-Wallis test followed by post-hoc Mann-Whitney U test.

When logMSI was correlated against GHQ, PSQI, CFQ, MSFsc, SJL and sleep duration, no significant associations were detected (Figure 3.6). Further, when MSI was used to assign participants into four groups based on MSI quartiles (quartile 1 median MSI = 0, quartile 2 median MSI = 0.006, quartile 3 median MSI = 0.027, quartile 4 median MSI = 0.147), PSQI, CFQ, GHQ, MSFsc, SJL and sleep duration did not significantly differ across these groups (Figure 3.7).

Figure 3.6

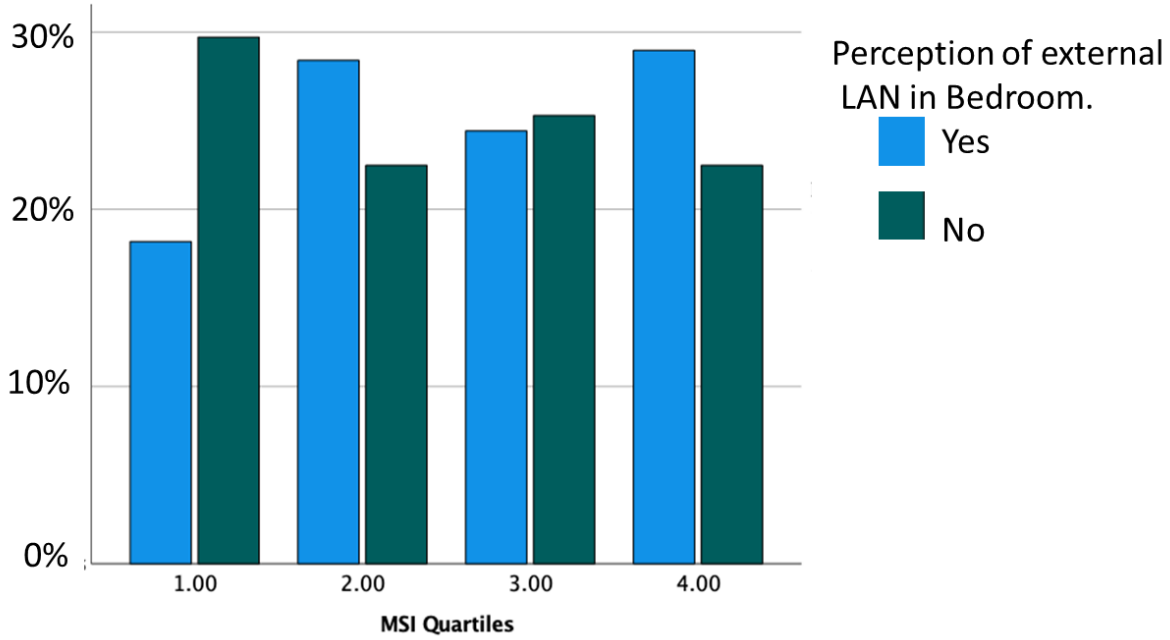
Scatterplots showing the lack of significant correlations between log transformed MSI scores and GHQ, PSQI, CFQ, MSFsc, SJL, and sleep duration.



Note. Correlation coefficients indicated are Spearman's rho.

Figure 3.7

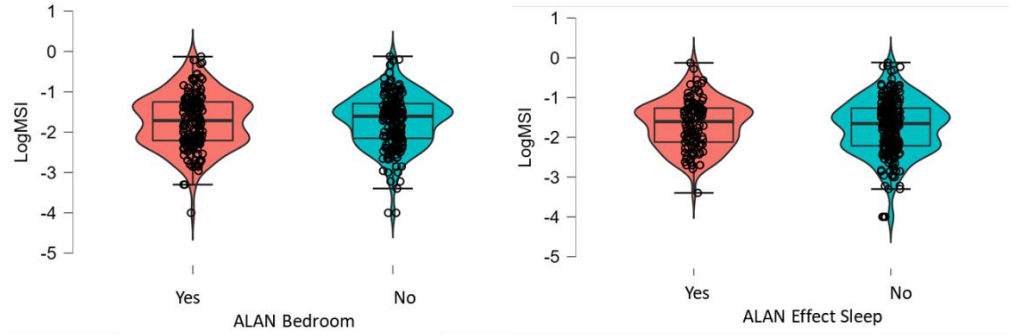
Association between residence MSI quartiles and responses to the question "Does artificial outside light (i.e. street lights, traffic lights, headlights etc.) enter the bedroom when you are sleeping?"



When Chi-Square tests for independence were carried out between the logMSI quartiles and self-report of external ALAN disrupting sleep, no significant associations were detected (p = .66), nor was there a statistically significant association between logMSI quartile with perception of ALAN entering the bedroom (p = .536). When assessed as a continuous variable, logMSI did not vary according to perception of external ALAN entering the bedroom (p = .911) or according to perception of external ALAN being disruptive to sleep (p = .827; see Figure 3.8).

Figure 3.8

Box and violin plots showing that logMSI at residence level does not vary between participants who report external ALAN entering the bedroom or that ALAN impacts on the quality of their sleep.



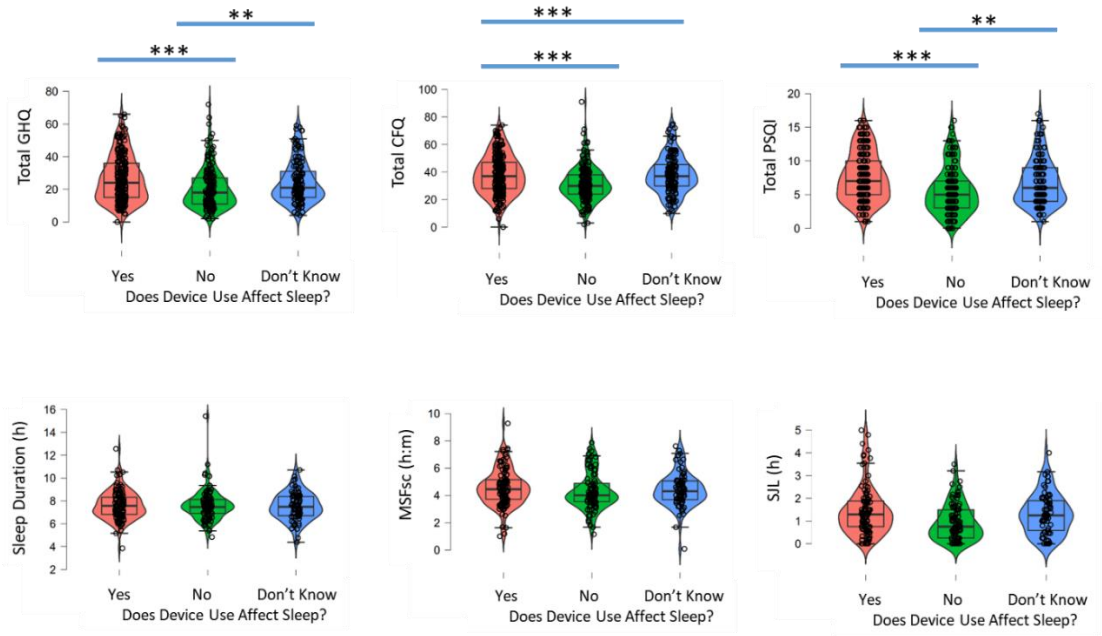
3.3.4 Perception of the effect of electronic devices affecting sleep

41% of participants perceived disruption to sleep by the use of electronic devices. Compared to those who reported no such effects (34% of respondents; the rest endorsing "Don't Know"), those who perceive negative sleep impacts of night-time device usage displayed poorer sleep quality after controlling for covariates of age, sex, house location and MSI (median PSQI scores 7, 95% CI [6; 8] vs 4.5, 95% CI [4; 5], $p < .001$, partial eta squared = 0.053), higher cognitive failures (median CFQ scores 35.5, 95% CI [32; 40] vs 28 95% CI [26; 31], $p < .001$, partial eta square=0.55), higher scores on the GHQ (median scores 23, 95% CI [20; 27] vs. 15, 95% CI [13; 19], $p = .016$, partial eta squared = 0.025). There were no effects of perceived sleep effects of device usage on MSFsc or sleep duration, and the unadjusted association with higher levels of social jetlag did not survive adjustment for covariates (median SJL 1.31h, 95% CI [1; 1.46] vs 0.73h, 95% CI [.58; 99] $p = .219$; Figure 3.9). 80.8% of respondents reported using electronic devices one hour before bed. Respondents who used light-emitting technology before sleep reported more cognitive failures (median score 35, 95% CI [31; 38] vs 27, 95% CI [24; 33]) and higher scores on the GHQ (median scores 22, 95% CI [18; 25] vs 16, 95% CI [13; 20]), but these differences were not statistically significant after adjustment for the covariates of age, sex, house location and MSI ($p = .488$ and $p = .272$ respectively). There were no unadjusted statistically significant differences between those that used devices in the run-up to sleep on PSQI (median scores 6 vs 5, $p =$

.091), sleep duration (7.54h vs 7.47h, p = .83), MSFsc (4.29 vs 4.10, p = .361) and SJL (1.13h vs 1h, p = .205). See Figure 3.9.

Figure 3.9

Box-and-violin plots of scores on the GHQ, CFQ, PSQI and sleep duration, MSFsc and SJL from the MCTQ, split by responses to the question about electronic device usage impact on sleep ("Do you feel that use of these devices affect your sleepiness/quality of sleep in a negative manner?")



Note. *** indicates p < .001, and ** p < .01 by ANCOVA, adjusting for the covariates sex, age, MSI and house location.

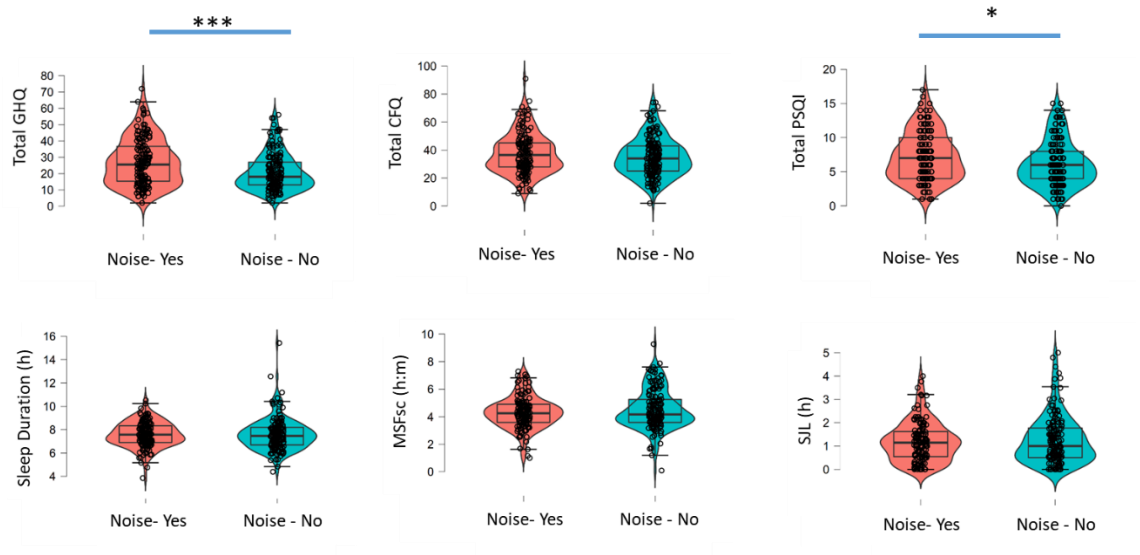
3.3.5 Perception of noise pollution in the bedroom.

46% of respondents reported being sensitive to external noise intruding into the bedroom. Those who endorsed noise sensitivity scores significantly higher on the GHQ (median score 25, 95% CI [22; 28] vs 18, 95% CI [16; 20], p < .001 by ANCOVA controlling for age, sex, and house location) and PSQI (median score 7, 95% CI [5; 7] vs 6, 95% CI [5; 6], p = .018 by ANCOVA; Figure 3.10). CFQ showed a difference on adjusted analysis (median score 38, 95% CI [33; 42] vs 31, 95% CI [29; 34]) but this did not persist after adjustment for covariates (p = .367). There were no significant differences, sleep duration, MSFsc or SJL (Figure 3.10). Similarly, respondents who endorsed that noise pollution negatively impacted on

sleep quality scored significantly higher on the GHQ (median 26, 95% CI [20; 28] vs 17, 95% CI [16; 20]; $p < .001$ by ANCOVA, and also scored higher on the PSQI (median 7 , 95% CI [6; 8] vs 5, 95% CI [5; 6]; $p < .001$ by ANCOVA. After adjusting for covariates, there was no significant difference on CFQ (median 36, 95% CI [30;40] vs 32, 95% CI [30;36], $p = .051$), SJL (median .96h, 95% CI [.79;1.25h] vs 1.25h, 95% CI [1.00;1.38h], $p = .572$), on sleep duration (median 7.48h vs 7.57h, $p = .914$) or on MSFsc (median 4.18 vs 4.36, $p = .126$). There was a statistically significant association between external outdoor ALAN entering the bedroom and whether individuals perceived noise pollution during the night ($x^2 = 9.51$, $p = .002$, phi = 0.183) with those reporting no noise during the night also typically reporting no outdoor LAN entering into their sleeping environment (57% of participants who perceive ALAN intruding into their bedroom also reported noise annoyance at night, whilst 61% of participants who did not report ALAN intrusion also did not report night-time noise annoyance. Results from supplementary analysis carried out on the survey can be found in appendix F.

Figure 3.10

Box-and-violin plots of scores on the GHQ, CFQ, PSQI and sleep duration, MSFsc and SJL from the MCTQ, split by perception of noise pollution in the bedroom assessed by the question "Does any particular noise annoy you at night?"



Note. *** indicates $p < .001$, * indicates $p < .05$, assessed by ANCOVA, adjusting for age, sex, and house location.

3.4 Discussion

The current results indicate that perceptions of ALAN in the bedroom associate with psychological distress, cognitive failures and subjective sleep quality. Interestingly, the perception of ALAN in the bedroom appears to be mostly independent of the level of external illuminance at the level of the individual residence. Subjective perception of ALAN affecting sleep quality is consistent with previous studies reporting that exposure to ALAN in the sleeping environment is associated with shallow sleep, more frequent arousals and sustained effects on EEG oscillations implicated in sleep depth and stability (Cho et al., 2013; 2016) and with poorer sleep and shorter sleep duration (Ohayon & Milesi, 2016). Obayashi et al. (2014a; 2014b) reported that in older adults, higher levels of photometer measured LAN exposure in the home setting was associated with a higher chance of self-reported insomnia, delayed sleep-onset latency and poorer objectively measured sleep quality. Our findings that perception of ALAN is associated with poorer psychological wellbeing is consistent with community-setting observational studies (Obayashi et al., 2013) and longitudinal studies wherein higher-level ALAN exposure was a predictor of subsequent diagnosis of depression (Obayashi et al., 2018). However, given the cross-sectional design of this study, causality in demonstrating that ALAN exposure affects sleep quality and psychological well-being cannot be provided. Furthermore, as luminance levels in the bedroom were not objectively assessed in the current study, it is not clear what the relationship between external ALAN, in-bedroom ALAN and subjective perceptions of the same are; future studies should address these relationships.

Previous studies have reported associations between ALAN (measured at the ecological level) and levels of subjective sleep quality. Such studies have reported that participants residing in areas with high external ALAN have delayed bedtime, shorter sleep duration, increased daytime sleepiness and increased daytime sleepiness (Koo et al., 2020; Patel et al., 2018). Such associations are reported to persist after controlling for population density and environmental noise (Xiao et al., 2020), although recently Helbich et al (2020) have reported that ALAN associations with depression are highly confounded by factors such as socioeconomic status and air pollution. Koo et al. (2020) report that outdoor light was associated with insomnia in middle age (47-58 years) and older (59-70 years) adults, but not in younger adults. Min and Min (2017) report that high outdoor ALAN was associated

with increased rates of depression and suicidality, and another recent study also reported an association of ALAN with increased risk of depression and specific anxieties in adolescents (Paksarian et al. 2020). A number of studies have found no association between photometer measured LAN inside the sleeping environment and satellite image data (Huss et al., 2019; Rea et al., 2011). In addition, Garcia-Saenz et al. (2018) reported no association between the subjective perception of indoor LAN and outdoor satellite measures of LAN.

The majority of extant studies to date have measured ecological ALAN by using satellite imaging with the main sources of satellite image data coming from the Defense Meteorological Satellite Program (DMSP) or the Day-Night Band instrument in the Visible Infrared Imaging Radiometer Suite (VIIRS) on the SUOMI satellite (Koo et al., 2016; Ohayon & Milesi, 2016; Paksarian et al., 2020; Xiao et al., 2020). There are three important limitations which affect the use of their data as true proxies for ALAN in such studies which should be borne in mind. Firstly, the data from these sensors provides a measure of the radiance in the general environment at relatively coarse resolution of approximately 2.7 km for DMSP and 750 m resolution for the VIIRS DNB instrument ; secondly, the timing of the satellite data varied between approximately 7pm local time for the DMSP series of satellites and approximately 3am local time for the SUOMI satellite; thirdly, both instruments are panchromatic detectors and hence the spectral wavelength of the light and the response of the instruments differ (Gatson et al., 2015). The identification of the spectral composition of light is of critical importance given that short wavelength visible light has more potent effects on the circadian system by reducing melatonin secretion, delaying the latency/onset of sleep and blood pressure regulation compared to long wavelength light (Brainard et al., 2011). The relatively coarse resolution of satellite data limits it as a proxy for an individual's exposure to light as it is based on the average light intensity of a region of interest as defined by the resolution available, typically with pixel sizes of the order of a square km (e.g. Paksarian et al (2020)) and may not reflect the true values, amounts, and patterns of ALAN at the levels of individual residences. Furthermore, a number of studies have found no associations between photometer measured ALAN inside the sleeping environment and satellite image data (Huss et al., 2019; Rea et al., 2011), and Garcia-Saenz et al. (2018) reported no association between the subjective perception of indoor LAN and outdoor satellite measures of LAN. To overcome an important

limitation of satellite data, that of the spatial resolution of ecological-level ALAN assessment, we estimated ALAN at the level of individual residences using a database of public lighting in Ireland. The use of MSI as a measure of light exposure is superior to the collection of light measurements in luminance by taking into account the amount of blue wavelength light most likely to exert biological effects and making an attempt to estimate the incidence of light in the vertical plane in comparison to a horizontal illuminance estimate provided by satellite imagery data, as evidence suggests that upward light remains weakly correlated to the light that may enter the house windows (Garcia-Saenz et al., 2018). As such, we believe that the approach taken in the current study is superior to approaches based on satellite data. However, future work should compare how exterior levels of ALAN corresponds to ALAN within the bedroom as directly experienced by participants during the sleep period.

An important feature of the current results is the apparent mismatch between the levels of ALAN estimated at the level of the individual residence and the subjective perception of either the presence of ALAN in the bedroom during sleep or the perceived disruptive effect of ALAN on sleep. Although our study cannot determine exactly why poor sleepers identify perceiving external light while residing in areas with equally comparable levels of outdoor ALAN to those that do not perceive it, a possible interpretation of these findings is that poor sleepers and/or those with higher levels of psychological distress are hyper-aware of the quality of their sleep and display sleep-related attentional bias to facets of their sleeping environment (Espie et al., 2006). Evidence of sleep related attentional bias (SAB) has come from subclinical populations of poor sleepers and insomniacs using cognitive paradigms (Harris et al., 2015). Tang et al. (2006) found that clock monitoring resulted in higher self-reported levels of worry and disturbed sleep along with delayed onset of sleep latency. Woods et al. (2009) used a modified version of the Posner paradigm whereby alarm clock displaying sleep times were presented and reported that participants with insomnia disorder were quicker in identifying valid trials compared to invalid trials, suggesting that the salience of the alarm clock cue resulted in participants with insomnia directing their attention to the sleep-salient stimulus (alarm clock). Therefore participants who have existing poor sleep quality display SAB towards ALAN entering the bedroom and as such are more likely to report the presence of bedroom ALAN and its disruptive impacts on sleep. Given the

high level of association between subjective sleep quality and psychological distress, SAB may also mediate the associations of ALAN perception and greater GHQ scores reported here. Another possible suggestion is that poor sleepers have a higher sensitivity towards light and as a result are more vulnerable to disruption to sleep. Philips, et al. (2019) found that while humans are sensitive to evening light there is significant inter-individual differences in sensitivity to light with some having greater than 50% reduction in melatonin suppression when exposed to light levels as low as 10 lux. However, the relative lack of associations between MSFsc and SJL and ALAN perceptions may argue against a biological effect of ALAN in impacting on the circadian system, and alternatively may favour an attentional or another psychological explanation. Another finding that may support the subjectivity of perception of bedroom ALAN is our finding that perception of light coming into the bedroom from indoor sources like bathrooms and landings was not associated with differences in GHQ scores and showed smaller associations with CFQ and PSQI scores than external ALAN did. This differential may be explained by locus of control; external sources of ALAN (e.g. streetlights, skyglow) cannot be "switched off" by participants, whilst sources such as bathroom lights could be if deemed to be of nuisance value. The current result therefore may generate testable hypotheses linking ASB and perceptions of bedroom ALAN, and future work may examine such.

Our findings indicate that 80% of participants indicated using light emitting technology before bed, whilst 46% of respondents perceived a negative effect of device usage on their sleep. Those that reported negative impacts of device use on sleep had higher scores on the PSQI, GHQ and CFQ than those who did not, a finding that may be consistent with previous reports that light emitting technology usage is associated with greater onset to sleep latency, poor sleep quality and quantity, and excessive daytime sleepiness (Gringras et al., 2015; Carter et al., 2016). Further, Christensen et al. (2016) report that smartphone screen time measured objectively was associated with poorer sleep quality, decreased sleep efficiency and longer sleep onset latency. An important caveat is that self-report of technology before sleep may not reflect actual device use; for example, Reddy Katapally and Chu (2019) found that individuals consistently underreport their screen time when compared to objective measures. With regards to the influence of external ALAN, those participants with poorer psychological and sleep health may display more

negative attributions towards device usage, and as such the reported association between perceived sleep impacts of device usage and both psychological and sleep health may not be in the direction of device use to psychological distress/poor sleep, but vice versa.

If there is an impact of ALAN on sleep and psychological health, the mechanism underlying such associations remains underexplored. Circadian rhythm dysfunction may be associated with poor psychological health as indicated by experimental studies of animal models (e.g. circadian clock gene knockouts) and some human studies (McClung et al., 2013). Night-time ALAN may supress the secretion of the pineal hormone melatonin, and multiple lines of evidence indicate that exposure to light at the biological night, even at low levels, can supress melatonin production resulting in delayed sleep onset, impaired sleep maintenance and poor quality of sleep (Scheer & Czeisler, 2005; Vartanian et al., 2015). In animal studies, ALAN which induces circadian disruption leads to changes in brain regions which contribute to depression and mood disruption (Fernandez et al., 2018; Germain & Kupfer, 2008). Therefore, it is biologically plausible that night-time ALAN exposure in humans could cause increased risk of psychological distress and/or poorer sleep; however, the current findings neither fully support nor reject that hypothesis, but rather highlight the importance of considering psychological factors that may influence perception of ALAN and its impacts.

There are a number of limitations to this study which impact on the generalisability of the findings. Firstly, this is a cross-sectional study that did not seek to assess causality in the explored relationships. Secondly, perceptions of ALAN in the sleeping environment were determined through self-reported measures and we did not measure ALAN in the bedroom environment in terms of illuminance levels, spectral composition and timing. As noted earlier it is not clear how ALAN assessed at the level of the residence corresponds to levels within the bedroom, and a number of studies have found no association between outdoor ALAN and levels of sleeping environment LAN (Huss et al., 2019; Rea et al., 2011). Further, the questionnaires employed only asked about ALAN in the bedroom and did not assess ALAN outside of the bedroom in the hours before bedtime which could impact on sleep. The method used to calculate ALAN at the level of the residence was based on the use public lighting databases, and we believe this approach has significant advantages over the use of satellite-based data in terms of spatial resolution, though

it may also under-estimate ALAN exposure due to other lighting sources, such private and commercial lighting, which were not included in the MSI calculations. The calculation of MSI is based upon the assumption that public lighting databases are accurate and complete and appropriate to the date of the survey, that public lighting is the sole contributor to the light level at the residence and that the calculated light level is appropriate for the nominal location, i.e. that an unobstructed view of the light source is present and that there is no loss of light output due to the aging of the lamp or cleanliness of the optics (the maintenance factor). The expected light output assumes a uniform emitter while, realistically, lantern photometry shows that light output varies with both azimuth and elevation angles.

3.4.1 Conclusions

Our study indicates that perception of ALAN in the bedroom and its impact on sleep is associated with more psychological distress, more daily cognitive difficulties and poorer subjective sleep quality, although it is not clear how these associations are linked (e.g. poorer sleep precipitating more distress and more cognitive failures). Further, the perception of bedroom ALAN and its impacts do not appear to be strongly correlated with objectively assessed ALAN at the level of the individual residence based on public street lighting, indicating that psychological factors that amplify the perceived nuisance value of ALAN may be important in mediating its impacts on psychological and sleep health. We believe that this work represents a first approach to estimate quantitatively the impact of external public lighting conditions on sleep inside the home. A recent Citizen Science study on public perception of ALAN in Ireland indicated that urban dwellers rated public lighting as the most important source of ALAN, although rural dwellers reported the main source being other residences in the locale (Coogan et al, 2020). As such, future work is warranted for the granular examination of ALAN at the level of the residence and the individual in order to better inform public lighting and health policy.

Chapter 4

Examining the effect of the perception of LAN on sleep-related attention bias using the Emotional Stroop Test

Abstract

Cognitive models of insomnia have suggested that an attention bias towards sleep-related information may play an important role in the development and maintenance of insomnia. Evidence of attention bias has also been observed in non-clinical poor sleepers. This study examined whether individuals who perceive LAN in their sleeping environment display an attention bias towards sleep-related word stimuli. A total of 177 participants aged between 18-70 (*M*=24; *SD*=8.98) completed the Emotional Stroop Test. Participants also completed the perception of LAN in the sleeping environment survey along with PSQI, MCTQ, DBAS and psychological health questionnaires. Our findings indicated that the perception of LAN in the sleeping environment is not associated with either differences in response time latencies to sleep related stimuli or attention bias scores derived from the EST. No association was observed between PSQI scores and attention bias indices. We discuss that the EST may not be a suitable measure to examine the effects of LAN perception on attention bias.

4.1 Introduction

Two cognitive models have been proposed to explain the mechanisms involved in the maintenance of chronic insomnia. These theories are Espie's (2002) attention-intention effort (AIE) pathway model and Harvey's (2002) model. A critical factor central to both theories is that individuals with insomnia are excessively pre-occupied with sleep which, in turn, biases attention towards sleep-related information leading to the reinforcing of both sleep and daytime impairment. This pre-sleep worry and preoccupation is a significant predictor of delayed latency to sleep onset and night-time arousal (Takano et al., 2014; Wicklow & Espie, 2000; Wuyts et al., 2012). Sleep disturbed/insomniac individuals monitor their internal (e.g. heart rate, temperature and fatigue) and external environment (e.g. noise, light, the time) for sleep-related threat cues to confirm their bias that they have had disturbed sleep and that next-day functioning is negatively impacted. Studies indicated that monitoring of a clock resulted in higher self-reported levels of worry and disturbed sleep along with delayed onset of sleep latency (Tang et al., 2006). These sleep-related cues became the focus of attention and attempts to control that automaticity of sleep lead to the inverse of intent such as disturbed sleep/insomnia. This process is referred to as sleep-related attentional bias (SAB), where individuals focus a disproportionate amount of their attention to sleep-related information in comparison to neutral information and is hypothesised to play a central role in the maintenance of insomnia (Espie, Broomfield, MacMahon, Macphee & Taylor, 2006).

The premise that SAB represents an important factor in the maintenance of insomnia comes from contemporary theories of psychopathology where attentional bias is seen as fundamental to the development and maintenance of anxiety, depression, PTSD and substance misuse (Mathews & MacLeod, 2005). It is suggested that those with clinical diagnoses selectively allocate processing resources to stimuli which are related to the key concerns of their disorder. For example, there is a bias towards anxiety-related stimuli in anxiety disorders (Mogg & Bradley, 2005) and a bias towards drug-related stimuli in those with substance-abuse disorders (Cox et al., 2006). Individuals who are clinically anxious and high anxious selectively attend to anxiety specific threatening information compared to neutral information. While attending to these potential threats can be protective, undue

biases towards threat are a fundamental component of anxiety disorders (Bar Haim et al., 2007). Cognitive models of psychopathology propose that high anxiety individuals are vigilant towards detecting threat (Koster, et al., 2004). The increased vigilance may lead to heightened sensitivity for negative information resulting in increased anxiety.

Attention bias has been measured objectively using computerised reaction time-based cognitive tasks where information processing speed is taken as a proxy for selective attention. (Emotional Stroop-, Dot-probe-, Flicker- and Posner Task (See Harris et al., 2015 for review)) These tasks involve the presentation of neutral cues (i.e. words/images) and cues relevant to the psychopathology of the individual (i.e. sleep-related word/images for insomnia disorder). The difference in reaction times between disorder specific cues and neutral cues indexes the magnitude of attentional bias with longer reaction times indicating biased attention (Harris et al., 2015). One commonly used task to measure selective attentional processes is the Emotional Stroop Task (EST) (Williams et al., 1996). In this task, neutral words and emotionally salient words (sleep-related) are presented on screen in one of four colours. As fast as possible, the participant presses the coloured response key which corresponds to the colour of the word. It is argued that in comparison to neutral words, response latencies are longer for sleep-related words which represents an index of Stroop interference. That is, the content of the sleep-related word, takes up more attentional resources resulting in performance being slower.

Lundh et al. (1997) were the first to investigate the presence of sleep-related attentional bias using the EST in individuals with persistent insomnia and good sleepers. Although people with insomnia displayed interference effect with longer response latencies for sleep-related stimuli in compared to physical threat words and control words, the effect, were also found in good sleepers. These results indicate that sleep-related stimuli may be salient to both groups. Taylor et al. (2003) provided evidence of the role that sleep-related attentional bias plays in insomnia by examining the role of sleep-related attentional bias in the development of persistent insomnia in recovering cancer patients who either had acute- (0-3 months) or persistent insomnia (12-18 months). Although Stroop interference was comparable in both groups on cancer-related words, only the persistent insomnia group displayed an interference effect for sleep-related words. These findings suggest that sleep-related attention bias may be related to the maintenance, rather than the

onset/development, of insomnia. Spiegelhalder et al. (2009) found that in a non-clinical population both sleepiness and sleeplessness resulted in sleep-related attentional bias as measured by the EST. A negative interaction between sleepiness and sleeplessness was found with attention bias scores being reduced when poor sleep quality was associated with high sleepiness and high sleep quality was associated with low sleepiness. These findings provided evidence that subclinical sleep disturbance is linked to attentional bias towards sleep related material. This evidence of attentional bias in a non-clinical sample suggests that attention bias is present in nonclinical sleep disturbance.

Although a number of studies have provided evidence of no differences between insomniacs/poor sleepers on measures of attentional bias (Lund et al., 1997; Lancee et al., 2017; Spieglhalder et al., 2010, Spieglhalder et al., 2016) Harris et al. (2015) in their meta-analysis of sleep-related attentional bias in good and poor sleepers/insomniacs found that the majority of studies provided statistical evidence for differential sleep-related attentional bias in insomnia, with medium to large effect sizes.

Jones et al. (2005) provided evidence of pre-occupation with sleep and sleep-related attentional bias by using a novel "flicker" paradigm for inducing change blindness to examine sleep-related attentional bias in good, moderate and poor sleepers. The task involves a brief presentation of a visual scene and is then briefly replaced with a similar image where either a sleep-related object or neutral object change has occurred. Their results found that poor sleepers were significantly faster in detecting changes to a sleep-related object compared to the neutral object, revealing sleep-related attentional bias in poor sleepers. In addition, good sleepers displayed attentional bias towards neutral stimuli. Marchetti et al. (2006) argued that that the results from Jones et al.'s (2009) study may be due to only one change to stimuli in both the neutral and sleep-related material. Marchertti et al. (2006) used a more complex flicker task along with a sample of insomniacs, good sleepers and a second control group comprising of delayed sleep phase syndrome (DSPS). They found that insomniacs detect sleep-related change significantly quicker than DSPS and good sleepers along with detecting change significantly quicker than neutral change.

Although, the aforementioned studies have provided evidence of sleep-related attention bias in sub-clinical poor sleepers and individuals with insomnia,

studies have also found no evidence of attentional bias in 'conceptual' control groups. Williams et al. (1996) proposed that while attentional bias may not be found in healthy controls when comparing clinical populations, such attentional bias may exist in experts who have expertise in the concepts. That is, the familiarity with, and usage of, the concept of sleep may be considered greater in those with primary insomnia along with sleep researchers than it would be with the rest of the population. Spiegelhalder et al. (2008) examined whether sleep experts who have high frequency concept usage towards sleep-related material have similar attention bias as primary insomniacs. Their results found that those with primary insomnia had significantly higher sleep-related attentional bias scores than the sleep expert group (but not with the good sleepers group). Spiegelhalder et al. (2008) argue that these results provide evidence that sleep-related attentional bias is due to differences in emotional, cognitive and procedural processing than due to differences in the frequency of concept usage. In order to ascertain that attention bias is specifically associated to insomnia and not simply to sleep disturbance, a control group of those with delayed sleep phase syndrome (DSPS) were examined. Similar to insomniacs, DSPS patients have difficulties in the initiation of sleep however, DSPS is a circadian rhythm disorder with no cognitive pathway to explain the development and maintenance of DSPS. MacMahon et al. (2006) used the dot probe test to examine group differences between good sleepers, primary insomniacs and those with DSPS Their results found that in comparison to good sleepers and those with DSPS, insomniacs displayed sleep-related attentional bias with shorter response latencies. No differences were found between good sleepers and the DSPS group. Similar results were found by Marchetti et al. (2006), who also utilised a DSPS group and found that in the ICB flicker task, those with insomnia detected sleep-related change significantly quicker than DSPS and GS and significantly faster than the change in sleep neutral images. The absence of a sleep-related attentional bias effect in the DSPS condition suggests that a sleep disturbance alone is insufficient to elicit a response in terms of biased information processing.

There have been mixed findings surrounding the therapeutic effects of attentional bias modification training in reducing attentional bias in insomniacs. Espie, Fleming and Paul (2008) found that CBT-I lead to the reduction of sleep-related attentional bias relative to those on the wait list control group. Attentional bias modification administered close to sleep was associated with improved sleep

quality and reduced pre-sleep arousal and sleep onset latency across a single sleep episode relative to nights where only a control task was employed (Milkins et al., 2016). However, the therapeutic effects of therapy in reducing sleep related attentional bias were not found in in a double-blind placebo controlled trial using attentional bias modification training for insomnia (Lancee et al., 2017). However, it is important to note that there was no differences between groups on attentional bias scores at baseline. Other studies have found no therapeutic effects of the reduction of attentional bias in insomniacs but have found decreases in sleep-related worry and reductions in objective sleep onset latency (Clarke et al., 2016; Milkins et al., 2016). These inconsistent findings call into question whether sleep-related attentional bias is a critical feature in insomnia.

While a number of studies support the hypothesis that poor sleepers/individuals with insomnia have an attentional bias for sleep-related stimuli relative to normal sleeping controls, the research on how sleep impacts on attention allocation to sleep stimuli is not as straightforward. The main cognitive models of insomnia propose that individuals with insomnia are pre-occupied with sleep and their bias towards sleep-related stimuli coupled with their attempt to control the automaticity of sleep results in insomnia (Espie, 2006; Harvey, 2002). However, Spiegelhalder et al. (2010) found opposite results whereby higher attentional bias scores as measured by the Visual Dot Probe Task were associated with increases in the polysomnography sleep parameters of total sleep duration, sleep efficiency and percentage of slow wave sleep and negatively correlated with the number of awakenings on the subsequent night of testing. These findings were unexpected and are in contrast to cognitive models of insomnia that higher levels of attentional bias and related arousal leads to disruptions of subsequent sleep. Spiegelhalder et al. (2010) proposes that a possible explanation for the positive association between sleep-related attentional bias and the objective sleep measures is due to insomniacs underlying craving for sleep. The disrupted sleep or non-restorative sleep in the previous nights may have accumulated into sleep debt resulting in increased attentional bias due to cravings for sleep. While this argument may be plausible in the same study, there was no association between attentional bias scores as measured by the EST and the objectively defined sleep parameters.

Barclay and Ellis (2013) propose that inconsistent findings pertaining to SAB and the EST may be due to the affective properties of the sleep-related word stimuli

used. Previous studies have used sleep-related word lists which are inherently negative in nature (e.g. nightmare, exhausted etc.). and such stimuli may be threatening to both individuals with and without sleep disturbance. Zhou, Zhao, Yang, Du, Yu & Shen (2018) found that in insomniac patients, the event-related potential amplitudes of the P1 and NI in the occipital area of the brain were larger for sleep-negative words compared to sleep positive and sleep unrelated words. This suggests early cognitive processing of sleep-negative stimuli. However, behaviourally, performance on the EST when compared separately found that sleep-positive words and sleep negative words effects were not similar, with a significant inference effect for sleep-positive words between insomniacs and good sleepers but similar effects not observed for sleep-negative words. However, Woods et al. (2013) reported longer gaze duration for both sleep positive and negative words compared to non-specific neutral words in insomniacs. These findings suggest that attention bias can occur towards stimuli which are sleep related in a positive and negative manner. Barclay and Ellis (2013) controlled for the affective valence of sleep-related words by using non-affective sleep words. Group differences were found with poor sleepers responding significantly slower to the affectively neutral sleep-related words compared to the non-specific threat words.

While cognitive paradigms have provided behavioural evidence of sleep-related attentional bias as a critical feature in the maintenance of insomnia, these tasks are reaction time tasks, which, in isolation only provide a 'snapshot'/indirect measure of attention. The addition of eye tracking provides objective measurement on the continuous visual attention of the eye throughout the duration of the stimulus presentation by generating data on eye movement, fixation direction and duration (Schütz, Braun, Gegenfurtner et al., 2011). It is typically expected that individuals will focus their eye gaze towards stimuli which specifically attract their attention (Jonides, 1981). The data provided is richer by producing more detail than the 'snapshots' which are provided by reaction times alone. In their seminal study providing a timeline of attention allocation for insomniacs, which was measured by eye tracking, Woods et al. (2013) found conflicting results. Such conflict related to the previous behaviour paradigms with insomniacs having longer delays in first fixation towards target words (sleep positive, sleep negative and neutral words) compared to pseudowords and took longer to discriminate between the target and pseudowords. However, insomniacs had shorter gaze duration towards target words

compared to pseudowords. The no significant difference within the insomnia group on target words on gaze and discrimination suggests no sleep-related attentional bias but instead neurocognitive deficit.

Further support that sleep-related attentional bias is present in insomniacs has been provided by research using neuroimaging. Baglioni et al. (2014) found evidence of emotional bias to sleep-related material, demonstrated by higher amygdala responses to insomnia-related material compared to good sleepers. In addition to this, when images were re-presented a mixed pattern of amygdala responses occurred in the insomniac group whereas, habituation of amygdala responses occurred in good sleepers. It is unclear what drives sleep-related attentional bias, with some studies arguing that poor sleepers may view sleep-related stimuli in terms of craving sleep (Espie et al., 2006; Spiegelhalder et al., 2009, Spiegelhalder et al., 2010) whereas some argue that sleep cues may represent a thread to poor sleepers, thus invoking anxiety-like responses (Baglioni et al. 2010; Harvey, 2002; Sagaspe et al. 2006; Spiegelhalder et al., 2009). Threat and craving have distinguishable activation patterns in the brain. However, a study using fMRI found no differences in brain reactivity between individuals with insomnia and controls when presented with sleep-related words (Spiegelhalder et al., 2016). These findings leave unanswered what drives sleep-related attentional bias.

Although the cognitive paradigms discussed above have provided evidence that sleep-related attentional bias is implicated in the development and maintenance of insomnia, these paradigms, lack ecological validity as they are derived from highly controlled visual displays/scenes which are abstracted from the experience from insomnia. Ecological valid images of sleep related stimuli such as alarm clocks and bedrooms may provide a more proximal stimulus which is more closely related to the insomniac experience compared to the words and images presented on the other paradigms. Woods et al. (2009) used a modified version of the Posner paradigm whereby the ecologically valid stimulus of an alarm clock displaying sleep times were presented. Woods et al. (2009) found that insomniacs were quicker in identifying a valid trial compared to invalid trials; that is to say, the salience of the alarm clock cue resulted in insomniacs holding their attention, which allowed them to process the target faster when it appears in the position vacated by the alarm clock. This results in a delaying effect on invalid trials as it slows the movement of attention towards the target on the opposite side of the screen.

Using eye-tracking, research has indicated that individuals with insomnia monitor tiredness in their social environment by spending more time fixating on and observing tired faces relative to neutral faces. In particular, insomniacs spend more time observing the eye region of the tired faces than the eye region of the neutral faces (Akram et al., 2017; Akram, Robson & Ypsilanti, 2018a). However, insomniacs display differences in both vigilance and disengagement compared to normal sleepers by taking longer to focus attention towards tired faces and taking longer to shift attention away from tired faces (Akram et al. 2018b). Similar results were found by Beattie et al. (2017) who found that when insomniacs free-viewed bedrooms, once they became fixated on the bed they maintain attention for longer on the bed. However, insomniacs do not differ from normal sleepers in their initial exploration of the bedroom. This provides further evidence that insomnia is characterised by sleep-related attentional bias in the social environment.

At the core of the cognitive theories of insomnia, it is proposed that individuals become so pre-occupied with sleep and the confirmation of their wakefulness that they become highly vigilant of internal and external environment cues which are interpreted as sleep-related threats, which in turn drives pre-sleep worry and arousal leading to further wakefulness (Espie, 2002; Harvey, 2002). This pre-sleep worry is associated with longer latency to sleep onset (Weise et al., 2013) with a strong positive association between pre-sleep worry and increased levels of insomnia-like symptoms being found (Lancee et al., 2017; O'Kearney & Pech, 2014). However, to date, it is unclear what the specific sources in the sleeping environment are that may drive pre-sleep worry. Experimental studies have provided evidence that increased self-focus and external monitoring of specific stimuli (alarm clock) in the bedroom environment can have deleterious effects by increasing pre-sleep worry, delaying the onset of sleep and on the misperceiving of sleep and daytime function (Samiet & Harvey, 2006; Tang et al., 2007) along with driving an attention bias (Woods et al., 2009). However, it is unclear whether specific sources such as LAN, which are routinely found in the sleeping environment and may disrupt sleep (Cain et al., 2020; Cajochen et al., 2011; Obayashi et al., 2014), are attended to in the sleeping environment and is their perception associated with both sleep disturbance and dysfunctional beliefs and attitudes towards sleep. Additionally, while previous studies have examined attentional bias using ecologically valid environmental cues such as free-viewing of the bedroom (Beattie et al., 2017) and

attentional focus on tired faces (Akram et al., 2017, Akram et al., 2018, & Akram et al., 2018b), these studies fail to investigate how environmental cues in the sleeping environment such as the light are taking up attentional resources and processing light as a sleep-related threatening cue, resulting in heightening the anxious state and leading in failure to inhibit wakefulness. Individuals that perceive external environmental cues as threatening towards sleep may have associated the pairing of negative thoughts of light (unconditioned stimulus), its presence in the bedroom at night (conditioned stimulus) leading to negative affective responses towards the sleeping environment (conditioned response). Korany et al. (2017) found that insomniacs have a stronger negative affective response towards the sleeping environment compared to good sleepers. This study will directly assess whether the specific perception of LAN is associated with an attention bias to sleep-related stimuli.

The aim of the current study is to investigate whether the subjective experience of perceiving LAN in the sleeping environment is associated with higher attentional bias scores as measured by the EST. This is guided by the results from chapter 3 which indicate that subjective appraisal of LAN and it deleterious impacts on sleep do not correspond well with objective measurements of LAN. The EST will be employed given that several studies have found behavioural effects of sleep related attentional bias in both sub-clinical and those with insomnia. The use of the EST has led to inconsistent findings about whether attention bias is present in those with sleep disturbance. It has been argued that one possible reason for this inconsistency is that some of the sleep stimuli could have negative connotations which could be perceived as threatening to both poor sleepers and good sleepers (Barclay & Ellis, 2013). For example, the sleep words employed in some EST studies have included the words “tired, lethargic, fatigue, exhausted” (Spiegelhalder et al., 2008; 2009; 2018) These words may be perceived as threatening and may blur the presence of stroop interference for sleep-related words between good and poor sleepers. For this reason, this study will employ the same word set as Barclay & Ellis (2013), to examine whether those that perceive LAN in their sleeping environment or have poorer sleep quality, allocate preferential attention to sleep stimuli which are affectively neutral compared to neutral and non-specific threat words.

This study will utilise a non-clinical sample as there is no consistent pattern linking level of clinical insomnia severity to attention bias magnitude. Harris et al.,

(2015) in their meta-analysis report that effect size differences are not any greater between those who are clinically assessed and diagnosed with insomnia relative to those who are categorised as of poor sleepers (Harris et al., 2015). The symptoms and processes which are involved in the aetiology of insomnia exist along a continuum which varies between clinical and subclinical in regards to severity and intensity (Morin et al., 2009). The hypothesis of the current study is that:

1. The perception of LAN from specific sources in the sleeping environment is associated with increased response latencies to sleep stimuli and an attention bias towards sleep related information.
2. Dysfunctional beliefs about sleep and sleep quality and associated with sleep related attention bias.

4.2 Method

4.2.1 Participants

177 participants (70.6% female) with a mean age of 25.24 (*SD* = 8.98, range 18-70) and residing in Ireland partook in the study. Data was collected between October 2018 and January 2020. Ethical approval was obtained from Maynooth University Research Ethics Committee. Recruitment was through a mixture of snowball and convenience sampling via flyers, emails, and personal contacts. The inclusion criteria were that participants were aged 18 years and above and were not shift workers. All participants gave their electronic informed consent before participating in this study and were informed that all data collected would be stored anonymously. Participation was voluntary and unpaid, however, some participants received course credit for taking part in the study.

4.2.2. Materials

Participants completed the same demographic and subjective measures as were in completed in chapter 3 (demographic questionnaire, light habits/exposure, and attitude to light questionnaire, CFQ, PSQI, MCTQ and GHQ). In the current sample each of the subjective scales displayed good to excellent internal consistency as indicated by the Cronbach alpha coefficient (CFQ = .92; PSQI = .70; GHQ = .93). In this study an increased cut-off score was employed for the PSQI. Although scores greater than 5 on the PSQI indicate poor quality sleepers the current study used a cut off range greater than 7 to indicate poor quality sleeper. This cut-off point has been argued to appropriately distinguish healthy from problematic sleepers. The increased range provides superior range of sensitivity and specificity for detecting sleep problems among healthy populations (Backhaus et al., 2002; Manzar et al., 2015; Salahuddin et al., 2017; Sun et al., 2014; Zhao et al., 2017).

In addition to these measures, participants completed the brief version of the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16; see appendix G). This 16 item self-report measure is used identify and asses various sleep/insomnia-related cognitions (e.g., beliefs, attitudes, expectations, appraisals, attributions) and is sub-grouped into 4 subscales: (a) perceived consequences of insomnia; (b) worry/helplessness about insomnia; (c) sleep expectations and (d) medication. Each

question has a 10-point Likert response with responses ranging from 0 (strongly disagree) to 10 (strongly agree). There is no absolute right or wrong answer for a single item. Rather the degree to which a particular item is endorsed by participant is a reflection of the nature of the dysfunction. Responses for each item are within a subscale are averaged, with higher scores indicating greater dysfunctional beliefs about sleep. The DBAS-16 scale has high internal consistency (Cronbach alpha 0.77 for clinical and 0.79 for research samples; Morin et al., 2007). The internal reliability based on the current sample was .83 indicating excellent consistency.

Participants also completed the computerised EST to measure attentional bias. The task was administered using Inquisit software (https://www.millisecond.com/). In each trial, either a sleep-related word stimulus, neutral word stimulus or non-specific threatening word stimulus was individually presented in the center of a computer screen, in one of possible four colours (red, green, blue, and yellow). Participants were instructed to place their index and middle fingers over the keys "D" "F" "J" "K" and to press the correctly coloured key on the computer keyboard corresponding to the colour of the word in screen as quickly as possible. A fixation cross was presented for 500 ms prior to the word stimuli and between each word. The words were displayed on screen until a response was made (see Figure 4.1). Participants were first presented with practice trial which comprised of 10 neutral words. Participants then completed the experimental task which comprised of 60 trials. The word list (see Table 4.1) contained 20 sleep related words, 20 neutral words and 20 non-specific threat words. The sleep-related words were neutral in nature having no negative connotation to sleep. These non-specific threat words had been used in a previous study controlling for the affective valence of sleep-related words (Barclay & Ellis, 2013). Each of the word types in the experimental trials were matched in terms of word length and the number of syllables. In the experimental trial, the word stimuli were randomly presented with constraints. These were that the same word or colour could not appear twice in a row, that the same word would not appear twice in the same block and sleep related words would not appear together in the same block. Participants were asked to respond as quickly and as accurately as possible. All words along with their individual reaction time (measured in milliseconds) were automatically downloaded to an excel file.

Figure 4.1

Example of a sleep-related word printed in colour and presented on a computer screen. The participant is asked to match the colour of the word with to a colour coded-response box as quickly as possible, while ignoring the word's meaning.

Table 4.1

Word type and stimuli used in the emotional stroop task (EST).

Sleep-Related Words	Neutral Words	Non-specific threat words
Sleep	Plate	Cruel
Dream	Chord	Dread
Bed	Cat	Ill
Night	Crown	Shame
Snooze	Change	Grieve
Pillow	Number	Scared
Duvet	Towel	Upset
Blanket	Between	Useless
Mattress	Keyboard	Helpless
Quilt	Grasp	Shock
Pyjamas	Through	Jealousy
Bedtime	Camping	Hateful
Slumber	Jumping	Worried
Nap	Now	Bad
Doze	Once	Glum
Nightgown	Something	Disgraced
Shuteye	Fishing	Hostile
Alarm Clock	Housework	Vulnerable
Awake	Table	Panic
Asleep	Travel	Rotten

The time taken to pair the colour of the word to the appropriate colour was calculated for each trial. These individual trial times were then aggregated to their specific trial condition where the average mean response time (MRT) was calculated. This resulted in 3 MRT conditions (MRT Sleep, MRT Neutral and MRT Non-Specific Threatening). Each respective mean response time acted as the response latency for their respective trial condition. Words which had longer response latencies are interpreted as being perceived as threatening. Two attention bias scores were also calculated (Sleep Attention Bias and Negative Attention Bias). An outline for how the attention bias score for sleep attention bias was calculated and its interpretation is presented in Table 4.2.

Table 4.2

Outline of the calculation and interpretation of the attention bias score for sleep attention bias.

Attention Index	**Attention Bias (AB)**	**Response Latency**
Formula	AB = Sleep MRT – Neutral MRT	MRT Trial Type
Interpretation of positive scores	+AB = Vigilance for sleep stimuli (Slower MRT for threatening stimuli compared to neutral stimuli)	Higher MRT = (Slower responding to sleep stimuli indicating attention bias)
Interpretation of negative scores	-AB = Avoidance of sleep stimuli (Faster MRT for threatening stimuli compared to neutral stimuli)	

4.2.3 Procedure

Recruitment of participants for this study was through a mixture of snowball and convenience sampling via flyers, emails, and personal contacts. Some participants were recruited through the Department of Psychology Participant pool. Participants collected from the pool received course credit for taking part in the study. The computerised Emotional Stroop Task (EST) was carried out in a well-lit, quiet experimental cubicle in the Department of Psychology at Maynooth University between 12.00 and 16.00 hours. This time was chosen as it is the most neutral time for all chronotypes and reduces performances being impacted by time of testing (Facer-Childs et al., 2018). Before starting the study, participants were required to give their informed consent complete the demographic and subjective measures which was hosted on survey hosting platform Qualtrics. After this was completed participants were given standardised written instructions on the EST as well as verbal instructions. Once the participant indicated that they understood the instructions they completed the 20 practice trials to familiarise themselves with the task. After the practice trial was completed, written instructions appeared on screen again. Once the participants read these instructions they pressed the spacebar and the experimental task commenced. The researcher stayed outside of the research cubicle for the duration of the study. Once the participant had completed the study, they were debriefed on the aims and purpose of the research study.

4.2.4 Data Analysis

This design of this study is quasi-experimental. All time-based variables from the MCTQ (MSFsc, average sleep duration and SJL) were decimalised (i.e, 8:30 became 8.50, 30 min became .50). As guided by Spiegelhalder et al. (2018) data from the EST was excluded if there were errors in trials or omissions. Response times less than 250ms were excluded. Response times that deviated from the participants mean score by more than 3 standard deviations were eliminated as outliers. This exclusion resulted in 6 (3.82%) participants being removed from analyses. In order to account for missing data and to minimise removal of whole participant data, pairwise deletion was employed when carrying out inferential testing. Independent variables were the perception of LAN and the perceived disruption of LAN. The dependent variables were the attention bias indices derived

from the EST. Welch's t-test were performed to examine between groups differences on the perception of LAN being disruptive to sleep and the perception of LAN in the sleeping environment on attention bias indices. Welch's t-test was employed due to the sample sizes being unequal between groups. The Welch's t-test also gives the same result when samples variances are equal. Eta squared was used to determine effect sizes. The guidelines for interpreting eta squared values were .01 = small effect, .06 = moderate effect and .14 large effect. Pearson product moment correlations were employed to examine the association between continuous subjective measures (PSQI, DBAS, CHQ, GHQ) and attention bias indices. One-way repeated measures ANOVA was employed to examine differences in response latencies across the three stimuli groups. Post hoc comparisons were employed using Bonferroni test.

The study sample size was estimated using a-priori power calculations based on it being important to detect effect sizes of moderate size (d=.5). These calculations indicated a required study sample of approximately 210 would be required to detect differences of a likely-to-be-meaningful magnitude. All statistical analysis was conducted using IBM SPSS (V25, IBM Corporation) and graphs were conducted either on SPSS or Jasp (V 0.9.1.0, https://jasp-stats.org/).

4.3 Results

The mean, median and standard deviation scores for the subjective data scales are presented in Table 4.3.

Table 4.3

Mean, median and standard deviation for scores on the psychometric instruments used to access psychological health, subjective sleep quality, sleep timing, attitudes towards sleep and psychological health.

	Mean	95%CI	Median	SD	Range	N%
Age (yrs)	25.23	[23.90-26.58]	21	8.99	18-70	
Psychological Health						
Total CFQ	44.79	[42.44-47.14]	45	15.82	2-95	
Total Score GHQ	29.61	[27.40-31.82]	28	14.75	0-84	
Somatic Symptoms	7.29	[6.63-7.96]	7	4.44	0-21	
Anxiety & Depression	9.17	[8.41-9.93]	9	5.06	0-21	
Social Dysfunction	9.16	[8.56-9.77]	9	4.02	0-21	
Severe Depression	3.96	[3.24-4.67]	2	4.81	0-21	
Sleep Parameters						
Global PSQI	7.71	[6.67-7.68]	6	3.36	1-17	
PSQI >7						85(48.9%)
Circadian Parameters						
Mean sleep duration (h)	8.05	[7.86-8.23]	8	1.25	2.79-11.83	
MSF*sc* (H.MM)	4.99	[4.78-5.20]	4.91	1.38	1.01-9.16	
Social Jetlag (h.mm)	1.15	[1.03-1.27]	1.16	.76	.00-3.62	
Sleep Attitudes						
Global DBAS	4.24	[4.01-4.47]	4.18	1.51	.69-8.44	
Consequences of Insomnia	5.07	[4.79-5.36]	5.20	1.90	.4-9.4	
Worry/Helplessness about sleep	3.75	[3.44-4.06]	3.50	2.06	.00-9.33	
Expectations for sleep	6.34	[5.99-6.68]	6.50	2.33	.5-10	
Medication	2.43	[2.18-2.67]	2.33	1.64	0-8.33	

Note. n = 177

4.3.1 Examination of the impact of sleep quality on each of the subjective measures

Welch t-tests were carried out to compare sleep quality scores, circadian parameter scores, attitudes towards sleep and physical/psychological health scores for good and poor quality sleepers. Sleep quality type was categorised by scores obtained on the PSQI. There was no effect of sleep quality type on chronotype (p =.136). With regards to everyday cognitive functioning poor sleepers reported higher level of cognitive errors (M = 50.33, SD = 16.34) compared to good sleepers (M = 39.45, SD =13.58), $F(1, 163.50) = 22.70$, p <.001. However, the effect size was small (.12) as indicated by the eta squared. Poorer sleepers also reported poorer psychological health (M = 36.17, SD = 14.57) compared to good sleepers (M = 23.42, SD = 11.82) $F(1, 149.82) = 39.48$, p <.001 with a large effect size (.19) as indicated by eta squared. See Table 4.4.

Table 4.4

Descriptives and inferential analysis examining sleep quality type according to subjective measures

	Good N(89)	Poor N(85)		
Scale	Mean(SD)	Mean(SD)	*F*	*p*
SD week (h.mm)	8.28(1.02)	7.80(1.43)	6.38	**.013**
MSFsc (hh.mm)	4.84(1.32)	5.15(1.43)	2.24	.136
Social Jetlag (h.mm)	1.01(.68)	1.29(.81)	5.78	**.017**
DBAS	3.63(1.31)	4.90(1.44)	36.92	**<.001**
CFQ	39.45(13.58)	50.33(16.34)	22.70	**<.001**
GHQ	23.42(11.82)	36.17(14.57)	39.48	**<.001**

Further analysis was carried out to specifically examine the effect of sleep quality type on each of the subscales from the GHQ. Between groups descriptives and their associated inferential analyses can be seen in Table 4.5. From this table it can be observed that poor sleepers reported higher levels of somatic symptoms and anxiety and depression compared to good sleepers with the magnitude in mean differences being large as indicated by the eta squared. Additionally, poor sleepers reported higher levels of social dysfunction and severe depression compared to good

sleepers with the magnitude of the mean differences being of medium effect size as indicated by the partial eta squared.

Table 4.5

Descriptives and inferential analysis examining sleep quality type according to the subscales of the GHQ.

	Good N(84)	Poor N(89)			
GHQ Sub-scales	Mean(SD)	Mean(SD)	*F*	*P*	*Eta Squared*
Somatic Symptoms	5.60(3.68)	9.11(4.44)	32.06	**<.001**	.16
Anxiety& Depression	7.09(4.71)	11.32(4.27)	38.12	**<.001**	.18
Social Dysfunction	8.17(3.65)	10.35(4.07)	13.52	**<.001**	.07
Severe Depression	2.57(3.77)	5.38(5.33)	15.81	**<.001**	.08

Correlational analysis was carried out to examine the relationship between each of subjective measures using the continuous score. With regards to global PSQI scores, a medium negative association was observed with average weekly sleep duration, r = -.334, n = 172, p <.001 with poorer sleep associated with lower average weekly sleep duration. A small positive association was observed between global PSQI scores and social jetlag, r = .176, n = 172, p = .021 with poorer sleep associated with increased levels of social jetlag. A large positive association was observed between PSQI scores and scores on the DBAS, r = .518, n = 174, p < .001 with poorer sleep quality associated with higher levels of poor attitudes and beliefs towards sleep. A moderate positive association was observed between PSQI scores and CFQ scores, r = .354, n = 174, p < .001. A large positive association was observed between PSQI scores and GHQ scores r = .514, n = 172, p < .001. In both cases higher PSQI scores were associated with higher scores on the GHQ and CFQ.

A moderate positive association was observed between chronotype and absolute social jetlag, r = .332, n = 171, p < .001 with later MSF*sc* associated with higher amounts of social jetlag. Additionally, a small positive association was observed between social jetlag and scores on the CFQ, r = .223, n =172, p = .003,

with higher levels of social jetlag associated with increased frequency of cognitive errors. A small negative association was observed between average weekly duration and DBAS scores, r = -.160, n = 174, p = .036 with lower average weekly sleep duration associated with increased poor attitudes and beliefs towards sleep. Higher scores on the DBAS were positively associated with both the GHQ (r = .384, n = 176, p <.001) and CFQ (r = .548, n = 174, p <.001). All correlation coefficients can be observed in Table 4.6.

Table 4.6

Associations between sleep and health variables.

Scale	1	2	3	4	5	6	7
1. Global PSQI	-						
2. MSFsc	.127	-					
3. SD Week	-.344**	-.078	-				
4. Social Jetlag	.176*	.332**	-.086	-			
5. DBAS	.518**	.062	-.160*	.107	-		
6. CFQ	.354**	.100	.000	.223**	.384**	-	
7. GHQ	.514**	.124	-.117	.131	.548**	.535**	-

Note. * = p < .05, ** = p < .01, *** = p < .001.

4.3.2 Examination of attitudes towards LAN exposure and sleep disruption to sleep

72.2% (n = 108) of the sample perceive LAN exposure to be disruptive to sleep. Welch t-test analysis found that those that perceive LAN to be disruptive to sleep report poor sleeper quality (M = 7.65, SD = 3.31) compared to those that do not perceive LAN to be disruptive (M = 5.89, SD = 3.20), F(1, 84.84) = 10.192, p = .002. However, the magnitude in the differences was small (.05) as indicated by eta squared. No differences were found between those that perceive LAN to be disruptive to sleep compared to those that do not on chronotype (p = .183), average sleep duration (p = .615) or social jetlag (p = .842). However, between group differences were found towards attitudes and beliefs towards sleep. Those that perceived LAN to be disruptive to sleep reported higher scores on the DBAS (M = 3.58, SD = 1.31) compared to those that did not (M = 4.49, SD = 1.51), F(1, 99.63) =

15.81, p <.001, with a moderate effect size (.07) as indicated by eta squared. Examination of the specific subscales from the DBAS found that those that perceived LAN to be disruptive to sleep quality reported perceived that the consequences of insomnia were higher (M = 5.36, SD = 1.86) compared to those that did not perceive impacts (M = 4.33, SD = 1.78), F(1, 91.29) = 11.59, p = .001 with a small effect size (.06) as indicated by eta squared. Similarly, those reporting LAN to be disruptive reported higher worry/helplessness about sleep (M = 4.11, SD = 2.07) compared to those that did not (M = 2.81, SD = 1.71), F(1, 104.98) = 18.04, p <.001 with a moderate effect size (.08) difference in the means observed as indicated by eta squared. Perception of LAN reported higher use of sleep medication (M = 2.59, SD = 1.75) compared to that that did not (M = 2.01, SD = 1.26), F(1, 120.14) = 5.91, p = .016. However, a small effect size (.02) was observed. No significant between group differences were observed for expectations for sleep (p = .923).

24.40% (n = 43) of respondents reported that LAN was disruptive after falling asleep. Those that perceived LAN to be disruptive after falling asleep reported higher levels of social jetlag (M = 1.42, SD = .90) compared to those that did not (M = 1.07, SD = .68), F(1, 56.850) = 5.51, p = .002 with a small (.04) effect size observed as indicated by eta squared. Additionally, those that reported LAN to be disruptive after falling asleep reported poorer attitudes towards sleep (M = 4.86, SD = 1.43) compared to those that did not (M = 4.04, SD = 1.48), F(1, 73.07) = 10.51, p = .022 with a medium effect size observed (.06). Further analysis of the subscales of the DBAS found that those that perceived LAN to be disruptive after falling asleep reported higher worries about the consequences of insomnia (M = 5.86, SD = 1.75) compared to those that did not (M = 4.82, SD = 1.88), F(1, 72.72) = 1.91, p = .001 with a small effect size as indicated by eta squared. Similarly, statistically higher levels of worry/helplessness about sleep were found in those that perceive LAN to be disruptive to during sleep (M = 4.54, SD = 2.09) compared to those that do not (M = 3.49, SD = 1.99), F(1, 72.72) = 1.91, p = .005. However, the effect size was small (.04) as indicated by eta squared. No between group differences were found for expectations for sleep (p = .522) or medication (p = .171). However, no differences were observed between those that perceive that LAN interferes with their sleep quality after falling asleep and those that do not on sleep quality (p = .071), average weekly sleep duration or (p = .660), chronotype (p = .812).

35.8% of respondents in the sample reported that external LAN entered the sleeping environment. However, no between group differences were observed between those that perceived external LAN entering compared to those that do not on sleep quality (p = .082), chronotype, (p = .555), average weekly sleep duration (p = .623), social jetlag (p = .871) or on attitudes and beliefs towards sleep (p = .764).

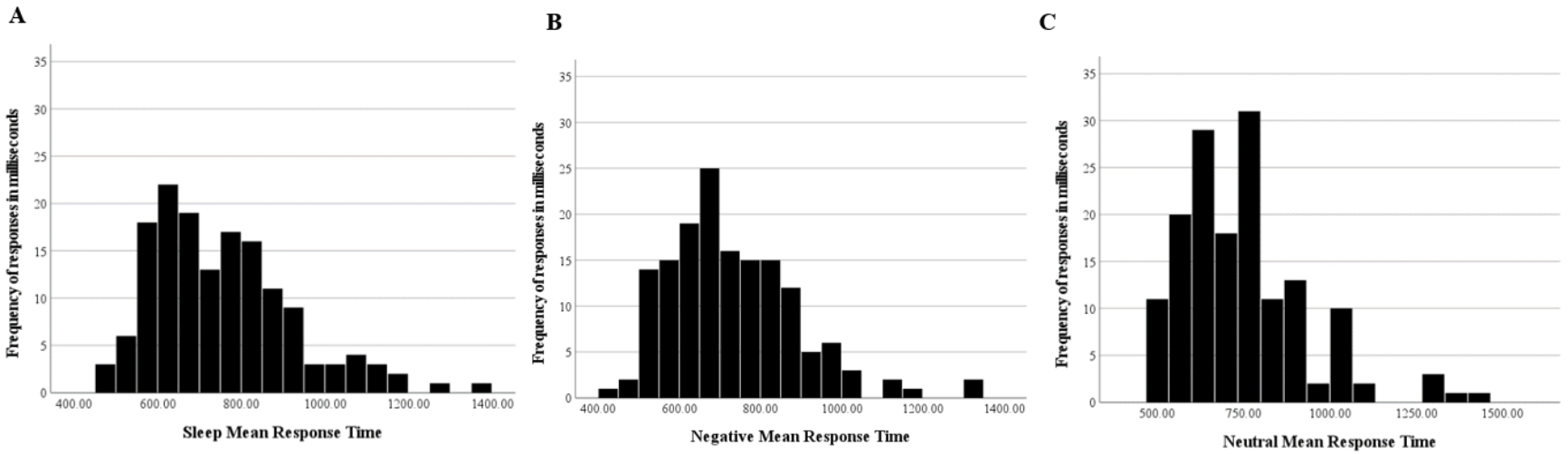
28.8% (n = 51) of respondents reported that internal LAN from outside the sleeping environment trespassed into the bedroom. Those that reported perceiving internal LAN trespassing into the sleeping environment reported poorer sleep quality (M = 8.20, SD = 3.55) compared to those that did not (M = 6.78, SD = 3.21), F(1, 80.34) = 5.98, p = .017. However, eta squared indicated a small effect size (.03) in the differences in the means. Additionally, those that perceive internal LAN reported shorter sleep duration (M = 7.62, SD = 8.22) compared to those that did not (M = 8.21, SD = 1.26), F(1, 94.190) = 8.930, p = .004, however, a small effect size (.03) was found as indicated by eta squared. No between groups differences were observed for chronotype (p = .946), social jetlag (p = .821) or to attitudes and beliefs towards sleep (p = .376).

4.3.3 Examination of the effect of word type on response time latency

As can be seen from the histograms (Figure 4.2), for the overall sample, the distribution of scores for mean response time to word types indicates that there was a wider range of scores for sleep related words compared to both negative words and neutral words. Neutral words displayed a closer frequency of scores compared to both sleep related and negative words. Each of the word types were positively skewed.

Figure 4.2

Histograms illustrating the distribution of mean response time scores for sleep-related words (A), negative words (B), and neutral words (C).



A one-way repeated measures ANOVA was conducted to compare reactions times across word types (neutral, sleep and negative) on the EST. The means and standard deviations are presented in Table 4.7. There was a significant main effect for word type on reaction times, Wilks Lambda = .88, $F(2, 149) = 9.41$, $p < .001$, multivariate partial eta squared = .11. Post hoc comparisons using the Bonferroni test indicated that the mean reaction time for negative stimuli ($M = 731.55$, $SD = 159.45$) was significantly faster than response times for both sleep related words ($M = 752.48$, $SD = 169.50$, $p < .001$) and neutral stimuli ($M = 750.19$, $SD = 181.38$, $p = .006$). The reaction times for sleep related stimuli and neutral stimuli did not significantly differ (Figure 4.3).

Table 4.7

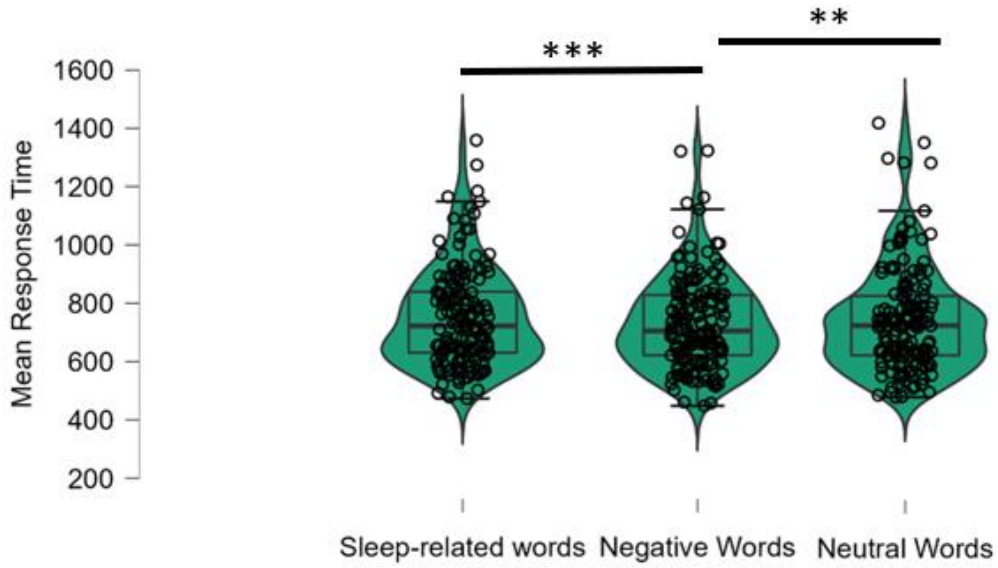
Descriptives for response time latencies across word types and attention bias indices.

	M	M(95%)	SD	Median	Range
MRT sleep related words	752.48	725.23-779.74	169.50	722.37	472.84-1359.10
MRT negative words	731.55	706.09-757.02	159.45	705.30	448.32-1323.21
MRT neutral words	750.19	721.12-779.26	181.38	729.03	483.00-1423.12
AB Negative	-.39	-18.42-17.64	112.90	5.50	-277.50-712.31
AB Sleep	-11.71	-26.75-3.31	93.48	-14.80	-281.50-220.73

Note. MRT = Mean Response Time; AB = Attention Bias

Figure 4.3

Bar graph illustrating mean response times for each of the word types on the EST.



Note. *** = $p < .001$, ** = $p < .01$

4.3.4 Attention bias indices for sleep and non-specific threat

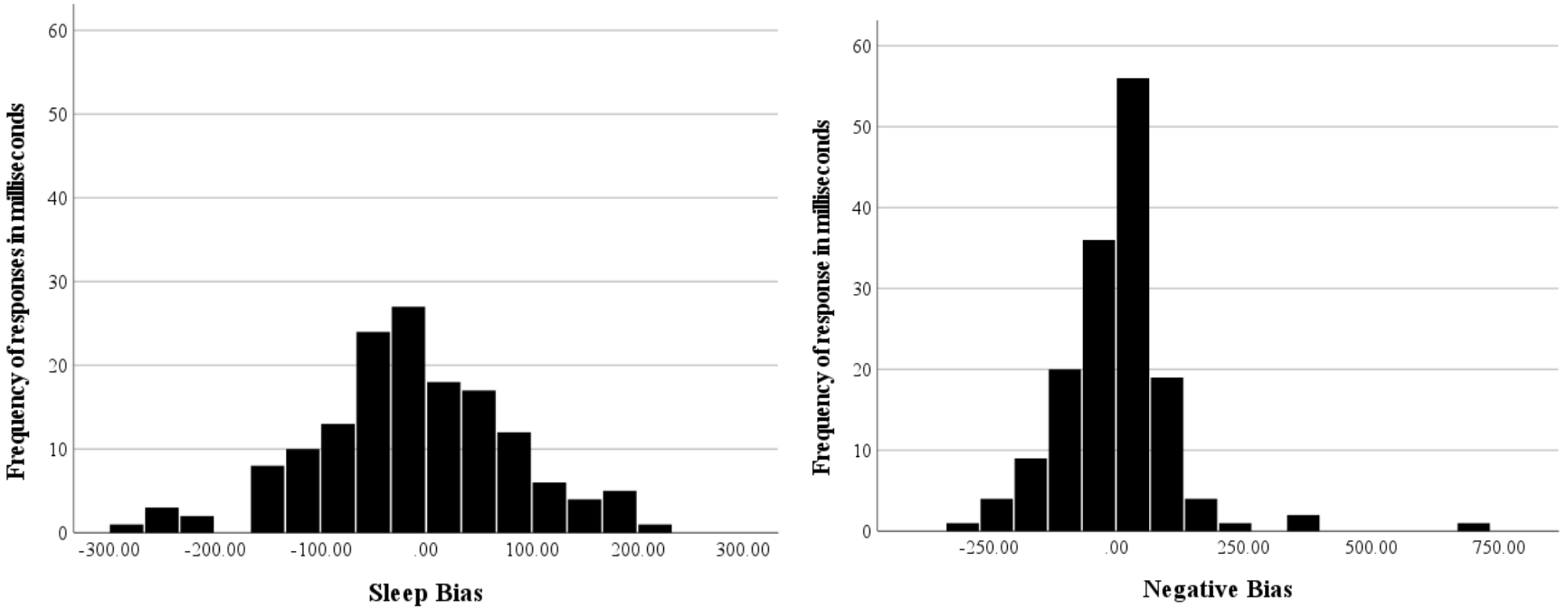
In the EST, attention bias is calculated by subtracting the mean reaction time of threatening stimuli from the mean reaction time of neutral stimuli. In this study two attention bias scores were calculated (sleep and negative). A positive attention bias score indicates that reaction times are longer for threatening stimuli compared to neutral stimuli. A negative attention bias score indicates that reaction times are faster for threatening stimuli compared to neutral stimuli. In this study two attention bias scores were calculated. Figure 4.4 illustrates the distribution of attention bias scores. As can be seen from the distribution of scores there is a platykurtic distribution of scores for attention bias, but a leptokurtic distribution for negative bias.

Of the 151 participants, 41.7% (n = 63) reported an attention bias towards sleep related information. 53.9% (n = 82) reported an attention bias towards non-specific threatening stimuli. A chi-square test for independence (with Yates' Continuity correction) indicated a significant association between type of attention bias, X^2 (1, 151) = .34, p = <.001, phi = .490. Individuals who had faster responding

to sleep related stimuli (67%) relative to neutral stimuli (i.e. indicating no attention bias) more frequently displayed faster responding to threatening stimuli relative to neutral stimuli. Conversely, individuals who display longer response latencies to sleep related stimuli (82%) relative to neutral stimuli more frequently displayed slower response latencies to threatening stimuli. A paired samples t-test examined differences in attention bias scores for sleep related word stimuli compared to attention bias for non-specific threatening word stimuli. No significant difference in bias scores were found between attention bias score for sleep related word stimuli (M = -11.71, SD = 93.48) compared to attention bias for non-specific threatening words (M = -4.19, SD = 87.54), $t(150)$ = .-588, p = .557.

Figure 4.4

Histograms illustrating the distribution of scores for sleep related attention bias (left panel) and negative bias scores (right panel).



4.3.5. Examination of the association between response time latencies across word types and subjective measures of sleep, circadian parameters, and psychological/physical health.

A weak negative correlation was found between MSF*sc* scores and sleep bias scores, r = -.208, n = 148, p = .011 (see Figure 4.5), with later chronotype associated with lower levels of sleep attention bias (faster responding to sleep stimuli relative to neutral stimuli). Additionally, a weak positive correlation was found between the average sleep duration for the week and sleep bias scores, r = .189, n =149, p = .021 (see Figure 4.5), with longer sleep durations associated with higher levels of sleep attention bias (slower responding to sleep-related information compared to neutral information). As can be seen from Table 4.8 no other significant associations between sleep bias scores were found on quality of sleep, attitudes towards sleep and social jetlag.

Figure 4.5

Association between MSFsc (left) and average weekly sleep duration (SDweek; right) with sleep bias.

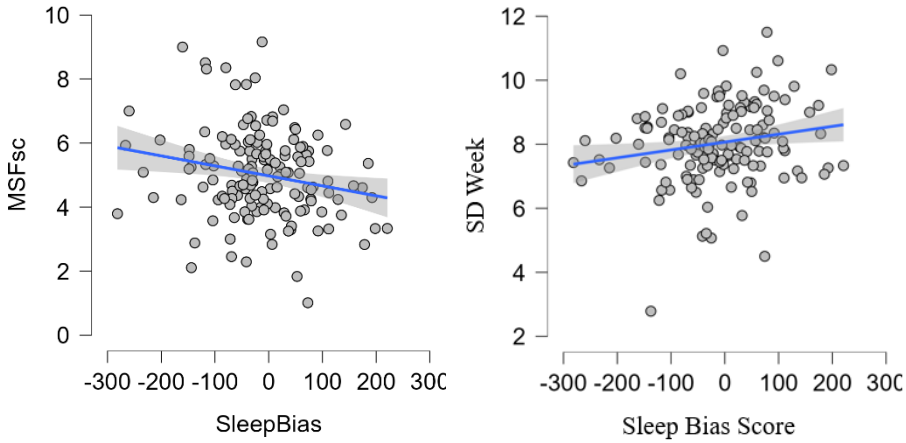


Table 4.8

Pearson Product-moment Correlations between Reaction Times Latencies across word type and sleep quality, circadian parameters, and attitudes towards sleep.

	Sleep RT	Neutral RT	Negative RT	Sleep Bias	Negative Bias
PSQI	-.008	.008	.007	.420	.535
MSF*sc*	-.111	-.004	-.114	-.208*	-.103
SD Week	-.099	-.145	-.101	.189*	.044
Absolute Social Jetlag	.089	.116	.033	-.076	-.096
DBAS	.026	.028	.034	-.029	.071

Note. * = $p < .05$

Further correlational analysis was carried out to investigate whether there were associations between the measures of psychological health and response times across word type on the EST and separately for attention bias scores for sleep or non-specific threatening words. As can be seen from Table 4.9 no significant association between psychological health subscales and any of the indices from the EST.

Table 4.9

Pearson Product-moment Correlations between Reaction Times Latencies across word type and measures of psychological health.

	Sleep RT	Neutral RT	Threatening RT	Sleep Bias	Negative Bias
CFQ	.095	.082	.048	-.014	-.015
GHQ	-.011	.055	.012	-.112	-.036

4.3.6 Effect of Sleep Quality on reaction time to word categories and AB indices

A series of Welch t-tests were carried out to examine whether poor sleepers differed from good sleepers on response time latency to sleep-related words, negative words, and neutral words. As can be seen from Table 4.10 no between group differences were observed. Additionally, no statistically significant between

group differences were observed when examining level of attention bias for sleep-related stimuli relative to neutral stimuli or for attention bias to threatening affective stimuli relative to neutral stimuli (see Table 4.10; Figure 4.6). Separately, Chi-square analysis was carried out to investigate whether there was an association between sleep quality type and the presence of a sleep attention bias. Using the Yates Continuity correction results found no association between sleep quality type and presence of sleep attention bias, x^2 (1, n = 149), .00, p = 1, phi = .01. Similarly, no association was found between sleep quality type and the presence of attention bias towards non-specific threatening information x^2 (1, n = 150), .49, p = .482, phi = .07.

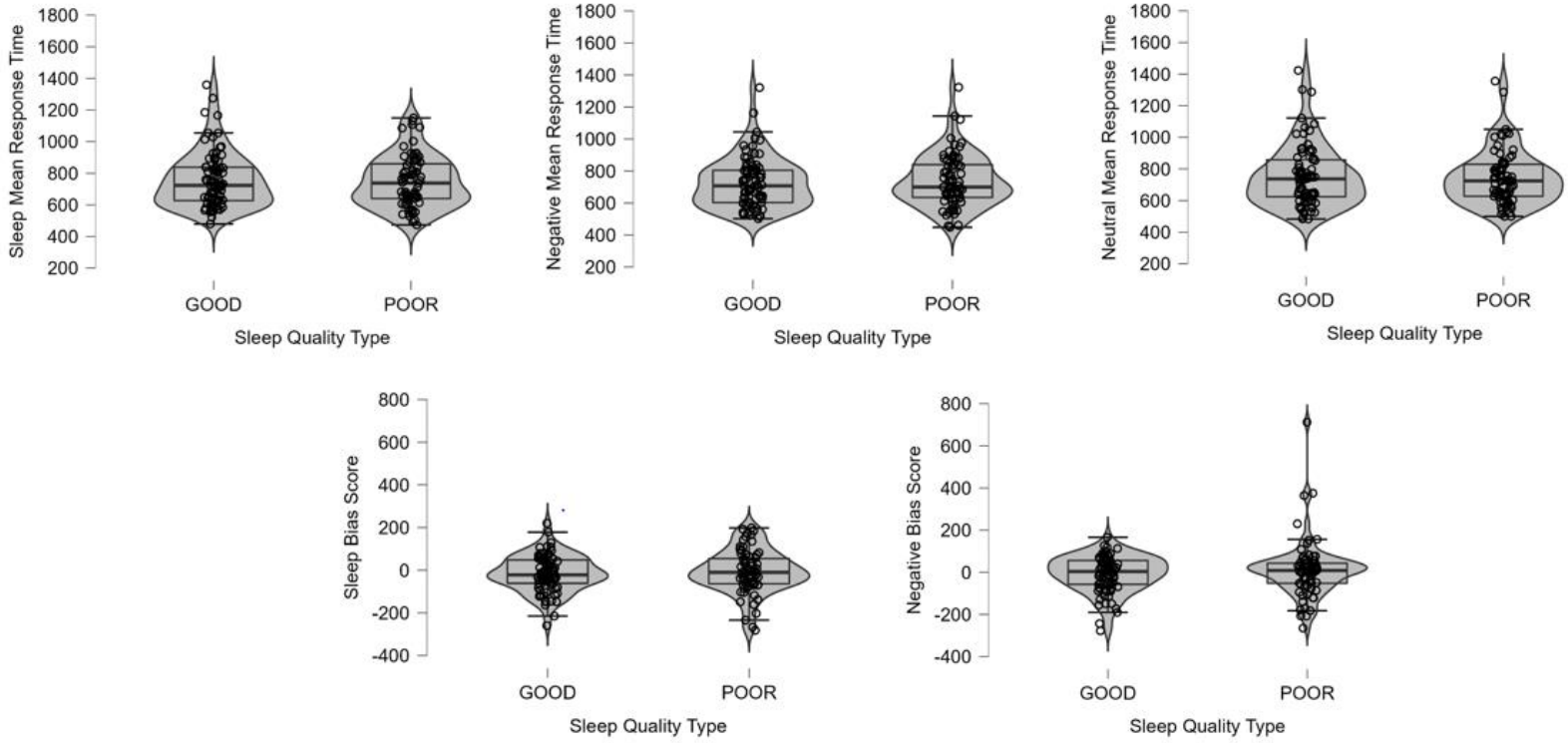
Table 4.10

Response time for sleep-related, negative, and neutral words in the Emotional Stroop Task.

	Good N(78)	Poor N(71)		
EST Indices	Mean(SD)	Mean(SD)	F	P
Mean RT sleep related words	758.66(178.14)	747.50(162.68)	.160	.690
Mean RT negative words	725.72(157.64)	164.10(19.21)	.236	.628
Mean RT neutral words	754.40(193.80)	745.48(170.79)	.090	.765
AB Negative	-15.87(.85)	-6.13(102.16)	.395	.531
AB Sleep	-8.55(86.02)	10.17(136.46)	1.002	.319

Figure 4.6

Box-and-violin plots of mean reaction times (measured in milliseconds) for each category type (presented in top panel) and attention bias scorers (presented in lower panel) illustrating the distribution of scores for good and poor sleep as categorised by the PSQI.



4.3.7. Examination of attitudes towards LAN exposure and sleep disruption to sleep and attention bias.

Welch t-tests were carried out to investigate whether the perception of LAN being disruptive to sleep quality resulted in differences in response time latencies across word types or towards attention bias scores for either sleep related stimuli and/or non-specific threatening stimuli. Between groups descriptives and their associated inferential analyses can be seen in Table 4.11. It was observed that those that perceived LAN to be disruptive towards sleep quality displayed an attention bias towards non-specific threatening information (M = 9.96, SD = 117.80) compared to those that did not perceive LAN to be disruptive (M = 26.87, SD = 95.47), F(1, 94.04) = 4.01, p = .048, however, the eta squared indicated a small effect size (.02). As can be seen from Table 4.11 and Figure 4.7, no other significant differences were observed between groups on response time latencies to word types or for attention bias towards sleep related information.

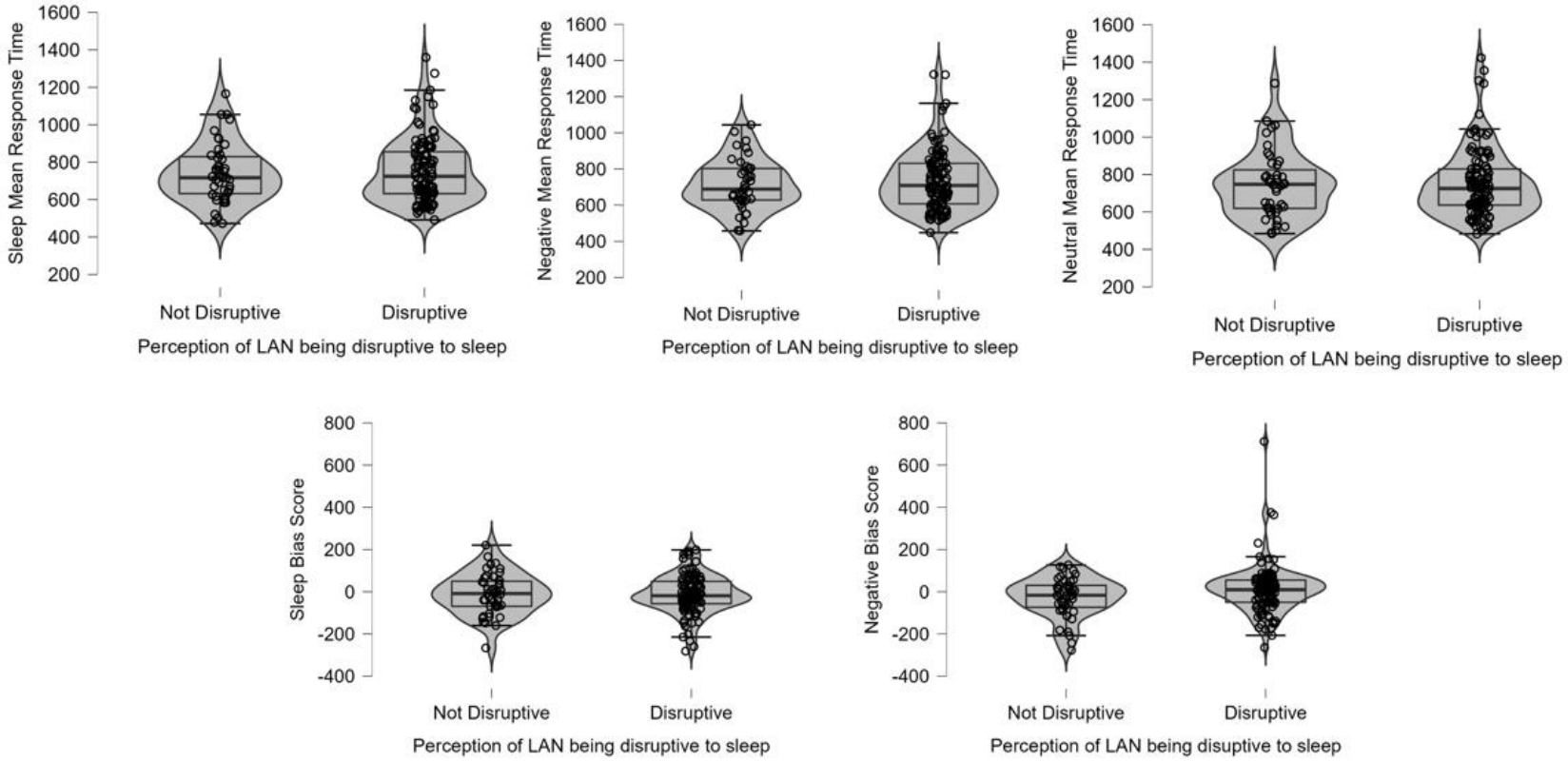
Table 4.11

Descriptives and inferential analysis examining between group differences of the perception of LAN being disruptive to sleep quality on each of the EST attention bias indices.

	Not Disruptive N(43)	Disruptive N(108)		
EST Indexes	Mean(SD)	Mean(SD)	F	P
Mean RT sleep related words	742.53(161.04)	756.44(173.33)	.220	.641
Mean RT negative words	713.69(139.70)	738.54(166.61)	.875	.352
Mean RT neutral words	746.80(179.48)	751.53(182.94)	.021	.885
AB Negative	-26.87(95.47)	9.96(117.80)	4.01	**.048**
AB Sleep	-10.51(98.15)	-12.20(92.03)	.009	.923

Figure 4.7

Box-and-violin plots of mean reaction times (measured in milliseconds) for each category type (presented in top panel) and attention bias scorers (presented in lower panel) illustrating the distribution of scores for those that perceive LAN to be disruptive to sleep and those that perceive it not to be disruptive.



Further analysis using Welch t-tests were carried out to investigate whether those that perceive LAN to be disruptive to sleep quality after falling asleep had different response times to word types or displayed an attention bias towards sleep related information. Descriptives and associated inferential analysis are located in Table 4.12. As can be seen there were no between group differences between those that perceived LAN to be disruptive to sleep quality after falling asleep.

Table 4.12

Descriptives and inferential analysis examining between group differences of the perception of LAN being disruptive during sleep on each of the EST attention bias indices.

	Yes N(39)	No N(112)		
EST Indexes	Mean(SD)	Mean(SD)	F	P
Mean RT sleep related words	778.58(187.27)	743.39(162.78)	1.09	.301
Mean RT negative words	769.64(184.78)	718.148(148.03)	2.53	.117
Mean RT neutral words	771.44(184.86)	742.86(180.42)	.702	.405
AB Negative	7.83(120.82)	-3.30(110.37)	.263	.610
AB Sleep	-7.56(91.64)	-13.16(94.48)	.106	.745

4.3.8 Examination of the perception of external LAN trespassing into the bedroom environment on attention bias indices.

Further Welch t-test analysis was carried out to investigate whether there were differences between that those that perceive LAN entering the sleeping environment on response time latencies to words type and on attention bias score for sleep related stimuli and non-specific threatening stimuli. The between group descriptives and their associated inferential *p* values are provided in Table 4.13. As can be seen from Table 4.13 and Figure 4.8 no differences were observed between groups on response times to word types or to the attention bias indices.

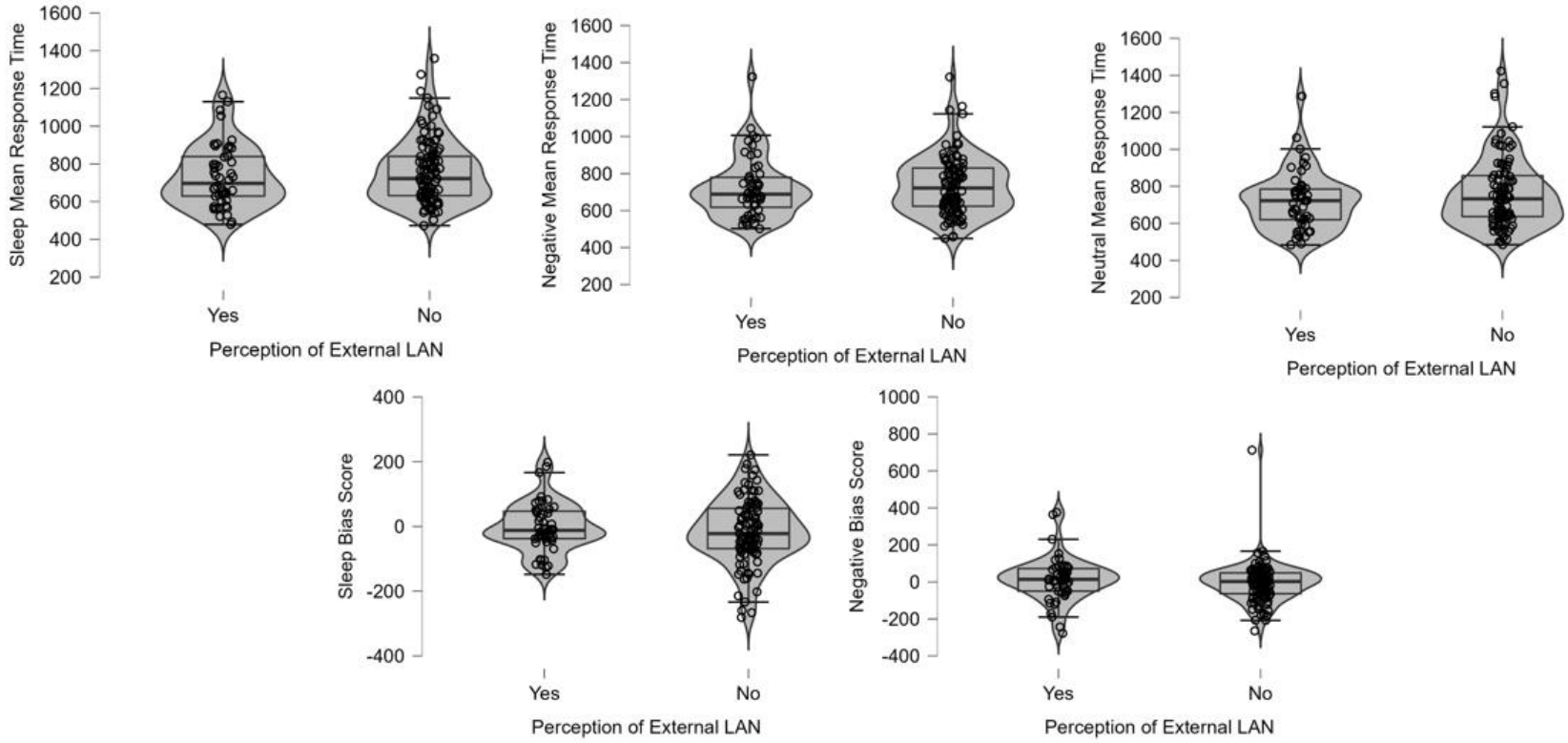
Table 4.13

Descriptives and inferential analysis examining between group differences of the perception of external LAN entering the sleeping environment on each of the EST attention bias indices.

	Yes N(50)	No N(101)		
EST Indexes	Mean(SD)	Mean(SD)	F	P
Mean RT sleep related words	736.41(164.51)	760.44(172.17)	.691	.408
Mean RT negative words	722.06(163.27)	736.30(158.11)	.264	.608
Mean RT neutral words	721.45(158.51)	764.29(190.76)	2.13	.147
AB Negative	13.70(119.42)	-7.44(109.42)	1.13	.291
AB Sleep	-3.14(76.88)	-15.96(100.79)	.751	.388

Figure 4.8

Box-and-violin plots of mean reaction times (measured in milliseconds) for each category type (presented in top panel) and attention bias scorers (presented in lower panel) illustrating the distribution of scores for those that perceive external LAN trespassing into the sleeping environment.



4.3.9 Examination of the perception of internal LAN trespassing into the bedroom environment on subjective measures and attention bias indices.

Further examination was carried out to investigate whether those that perceive internal LAN entering the sleeping environment exhibit differences in response times to word type or display an attention bias to either sleep-related information or non-specific threatening information. Table 4.14 illustrates between groups descriptive differences along with their associated inferential analysis. Welch t-test found that those that perceive internal LAN trespassing into the sleeping environment display an attention bias to sleep related information (M = 11.61, SD = 94.91) compared to those that do not (M = -21.94, SD = 91.44, $F(1, 83.09) = 4.087$, p = .046. However, the effect size was small (.02) as indicated by eta squared. As can be seen from Table 4.14 and Figure 4.9 no other between group differences were observed between those that perceived internal LAN trespassing into the sleeping environment and those that did not on response time latencies to word types or attention bias scores towards non-specific threatening information.

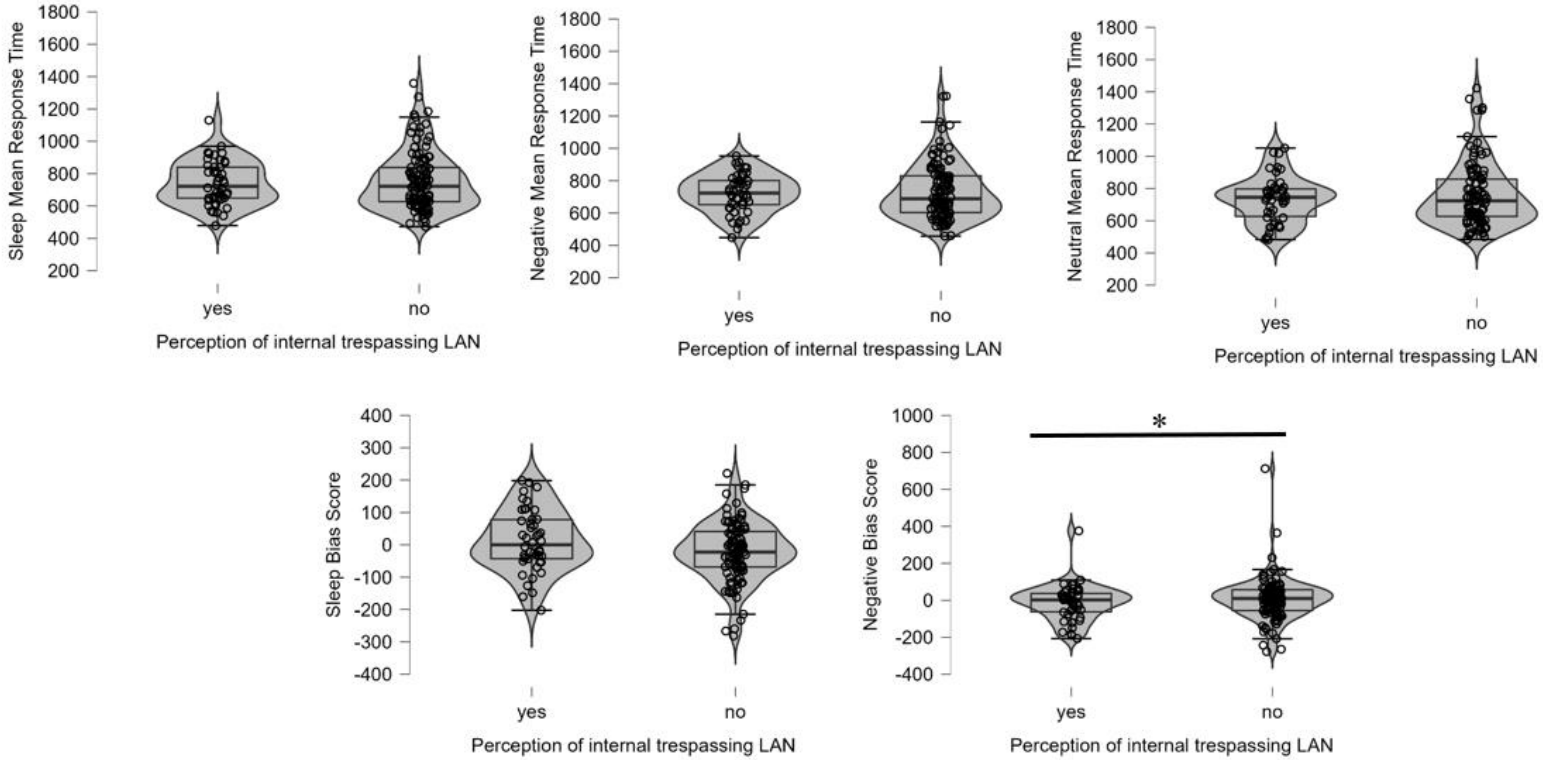
Table 4.14

Descriptives and inferential analysis examining between group differences of the perception of internal LAN entering the sleeping environment on each of the EST attention bias indices.

	Yes N(46)	No N(105)		
EST Indexes	Mean(SD)	Mean(SD)	F	P
Mean RT sleep related words	741.48(136.41)	757.30(182.52)	.347	.557
Mean RT negative words	722.95(119.37)	735.26(174.25)	.255	.614
Mean RT neutral words	733.40(141.00)	757.48(196.51)	.728	.395
AB Negative	-13.98(99.91)	5.45(118.36)	1.10	.297
AB Sleep	11.61(94.91)	-21.94(91.44)	4.09	**.046**

Figure 4.9

*Box-and-violin plots of mean reaction times (measured in milliseconds) for each category type (presented in top panel) and attention bias scorers (presented in lower panel) illustrating the distribution of scores for those that perceive internal LAN trespassing into the sleeping environment. * = p <.05*



4.4 Discussion

The aim of this study was to examine whether the perception of LAN in the sleeping environment was associated with increased response latencies to sleep related stimuli and increased attention bias towards sleep stimuli using the EST. The current study found that, independent of any between groups analysis, that the overall sample displayed faster response latencies for threatening stimuli compared to both sleep-related information and neutral stimuli. These findings are consistent with Barclay and Ellis (2013) who, using the same stimuli, found that response time latency was faster for threatening word stimuli compared to sleep stimuli. These finding have important conceptual implications as it suggests that independent of the specific content, non-specific threatening words can impact on both impact on both cognitive and behavioural perception of a stimulus. The current study's findings are consistent with one other study which also found no differences between good and poor sleepers in their response latency to sleep related words (Barclay & Ellis, 2013). Furthermore, our results are consistent with several other studies which found no evidence of attention bias between insomniac/poor sleepers and good sleepers using the EST (Lundh et al., 1997; Spiegelhalder et al., 2008; Spiegelhalder et al., 2017). It was originally suggested that the reason for not observing sleep-related Stroop interference between good and bad sleepers was due to sleep-related words having an emotional valance irrespective of quality of sleep. The current study's findings along with the aforementioned studies however, are in contrast to other studies which have found evidence of attention bias using the task in both clinical (Spiegelhalder et al., 2010) and subclinical groups (Spiegelhalder et al., 2009). Additionally, Barclay and Ellis (2013) while not observing attention bias found that poor sleepers had slower response latencies to sleep-related words compared to threatening word stimuli. The possible reason for the difference between the current study findings and previous studies are methodologically unclear. In each of these studies the PSQI was employed to categorise individuals into either poor or good sleepers and the stimuli employed were the same as used by Barclay and Ellis (2013).

Most studies to date examining sleep related attention bias have only examined between group differences based upon the classification of insomniac/poor

sleeper and good sleeper classification. However, these studies have not examined the specificity or the antecedent driving attention bias (e.g., monitoring of LAN). This builds upon previous work which observed that increased self-focus and monitoring of bedroom environment sources can increase pre-sleep worry and latency of sleep onset (Semler & Harvey, 2006; Tang et al., 2007; Woods e al., 2009). This study is the first to examine whether the perception of LAN in the sleeping environment and the perceived disrupting impact of LAN on sleep is associated with increased latencies to sleep stimuli and attention bias scores. Our results reported that the perception of internal LAN from inside the dwelling entering the sleeping environment had an effect on attention bias scores. With this group reporting statistically longer response latencies to sleep related words compared to neutral words. However, it is important to note that the effect size in the magnitude of the means was small. The perceived disruptive impact of LAN exposure on sleep or the perception of external LAN had no effect on attention bias scores or response latencies to sleep related information. It is plausible that although individuals perceive LAN in their sleeping environment, this may not be directly disruptive to their own sleep due to individuals employing strategies to minimise the perception of the LAN source. It is plausible that those that perceive external LAN entering the sleeping environment may engage in minimising LAN exposure by sleeping in the opposite direction of the light source or wearing an eye-mask. This could result in LAN not being perceived as a threatening source and ultimately not becoming a driver of attention bias. This is also supported by experimental evidence which have demonstrated that light exposure to the lower retina leads to greater suppression of melatonin compared to the upper retina (Glickman et al., 2003) and light exposure at the nasal side of the retina leads to greater non-image forming responses compared to temporal side of the retina (Visser et al., 1999; Ruger et al., 2005). Future studies examining sources of LAN exposure in the sleeping environment should also examine if individuals employ strategies to minimise their perception of LAN. For instance, in the monitoring studies which found that self-focus of sleep environmental stimuli increased pre-sleep worry and latency to sleep onset individuals were directed to monitor the source (Tang et al., 2007; Woods et al., 2009) while those that did not monitor their clocks in the sleep environment had less sleep-worry and reduced sleep onset.

Cognitive theories of insomnia propose that the aetiology of insomnia is due to a preoccupation with sleep and misperception of sleep and daytime impairment (Espie et al., 2006; Harvey, 2002). Some empirical studies have reported an association between pre-sleep worry and insomnia/sleep disturbance, but specificity of this association is unknown (Gerlach et al., 2019; Lancee et al., 2017, REF). Our study reports that the perception of LAN being disruptive to sleep is associated with increased dysfunctional beliefs and poor attitudes towards sleep. In particular, the associations are observed in the domains of the consequences of insomnia and increased levels of worry/helplessness about sleep. This supports previous work which has demonstrated that light exposure during sleep leads to more awakenings during the night (Cho et al., 2016; 2018) and the habitual presence of LAN exposure in the home setting before sleep is associated with higher dissatisfaction with sleep and poorer subjective sleep quality (Cain et al., 2020). However, the specific sources of LAN in the sleeping environment either trespassing into the room externally or internally was not associated with pre-sleep worry. Further, our study finds that the specific perception of sources on LAN in the sleeping environment are not associated with dysfunctional beliefs and attitudes towards sleep. Our findings are also in contrast to the findings from chapter 3 which found that the perception of specific sources of LAN exposure in the sleeping environment was associated with poorer sleep quality. This research also finds that DBAS scores are not associated with attention bias scores or response latency to sleep related stimuli.

A later chronotype was associated with sleep attention bias. Specifically, later chronotypes displayed lower levels of attention bias as indicated faster responding to sleep-related stimuli compared to neutral stimuli. While, a positive correlation was observed between longer sleep duration associated and higher levels of attention bias as indicated by faster reaction times to neutral stimuli compared to sleep-related stimuli. This direction of this association is surprising as Spiegelhalder et al. (2009a) reported a positive linear relationship between sleep-related attention bias and state sleepiness along with high sleep quality and low sleepiness being associated with lower attention bias. Although the current study did not measure sleepiness, it has been found that sleep duration and sleepiness are negatively associated (Akerstedt et al., 2017). It is plausible that our findings are inconsistent with Spiegelhalder et al. (2009a). Our findings indicating an association between

higher sleep duration and higher levels of sleep bias may be driven by craving for sleep instead of threat of sleep. Spiegelhalder et al. (2009b) reported that attention bias scores were positively associated with objective measures of sleep. In this case what drives the sleep-related attention bias may not be threat towards sleep-related information but instead a craving for sleep (Spiegelhalder et al., 2017). This may suggest that higher levels of sleep duration may be indicative of homeostatic craving for sleep and being positively associated with high attentional bias. Examining duration of sleep in attention bias is important given that previous studies have reported that the association between sleep quality as measured by the PSQI is weakly associated to sleep duration (Pilcher, Ginter & Sasiwsky, 1997). These results must be taken with caution however given the small effect size. It is plausible that this could be a type one error.

A plausible reason for no differences in the latency of responding towards non affective sleep-specific words and neutral words is that they did not vary sufficiently in affective intensity to elicit a threatening response. Yiend and Matthews (2001) argue that threatening words (sleep-related words) may not sufficiently vary in affective intensity from neutral words to elicit a different threat response. Although the study found that the whole sample displayed faster response times to non-specific threat words, this was not specific to any group. The words employed in the non-specific threat condition (e.g., shame, upset, hateful) were negatively valanced and would be perceived as threatening to all individuals resulting in eliciting the same cognitive and behavioural response. The current study employed non-affective sleep related words so to ensure that individuals were not simply responding based upon the salience of the emotionally threatening stimuli. Future studies must take caution when choosing which word stimuli to employ within word types. Barclay and Ellis (2013) provided evidence for this when they found that non-specific threat words facilitated performance but non-affective words hindered performance. It is plausible that individuals may attend differently to sleep-related content based upon the affective valance of the stimuli. This suggests that studies should avoid using both emotionally valanced and non-affective sleep-related stimuli within specific word type conditions, as this may mask and counteract the attention bias to sleep-related stimuli. Future studies should employ the EST having two sleep related stimuli word types, which are affective sleep-related stimuli and

non-affective sleep related stimuli, to examine if differences in attention bias exist. It is also plausible that the word stimuli employed should have a lower potential to attract attention compared to pictorial stimuli (Moritz et al., 2008). In this case, the written presentation of a word may not elicit the same threatening response as an image of the same stimulus. Evidence to support this claim comes from Roelofs and colleagues (2009) who reported evidence of attention bias towards images but not words when examining pain related attention bias. The use of images in attention tasks could potentially be attributed to the tasks appearing to be more sensitive to group effects with larger effect sizes (Harris et al., 2015). In addition, the sleep-specific word stimuli employed may be too general to elicit an attention bias. It may be plausible that attention bias is driven by a specific monitoring of a sleep specific stimulus. While the current study found that LAN was perceived to be disruptive to sleep, the stimuli in the EST may not have been of personal relevance to be perceived as threatening/or what is monitored in the sleeping environment. Future studies should examine whether images of LAN in the sleeping environment may be of salience to those that perceive LAN to be disruptive.

The results demonstrating no evidence of attention bias between good and poor sleepers or between those with poor attitudes towards sleep calls into question whether the EST has the ability to be a reliable and valid measure in detecting sensitivity to sleep-related attentional bias in both clinical and non-clinical populations. This comes from the ratio of studies finding no evidence of attention bias using the EST being higher than the number of studies finding between group differences on attention bias in both clinical and subclinical populations. Further evidence that the EST may not be a suitable measure for examining attention comes from studies which have found between group differences using other cognitive paradigms such as the flicker, dot-probe, and Posner tasks. Harris and colleagues (2015) reported in their systematic review that between group differences in sleep-related attention bias using cognitive tasks found that the EST provided the lowest effect size (d = .26) compared to other paradigms

There are several strengths to this study. Firstly, the study moves beyond simply examining associations between sleep disturbance and attention bias to instead trying to understand whether LAN exposure is specifically associated with

poor attitudes towards sleep and are both of these specifically driving an attention bias. This allowed for a better understanding of the specificity of the bias along with whom and under what conditions attention bias is elicited. The study also separately examined the specific sources of LAN exposure in the sleeping environment to investigate their associations with the outcome variables. The study also examined whether sleep timings are associated with attention bias by examining social jetlag, sleep duration, chronotype and sleep quality on attention bias. This expands upon past research which examined attention bias differences solely based upon quality of sleep. Examining differences based upon other sleep and circadian parameters is of importance given that sleep quality as defined by the PSQI has been found to be weakly associated with these parameters (Takeuchi et al., 2018; Ramen & Coogan, 2019). This current study controlled for psychological wellbeing. As is observed in many studies, depression and trait anxiety are higher in those with poor sleep (Fortier-Brochi & Morin, 2014). The analysis of the current study ensured to control for psychological wellbeing when investigating attention bias.

Given that this study was employed using a non-clinical sample, we cannot generalise whether the perception of LAN is associated with increased attention bias towards sleep related information in individuals with insomnia. Although not employing a clinical group limits the generalisability of the study, there is strength in examining attention bias differences between subclinical poor sleepers and good sleepers. Mainly being that the processes which underlie insomnia possibly exists along a continuum which varies in terms of severity and intensity. However, processes which drive the clinical and subclinical disturbances remain the same. It is important to note that very few studies compare differences in preferential attention allocation between good and poor subclinical sleepers. This is problematic given findings which show that as sleep disturbance increases to clinical thresholds, so to does the presence of attention bias (Taylor et al., 2003; Jones et al., 2005). Should attention bias to sleep-related stimuli be a core feature in insomnia, then it is important to understand at what point in the progression to clinical diagnosis of sleep disturbance does preferential attention allocation occur and what is the magnitude of this attention bias. However, in Harris and colleagues' (2015) systematic review, they argue that the effect sizes are not greater in clinical populations relative to subclinical populations. The current study did not use an objective measurement of sleep. This may be a limiting factor in the reliability of findings and for poor

sleepers. Poor sleepers have been found to misperceive attributes of sleep with subjective measures of the timing and duration of sleep being overestimated and total sleep time underestimated (Tang & Harvey, 2006; Van der Berg et al., 2008). This misperception may also extend to the perception of LAN being disruptive to sleep. This reasoning comes from Akram and colleagues (2016) who observed that those with insomnia perceive their facial appearance to be exhibit more features of tiredness than actually are present. The current study employed a student population where pre-sleep worry may be potentially lower due to the perceived impacts of a poor night's sleep being less severe both in terms of the consequences of delayed sleep timing and daytime impairment the next day. This lower pre-sleep worry may lead to sleep ensuing faster and less attention monitoring of sleep environment threats which would ordinarily be perceived as being disruptive to sleep or confirming wakefulness. While conversely for those in more traditional forms of employment, the thought of poor sleep may be amplified leading to higher arousal and searching for sources that confirm the inability for sleep to commence.

In conclusion, our findings report no evidence of attention bias towards sleep related stimuli in those that perceive LAN in the sleeping environment. However, the lack of effect may be due to the stimuli used. It is plausible that images of a LAN sleeping environment may elicit more attentional resources in those that perceive LAN compared to stimuli of items found in the sleeping environment.

Chapter 5

The effect of perceiving light in the sleeping environment on attention bias using the Dot Probe Test

Abstract

Cognitive models of insomnia have suggested that an attention bias towards sleep-related information may play an important role in the development and maintenance of insomnia. Evidence of attention bias has also been observed in non-clinical poor sleepers. This study examined whether individuals who perceive LAN in their sleeping environment display an attention bias towards sleep-related image stimuli depicting LAN sleeping environments. A total of 149 students aged between 18-51 (M=26; *SD*=8.10) completed a Dot Probe Test (DPT). In this task individuals were presented with images comprising of LAN sleeping environments, dark sleeping environments and neutral stimuli. Participants also completed the perception of LAN in the sleep environment survey along with other self-report measures (PSQI, MCTQ, DBAS, CFQ and GHQ). Our results found that there was no association between sleep quality scores with any of the attention bias indices derived from the DPT. There was no effect of the perception of LAN in the sleeping environment on any of the attention bias indices. We outline possible limitations of the task.

5.1 Introduction

As previously outlined, disruption to sleep and insufficient sleep are associated with a number poor physical and psychological health outcomes (Seow et al., 2020). Higher levels of worry and increased rumination about prospects of poor sleep have been observed in individuals with sleep disturbances compared to good sleepers (Thomsen et al., 2003). This sleep or insomnia-related cognition is particularly problematic when it occurs in pre-sleeping hours. Selected attention towards thinking about sleep and the possible consequence of poor sleep are significant predictors of increased latency to sleep (Takano et al., 2014) and night-time physiological arousal (Takano et al., 2014). Our findings from chapter 3 found that the perception of external LAN is not associated with objectively measured external LAN. However, the perception of external LAN was associated with both poor sleep quality and psychological health. In chapter 4 we attempted to examine whether attention bias towards sleep-related stimuli was a possible reason for understanding why poor sleepers identify perceiving external light while residing in areas with equally comparable levels of outdoor ALAN to those that do not perceive it. Our findings did not report any association between the perception of LAN and attention bias using the EST, however, this lack of association may be due to the general nature of the task and the stimuli used.

Cognitive models (Espie, 2002; Espie et al., 2006; Harvey, 2002) of insomnia propose that insomnia is partly maintained by a disproportionate selective attention bias towards sleep-related 'threat' cues which may be internal (i.e., body sensations) or external (i.e. environmental LAN in the sleeping environment). Selective attention towards these cues further enforces that the individual is not initiating sleep which lead to sleep becoming both a threat and anxiety generator (Espie, 2002). As a result, poor sleepers becoming selectively vigilant towards stimuli indicative of wakefulness at night (possibly sources of LAN) and towards symptoms of fatigue during the daytime (MacMahon et al., 2006; Semler & Harvey, 2004). These sleep-related 'threat' cues may be the product of sleep-specific anxiety which is maintained because of high levels of arousal, distress, negative catastrophising thoughts about sleep and the belief that disturbances to sleep will lead to impaired functioning the following day (Harvey & Greenall, 2003). As a result of this anxious state, attentional resources are preferentially allocated to the

processing of sleep related cues. Once the individual detects these cues they may be then interpreted in an insomnia consistent manner. This leads to further increases in arousal, distress, and negative thought concerning sleep and daytime function. This leads to an increased effort to sleep which can have the opposite effect by reducing the probability sleep will ensue (Broomfield et al., 2006; Harvey & Greenall, 2003). This vicious thought cycle is in part maintained by the sleep-related attention bias. Additionally, from an operant conditioning perspective specific sleep-related objects may exert control over sleep behaviour (Bootzin, 1972). Within conditioning framework specific sleeping environment objects may become discriminative stimuli for sleep however, when the bedroom-sleep contingencies are broken they may become discriminative stimuli for wakefulness (Boozin et al., 1991).

Several studies have examined the presence of sleep-related attention bias using cognitive paradigms in those with either insomnia or disturbances to sleep. These include the flicker paradigm (Jones et al., 2005; Marchetti et al., 2006), the Posner paradigm (Woods et al., 2009), free-viewing tasks (Beattie et al., 2017) and the Dot Probe Test (Akram et al., 2018; MacMahon et al., 2006; Takano et al., 2018). In the previous chapter we employed the EST to examine whether the perception of LAN was associated with sleep-related attention bias. However, it is unclear whether this task measures increased vigilance or simply reflects the impact of heightened arousal interfering with information processing when threatening stimuli are presented (MacLeod et al., 1986). The Dot Probe Task (DPT) developed by Macleod et al. (1986) is argued to overcome these limitations and is frequently used as a measure of attention allocation towards threatening stimuli (Harris et al., 2015). Research from the DPT has led to numerous randomised clinical trials to modify attentional bias as a potential treatment for internalizing disorders (Hakamata et al., 2010). The premise of the task is that individuals respond faster to a probe that takes the position of where a previous threatening stimulus was. During the task, a series of either word or image stimuli are paired and presented briefly on a computer screen at two different spatial locations. In critical trials, one of the stimuli pair is threat related and the other neutral. When the stimuli pair disappears, a probe appears in the position formerly occupied by one of the stimuli. If the probe appears in the location previously occupied by the threatening stimulus this is referred to as congruent presentation. Conversely if the probe appears in the location of the neutral stimulus this is referred to as an incongruent presentation. Participants are asked to

respond as quickly as possible to location of where the dot appears. Attention biases are inferred from different response times towards probes that replace threatening stimuli compared to probes that replace neutral stimuli. Attention allocation to threat is measured indirectly by the reaction times to the dot. Fast reaction when the probe replaces threat words and slow reaction times when the probes replace neutral words indicate an attention bias to threat. Evidence has suggested that those with anxiety disorders show an attention bias toward threat stimuli compared to incongruent trials in the DPT (Bar-Haim et al., 2007). These findings are interpreted as vigilance towards threat.

Several studies have used the DPT to examine sleep related attention bias (see Table 5.1 for an overview of studies and findings). However, an issue with some of the studies using the DPT is that the stimuli employed are word stimuli (Lancee et al., 2017; MacMahon et al., 2005; Takano et al., 2018). Evidence suggests that the dot-probe may not be optimal when word stimuli with mild threat value are used (Mogg et al., 2000). Along with being more ecologically valid, picture stimuli are more salient and threatening compared to words (Mogg & Bradley, 1999; Schmukle et al., 2005). Evidence from other tasks have found that the use of images elicits evidence of attention bias in those with insomnia or severe sleep disturbance (Jones et al., 2005; Marchetti et al., 2006; Woods et al., 2009). To date, four studies have examined evidence of attention bias in insomnia participants by utilising sleep-related images when using the dot-probe task (Akram et al., 2018; Baglioni et al., 2010; Jansson-Frojmark et al., 2013; Spiegelhalder et al., 2010) to investigate evidence of attention bias. Mixed results have been yielded with some studies illustrating attention bias towards tired faces (Akram et al., 2018) and images depicting daytime fatigue/malaise (Jansson- Frojmark et al., 2013). However, Spiegelhalder et al. (2010) reported no evidence of attention bias when employing the dot probe test with images of bedrooms. However, bias scores were correlated with other objective sleep parameters such as total sleep time, sleep efficiency and amount of slow wave sleep. See Table 5.1.

Table 5.1

Overview of previous studies which employed the DPT in clinical and non-clinical groups.

Study	Participant Allocation	Sleep Measures	Stimuli Type	Key Findings
MacMahon et al. (2006)	DSM-IV, ICSD-R, actigraphy and PSQI > 6 criteria for Primary insomniac & DSPS Good Sleeper – PSQI < 5	Actigraphy & PSQI	Word Stimuli sleep words and neutral words	PI sig faster responses for sleep-related stimuli relative to GS and DSPS. No significant difference between GS & DSPS
Takano et al. (2018)	Non-clinical Poor sleeper PSQI > 5	PSQI	Word Stimuli sleep words (non-affective) and neutral words	No association between PSQI Scores and RT scores on the task.
Akram, et al. (2018)	DSM-IV criteria for Insomnia. Criteria for normal sleepers – no problems with sleep or history of a sleep-wake disorder. Use of SLEEP-50 to confirm absence of same.	Stanford Sleepiness Scale	Images of tired faces and non-tired faces	Individuals with insomnia display slower orienting and disengagement from tired faces compared to normal sleepers.

Jansson-Frojmark et al. (2013)	ISI, ICSD & DSM-IV-TR criteria for primary insomnia. Normal Sleepers use of SLEEP-50, PRIME-MD,	ISI & Epworth Sleepiness Scale	Image of stimuli depicting daytime fatigue/Malaise & Neutral Stimuli.	No difference in attention bias scores between normal sleepers and those with primary insomnia. PI displayed significant delay in disengaging from tired faces compared to normal sleepers.
Lancee et al. (2017)	Insomnia classification DSM-IV, ISI > 10,	ISI, PSQI & Anxiety & Preoccupation with Sleep Scale	Word Stimuli: Sleep Words & Neutral Words	No indication for the presence of a sleep-related attention bias (at baseline and after Attention Bias Modification)
Spiegelhalder et al. (2010)	PI meeting criteria based on DSM-IV-TR & Polysomnography	PSQI, SSS,	Word Stimuli: Sleep related stimuli & neutral stimuli	No significant difference between PI and normal sleepers on attention bias scores. Attention bias scores were associated with total sleep time, sleep efficiency and amount of slow wave sleep. Attention bias scores were negatively associated with number of awakenings.

Note: DSM-IV = Diagnostic Statistics Manual; PSQI = Pittsburgh Sleep Quality Index; DSPS = Delayed Sleep Phase Syndrome; GS = Good Sleeper; PI = Primary Insomnia; RT = Response Time; SSS = Subjective Sleepiness Scale; ISI = Insomnia Severity Index; ICSD = International Classification of Sleep Disorders; Prime-MD = Primary Care Evaluation of Mental Disorders.

Although previous studies have provided evidence of attention biases towards sleep-related images in individuals with insomnia (Jones et al., 2005; Marchetti et al., 2006; Spiegelhalder et al., 2010), these images, although sleep-related, have lacked specificity by being based on general images from the sleeping environment (i.e. teddy bears, slippers or pillows). The findings from these studies may underestimate the true magnitude of attention bias as the images may not be perceived as threatening to sleep quality. As a result, it is unclear from these studies what specific sleep-related cues drives attention bias. This needs to be examined given that theories of insomnia propose that individuals attend and specifically monitor specific threat cues to confirm they are not sleeping (Espie, 2006). Identifying which specific sleep-related stimuli are perceived as threatening may allow for investigation of the true magnitude of attention bias and what stimuli in the sleeping environment are selectively monitored and perceived as threatening (Harvey, 2002). The images employed in previous tasks have also been based on contrived visual scenes such as the brief removal of a sleep-related stimulus from an image which may not be ecologically valid. To overcome some of the limitations in the current study the DPT will comprise of two sets of sleep-specific stimuli (LAN sleeping environment and dark sleeping environment) and neutral stimuli. This will allow us to determine whether those that perceive LAN are selectively biased towards sleep-related stimuli more generally or allocate more attention resources to LAN sleeping environments.

Past research examining attention bias to sleep-related information have not fully examined the nature of attention bias in those with insomnia/poor-sleepers (MacMahon et al., 2006; Takano et al., 2018). From these studies it is unclear if the attention bias is due to vigilance towards sleep-related information or due to showing difficulties disengaging from sleep-related information. To fully examine possible attention bias towards LAN stimuli this study will examine attention bias using the traditional index of attention bias along with the indices of orientation and disengagement (Cisler & Koster, 2010; Koster et al., 2006; Koster et al., 2007). By examining the possible nature of attention bias towards LAN in the sleep environment this allows for an understanding on whether individuals direct more attention towards LAN or try to experientially avoid directing attention towards LAN.

We hypothesised that:

1. Those that perceive LAN in the sleeping environment will display faster response times to congruent stimuli which are LAN specific relative to neutral stimuli and dark bedroom environment stimuli.
2. Those that perceive LAN will have difficulty will slower disengagement from images displaying LAN sleeping environments compared to dark bedroom environments and neutral stimuli.

5.2 Method

5.2.1 Participants

149 participants (67.8%, *n* = 101 female) with a mean age of 25.63 (*SD* = 8.10, range 18-51) and residing in Ireland took part in the study. Data was collected remotely between February 2021 and April 2021. Ethical approval was obtained from Maynooth University Research Ethics Committee. Recruitment was through a mixture of snowball and convenience sampling via flyers, emails, and personal contacts. The inclusion criteria were that participants were aged 18 years and above and were not shift workers. All participants gave their electronic informed consent before participating in this study and were informed that all data collected would be stored anonymously. Participation was voluntary and unpaid however, some participants received course credit for parking in the study.

5.2.2 Materials and Measures

5.2.2.1 Light at Night Survey

Participants completed a number of questions assessing their attitudes towards to light at night exposure on their sleep quality. The survey also examined what are the specific sources of LAN exposure in the sleeping environment and to what degree these specific sources are perceived as disruptive to sleep.

5.2.2.2 Sleep quality & chronotype measures

The PSQI and the MCTQ both previously described in chapter 3 were completed by participants. A global PSQI score was derived from the PSQI measuring a participant's level of self-reported sleep disturbances over the previous months. A PSQI score >7 was used to differentiate between 'poor sleeper' and 'good sleeper.' In the current study, the PSQI displayed good internal reliability with a Cronbach alpha coefficient of .737. From the MCTQ individuals average sleep duration was calculated. Individual's sleep debt corrected mid-point of sleep on free days (MSFsc) was derived as a marker of circadian phase of entrainment. Social jetlag was calculated as a typical measure of recurring circadian misalignment as previously described.

5.2.2.3 Attitudes towards sleep

Sleep related cognitions were measured with the DBAS-16 which has been described in the previous chapter. The DBAS-16 consists of 4 subscales representing types of sleep-related cognitions: perceived consequences of insomnia, worry/helplessness about insomnia, sleep expectations and medication. Higher scores on the DBAS indicate greater dysfunctional beliefs about sleep. In the current study the Cronbach alpha coefficient was .84.

5.2.2.4 Physical and psychological wellbeing

The GHQ-28 which has been previously described was employed to measure psychological well-being. The GHQ comprises of 4 subscales which measure: Somatic symptoms, anxiety and insomnia, social dysfunction, and severe depression. Higher scores indicated poor levels of psychological wellbeing. In the current study the Cronbach alpha coefficient was .94 for the GHQ. The CFQ was employed to evaluate individual's propensity towards mistakes or errors in cognition. Higher scores indicate higher levels of errors in everyday cognition. In the current study, the Cronbach Alpha coefficient was .91.

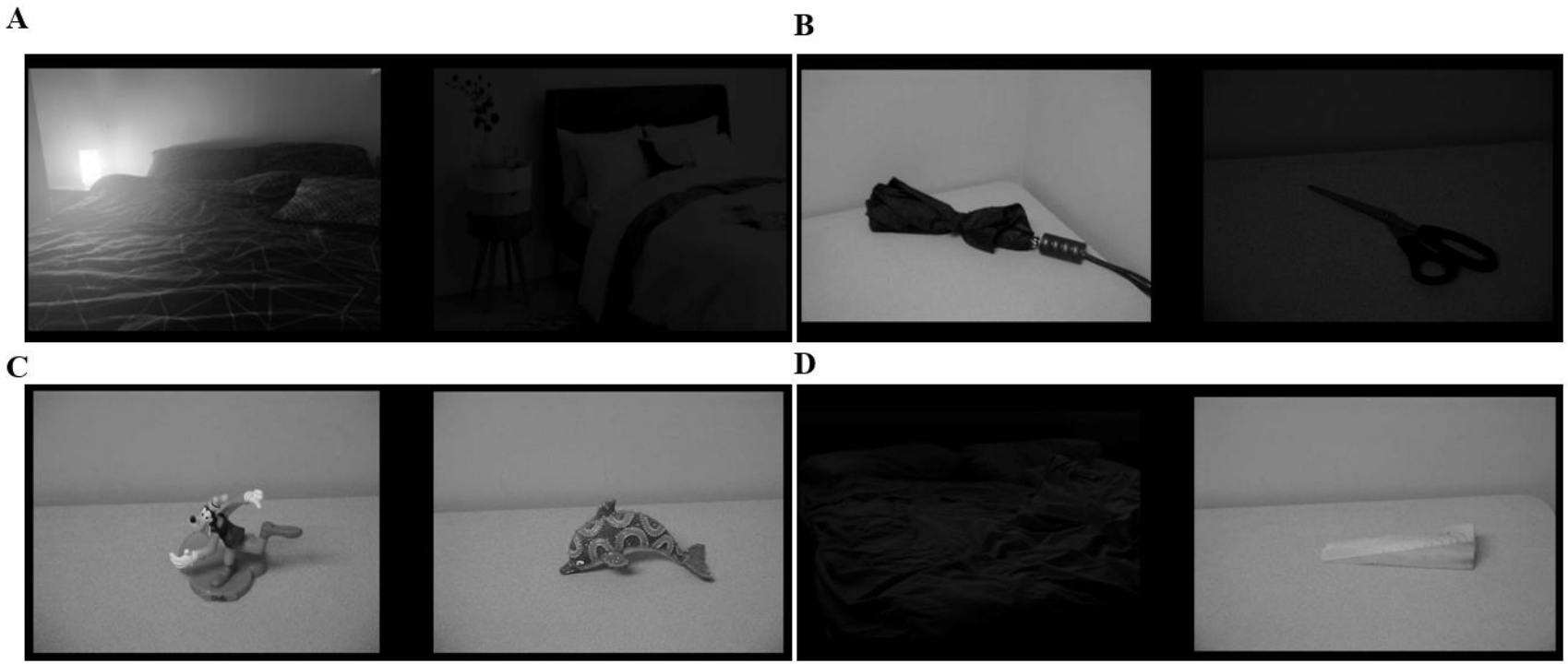
5.2.2.5 Visual Dot Probe Task

Attention bias was measured using a modified version of the visual dot probe task which was programmed and presented using the INQUISIT Millisecond software package (INQUISIT 6.3.1, 2020). In this modified version of the visual dot probe task five types of stimulus pairs were created: (i) LAN-Dark (Figure 5.1A); (ii) Dark-Neutral (Figure 5.1D); (iii) LAN/Neutral; (iv) Neutral/Neutral (Figure 5.1C) and (v) Neutral/DarkNeutral (Figure 5.1B). In total this task used 30 picture stimuli 5 LAN sleeping environment stimuli (LAN), 5 Dark sleeping environment stimuli (DARK) and 20 neutral stimuli (Neutral; e.g. "flag"). Furthermore, 20 non-threatening stimuli were selected for the practice trials which comprised of 10 trials. In the test block 10 of the same neutral stimuli pairings appeared in 2 blocks. Dark images depicted a sleeping environment with no light sources (e.g., a dark room with no electrical devices). LAN stimuli depicted sleeping environments with light sources (e.g., individuals in bed using light emitting technology or external light trespassing into the sleeping environment. In the LAN-Dark condition, the images

were matched (e.g., individual in bed using a light emitting technology vs individual in bed with no light sources). In one block the neutral/neutral pairing occurs with both neutral stimuli having the same colour contrast of black and white. In the second pairing, 5 of the neutral images were made darker. This was to examine whether differences in reaction times scores was due to the content of the stimuli rather than contextual factors such as the darkness of the image. The neutral stimuli were selected from a previous study (Miller & Fillmore, 2010). As a result of there currently not being an existing picture set depicting dark sleeping environments and LAN sleeping environments, a new set was created within the context of this study. Stimuli were selected from the internet and by photographs taken by the researcher. The sleeping environment stimuli depicted a dark sleeping environment where no light source could be perceived, and images where LAN sources were perceived. The pictures were included in the task after a thorough discussion between the researcher and the principal supervisor with the decision for inclusion based upon this. The approach to deciding the picture stimuli were similar to that of Jansson-Frojmark and colleagues (2013) study which had 4 main criteria. These were to ensure that the stimuli selected had: (i) matching the type of everyday situations in the two picture sets, (ii) matching of age and gender in the two picture sets (iii) similar qualitative aspects of the pictures and (iv) identical size of the pictures. Once LAN and Dark stimuli were approved, they were greyscaled. The justification for greyscaling all images were due to findings from Bekhtereva and Muller (2017) study where they found that participants judged unpleasant scenes presented in colour as slightly more negative and more arousing than the greyscale versions of the same images. This suggest that colour information may contribute to the perceived emotional intensity of threatening information. The photos were also adjusted so that the width, length, and orientation of the images were the same. The adjustments to the images were carried using an image editor (Gimp 2.10.28, 2020). All pictures presented were 13cm x 18cm.

Figure 5.1

Examples of the stimuli used in the 4 conditions employed in the Dot Probe Task. Figure A displays an image of a LAN environment on the left and dark sleeping environment on the right. This is the LAN/Dark condition. Figure B displays two neutral stimuli. The image on the right is a dark neutral image. This is the Neutral/DarkNeutral condition. Figure C comprises of two neutral images. This is the Neutral/Neutral condition. Figure D comprises of dark sleeping environment on the left and a neutral image on the right. This is the Dark/Neutral condition.



Three attention bias indices were calculated for each participant. The first index was the conventional bias index (ABI). The ABI for each participant was calculated by taking the means response of trials in which the probe appeared at the location on neutral stimuli (incongruent trial RT) and subtracting the mean RT in which the probe appeared in the location of the threatening stimulus (congruent RT). A positive ABI score indicates faster RTs on congruent stimuli and longer RTs on incongruent relative. This finding is interpreted as vigilance for threat as an individual's attention is systematically drawn to the threat stimulus. A negative ABI score indicates longer RT congruent trials with shorter RT on incongruent trials indicating an avoidance of threatening stimuli.

Two other attention bias indices were calculated from dot probe test which are the orientation score and disengagement score. These scores are calculated using the average mean on Neutral/Neutral trials. It is argued that the Neutral/Neutral trials act as a baseline measure in that there is no threatening information to shift the participant's attention. Comparing trials which contain threatening information to trials which contain neutral only information allows for evaluation of components of visual attention such as orientation and disengagement, which cannot be found by using the ABI score**.** Orientation refers to the relative speed at which attention is drawn towards a threatening stimulus (Cisler & Koster, 2010). For orientation scores, the average overall mean for neutral/neutral trials was subtracted from the mean RT on congruent trials. A positive orientation score indicates that individuals were faster to respond to congruent trials compared to neutral trials indicating their engagement towards threatening stimuli whereas a negative score indicates slower engagement to threatening stimuli compared to neutral stimuli (Cisler & Koster, 2010). To calculate disengagement scores the mean RT for incongruent trials are subtracted from the mean RT for neutral-neutral trials. Positive scores reflect difficulty disengaging, indicating that the participant was faster to respond to neutral-neutral pairings than to the incongruent trials. Negative scores reflect eased disengagement, indicating that participants have no difficulty disengaging (see Table 5.2).

Table 5.2

Calculation and interpretation of indices of Attention Bias.

Attention Index	Attention Bias (AB)	Orientation (O)	Disengagement (D)
Formula	AB = IRT - CRT	O = NRT - CRT	DD = IRT - NRT
Interpretation of positive scores	+AB = Vigilance for threatening stimuli (Faster to respond on congruent trials than incongruent trials)	+O = Facilitated engagement for towards threatening stimuli (Faster to respond on congruent trials than neutral trials)	+D = Difficulty disengaging from threatening stimuli (Faster to respond on neutral trials than incongruent trials)
Interpretation of negative scores	-AB = Avoidance of threatening stimuli (Slower to respond on congruent trials than incongruent trials)	-O = Slow engagement towards threatening stimuli (Slower to respond on congruent trials than neutral trials)	-D = Eased disengagement from threatening faces (Slower to respond neutral trials than incongruent trials)

Note: IRT = mean reaction time on incongruent trials; CRT = mean reaction time on congruent trials; and NRT = mean reaction time on neutral trials.

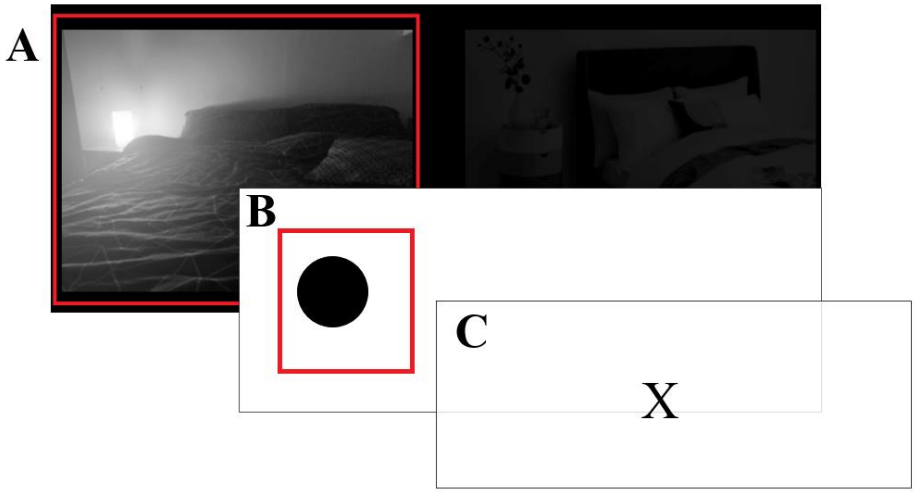
5.2.3 Procedure

Ethical approval was granted by Maynooth University Research Ethics Committee. Participants were recruited through the Department of Psychology's student participant pool. In this pool individuals partake in a number of studies across the academic year in return for course credits. A brief information sheet was posted on the participant pool information channel and interested students emailed the researcher to indicate their interest to participate. The researcher then sent an email to the students informing them of their participant ID, a link to the Qualtrics form which hosted the surveys and a link to the dot probe task. Participants were asked to complete the questionnaires first and then complete the dot probe task. Before participants completed the survey they had to read and agree to participate in the study. Participants were asked to complete the dot probe task in a quiet environment which did not have any background noise and were informed that they could only complete the cognitive task on a laptop. Participants were instructed through written instructions to be seated upright in front of a laptop/computer screen. Each trial began with a centred fixation cross that was presented for 500ms in the middle of the screen (see figure 5.2 for a schematic example of a trial from the DPT). Then two picture stimuli were presented simultaneously side-by-side for 2000ms with one image appearing on the left of the screen and the second image appearing on the right of the screen. Both images appeared 3cm apart on a computer screen. Immediately after the offset of the two picture stimuli, a visual target appeared (an "X") for 2000ms which replaced the space where one of the pictures were. Participants then had to indicate the probe location by pressing one of two buttons which corresponded to the side of the screen as quickly and accurately as possible on the laptop's keyboard. Participants pressed the "E" key with the left index finger when the probe was presented on the left location and the "I" key with the right index finger when the probe was presented on the right location. The probe would disappear after a response was made or after 2000ms. Before the test block individuals had to partake in practice block consisting of 10 trials. In this block direct feedback was be provided, in that if they made an incorrect response a red X appeared in the middle of the screen. If they made the correct response, they proceeded to the next trial within the practice block. After completion of the practice block the instructions of the task were presented again and participants pressed the

spacebar when they wished to start the task. The test block did not provide direct feedback. The test block comprised of 100 trials with 20 trials for each of the 5 stimulus pairings. This is a balanced design with each of the 5 stimuli per category being presented a total of 4 times per block trial. Once on the right with the probe following, once on the right with the probe not following, once on the left with the probe following and once on the left with the probe not following. The location of the threatening stimuli was counterbalanced with the picture stimuli being presented equally often at the left and right position and the order of trials were randomised for each participant. The reaction time (RT) of the participants' detection of the dot's location was recorded in milliseconds**.** Within each trial type there were two conditions of RT: congruent (threatening vs neutral, with dot replacing threatening) and incongruent (threatening vs neutral with dot replacing neutral). After completion of the task responses were automatically uploaded and stored by Qualtrics and Inquisit.

Figure 5.2

Schematic example of a trial from the DPT. Participants are presented two images. As denoted in A one image appears on the left and one image on the right. The example presented here is from the LAN/Dark condition. The LAN sleeping environment appears on the left and the dark sleeping environment appears on the right. After the presentation of the two images, a new screen appears with a dot appearing on either the left of the screen or the right of the screen. If the dot follows after where the threatening stimulus previously appeared this would be referred to as a congruent trial. Image B denotes a congruent trial. After a response has been made a fixation cross appears (denoted in c) and another trial is presented.



5.2.4 Data Analysis

The design of this study is quasi-experimental. All time-based variables from the MCTQ (MSFsc, average sleep duration and SJL) were decimalised (i.e, 8:30 became 8.50, 30 min became .50). As guided by Jansson -Frojmark (2013) data from the DPT was excluded if there were errors in trials or omissions. Response times that deviated from the participants mean score by more than 3 standard deviations were eliminated as outliers. This exclusion resulted in 6 (3.82%) participants being removed from analyses. In order to account for missing data and to minimise removal of whole participant data, pairwise deletion was employed when carrying out inferential testing. Independent variables were the perception of LAN and the perceived disruption of LAN. The dependent variables were the attention bias indices derived from the DPT (RT between congruent and incongruent trials, attention bias score, orientation score and disengagement score). 4x2 between-within groups ANOVAs examined RTs across each trial condition. Repeated measures ANOVA were used to examine differences in scores of orientation and disengagement across conditions. Pearson Product-moment correlations were conducted to examine associations between subjective measures and attention bias indices. Welch t-tests were employed to investigate between group differences on the perception of LAN on attention bias indices. Welch's t-test was employed due to the sample sizes being unequal between groups

The study sample size was estimated using a-priori power calculations based on it being important to detect effect sizes of moderate size (d=.5). These calculations indicated a required study sample of approximately 210 would be required to detect differences of a likely-to-be-meaningful magnitude. All statistical analysis was conducted using IBM SPSS (V25, IBM Corporation) and figures were created either on SPSS or Jasp (V 0.9.1.0, https://jasp-stats.org/).

5.3 Results

Descriptive statistics are provided in Table 5.3 which provide an overview of the each of the subjective scale measures.

Table 5.3

Mean, median and standard deviations for scores on the psychometric instruments used to assess psychological health, subjective sleep quality, daily cognitive failures, and sleep timing.

	Mean	95%CI	Median	SD	Range
Age (yrs)	25.57	[24.43-27.10]	22	7.99	18-51
Total CFQ	44.95	[42.34-44.34]	44	15.57	14-95
Total Score GHQ	31.81	[29.27-34.36]	30	15.22	0-84
GHQ A - Somatic Symptoms	7.78	[7.03-8.54]	8	4.50	0-21
GHQ B - Anxiety & Depression	9.98	[9.17-10.79]	10	4.82	0-21
GHQ C - Social Dysfunction	9.68	[8.98-10.38]	9	4.20	0-21
GHQ D -Severe Depression	4.36	[3.51-5.21]	2	5.08	0-21
Global PSQI	7.54	[6.92-8.15]	6	3.67	1-18
Average Sleep Duration (hh.mm)	8.02	[7.81-8.23]	8.95	1.27	2.79-11.83
MSFsc (hh.mm)	5.21	[4.98-5.45]	5.17	1.39	1.50-9.16
Social Jetlag (hh.mm)	1.11	[.97-1.25]	1.00	.80	0-3.75

5.3.1 Investigation of the distribution of scores across stimulus category

In the dot probe task individuals were exposed to 3 stimulus types (neutral images, dark sleeping environment images and LAN sleeping environment images). There were two types of neutral images (neutral and dark neutral). The dark neutral stimuli acted as a second control to ensure that if changes in RT to dark sleeping environment occurred it was due to the specific content and not the darkness of the image. This led to 3 categories of image which were LAN, Dark and Neutral. The

overall mean RT for each of these were calculated and can be seen in Table 5.4. These descriptives are derived from adding together the RT for each stimulus type and dividing by the number trial types they appeared in. For example, neutral stimuli appeared in four trial types ((i) Dark/Neutral trials, (ii) LAN/Neutral trials, (iii) Neutral/Neutral trials and (iv) Neutral/DarkNeutral trials). For Neutral/Dark neutral trials only neutral stimuli were included to calculate the overall mean RT for neutral stimuli. This resulted in adding together 4 trial types together and dividing the total score by 5. For LAN trials, LAN stimuli (LAN/Dark & LAN/Neutral) were added together and divided by 2. For Dark trials, dark stimuli (LAN/Dark, Dark/Neutral and Neutral/Dark) were added together and divided by 3). T-test analysis reported that there was no statistically significant difference in reaction times for the overall RT score for the dark bedroom trials (from LAN/Dark & Dark/Neutral trials) and RTs from the specific DarkNeutral trials, t = -.788, p = .432. Given no difference was found between RTs on sleep specific dark images and dark neutral images, the average of these 3 trial groups were to get an average RT for dark trials.

Table 5.4

Descriptives illustrating overall reaction time responses across stimulus categories

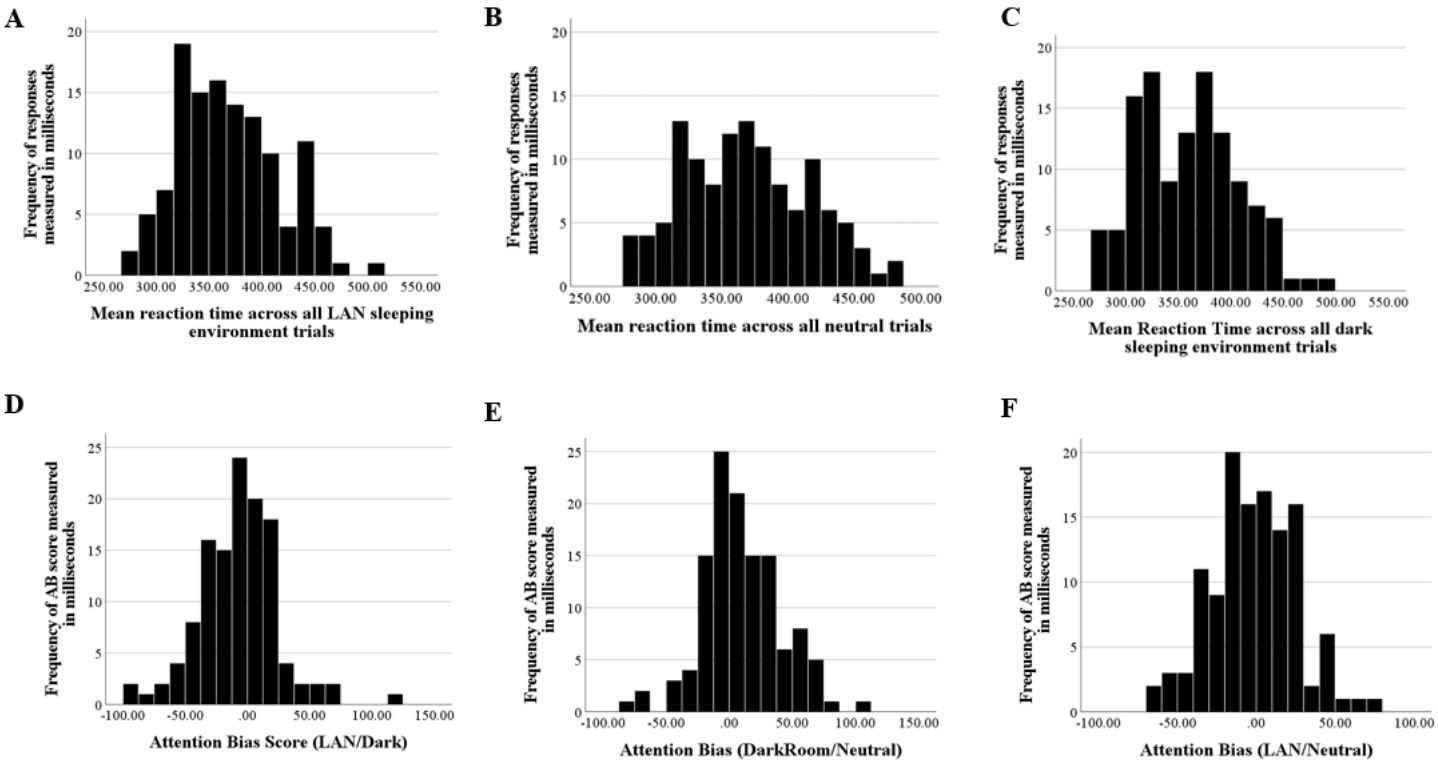
	Mean(SD)	CI(95%)	Median	Range
RT Neutral Trials	369.67(47.85)	361.06-378.28	367.73	277.36-485.40
RT LAN Trials	369.23(47.39)	360.73-377.72	363.31	280.80-512.55
RT Dark Trials	360.00(47.79)	351.44-368.57	364.03	270.89-497.07

Note. RT = Response time.

The mean RT for each of these stimulus types are presented in Table 5.4. The average RTs across the different stimulus types indicate that on average RTs are faster for dark stimuli while RT response times are similar for LAN and Neutral stimuli. The distributions of response times scores across each stimulus type are presented in Figure 5.3. Inspection of the histograms (see Figure 5.3) illustrates that across all stimulus types there is minor positive skew in the data. The level of kurtosis across each category type is slightly platykurtic indicating a flatter distribution of scores with this being more pronounced across neutral trials (-620). The range of scores appears similar across category types.

Figure 5.3

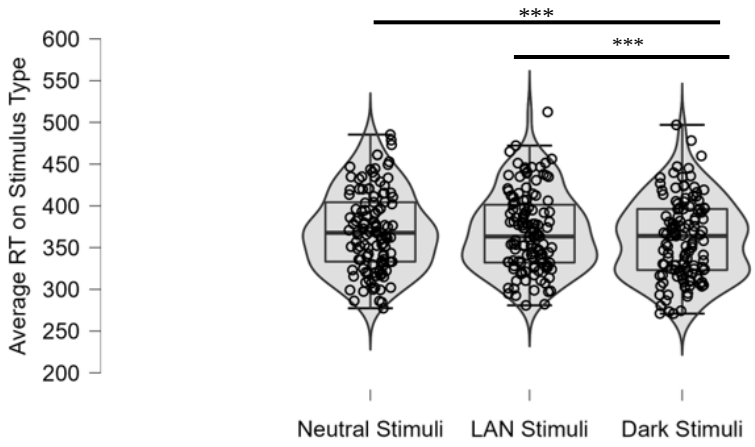
Frequency of reaction time across: all LAN sleeping environment trials (A), neutral trials (B) and dark sleep environment trials (C). Attention bias score for LAN/Bark (D), Dark room/ Neutral (E), and LAN/Neutral (F).



A one-way repeated measures ANOVA was conducted to compare RTs across the three stimulus types (Neutral, LAN, Dark). There was a significant effect for stimulus type, Wilks' Lambda = .742, $F(2, 121) = 20.668$, $p < .001$, with a large effect, multivariate partial eta squared = .258. Post-hoc comparisons using the Bonferroni test indicated that dark type stimuli (M = 360.00, SD = 47.79) had significantly faster response times than both neutral trials (M = 369.67, SD = 47.85, $p < .001$) and LAN trials (M = 369.23, SD = 47.39 $p < .001$). There was no significant difference in response times between neutral stimuli and LAN stimuli (p = 1.00).

Figure 5.4

Box-and-violin plots comparing average RT across the three stimulus types (Neutral, LAN and Dark)



Note. *** = $p < .001$

5.3.2 Examination of response latencies within trials

Table 5.4 outlines the average reaction time across each of the trial types and attention bias. Response times were analysed using a 4 (threat value: LAN/Dark; Dark/Neutral; LAN/Neutral; Neutral/DarkNeutral) X 2 (congruency: congruent/incongruent) repeated measures analysis of variance (ANOVA). Both variables were within subjects' factors. Notably, in this analysis Neutral-Neutral cannot be differentiated as being congruent or incongruent. The interaction effect between threat value and congruency was significant, Wilks' Lambda .787, $F(3, 119) = 10.730$, $p < .001$ however, the effect size was small (partial eta squared = .213). To investigate this interaction three separate paired samples t-tests were carried out (see Figure 5.5). In LAN/Dark trials there was a statistically significant

difference in response times with longer response times on congruent trials (M = 367.19, SD = 49.65) compared to incongruent trials (M = 360.10, SD = 49.83), t = 2.52, p = .013. The mean difference in reaction times between congruency type was 7.08 with a 95% confidence interval ranging from 1.54-12.64. However, the effect size was small as indicated by the eta squared statistic (.05). In the Dark/neutral trials there was a statistically significant difference in response times with faster responses observed on congruent trials (M = 361.05, SD = 51.87) compared to incongruent trials (M = 370.51, SD = 54.23), t = -3.350, p = .001. The mean difference in reaction time between congruency type was -9.47 with a 95% confidence interval ranging from -15.06- -3.87. However, the effect size was small as indicated by the eta squared statistic (.05). There was no statistically significant difference in response times between congruent and incongruent trials in the LAN/Neutral condition, t = .577, p = .565. In the Neutral/DarkNeutral condition there was a statistically significant difference between reaction times with longer reaction times for congruent neutral stimuli (M = 369.24, SD = 49.83) compared to incongruent neutral dark stimuli (M = 359.07, SD = 49.47), t = 4.06, p < .001. The mean decrease in reaction times between categories was 10.17 with a 95% confidence interval ranging from 5.20-15.14. The eta squared statistic (.11) indicated a small effect size. See Figure 5.5 for illustration of all differences. There was a main effect of threat value, Wilks' Lambda = .899, F(2, 120) = 6.72, p = .002; however, the effect size was small (partial eta squared = .10). Post-hoc comparisons using the Bonferroni test indicated that there was no statistically significant difference between any of the threat values.

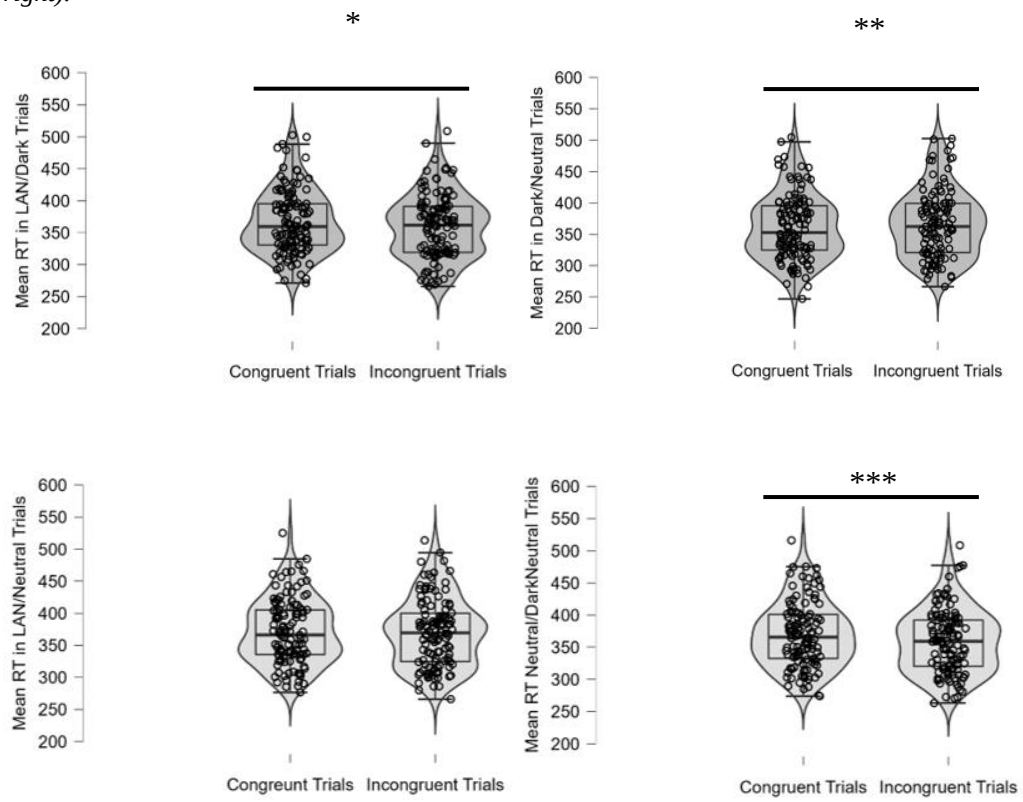
Table 5.4

Descriptive statistics for overall sample on reaction times for congruent and incongruent trials within each condition along with the attention bias index.

		Mean(SD)	CI(95%)	Median	Range
LAN/Dark	Congruent	367.19(49.65)	358.66-376.36	359.55	271.20-502.70
	Incongruent	360.10(49.83)	351.51-369.27	361.90	266.30-509.00
Neutral/Neutral	Congruent	372.88(50.74)	363.74-376.01	369.40	270.90-511.30
	Incongruent	366.92(48.58)	358.34-375.47	360.20	281.50-499.50
	Average	369.90(47.31)	361.38-378.41	364.11	276.55-484.35
Dark/Neutral	Congruent	361.43(51.83)	352.17-370.68	353.60	247-504.90
	Incongruent	370.93(54.20)	361.25-380.60	366.33	268.89-508.70
LAN/Neutral	Congruent	371.60(50.33)	362.62-380.58	366.70	276.56-525.20
	Incongruent	370.19(52.73)	360.77-379.60	370.30	266.0-513.80
Neutral/DarkN	Congruent	369.24(49.83)	360.35-378.14	366.90	274-516.10
	Incongruent	359.07(49.47)	350.24-367.90	359.30	263.20-508.40
LAN/Dark	ABI	-7.11(49.83)	-12.62- -1.61	-8.24	-91.30-118
Dark/Neutral	ABI	9.49(31.09)	3.94-15.05	6.7	-86.94-107.96
LAN/Neutral	ABI	-1.41(27.15)	-5.26-3.43	-2.79	-62.40-77.30

Figure 5.5

Mean reaction times in congruent and incongruent trials in the LAN/Dark trials (top left), Dark/Neutral trials (top right), LAN/Neutral trials (bottom left) and Neutral/DarkNeutral (bottom right).



Note. *** = $p < .001$, ** = $p < .01$, * = $p < .05$.

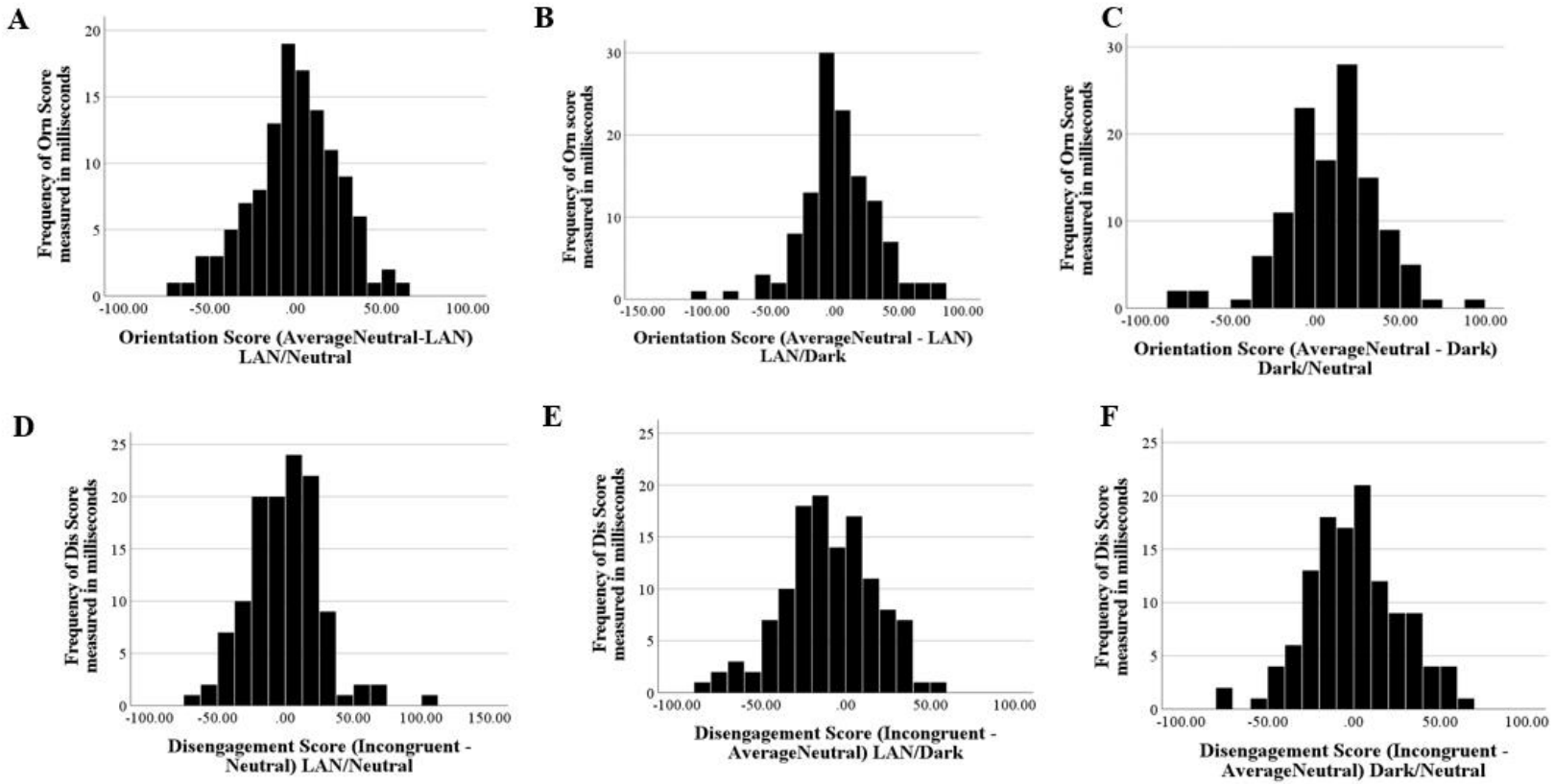
5.3.3 Examination of whether stimulus condition facilitates difficulty in disengagement or attentional avoidance of threat.

RTs on congruent and incongruent threatening trials were compared to the mean response times on the neutral trials to create an orientation score (average neutral response time – average congruent response time) and disengagement score (average incongruent response time – average neutral response time). The distribution of the orientation score and disengagement scores are presented in Figure 5.6. With specific references to the distribution of orientation scores it can be observed that in LAN/Dark trials (Figure 5.6B) the distribution of scores is positively skewed with individuals displaying a tendency to quickly draw attention to the LAN sleeping environments. This same response is not observed in

LAN/Neutral trials (Figure 5.6A). Inspection of Figure 5.6 indicates that orientation scores are more dispersed.

Figure 5.6.

Top row displays orientation scores for LAN/Neutral (A), LAN/Dark (B), Dark/Neutral (C). Bottom row shows disengagement scores for LAN/Neutral (D), LAN/Dark (E), and Dark/Neutral (F).



A one-way repeated measures ANOVA was carried out to compare orientation scores for congruency across the three conditions: (i) LAN/Darkroom, (ii) Darkroom/Neutral and (iii) LAN/darkroom. See Table 5.7 for descriptive details. Analysis found that there was a statistically significant effect of trial type on orientation scores, Wilks' Lambda = .882, $F(2,119) = 7.95$, p =.001 with a small effect size as indicated by the partial eta squared .052. Post-hoc comparisons using the Bonferroni test indicates that the mean orientation score for Darkroom trials (M = 8.94, SD = 28.59) is significantly greater than for LAN trials (M = -.92, SD = 25.22), p < .001 (Figure 5.7). This indicates that individuals facilitated their attention towards dark sleeping environment stimuli while individuals displayed slower engagement towards LAN sleeping environments. There was no statistically significant difference in orientation scores between LAN/Dark and Dark/Neutral (p = .096) and LAN/Dark and LAN/Neutral (p = .563).

Table 5.7

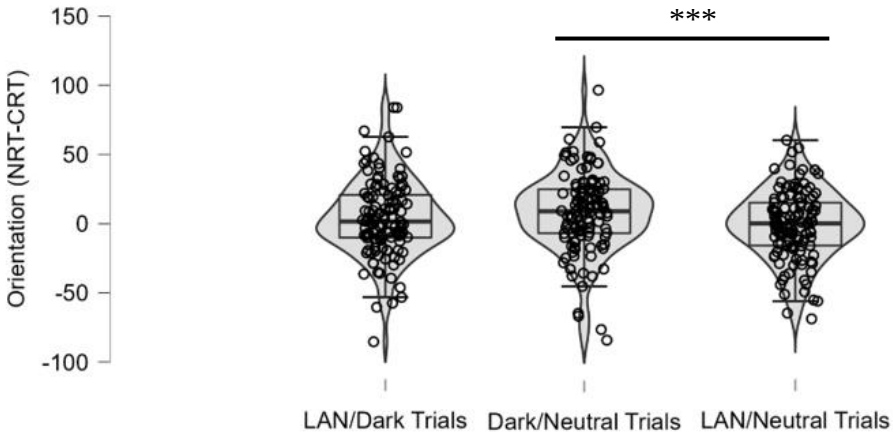
Descriptives of orientation scores

	Mean(SD)	CI(95%)	Median	Range
Orn-LAN/D	2.99(29.52)	-2.36-8.52	1.55	-110.20-84.05
Orn-Dark-	8.94(28.59)	3.79-14.08	9.95	-83.24-97.55
Orn-LAN	-.920(25.22)	-5.46-3.62	.15	-68.99-60.34

Note. Orn = Orientation

Figure 5.7

Box-and-Violin plot demonstrating the effect of trial type on orientation scores.



Note. *** = p < .001

A separate one-way repeated measures ANOVA was carried out to compare disengagement scores across the three conditions: (i) LAN/Darkroom, (ii) Darkroom/Neutral and (iii) LAN/darkroom. See Table 5.8 for descriptive details. Analysis found that there was a statistically significant effect of trial type on disengagement scores, Wilks' Lambda = .857, $F(2, 119) = 9.96$, $p = .001$ with a small effect size as indicated by partial eta squared (.143). Post-hoc comparisons using the Bonferroni test indicates that the mean disengagement score for LAN/Dark trials (M = -10.05, SD = 27.20) is statistically significantly different to Dark/Neutral trials (M = .0076, SD = 26.96, p = .001) and LAN/Neutral trials (M = -.17, SD = 26.77, p <.001). In the LAN/Dark trials the disengagement index was measured from the RTs of congruent LAN sleeping environment stimuli. This indicates that individuals displayed eased disengagement from LAN sleeping stimuli which was significantly different to disengagement scores on LAN/Neutral trials. Disengagement scores for Dark/Neutral and LAN/Neutral were not statistically different from each other (p = 1.00). See Figure 5.8.

Table 5.8

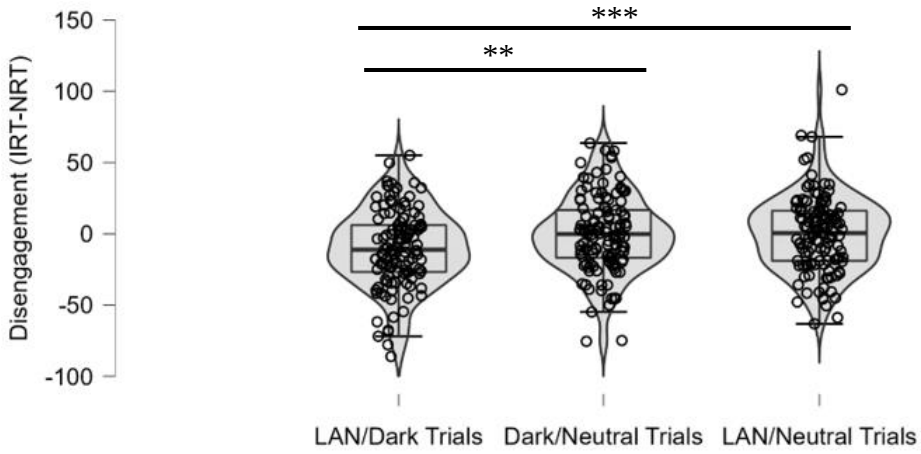
Descriptives of disengagement scores.

	Mean(SD)	CI(95%)	Median	Range
Dis-LAN/D	10.05(27.20)	-14.95- -5.15	-11.16	-86.05-55.07
Dis-Dark-	.0076(26.96)	-4.85-4.86	-.144	-75.45-63.71
Dis-LAN	-.17(26.77)	-4.98-4.65	.55	-63.15-101.14

Note. Dis = Disengagement

Figure 5.8

Box-and-Violin plot demonstrating the effect of trial type on disengagement scores.



Note. *** = $p < .001$, ** = $p < .01$.

5.3.4 Association between Attention Bias Index Scores, Orientation and Disengagement on sleep quality, circadian parameters & attitudes towards sleep.

Correlational analysis revealed no association between attention bias scores and scores on any of the sleep and circadian parameters. As can be seen from Table 5.9 no significant association were observed between attention bias score and the subjectively reported circadian parameters.

Table 5.9

Pearson Product-moment Correlations between ABI scores across trials and sleep quality, circadian parameters, and attitudes towards sleep

	AB LAN/Dark	AB Dark/Neutral	AB LAN/Neutral
Global PSQI	.018	.015	.012
MSFsc	.15	-.041	-.039
SD week	.027	.123	-.029
Absolute Social Jetlag	.036	.012	.012
DBAS	-.026	.103	-.071

Correlational analysis revealed no association between orientation scores and scores on any subjective sleep and circadian parameters (as can be seen from Table 5.10). Correlational analysis also revealed no association between disengagement scores and scores on any of the sleep and circadian parameters (as can be seen from Table 5.11).

Table 5.10

Pearson Product-moment Correlations between orientation scores across trials and sleep quality, circadian parameters, and attitudes towards sleep.

	Orientation Light/Dark	Orientation Dark	Orientation LAN
Global PSQI	.003	.004	-080
MSFsc	-.122	-.040	-.041
SD week	.019	.103	-.026
Absolute Social Jetlag	-.041	-.069	-.042
DBAS	.063	.113	.003

Table 5.11

Pearson Product-moment Correlations between disengagement scores across trials and sleep quality, circadian parameters, and attitudes towards sleep.

	Disengagement Light/Dark	Disengagement Dark	Disengagement LAN
Global PSQI	.825	.893	.343
MSFsc	.077	-.004	.000
SD week	.026	.031	-.005
Absolute Social Jetlag	.012	.088	.051
DBAS	-.084	-.004	-.073

5.3.5 Effect of physical and psychological wellbeing on indices of Attention bias

Based on the global GHQ score individuals were categorised as being either low psychological distress (<24) or high psychological distress (>24). A number of

Welch's t-tests were carried out to examine if there was a difference between those who were categorised as having psychiatric symptoms and those that did not on attention bias indices. As can be seen from Table 5.12 no statistically significant differences were observed between groups on attention bias indices.

Table 5.12

Descriptives alongside probability values from Welsh's t-test for the effect of psychological state on attention bias indices.

		Low Psychological Distress N(42)	High Psychological Distress N(92)		
		Mean(SD)	Mean(SD)	*F*	*P*
LAN/Dark	ABI	-6.07(35.39)	-7.75(29.36)	.064	.801
	Orientation	.02(25.06)	4.02(30.13)	.599	.457
	Disengagement	-5.86(28.44)	-11.81(26.69	1.18	.281
Dark/Neutral	ABI	9.57(30.55)	8.93(31.57)	.011	.916
	Orientation	7.95(25.04)	8.96(30.13)	.037	.848
	Disengagement	1.62(27.15)	-.80(27.17)	.208	.650
LAN/Neutral	ABI	5.05(26.55)	-4.21(27.37)	3.082	.083
	Orientation	1.03(24.81)	-2.01(25.58)	.385	.537
	Disengagement	3.97(28.04)	-1.73(26.11)	1.12	.293

5.3.6 Examination of attitudes towards LAN exposure and sleep disruption to sleep and attention bias indices

Within the current sample, nearly 74% (n = 112) perceive LAN to be disruptive to sleep. A series of Welch t-tests were carried out to examine whether there were differences between those that perceived LAN to be disruptive to sleep quality and reaction times for both congruent and incongruent trials across the 3 categories. As can be seen from Table 5.13 no significant differences were observed in reaction times between groups. Additionally, as can be seen from the Table 5.13 no significant differences were observed between groups on the index of attention bias (Figure 5.9), orientation, and disengagement (Figure 5.10).

Table 5.13

Descriptives alongside probability values from Welsh's t-test for the effect of light being perceived as disruptive to sleep on reaction times across trials and attention bias indices.

		LAN not disruptive to Sleep N(30)	LAN disruptive to sleep LAN N(92)		
		Mean(SD)	Mean(SD)	*F*	*P*
LAN/Dark	Congruent	365.53(40.12)	367.73(52.57)	.058	.810
	Incongruent	361.20(46.95)	359.74(50.97)	.021	.885
Dark/Neutral	Congruent	364.59(47.76)	359.89(53.35)	.207	.651
	Incongruent	368.30(47.63)	371.23(56.43)	.078	.781
LAN/Neutral	Congruent	372.38(41.49)	370.90(52.63)	.024	.878
	Incongruent	368.81(48.62)	370.18(54.35)	.017	.897
Neutral/DarkN	Congruent	367.26(45.33)	369.65(51.64)	.057	.811
	Incongruent	358.76(48.68)	358.91(50.19)	.000	.989
LAN/Dark	ABI	-4.56(35.95)	-7.98(29.54)	.217	.644
	Orientation	.125(23.62)	3.83(31.22)	.461	.500
	Disengagement	-4.43(25.09)	-11.82(27.73)	1.81	.183
Dark/Neutral	ABI	3.71(34.79)	11.35(29.92)	1.66	.286
	Orientation	.28(28.69)	11.67(28.16)	3.505	.061
	Disengagement	1.06(23.39)	1.06(23.39)	.070	.792
LAN/Neutral	ABI	-3.57(25.39)	.72(27.94)	.272	.604
	Orientation	-5.92(27.39)	.65(24.43)	.134	.253
	Disengagement	3.67(28.59)	-1.38(26.21)	.716	.402

Figure 5.9

Attention bias and the perception of LAN being disruptive to sleep in LAN/Dark, Dark/Neutral and LAN/Neutral trials

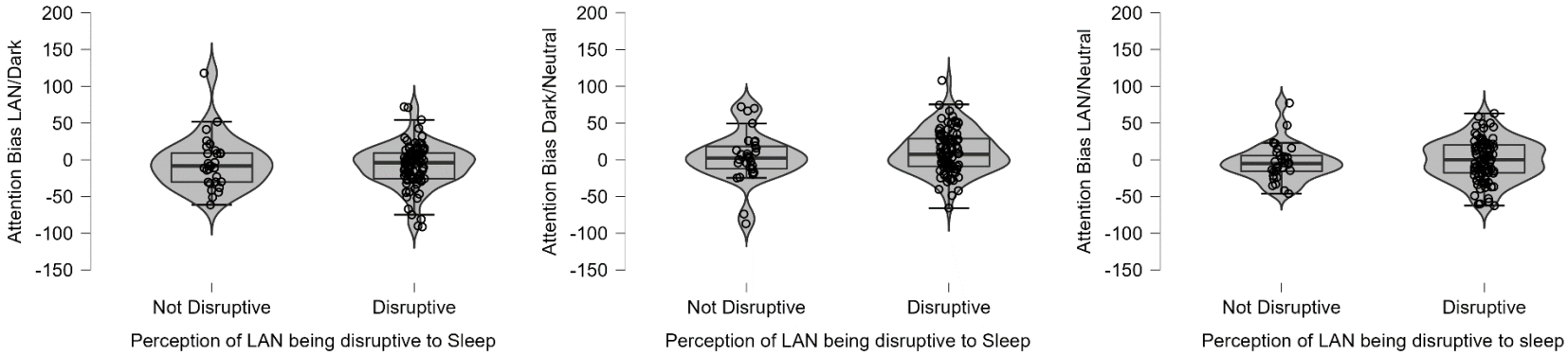
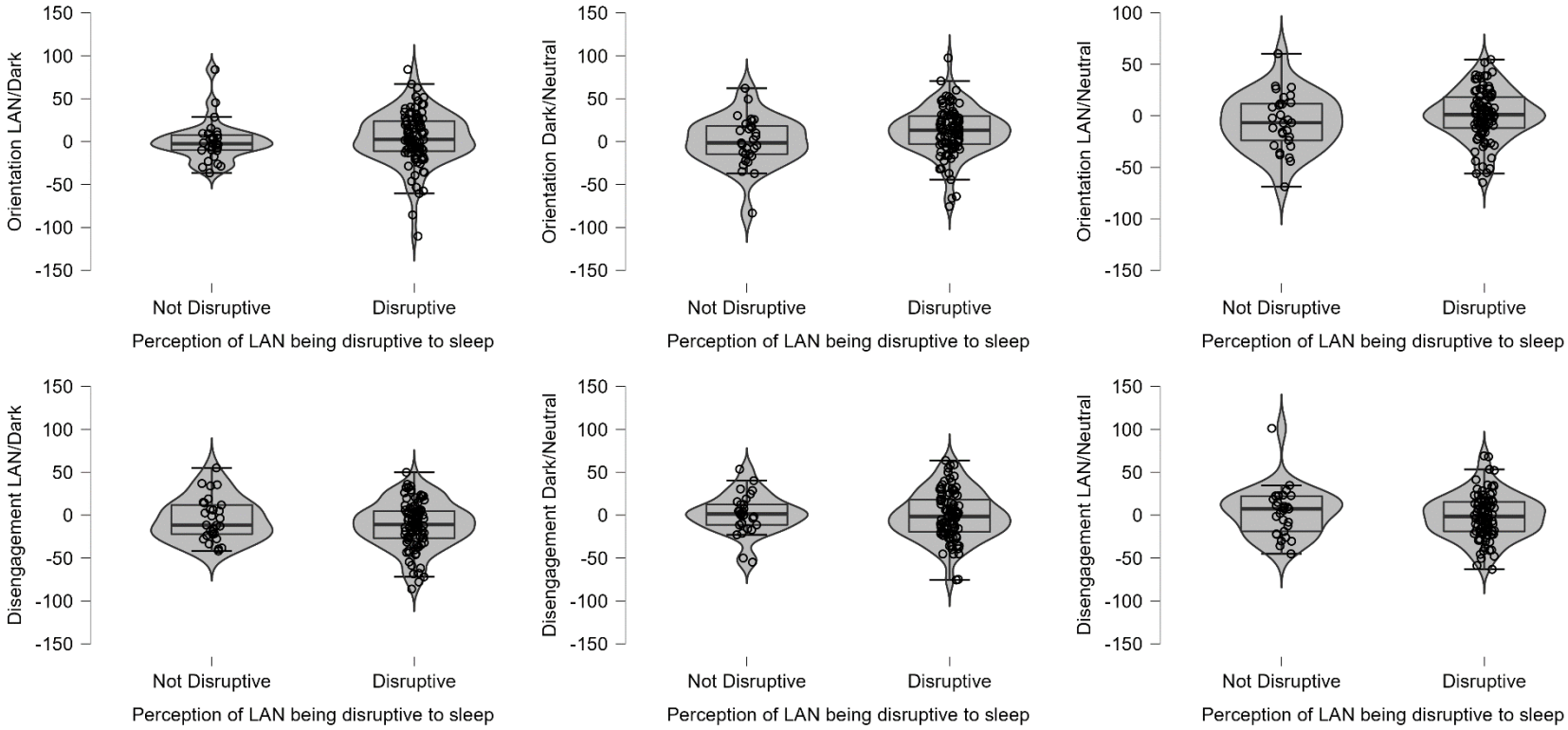


Figure 5.10

Orientation score (middle row) and disengagement score (bottom row), and the perception of LAN being disruptive to sleep in LAN/Dark, Dark/Neutral and LAN/Neutral trials.



5.3.7 Examination of the perception of external LAN trespassing into the bedroom environment on subjective measures and attention bias indices

Within the current sample, 38% (n = 41) reported that they subjectively perceived external light entering the sleep environment. A series of Welsh t-tests were carried out to examine whether there were differences between those that perceived external LAN entering the sleeping environment and reaction times for both congruent and incongruent trials across the 3 categories. As can be seen from Table 5.14 no significant differences were observed in reaction times between groups. Additionally, as can be seen from the Table 5.14 no significant differences were observed between groups on the index of attention bias (Figure 5.11), orientation, and disengagement (Figure 5.12).

Table 5.14

Descriptives alongside probability values from Welsh's t-test for the perception of external LAN entering the sleeping environment on reaction times across trials and attention bias indices.

		Perception of External LAN N(41)	No Perception of External LAN N(80)		
		Mean(SD)	Mean(SD)	*F*	*p*
LAN/Dark	Congruent	372.62(49.87)	364.33(49.61)	.763	.385
	Incongruent	364.84(49.85)	357.61(49.96)	.579	.449
Dark/Neutral	Congruent	365.19(50.18)	358.87(52.92)	.421	.518
	Incongruent	371.29(56.23)	370.10(53.50)	.013	.910
LAN/Neutral	Congruent	373.71(55.47)	369.98(47.84)	.137	.712
	Incongruent	373.91(52.14)	367.71(53.36)	.383	.538
LAN/Dark	ABI	-7.78(29.66)	-6.72(31.79)	.026	.872
	Orientation	-2.16(28.74)	5.56(29.76)	1.91	.170
	Disengagement	-5.68(27.17)	-12.29(27.11)	1.603	.209
Dark/Neutral	ABI	6.10(33.63)	11.23(29.950	.691	.408
	Orientation	4.87(24.13)	11.03(30.55)	1.46	.229
	Disengagement	-.38(25.30)	.21(27.93)	.014	.907
LAN/Neutral	ABI	.187(26.299)	-2.27(27.88)	.230	.633
	Orientation	-2.55(24.54)	-.08(25.67)	.266	.607
	Disengagement	3.77(22.61)	-2.19(28.59	1.56	.214

Figure 5.11

Attention bias and the perception of external LAN in LAN/Dark, Dark/Neutral and LAN/Neutral trials.

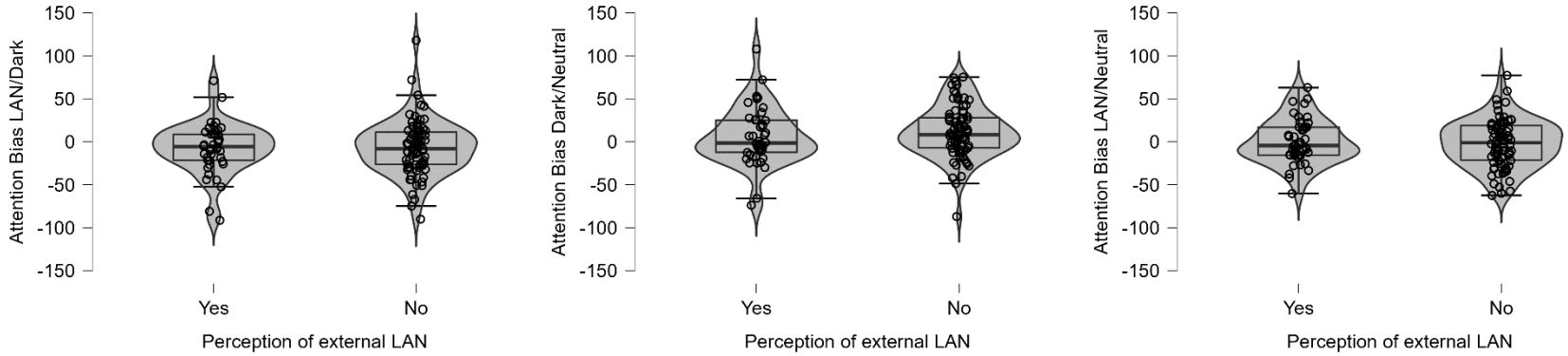
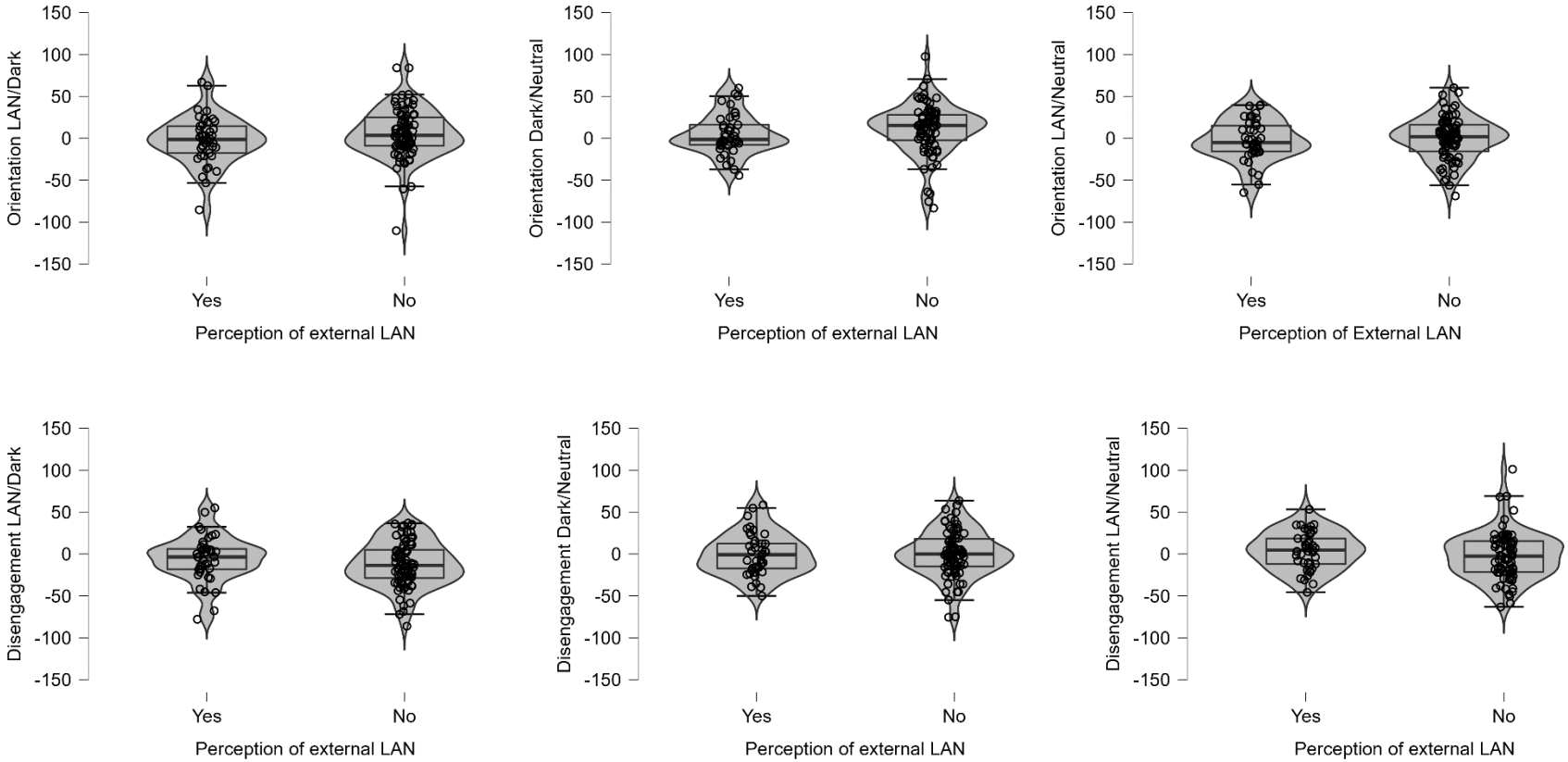


Figure 5.12

Orientation (top row) and disengagement (bottom row) and the perception of external LAN in LAN/Dark, Dark/Neutral and LAN/Neutral trials.



5.3.8 Examination of the perception of internal LAN trespassing into the bedroom environment on attention bias indices

27.50% (n = 34) of the sample indicated that they perceived LAN exposure trespassing into the sleeping environment from inside the house. A series of Welch tests were carried out to examine whether there were differences between those that perceived internal LAN entering the sleeping environment and those that did not on reaction times for both congruent and incongruent trials and attention bias indices across the 3 categories. As can be seen from Table 5.15 no significant differences were observed in reaction times between groups. Additionally, as can be seen from the Table 5.15 no significant differences were observed between groups on the index of attention bias (Figure 5.13), orientation, and disengagement (Figure 5.14).

Table 5.15

Descriptives alongside probability values from Welsh's t-test for the perception of internal LAN entering the sleeping environment on reaction times across trials and attention bias indices.

		Perception of Internal LAN N(34)	No Perception of Internal LAN N(88)		
		Mean(SD)	Mean(SD)	*F*	*p*
LAN/Dark	Congruent	363.23(45.97)	368.71(51.17)	.327	.569
	Incongruent	358.70(44.53)	360.64(51.96)	.042	.838
Dark/Neutral	Congruent	355.73(49.12)	363.07(53.04)	.526	.471
	Incongruent	362.47(43.31)	373.62(57.81)	1.334	.252
LAN/Neutral	Congruent	369.03(49.50)	372.13(51.00)	.094	.760
	Incongruent	366.86(50.77)	370.99(53.82)	.147	.693
LAN/Dark	ABI	-4.52(32.52)	-8.07(30.47)	.255	.616
	Orientation	1.19(31.86)	3.33(28.79)	.050	.823
	Disengagement	-6.42(22.06)	-11.41(28.89)	1.025	.315
Dark/Neutral	ABI	6.74(27.97)	10.52(32.48)	.410	.524
	Orientation	8.92(27.20)	8.95(29.24)	.000	.996
	Disengagement	-4.17(22.78)	1.57(28.33)	1.327	.253
LAN/Neutral	ABI	-2.17(23.92)	-1.14(28.57)	.41	.839
	Orientation	-3.15(24.16)	-.08(25.69)	.374	.543
	Disengagement	2.18(26.33)	-1.05(27.03)	.358	.552

Figure 5.13

Attention bias and the perception of internal trespassing LAN in LAN/Dark, Dark/Neutral and LAN/Neutral trials.

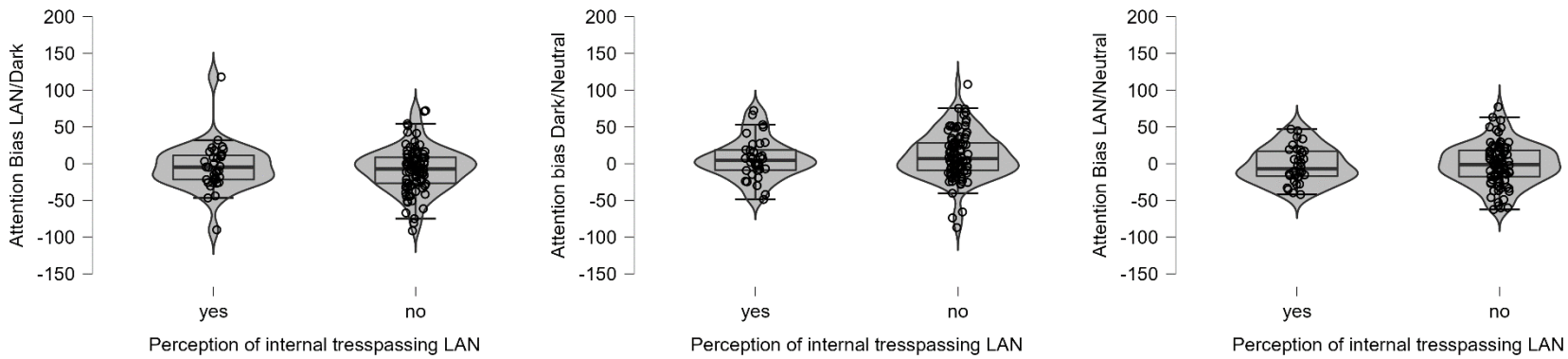
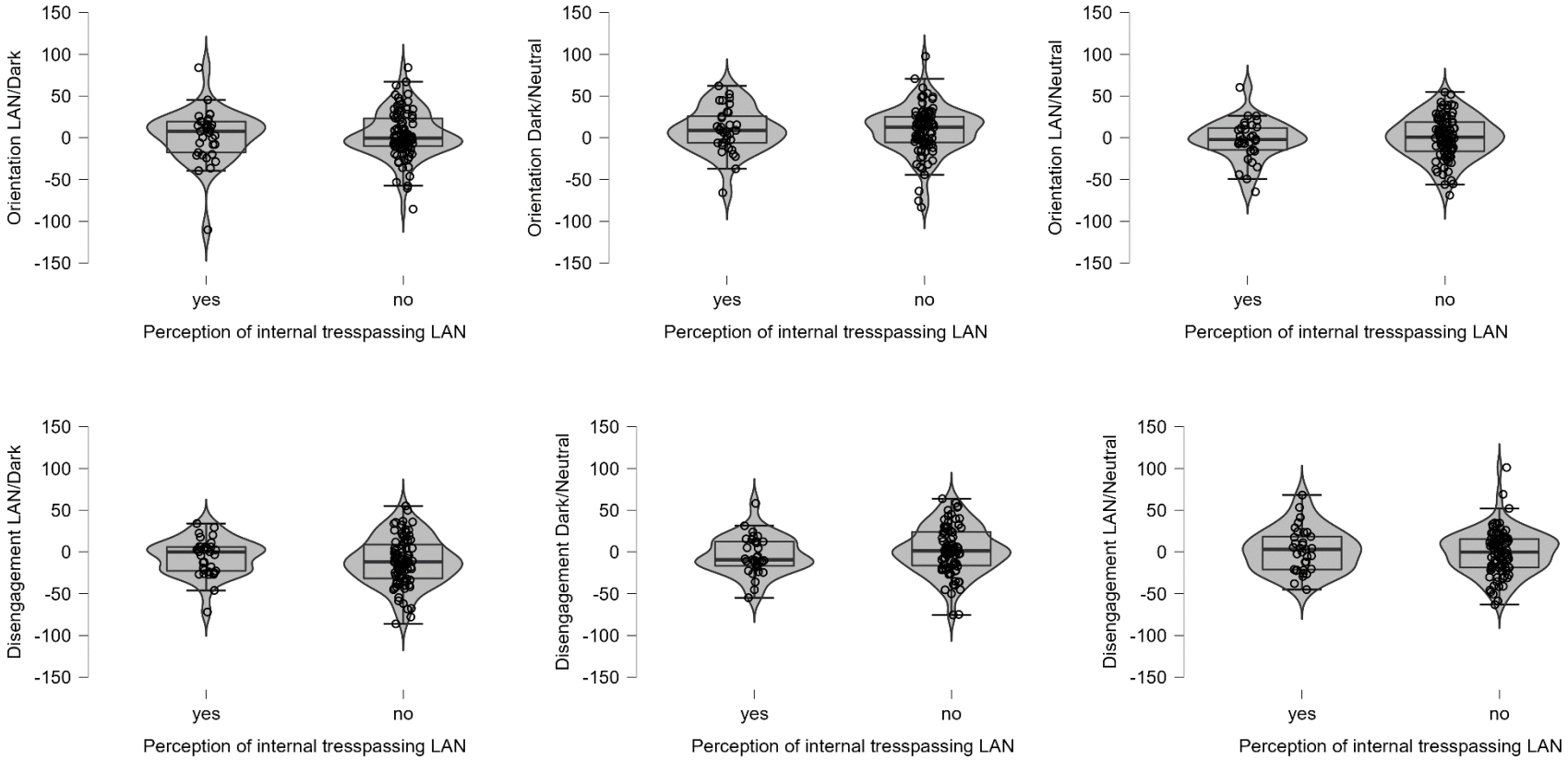


Figure 5.14

Orientation (top row) and disengagement (bottom row) and the perception of internal trespassing LAN in LAN/Dark, Dark/Neutral and LAN/Neutral trials.



5.4 Discussion

The present study examined whether the perception of LAN from specific sources in the sleeping environment was associated with an attention bias towards LAN sleeping environment stimuli compared to both neutral and dark room environment stimuli. Separately, this study attempted to localise and characterise the nature of attention bias with respect to vigilance for and disengagement from LAN stimuli. This was achieved by investigating scores of orientation towards and disengagement from sleep-specific stimuli compared to neutral-neutral images. Overall, our findings report that the perception of LAN from various sources in the sleeping is not associated with any indices of attention bias from the DPT.

The current study found no association between quality of sleep and attention bias scores either on LAN/Dark trials, Dark/Neutral and LAN/Neutral trials. These findings are consistent with other research studies which have reported no evidence of attention bias between individuals with insomnia/poor quality sleepers and normal sleepers while using the DPT (Jansson Frojmark et al., 2012; Lancee et al., 2017; Spiegelhalder et al., 2010; Takano et al., 2018). Our findings are however in contrast to previous research using the DPT which found evidence of attention bias towards image stimuli depicting tired faces (Akram et al., 2018) and sleep-related word stimuli (MacMahon et al., 2006). Although, Jansson-Frojmark et al. (2013) reported no evidence of attention bias using the traditional attention bias indices they did find evidence of difficulty disengaging away from stimuli depicting daytime fatigue/malaise relative to neutral-neutral picture presentations (Jansson-Frojmark et al., 2013). A plausible reason for the inconsistent findings between the current study and the aforementioned studies is due to participants being categorised as either primary insomniacs or being good/normal sleepers as measured by the ISI. This categorisation of insomniac symptoms may be the independent variable for between group differences on of sleep-related attention bias (Akram et al., 2018; Jansson-Frojmark et al., 2013; MacMahon et al., 2006). Evidence for this comes from MacMahon and colleagues (2006) who included a second control group comprised of individuals with delayed sleep phase syndrome (which is a result of a circadian timing disorder to which no cognitive pathway for is emergence and maintenance exists) to compare attention bias scores towards sleep stimuli. The results found that the DSPS group and good sleepers had comparable levels of attention bias however,

those with primary insomnia displayed significantly higher levels of attention bias compared to both control groups. This suggests that attention bias may be specific to clinical chronic insomnia and independent from sleep quality. In MacMahon et al.'s (2006) study although the PSQI score of individuals with DSPS were statistically different from that of good sleepers those with DSPS performed comparable to good sleepers. This indicates that attention bias may mediated independently of sleep quality as indexed by the PSQI.

Additionally, the current study found that neither DBAS scores, attitudes and perceptions towards LAN were associated with attention bias scores to dark or LAN sleep-related stimuli. The lack of association between pre-sleep worry and attention bias scores are in part consistent with Takono et al. (2018) who reported no association between pre-sleep arousal (somatic arousal and cognitive arousal) and attention bias scores using the dot-probe test. Our findings are inconsistent with the cognitive frameworks of sleep disturbance and insomnia (Espie et al., 2006; Harvey, 2002), which propose that individuals selectively monitor external cues in the sleeping environment that confirm wakefulness or are contributing to the disruption and initiation of sleep. In this study the specific perception of LAN in the sleeping environment is neither associated with poor attitudes and beliefs towards sleep or with attention bias indices to either dark or LAN sleeping environments.

The current study observed that for the overall sample, reaction times were significantly faster when the probe followed dark sleeping environment in dark/neutral and LAN/dark trials. However, no significant differences in reaction times were observed in LAN/Neutral trials. At first, these findings may align with Lundh and colleagues (1999) by suggesting that independent of sleep quality and attitudes towards sleep individuals exhibit an attention bias towards sleep-related stimuli. In the case of this study individuals may perceive dark sleeping environments as conducive for sleep. However, the observation that on neutral/dark/neutral trials there was a significantly faster reaction time when the location of the probe followed the dark neutral compared to neutral stimuli suggests that the darkness of the image may have a contributing factor for the placement of attentional resources and not the content. Previous research has argued that it is plausible that there is an interaction between the brightness of the visual stimuli leading to the misinterpretation of cognitive processes. In this sense dark images are not being attended to due to their threatening content but instead due to the darkness

of the image. Evidence for this argument comes Lankens and colleagues (2012) who reported that brighter pictures were evaluated more positively irrespective of the content of the picture in a phenomena they phrase “brightness bias”. Additionally, in a separate study brighter versions of neutral picture were evaluated more positively than darker version of the same picture (Lankens et al., 2013). Further evidence of brightness bias is provided by EEG recordings showing that apart from the content of the visual stimuli, the brightness of visual stimuli has an influence on neural activity (Eroglu et al., 2020; Eroglu et al., 2017). Redies et al. (2020) provide evidence that the properties of an image account for between 6 and 20% of the variance in the subjective ratings for valance and arousal. This indicates that affective pictures evoke emotions not only by what they show but they can also differ by how they show it. However, controlling for brightness bias for LAN sleeping environments stimuli is a methodological challenge given that LAN bedroom environment will by default be brighter in real life and brighter than an image of a dark sleeping environment. Taking into consideration the that the brightness of an image may interact with the cognitive processes of directed sustained attention and not emotional content calls into question whether the difference between groups in Akram and colleagues (2018) on tired faces is as extreme given that the tired faces had been purposively adjusted to have darkness around the eyes.

Another plausible reason for not observing an association between those that perceive LAN with indices of attention bias could be due to the current sample mainly comprising of moderately poor sleepers. It has been reported that within trials, moderately poor sleepers display no difference in time taken to detect sleep-related changes to an image compared to non-sleep changes to the same image (Jones et al., 2005). In comparison, those with extreme high scores on the PSQI display significantly faster times in detecting sleep-related changes to an image compared to non-sleep changes to the same image. For extreme good sleepers, the opposite is observed with them being statistically faster at detecting non-sleep specific changes to an image compared to sleep-specific changes (Jones et al., 2005). This indicates that attention bias may only exist at the extreme higher end of the PSQI and not in a subclinical categorisation. However, in Jones and colleagues (2005) study, between groups comparisons of only sleep-related change latencies

found a significant ordinal effect with poor and moderately poor sleepers being significantly faster at detecting sleep-related change compared to good sleepers.

Several studies specifically using image stimuli have provided evidence of sleep related attention bias in individuals with insomnia and those disturbed sleepers (Akram et al., 2018; Frojmark et al., 2013; Jones et al., 2005; MacMahon et al., 2006; Marchetti et al., 2006; Woods et al., 2009). However, the current study's findings are in contrast to the aforementioned studies with no evidence of attention bias towards either LAN sleeping environments or dark sleeping environments relative to neutral stimuli or to the LAN sleeping environment being perceived as more threatening than a dark sleeping environment. A possible reason for the lack of evidence could be due to the sleep-specific stimuli being non-affective or non-threatening. Two studies using the DPT and the EST have argued that the use of non-affective sleep-related word stimuli are possible explanations for no association between self-report sleep measures and attention bias scores being observed (Barclay & Ellis, 2013; Takano et al., 2018). This may suggest that the attention bias in those sleep disturbance is influenced more by affective valence of the stimulus than by the sleep-relatedness of the stimuli (image depicting a LAN sleeping environment). For instance, Akram and colleagues (2018) using the dot probe task observed that individuals with insomnia had greater attention bias for sleep-specific faces depicting tiredness compared to good sleepers. These depictions of tired faces may have been perceived as more negatively valanced compared to the sleep-related stimuli employed in the current study. In Akram and colleagues study individuals may attend to bodily sensations which provide evidence of impairment such as fatigue and tiredness as a result of having had a poor night sleep. Similarly, in Jansson-Frojmark and colleague's study (2013) the images may have elicited an emotional response about the lack of sleep through the use of images depicting daytime fatigue. The stimuli employed in this study may not have been considered as sleep-threatening. For example, a bed in a LAN sleeping environment may not appear as threatening. The potential impact of LAN exposure on delaying the onset of sleep or disrupting the maintenance of sleep may not be perceived as threatening or emotionally valanced by those who experience LAN in the sleep-environment however, the consequences of not having sleep may be perceived as more threatening. However, studies have reported evidence of sleep-related attention bias where the image stimuli content was specific to sleeping environment (e.g., a teddy

or a slipper) but would not be considered threatening or have a negative valence (Woods et al., 2009; Marchetti et al., 2006). However, it must be noted that in both of these studies the task employed were different to the task employed in the current study.

The stimuli employed in the current study aimed to be reflective of different bedroom environments due to limitations of previous research where the visual scenes were artificial (Marchetti et al., 2006; Woods et al., 2009). However, the current employed stimuli may still have lacked ecological validity as the images were static. Although, these images acted as a surrogate for the real-world sleeping environment, the viewing of static images may not be equally comparable with natural visual attention. In this sense, in the real-world individuals may allocate attentional resources differently when viewing a dark bedroom environment compared to a LAN sleeping environment. Tatler et al. (2011) argues that static images do not provide a space where the participant can act and as a result the attentional demands are likely to be different from actual natural behaviour. Furthermore, images may lead to systematic biases due to framing (the tendency to look in the centre of the image) and not being presented for a sufficient amount of time (Tatler et al., 2011; Tatler & Vincent, 2009). A number of research studies have provided evidence that getting participants to view video recordings of other eye-tracking recordings in different environments resulted in fixations in the continuous replay condition being a better predictor of gaze in the real world than static scenes (Foulsham et al., 2017; 't Hart et al., 2009). These findings indicate that static scenes do not stimulate attention in the same way as the real-world environment. Based on this, future studies may utilise recordings of bedroom sleeping environments which are separately dark or have LAN exposure and with the use of eye tracking to examine where individuals with poor sleep quality typically allocate their gaze and attention. This would build upon the previous work by Beattie et al. (2017) which provided evidence that those with insomnia displayed an attention bias to beds (greater number of fixations at the bed region and once fixated at the bed gaze remaining there for longer) when viewing various indoor scenes while their eye-movements were recorded in a free-moving task.

Participants in the current study undertook the tests during the daytime which may have impacted on finding no association between perception of LAN with attention bias indices. Evidence for this possibility come from Milkins et al. (2021)

who found that when individuals were tested during the daytime no relationship between attention bias scores and the experience of insomnia were found. However, when individuals were tested before sleep, it was found that attentional bias to sleep-related information predicted insomnia through its mediating influence on pre-sleep worry. Milkins et al. (2016) argue that a plausible reason for this difference in findings is that when examination occurs at pre-sleep the emotional salience of the sleep-related stimuli and the factors which contribute to poor sleep are more relevant to individuals when they are presented in close proximity sleep compared to during daytime when factors which are perceived to impact sleep are less threatening and the prospect of sleep is not problematic (Harvey & Greenall, 2003; Wicklow & Espie, 2000). This aligns with Harvey's (2002) cognitive framework of insomnia where the pre-sleep period is known to be critical with regard to cognitive arousal in insomnia and attentional bias for sleep-negative information is thought to be acutely problematic in the pre-sleep period (Harvey & Greenall, 2003). Future work examining the relationship between attention bias in LAN sleep environments and sleep quality may be best understood when attention bias is examined just before the individual's bedtime where the thought intrusions about the factors which impede on sleep are at their highest.

The non-clinical nature of the sample may explain the absence of attention bias. Many of the studies which have provided evidence of attention bias have been from samples which had individuals with primary insomnia (MacMahon et al., 2006; Woods et al., 2009). The sample included in this study was based on student population. Students may have greater flexibility to adjust work schedules compared to those that are in traditional forms of employment (Tankova et al., 1994). The impact of not sleeping at night may pose less of a threat to (and hence less of a focus of attention for) poor sleepers than would occur in a sample comprised of individuals in regular employment. As a result, this study may underestimate the degree of attention bias between good sleepers and poor sleepers and those that perceive LAN. Although there was a statistically significant mean difference in global PSQI scores between those that perceived external LAN and those that did not, the PSQI for good quality sleepers was high (M = 6.91) and near the cut-off point for categorising individual as a poor-sleepers. This may have meant that the between groups categorisation may not have been large enough to detect differences on attention bias. The high scores of PSQI may partly be due to the sample comprising of

university students whom generally have a high prevalence of significant sleep problems (Lund et al., 2010). In the current study, the presence of attention bias may be underestimated as the current sample may be more representative of having poor quality sleep leading to a lack of differentiation between true good quality sleepers and poor-quality sleepers which may impact on the potential to identify attention bias in the dot-probe task. However, the current study did find that there was a significant positive association between higher PSQI scores and heightened dysfunctional beliefs and attitudes towards sleep. However, DBAS scores are not associated with any of the attention bias indices in any of the sleep environment stimuli.

Our findings indicating no evidence of sleep-related attention bias are consistent with a small number of other studies (Lancee et al., 2017; Takano et al., 2018). This may suggest that the dot probe is insensitive to detect existing interindividual differences in attentional allocation to sleep related information (Schmukle, 2005). Additionally, the reliability of previous research findings observing differences between good sleepers and insomniacs on attention bias using the dot-probe is called into question given the small effect sizes found (Jansson-Frojmark et al., 2013). This could imply either a type 1 error or a means difference which is marginally small. Additionally, the poor psychometric properties of the dot-probe task may provide a possible valid reason for not observing attention bias scores between those that perceive LAN and or are poor quality sleepers. Some studies have indicated that the dot-probe task lacks reliability (Schmukle, 2005; Waechter et al., 2014) with Cronbach's alpha for attention bias scores in the range of .03-.25. Using sleep-related word stimuli Lancee and colleagues (2017) reported a split-half reliability of r = .06. In this case it may be possible that poor sleepers and those that perceive LAN in their sleeping environment may attend to sleep-specific stimuli over neutral stimuli, however, the dot-probe may have failed to detect it. However, it is important to note that Bar-Haim and colleagues (2007) in their meta-analysis observed that across different anxiety disorders that the dot probe task provided consistent evidence of anxiety-related biased processing with medium effect sizes. These findings are supported by additional meta-analysis specific to social anxiety which found that that the dot-probe test was sufficient to detect attention bias in clinically socially anxious individuals compared to healthy controls

(Bantin et al., 2016). However, one meta-analysis found no evidence of disorder-specific attention bias (Pergamin-Hight et al., 2015).

Meta-analysis examining attention bias in those with sleep disturbances and insomnia found that the dot-probe was one of the most sensitive attention paradigms for observing group effects of attention bias. Given the issues with reliability from the DPT some researchers have argued that the traditional attention bias index approaches attention bias as a static phenomenon providing only one summary index to represent allocation over the entire test (Kruijt et al., 2016). A number of researchers propose that attention allocation is a highly dynamic and highly variable with within trials individuals displaying phasic bursts towards and away from the threatening stimuli (Evens et al., 2020; Iacoviello et al., 2014; Meissel et al., 2021; Zvielli et al., 2014). However, the validity of these newer approaches has been called into question with Kruijt and colleagues (2016) arguing that these new indices may have both variability and measurement error combined with the possibility that significant group difference potentially being observed even if there is no attention bias present in the individual dataset. Kruijit et al. (2016) also argue that until it is clear what the dynamic measurement is precisely measuring that they should not be viewed as valid or reliable indices of attention bias (Harris et al., 2015).

There are several strengths to this study. Firstly, the research extends upon previous research which showcased that those with primary insomnia display an attention bias to general representations in the sleeping environment to specifically examining whether the perception of LAN is selectively monitored and perceived as threatening to the onset and maintenance of sleep. The study employed images which aimed to be ecologically valid and representative a typical sleeping environment. Secondly, the study examined attention bias in the general population instead of the clinical population meaning our findings are in part more reflective of the general population.

There are a number of limitations to the study. Although, the images employed aimed to be representative of sleeping environments the images employed were not validated. However, consideration was taken to ensure that the image were matched so the only content differences were in LAN and dark sleep environments was the presence of LAN sources. Sleep disturbances and quality was not measured objectively. The perceptual experience of LAN in the sleeping environment was assessed in a cross-sectional design where individuals had to think of their sleep

environments retrospectively. Future studies should employ objective measures of sleep environment LAN. All participants completed self-report measures prior to the DPT which might have introduced a priming effect, for example, by producing a greater sleep-related attention for the whole sample (Ellis et al., 2010). Although, the general sleep quality of the participants was measured it is unclear the level of sleepiness of the participant when completing the task. This may have impacted on the participants' response to the experiment. The DPT is typically employed to provide a measure of attentional allocation. This means that the reaction-time nature of this task is limited to an indirect measure of attention which assesses only indices of covert attention allocation (Marks et al., 2014). To overcome this limitation, future work should utilise eye tracking to examine when individuals examine dark and LAN sleep environments, do those that have sleep disturbance show heightened attention bias towards LAN stimuli as indexed by earlier and more frequent fixations compared to when viewing a dark sleeping environment. As discussed, later time of assessment may impact on establishing the presence of attention bias with greater saliency to sleep-related information pre-sleep compared to during the day. However, time of day may impact on levels of sleepiness. Sleepiness at the time when the task is being carried out could interfere with the responses on the task. Ree and Harvey (2006) report that those who were sleepy at the time of the experiment responded in an insomnia-consistent manner when presented with ambiguous stimuli.

In conclusion, the current study using a novel approach found no evidence that the perception of LAN with any of the indices of attention bias. This research builds upon an accumulating number of studies employing various cognitive paradigms which have failed to detect attention bias differences towards sleep-related information. Some researchers are suggesting that attention bias may not be a core feature of sleep disturbance/insomnia (Spiegelhalder et al., 2016). However, this study has highlighted some of the conceptual factors which may have led to no between groups differences in findings. When designing tasks significant consideration should be taken into the type of stimuli used, how stimuli are presented, and the type of participants recruited in order to examine attention bias differences.

Chapter 6

Examining the effect of window and bedside LAN intensity on sleep, rest-activity patterns and variance in mood and sleep.

Abstract

Exposure to LAN in the home-setting has now become commonplace. A small number of studies have demonstrated that the levels of LAN experienced in the home-setting exerts significant biological effects on the circadian system either through phase shifting of the circadian clock and melatonin suppression. Few studies have carried out ecological study designs to examine the effect of LAN intensity in the sleep environment on sleep and psychological health. This study examined whether higher levels of LAN intensity experienced in the bedroom environment were adversely associated with sleep timing, sleep quality, proxies of circadian disruption and psychological health. Additionally, this study examined whether higher levels of LAN intensity was associated with increased variance in day-to-day mood, subjective sleepiness and actigraphy derived sleep parameters. 30 individuals aged between 24-73 (*M*=32; *SD*=9.57) participated in this proof on concept study. Over a two-week period LAN intensity was objectively measured using light meters at both the window and bedside. Sleep and rest-wake activity was measured using actigraphy. Participants also completed daily monitoring of mood and subjective sleepiness along with completing a number of self-report questionnaires assessing psychological health and sleep quality. Our results found that higher levels of window measured LAN intensity was associated with delayed L5, sleep onset and MCTQ derived midsleep. We found that bedside LAN intensity during the in-bed period was not associated with timing of sleep, sleep quality or proxies of circadian disruption. Furthermore, we provide no evidence that LAN intensity experienced in the sleep environment is associated with negative consequences for psychological health. Our findings indicate that the level of LAN experienced at bedside may not be sufficient to elicit biological and behavioral responses in response to light.

6.1 Introduction

The results from chapter 2 indicates that LAN exposure occurs from a variety of sources in the sleeping environment. The intensity of LAN from these sources may be of a sufficient level to impact on sleep timing, sleep quality and circadian rhythmicity. Early experimental research primarily focuses on the effects of bright light on sleep and circadian rhythms. However, later findings demonstrated that in comparison to bright light, exposure to lower levels of LAN (100-200lux) was sufficient to induce half of the maximum phase delaying response and reduction in melatonin concentration (Gooley et al., 2011; Zeitzer et al., 2000). Recently, research has indicated that the human circadian system is highly sensitive to evening light at lower levels. Philips et al. (2018) showcased that on average exposure to LAN at levels of 24.60lux was sufficient to induce high levels of melatonin suppression. Additionally, LAN levels as low as 10 lux were sufficient to phase delay DLMO by 29 minutes with LAN intensity at 30 lux delaying DLMO by 68 minutes. This indicates that the human circadian system is highly sensitive to the dim evening light. Field studies have showcased that these are the levels of LAN intensity that are routinely found in sleeping environments (Obayashi et al., 2014).

Field studies have demonstrated that in comparison to living in natural environments where light exposure occurs from the solar cycle living in modern electrical environments is associated with delaying the timing and reducing the duration of sleep (Stohard et al., 2017; Wright et al., 2013). Cain and colleagues (2020) showcased LAN intensity remained consistent from the early biological evening up to 10pm. Studies have showcased that higher LAN intensity in the 3-4 hours before sleep can delay sleep onset and increased bout of wakefulness later that night (Cain et al., 2020; Obayashi et al., 2014a). These effects were observed independent of sleep quality, chronotype, sleepiness and bedtime. However, Cain et al. (2020) reported that these effects are observed only when LAN exposure are at higher levels relative to the average level of LAN for that evening. Other studies have provided evidence that higher levels of LAN intensity routinely present in home environment are associated with both a delay in objectively measured sleep and DLMO (Burgess & Molina, 2014; Esaki et al., 2021). Additionally, regardless of the spectral wavelength of the light, higher LAN intensity in the 4 hours before sleep were associated with lower levels of actual sleep during the night and increased awakening during sleep. This suggests that LAN intensity of light irrespective of

type of wavelength in the home-setting can adversely impact both the quality and timing of sleep. However, these findings contrast with a study which found that exposure to intermediate melanopsin activating light (which individuals are exposed to in home settings before sleep) is associated with increased melatonin suppression, decreased subjective sleepiness, increased latency to sleep onset, latency to REM sleep, latency to persistent sleep and latency to slow wave sleep compared to near darkness and blue depleted light (Santhi et al., 2012).

Although, observational studies have demonstrated that light intensity at levels of 25lux-30lux before sleep are sufficient to impact on sleep and circadian rhythmicity. Only a small number of studies have examined the effects of low-level dim LAN (5lux) during the in-bed period. The results from chapter 1 indicates that some individuals are exposed to LAN during the in-bed period either by external environment or internal dwelling LAN passing into the sleeping environment. Field studies which have both objectively measured LAN during the in-bed period or self-reported the presence of LAN sources while sleeping have indicated that higher LAN intensity is associated with insomnia, shorter sleep duration, increased awakenings after sleep, poorer actigraphic sleep quality, including decreased sleep efficiency and delayed mid sleep (Esaki et al., 2019; Esaki et al., 2020; Moon-Park et al., 2019; Obayashi et al., 2014). These effects were observed independent of bedtime, rising time, daytime physical activity and other demographics. However, it must be noted that two of the studies have been conducted on individuals with bipolar, and it has been reported that this population may have an increased sensitivity to light (Hallam et al., 2006).

To date, only a small number of studies have investigated the effects of low-level LAN during sleep, however, these studies have reported the level of light intensity on sleep. In two separate studies it was found that sleeping with either 5 or 10lux light during sleep impacted the quality, quantity and architecture of sleep as measured by PSG (Cho et al., 2016; 2018). Specifically, they observed that dLAN reduced total sleep time, sleep efficiency, stage R latency, increased WASO increase in stage N1 and stage R and a decrease in stage N2. However, exposure to 10lux light during sleep elicited a more pronounced increase in REM sleep compared to 5lux (Cho et al., 2018) This indicates that even at low light levels there are differences in the impact on sleep architecture. These increases in REM sleep due to low level LAN during sleep may be problematic for psychological health with

increased REM associated with depression (Palagini et al., 2013). Recently, Stebelova et al. (2020) reported that exposure to 1lux light during sleep impacted on sleep quality through increases in fragmentation while exposure to 5lux light also increased fragmentation and reduced melatonin biosynthesis.

The use of nocturnal animals has provided additional evidence indicating that dim LAN during sleep impacts on the molecular clock and rest and activity independent of sleep disturbance. Studies have indicated that chronic and acute exposure to dLAN is associated with alterations to circadian clock genes and proteins in the hippocampus, hypothalamus, and the liver (Bedrosian et al., 2013; Fonken et al., 2013; Shuboni et al., 2010; Walker et al., 2019) along with diurnal fluctuation of cortisol concentrations being blunted (Bedrosian et al., 2013). Specifically, dLAN exposure leads to suppression of Per1 and Per2 gene expression in the hypothalamus and protein expression in the SCN (Bedrosian et al., 2013; Fonken et al., 2013). However, Shuboni and Yan (2010) report that PER1 expression was increased in animals exposed to dLAN of 20lux. Chronic exposure to dLAN lead to most core clock genes being repressed in the liver (Foneken et al., 2013). However, the effect of dLAN on specific clock proteins in the hippocampus differed across studies. It is important to note that these alterations were observed independent of changes to locomotor activity rhythm (Bedrosian et al., 2013). However, Tams et al., (2021) argue that exposure to dLAN for 4 hours at the start of the biological night delays locomotor activity onsets, midpoints, and offsets, delays molecular rhythms in the hippocampus and other peripheral organs and modifies patterns of hypothalamic and cFos signals suggesting that at short period dLAN has adverse effects. Studies have reported that dLAN exposure can impact on the organization and architecture of sleep through decreasing the amplitude of daily rhythms of REM and NREM sleep and slow wave activity (Stenvers et al., 2016; Paanagiotou & Deboer, 2019).

Exposure to low level LAN in the sleeping environment has been found to be associated with depression. In two separate studies comprising of older adults it was separately observed that exposure to LAN greater than 5lux and increased duration of LAN greater than 10lux during the in-bed period was associated with higher risk of depression (Obayashi et al., 2013; Obayashi et al., 2018). These increased risks were observed after controlling for basic demographics, morning light exposure, evening light exposure, sleep disturbance and time in bed. The impact of exposure to

low-level LAN and its increased risk of depression is strongly articulated in Obayashi et al. (2018) findings which were based on a longitudinal design where individuals who displayed self-report depressive symptoms were excluded from participation. Separately, in individuals with bipolar, low-level LAN (<3lux) is associated with increased risk manic symptoms.

Both chronic and acute (<3 day) exposure to dLAN at levels of 5lux has been found to be associated with a depressive-like phenotype in animals (Bedrosian et al., 2013; Bedrosian et al., 2011; Bedrisoan et al., 2012; Fonken et al., 2013; Fonken et al., 2012; Fonken et al., 2013; LeGates et al., 2013; Taufigue et al., 2018; Walker et al., 2019). Either removal of dLAN exposure or administration of antidepressant medication has been found reverse depressive-like phenotypes elicited under dLAN exposure providing predictive validity that exposure to dLAN is a model for depression (Bedrosian et al., 2012; Taufigue et al., 2018). As indicated earlier several mechanistic causes have been proposed for effects of dLAN on eliciting depression which include reduction in neurotrophin signaling (Bedrosian et al., 2011; Fonken et al., 2011; Walker et al., 2019), increases in neuroinflammation (Bedrosian et al., 2013; Walker et al., 2019) and reduction in dendrite complexity, spine density and function in the brain (Bedrosian et al., 2011; Bedrosian et al., 2012; Taufigue et al., 2018).

Research has recently indicated that irregular sleep timing may be a significant predictor of poor health and quality of life outcomes (Chaput et al., 2020; Duncan et al., 2016). Variability in sleep timing may ensue due to impairment of circadian and homeostatic processes (Chaput et al., 2020). As previously indicated LAN may interfere with the homeostatic drive for sleep by increasing alertness (Cajochen et al., 2005; Chang et al., 2015; Vanderwalle et al., 200) which may in turn drive sleep to a later time or by phase delaying circadian phase markers (Gooley et al., 2011; Zeitzer et al., 2000). Research examining social jetlag has found that the greater the difference in midsleep timing between work and free days the greater the risk of developing poor psychological health (Levandovski et al., 2011; Taillard et al., 2021). There has been some evidence to suggest that evening LAN is a risk factor of increased levels of social jetlag (Wright et al., 2013; Skeldon et al., 2017). However, research to date has not investigate whether higher levels of in-bed light lighting is associated with increased variability in sleep timing, duration and efficacy

across the week. Additionally, it is unclear whether higher levels of LAN leads to increased variance in both subjective sleepiness and mood. In both clinical and non-clinical populations it has been observed that there can interdaily variation in mood which shift from periods of high mood to period of low mood. This contributes to variation in mood stability. Mood instability is a core feature of borderline personality disorder (Carr et al., 2018; Tsanas et al., 2016), however, mood instability has been observed in 14% of the general population (Anderson et al., 2012; McDonald et al., 2017). The possible risk factors and causes of mood instability are not well understood however, some research has suggested that disruptions to sleep which has been found to be prominent feature of psychiatric disorders may be a contributing factor to mood instability (Landgraf et al., 2014; Monteleone & Maj, 2008). Given the possible association between sleep disruption and mood (McCarthy & Welsh, 2012), it is surprising that no research to date has examined whether high levels of LAN experienced in the home setting are associated with variation in both daily mood and sleepiness. Chang et al. (2015) provided evidence that while exposure to e-readers reduced levels of sleepiness in the evening its use was associated with increased levels of sleepiness the following morning. As stated above it may be plausible that higher LAN intensity may lead to variation in sleep timing, duration and efficacy which may be associated with variation in mood and sleepiness. This comes from evidence which has found that in individuals with bipolar disorder diurnal variability in heart rate has been found to contribute to variability in subjective mood (Carr et al., 2018) and individuals with unstable rest-activity more likely experienced mood variability compared to those with stable rest-activity cycles (Krane-Gartiser et al., 2016).

While animal models have aided in providing a mechanistic understanding the effects of dLAN exposure on circadian biology and the possible direct and indirect effect LAN exposure has on psychological health these findings may not translate to humans. Firstly, photic sensitivity differs between humans and mice with dim light likely to be perceived relatively brighter. For instance, half maximal effective light intensity response for human phase-shifting response is more than one log-unit lower in mice despite duration of light exposure required to induce this response being significantly shorter in mice (Gooley et al., 2010). This difference photic sensitivity is more pronounced hamsters (Glickman et al., 2012) primarily due to these animals needing to be sensitive towards the photoperiod for survival. This

renders issues with the reliability of previous studies and may indicate that low-level LAN effects are more pronounced in animals compared to humans. Additionally, differences with mice are observed in the geometry of the eye, the number of cone photoreceptor types, the total number of photoreceptors and biology of the eye which may potentially impact on both the sensitivity of light at different intensities and wavelengths (Tams et al., 2021; Walbeek et al., 2021).

Previous work examining the effects of low-level LAN during sleep are not without the methodological shortcomings. Many experimental studies to date examined the effects of one or more monochromatic light exposures environments (Gooley et al., 2010; Thapan et al., 2001; Lockley et al., 2003) which is different to the polychromatic light exposure which individuals are exposed to in their home environments (Cain et al., 2020; Santhi et al., 2012). The proximity of the light source in relation to the participant is controlled (Cho et al., 2018; 2016) which in real life settings the light sources may be of some distance from the participant. The presentation of light stimulus occurs at times which may not be aligned to the timing of exposure in real life. For example, in some studies the presentation of light occurred after the habitual bedtime (Brainard et al., 2008; Lockley et al., 2003) or LAN exposure is ceased before sleep (Santhi et al., 2012). The effects observed may be oversensitive as in some cases individuals are exposed to very dim light for prolonged period before light exposure is administered (Gooley et al., 2010). This is problematic as if individuals are exposed to dim light for a significant period their non-visual response may be more extreme compared to if they were exposed to bright light during the day (Hebert et al., 2002; Smith et al., 2004; Chang et al., 2011). Studies may be conducted in a sleep laboratory environment for a short period with sleep monitoring devices which may be both more disruptive to individuals sleep routine and/or not reflective of individuals typical sleep patterns (van de Water et al., 2011). These shortcomings of previous experimental work may lead to reliability issues in understanding the effects of low-level LAN on sleep which individuals are routinely exposed to in their sleeping environments.

To date, only a small number of field studies have addressed the effects of low-level LAN during sleep in the home environment and its association with sleep and depression. These studies, however, have been predominantly confined to two research groups which focus on elderly adults (Obayashi et al., 2013; 2014a; 2014b 2018) or individuals with bipolar (Esaki et al., 2019, 2020; 2021). The

generalisability of the findings in older adults may underreport the effects of LAN on sleep as older adults may become less sensitive to light. Research indicates that non-image forming light responses are significantly diminished in older individuals compared to younger adults (Chellappa et al., 2021). Conversely, individuals with bipolar disorder may be highly sensitive to light (Lewy et al., 1985). Given the results from the previous chapters, along with the published field studies, low-level LAN commonly occurs during sleep and there is a need to examine whether this impacts on sleep and psychological well-being in a younger age cohort. Taking into consideration the limitation of self-report bias in chapter 2 and 3 of this thesis, the current study objectively measured bedroom environment light intensity over a two week period. In addition to this actigraphy was utilised to provide an objective charting of sleep patterns and the rest-activity cycle to examine whether LAN intensity is associated with adversely impacting sleep and concurrently negatively impacting on psychological wellbeing. The study also examined whether LAN intensity is associated with variation in rest-wake activity, sleepiness, and mood. The current study has three hypotheses:

1. Higher levels of bedside and window LAN intensity will be associated with delayed sleep timing, shorter duration, and poorer sleep efficacy.
2. Higher bedside and window LAN intensity will be associated with later L5 and M10, higher values of intra-daily variability and lower values of inter-daily stability.
3. Higher LAN intensity will be associated with increased variance in daily ratings of subjective mood and sleepiness.

6.2 Method

6.2.1 Participants

A total of 33 participants were recruited to participate in this study. Individuals who were shift workers, had a history of psychiatric illness, had a clinical sleep condition or were on medication were excluded from participation. At earlier stages of the study only those with an android smartphone were eligible to participate. Due to incomplete data (n = 2) and a participant having an atypical sleep-wake schedule (n = 1) only 30 participants were included in analysis. Of the 30 participants 53.3% (n = 16) were male with the age range of sample being 24-73 years (M = 32.33, SD =9.57). Participants were recruited through means of convenience and snowball sampling. This was achieved by recruiting through personal contacts who had not participated in previously participated in the previous studies. Participants were also recruited through contacts of those participating in the study. Participants received no renumeration for participation in the study. Ethical approval for this study was granted by the Ethics Review Board at Maynooth University.

6.2.2 Materials & Apparatus

6.2.2.1 Light at Night Survey

Participants completed a number questions assessing their attitudes towards to light at night exposure on their sleep quality. The survey also examined what are the specific sources of LAN exposure in the sleeping environment and to what degree these specific sources are perceived as disruptive to sleep.

6.2.2.2 Sleep quality & chronotype measures

The PSQI and the MCTQ both previously described in chapter 3 were completed by participants. A global PSQI score was derived from the PSQI measuring a participant's level of self-reported sleep disturbances over the previous months. A PSQI score >7 was used to differentiate between 'poor sleeper' and 'good sleeper.' In the current study, the Cronbach alpha coefficient was .56. From the MCTQ individuals average sleep duration was calculated. Individual's sleep debt corrected mid-point of sleep on free-days (MSFsc) was derived as a marker of circadian phase of entrainment. Social Jetlag was calculated as a typical measure of recurring circadian misalignment as previously described.

6.2.2.3 Attitudes towards sleep

Sleep related cognitions were measured with the DBAS-16 which has been described in the previous chapter. The DBAS-16 consists of 4 subscales representing types of sleep-related cognitions: perceived consequences of insomnia, worry/helplessness about insomnia, sleep expectations and medication. Higher scores on the DBAS indicate greater dysfunctional beliefs about sleep. In the current study, the Cronbach's alpha was .82.

6.2.2.4 Physical and psychological wellbeing

The GHQ-28 which has been previously described was employed to measure psychological well-being. The GHQ comprises of 4 subscales which measure: Somatic symptoms, anxiety and insomnia, social dysfunction and severe depression. Higher scores indicated poor levels of psychological wellbeing. In the current study, the Cronbach alpha coefficient was .933. The CFQ was employed to evaluate individual's propensity towards mistakes or errors in cognition. Higher scores indicate higher levels of errors in everyday cognition.

6.2.3 Light Measurement

Illuminance levels was measured using a TR-74Ui illuminance UV recorder (see Figure 6.1). An illuminance UV sensor is attached to the recorder which measures levels of illuminance. The recorder is a data recorder which simultaneously measures and records illuminance. The unit of measurement is lux with a measurement range of 0lux to 130,000lux. The accuracy of the device was ±5lux. The data logger has a storage capacity of up to 8,000 readings. Once the period of data collection is completed the data logger is connected to a windows computer via a USB which has the "Illuminance UV recorder software" installed. From here the raw light illuminance is downloaded into a Microsoft Excel file.

Figure 6.1

Image of the Illuminance UV recorder (far left) which logs the data and UV sensor (far right) which measures light illuminance.

Bedroom light intensity was measured during the night-time for the two-week period at 5-minute intervals using the illuminance recorder. For this study, two illuminance UV recorders were used for each participant. One of the UV sensors was attached at eye level (sensor facing outwards) either to the side of the bed frame or on the wall of where the participant usually slept at eye level. This position was chosen so to approximate the amount of LAN exposure that was reaching eye level. The recorder was placed on the ground with the display screen facing downwards so that light recordings could not be perceived by the participant and they would not adjust their lighting levels. A second sensor was placed at the bedroom window. Before attaching the meter, the researcher closed the blinds/curtains to see where trespassing light came from. Once the area was placed at that location with the sensor was placed facing outwards towards the window. Curtains and blinds were opened and closed a number of times to ensure that the sensor was affixed correctly and would not become detached. The recorder was placed facing downwards either on the window ledge or at the side of the window. The average light intensity between 12.00am and 4am for each night was the parameter a LAN exposure. These times were chosen to ensure that the illuminance levels collected were reflective of artificial LAN exposure and not solar light. Although, previous studies have measured average LAN intensity between bedtime and rising time (Esaki et al., 2020; Obayashi et al., 2015) this can include natural light exposure which has been

found to associated with a higher prevalence of sleep disturbances independent of post-bedtime light exposure with these effects most pronounced in evening types (Obayashi et al., 2019). The average light intensity for the duration of the study was calculated separately for both bedside light and window light. Each of these LAN sources were categorised as either low LAN or high LAN based on a median split. Previous studies have used categorised LAN exposure based on quartile groups (Obayashi et al., 2019; Obayashi et al., 2015) but given the small sample size of the current study a median split was utilised.

6.2.4 Objective Sleep and Circadian Measures

Actigraphy was used to provide an objective measure of sleep timing, duration, efficacy, and activity derived circadian rhythm parameters during the duration of the study. Actigraphy passively monitors activity via a wrist-worn device. This allows for a non-intrusive estimation of circadian patterns and sleep-wake parameters (Ancoli-Israel, 2003). Actigraphy affords several advantages. From the outset actigraphy is an objective, cost-effective, reliable and non-intrusive tool to measure sleep and circadian parameters. Actigraphy affords the collection of data over a long period of time from ecologically valid environments. Actigraphy has been validated as being as accurate as PSG in measuring sleep parameters including SE, SOL, WASO, and TST (Kaplan et al., 2012) and has been found to provide an accurate method for measuring sleep quality in healthy human studies (Littner et al., 2003). These estimates of sleep derived from actigraphy have been found to provide more reliable measure of sleep estimation compared to self-report sleep timing (Gerschik et al., 2012; Lauderdale et al., 2009). This is of importance given that those with either chronic insomnia or poor sleep quality may misperceive their sleep and overestimate the level of sleep disturbance (Harvey & Tang, 2012; Rioux et al., 2006).

The current study used two different versions of actiwatch (MotionWatch 8 and ProDiary Actiwatch AW4, CamNtech LTD., Cambridgeshire, UK). The actigraph is an electronic device which contains a piezo-electronic accelerometer which measures the intensity, amount and duration of physical movement. The accelerometer produces an electrical signal when the participant wearing the device is in motion. After motion is transduced into an analog electrical form, the samples are collected from this continuous signal at the rate of 50 hertz which is 50 samples per second. This means that movement being recorded as activity counts. The

extracted information is then stored as data points at 30 second intervals/epochs. This epoch was chosen as it is the recommended setting for sleep analyses when using the MotionWare algorithm to calculate sleep estimates. A medium activity threshold (20 activity counts) was selected as it is the validated threshold for the MotionWatch system. Participants are asked not to remove the device during the recording period except when exposed to water. Participants were asked to indicate in the sleep diary when and the duration they removed the actigraph for in the sleep diary. Additionally, activity free intervals greater than an hour that were not indicated in the removal diary were treated as suspicious for device removal and were also eliminated from the analysis. Each day participants also completed the Consensus Sleep Diary (CSD; Carney et al., 2012) which outlined the participants bedtime, wake-time, along with estimates of sleep quality, number of awakenings along with detailing other comments relating to their sleep. All analyses excluded the first and last day of day of study. Days which did not include the full 24-h period were not included in analysis.

6.2.5 Sleep Measurement

Sleep quality, timing and duration was examined with the Actiwatch sleep analysis program provided by CamNtech LTD (MotionWare version 1.2.5). This software has an algorithm which automatically scores sleep and wake. The scoring algorithm has been compared with polysomnography with the two measures strongly correlating with each other (Kushida et al., 2001). As indicated above participants competed a Consensus Sleep diary (CSD; Carney et al., 2012) each morning which indicated the time the participant fell asleep, their wake-up time. The CSD was used when scoring sleep timing on actigraphy on the Actiwatch sleep analysis program. Specifically, when examining the actogram sleep onset and offset were cross-referenced with the sleep-diary. Once alignment of sleep onset and offset was selected the MotionWare automatically performed a categorisation of each epoch of the period between ‘Fell Asleep’ and ‘Woke Up’ as either ‘Sleep’ or ‘Wake’. The estimates derived from the actigraph data were sleep onset, sleep end, total sleep time (TST), time in bed (TIB), sleep efficiency (SlpEff), number of wake bouts, mean wake bouts (WASO) and fragmentation. Table 6.1 provides an overview of how each of these estimates are operationalised. From this variability in sleep onset, sleep end, total sleep time, sleep efficacy was calculated by working out the standard

error of the mean over the number of days participants wore the actigraph for (Kelly et al., 2022).

Table 6.1

Overview of how estimates from actigraphy sleep analysis were operationalised.

MSF	The midpoint of sleep which is a proxy of chronotype. From actigraphy it is calculated as the midpoint between sleep onset and sleep end. The computation is time of sleep onset + total sleep time/2.
Assumed sleep	The total elapsed time between the "Fell Asleep" and "Woke Up" times.
Actual sleep time	The total time spent in sleep according to the epoch-by-epoch wake/sleep categorisation.
Wake bouts	The number of contiguous sections categorised as wake in the epoch-by-epoch wake/sleep categorisation.
Sleep efficiency (%)	Actual sleep time expressed as a percentage of time in bed.
Fragmentation Index	This is an indication of the degree of fragmentation of the sleep period and can be used as an indication of sleep quality (or the lack of it).

6.2.6 Circadian Measures

Non-Parametric Circadian Rhythm Analyses (NPCRA; Motionware 1.2.31, Cambridge Neurotechnology, Fenstanton, Cambridgeshire, UK) were performed using rest-activity data to assess actigraphy derived circadian rhythm parameters. The algorithms which determine these variables are outlined by Van Someron et al. (1999). To calculate NPCRA the full continuous 24-h days of monitoring were required. If the actigraph device was removed for greater than 2 hours that day was

no considered in the NCPRA. Both inspection of the sleep diary and visual inspection of the actogram indicated when the actigraph device was removed.

Intradaily variability (IV): provides information on the variability of the rhythm within a day by providing an estimation of the fragmentation of the 24-h rest-activity cycle within the 24-h day. This provides a measure of the consolidation of continuous sleep and wake states. Values range from 0-2. Low values indicate that the transition between rest and activity are tightly consolidated. Higher values are indicative of random fragmented transitions between rest and activity. High IV values occur as a result of large hourly differences in the activity rhythm such as rest and inactivity during the normal active period (i.e. napping during the day) or greater nocturnal activity from awakenings or other sleep disturbances (Witting et al., 1990).

Interdaily Stability (IS): provides an estimation on the day-to-day stability of the rest-activity rhythm across the period of the study. IS values range from 0 to 1. Higher IS value indicates that the pattern of stability is repeated perfectly each day and indicates a more stable rhythm. A low IS may occur due to poor between day consistency if the rest-activity rhythm due to an entrainment deficit or pressure from the social clock. For instance, individuals who work alternating shift work schedules show decreased rhythm stability (Rea et al., 2008).

Relative Amplitude (RA): indicates the amplitude/robustness of daily rest-activity rhythms. It measures the difference in mean activity levels over the most active consecutive 10h period (M10) compared to the least active 5h period (L5). Values range from 0-1. Higher values indicate greater amplitude while lower values indicate little differentiation between periods of rest and activity suggesting a weaker circadian rhythm of rest-activity pattern.

L5 and M10 indicate arousal patterns during the day and night. L5 refers to the least active consecutive 5h interval of daily activity. M10 indicates the most active consecutive 10h interval of daily activity. High M10 values indicate activity during the day while high L5 values suggest disturbed sleep. The onset times of M10 and L5 indicate phase markers of circadian function which correspond to the onset of activity during the day and offset of activity during the night. Assessing activity this way has been found to be a validated measure of circadian phase in predicting the circadian rhythm of melatonin secretion (Bonmati-Carrion et al., 2014).

6.2.7 Daily Questionnaire of Subjective Mood and Sleepiness

The current study used electronic technology to monitor daily mood and sleepiness variability in individuals. Specific applications such as the Automated Monitoring of Symptom Severity (AMoSS) have been developed and utilized to measure mood variability in individuals in clinical populations (Carr et al., 2018; Tsanas et al., 2016) with one item questions reporting to be associated with validated measures of depression (Tsanas et al., 2016). The advantage of this real-life monitoring approach is that it may facilitate passive sensor-based monitoring and active real time monitoring through time-stamped prompts. This may provide a more accurate representation of current mood and sleepiness state along with examination of variation of states throughout the day and across the study period. Specifically, while self-report measures employed in this thesis demonstrate high internal and test-retest reliability an individual's assessment of mood or sleep quality at one time point may be primarily related the individuals state on that day of assessment rather than a true average over a longer a period of time.

Mood was examined by asking individuals to rate their current mood on a 5-point Likert scale ranging from 1 - very happy to 5 - very sad. Subjective sleepiness was measured using the Karolinska Sleepiness Scale which assesses an individual's current level of sleepiness on a 9-point Likert scale with 1 indicating extremely alert and 9 indicating extremely sleepy. Participants were asked to assess how they rated their mood and subjective sleepiness in relation to the Likert responses. The individuals were prompted to report their mood and sleepiness at 4 points throughout the day (10:00, 14:00, 18:00, 22:00) on a daily basis. These times were chosen so adherence to participation was not reduced by those that have a later wake-up time and earlier bed-time. In the early part of this study daily data was implemented using a customised Android application developed for this study (n = 10). This resulted in individuals who did not have an android being excluded from participating in the study. The application was also to provide an objective measurement of screen time which was to be used as a proxy measure of light exposure at sleep times. Unfortunately, as android software advanced/updated and privacy laws become more stringent the application became incompatible with certain mainstream devices and data became lost. To maintain assessment of daily self-monitoring of mood and sleepiness the remainder of participants in this study (n = 21) used the CamnTech Pro-Diary (see Figure 6.2). This device is a wrist worn device which combines actigraphy while simultaneously collating self-report data. Like the smartphone

application individuals were prompted by audible alerts to provide subjective responses. With the Pro-Diary the question and responses appeared on a touch sensitive user interface where individuals indicated their preferred response. These responses were stored with daily ambulatory accelerometer monitoring and were downloaded using the CamTech Pro-Diary software onto an excel document.

Figure 6.2

Image depicting the interface of the Pro-Diary which prompted participants to record their current mood and subjective sleepiness. In the above image participants are being asked to record their current mood state ranging from very happy to very sad.

To quantify the variability of the time series we separately calculated the Root Mean Squared Successive Differences (RMSSD) for mood and sleepiness across the 14-day period. The use of the RMSSD (see Figure 6.3) has been employed to quantify variability in other studies examining daily variability (Gershon & Eidelman, 2014; Tsanas et al., 2016).

Figure 6.3

Formulae for calculating the Root Mean Square Successive Differences (RMSSD)

$$= \sqrt{\frac{1}{N}\left(\sum_{i=1}^{N-1}\left(x_{i+1} - x_i\right)^2\right)}$$

6.2.8 Procedure

Individuals who expressed interest in participating were rang and were informed about the nature of the study, the duration, what was required of them and that the researcher would need access to the sleeping environment. Once individuals agreed to participate a time was chosen that was mutually suitable for the participant so that the researcher could visit the participants dwelling to install the devices and give the participants the actigraph. When the researcher arrived at the individuals dwelling, the participant was provided with an instruction sheet and consent form. Once the participant signed the consent form the researcher asked could they install the light meters in the sleeping environment. In the sleeping environment one light meter recorder and sensor was attached to the wall or bedpost at the side of the bed that the participant indicated they slept most frequently. Care was taken to ensure that the sensor was attached to the wall/bedpost at a level where the participants head rested. A second light data recorder and sensor was placed at the window with the sensor placing outwards. Care was taken to place the sensor as the area of the window where trespassing light from the external environment entered into the sleeping environment. Once both meters were installed the research ensured that the participant was aware that the sensor only recorded light illuminance and did not record any other data.

In earlier stages of the study participants were sent an email containing a link to the app to which they were required to download. Once participants downloaded the app they had to change some settings on their smartphone to allow the app to access their phone and collect data on the participants mood and sleepiness. Participants also had sign into the app using their google account. Once they completed this step, the participant had to give the researcher access to the google form file which was created when the participant signed into the application. The google form logged participants responses to the daily questionnaires and logged data on screen time. The researcher ensured that the application was downloaded correctly and that a google form had been created to which the researcher had access too. In later stages of the study participants were provided with the Pro-Diary and were instructed on its use to record daily ratings of mood and sleepiness. Participants were instructed both verbally and through written instructions that they would be prompted by the smartphone or the Pro-diary to indicate rate their mood and

sleepiness state at 4 time points throughout the day. Participants who were using the Pro-Diary were also sent a video on how to record their response.

Participants were sent a link to a Qualtrics survey which contained a link to the surveys and were provided with a paper copy of the Consensus Sleep Diary. The researcher provided instructions that the sleep diary was to be competed each morning and was reflective of the previous night sleep. Participants were asked to ensure that the diary was completed daily and not sporadically. When the two-week period had ended the researcher contacted the participant to arrange a suitable time to collect the materials. The researcher removed the light meters.

6.2.9 Data Analysis

This was an ecological design study. All time-based variables from the MCTQ (MSFsc, average sleep duration SJL), and all sleep measurements derived from actigraphy were decimalised (i.e, 8:30 became 8.50, 30 min became .50). Sleep measurements from actigraphy were averaged across the duration of the study. Averages were calculated for workdays and free days based upon the participants indication. As guided by Rea et al. (2011) average window LAN intensity was calculated between 00.00 and 04.00. The same process was used to calculate bedside LAN intensity. Daily average LAN intensity was then averaged across the week. From here a median split was performed with high LAN being categorised as any value above the median and low LAN being categorised as values below the median. A second bedside LAN score was also computed from the hours between 00.00-2.00 and 04.00. The same process to compute the score was provided. Variance in sleep timing was calculated by using the standard error of the mean and variance in daily subjective mood and sleepiness was calculated by using the Root Mean Square Successive Differences. Additionally, the average was calculated for each time point and overall average for daily mood and sleepiness scores.

Due to the small sample size Spearman Rho was used to examine correlations between continuous variables. Mann-Whitney U-tests were employed to examine between group differences of light intensity on sleep and circadian parameters. Friedman Test was examined the effect of timepoint on mood and sleepiness. Post-hoc analysis employed Wilcoxon Signed Rank Test. For post-hoc tests a lower alpha level was used ($p < .008$). This was to correct for multiple comparisons. Effect size used was r. The threshold used to determine the effect of the effect size was .1 = small effect, .3 = medium effect and .5 = large effect.

The study sample size was estimated using a-priori power calculations based on it being important to detect effect sizes of moderate size (d = .5). These calculations indicated a required study sample of approximately 210 would be required to detect differences of a likely-to-be-meaningful magnitude. However, due to COVID-19 it was not possible to continue with this study and as a result this study reflect a proof of concept. All statistical analysis were conducted using IBM SPSS (V25, IBM Corporation) and graphs were produced either on SPSS or Jasp (V 0.9.1.0, https://jasp-stats.org/)

6.3 Results

Descriptive statistics, and initial correlations among actigraphy and questionnaire data can be observed in Table 6.2, Table 6.3, and Table 6.4.

Table 6.2

Overview of descriptive statistics for subjective measures, LAN intensity and variance in daily mood and sleepiness.

	Mean(SD)	**CI(95%)**	**Median**	**Range**	**%(N)**
PSQI	6(2.41)	[5.10-6.90]	5	3-13	
Poor Sleeper >7					33.3(10)
Sleep Duration (hh.mm)	7.83(.86)	[7.518.15]	7.67	6.69-10	
MSF*sc* (hh.mm)	4.71(1.41)	[4.19-5.24]	4.64	1.01-7.17	
Social Jetlag (hh.mm)	1.41(.91)	[1.08-1.75]	1.50	0-3.79	
GHQ	25.53(13.74)	[20.40-30.66]	24	4-56	
CFQ	42.13(15.61)	[36.30-57.96]	44	11-72	
Window LAN Intensity (lux)	.75(1.34)	[.20-1.30]	.11	.00-6.10	
Bedside LAN Intensity (lux)	2.02(5.49)	[.03-4.07]	.08	.00-29.54	
Sleepiness Variability	2.07(.78)	[1.77-2.36]	1.87	.70-3.74	
Mood Variability	.93(.23)	[.85-1.02]	.93	.46-1.40	

Table 6.3

Correlation Matrix depicting correlations between actigraphy derived estimates of rest-activity and questionnaire derived of self-reported measures.

	PSQI	MSF*sc*	SJL	GHQ	CFQ	DBAS	IS	IV	RA	M10	$M10_0$	L5	$L5_0$
PSQI	-												
MSF*sc*	.183												
SJL	-.171	.023											
GHQ	**.650****	.197	-.029										
CFQ	**.491****	.297	.239	**.820****									
DBAS	.370	.114	-.175	**.510****	.375								
IS	.044	.037	-.350	-.216	-.199	**-.398***							
IV	.035	-.156	-.157	.084	-.106	**.427***	**-.393***						
RA	-.403	-.080	.050	**-.483****	**-.437***	**-.405***	**.435***	.174					
M10	-.131	-.054	.330	-.099	.068	**-.512****	.025	-.530	.158				
$M10_0$	-.010	.352	.175	**.371***	**.393***	.230	**-.483****	-.057	**-.472****[1]	.070			
L5	.062	.014	.238	.080	.250	-.254	-.189	-.325	**-.475****[1]	**.740****	.315		
$L5_0$	.000	**.392***	.041	.172	.246	.077	-.203	-.202	.051	-.118	.321	.149	-

Note. PSQI = Pittsburgh Sleep Quality Index; MSF*sc* = Midsleep on free days corrected for sleep debt; SJL = Social Jetlag; GHQ = Global Health Questionnaire; CFQ = Cognitive Failures Questionnaire; DBAS = Dysfunctional Beliefs and Attitudes towards Sleep; IS = Inter-daily Stability; IV = Intra-daily Variability; RA = Relative Amplitude; M10 = Activity counts for most active 10h; $M0_0$ = time of onset of most active 10h; L5 = Activity counts for least active 5h; $L5_0$ = Time of on onset for least active 5 hours. **p*<.05; ***p*<.01; ****p*<.001

[1] Although a moderate negative association was separately observed between the relative amplitude of the rhythm the L5, the computation of if the relative amplitude score is derived from calculating the difference between the average activity level in the M10 and that in the L5. For a more detailed overview of the computation of RA see Van Someren et al (1999). While a moderate negative association was observed between $M0_o$ and the relative amplitude, the $M0_o$ is derived from starting hour of the most active hours and provides an indication of the most active hours. The association between the variable $M10_o$ and RA is due to the RA being derived from M10 which is a similar measure to $M10_o$.

Table 6.4

Correlation Matrix depicting correlations between actigraphy derived estimates of sleep quality, duration and timing and questionnaire derived self-report measures.

	PSQI	**MSF*sc***	**SJL**	**GHQ**	**CFQ**	**DBAS**	**TIB**	**TST**	**SlpEff**	**#Wake**	**WASO**	**Frag**
PSQI	-											
MSF*sc*	.183											
SJL	-.171	.023										
GHQ	**.650****	.197	-.029									
CFQ	**.491****	.297	.239	**.820****								
DBAS	.370	.114	-.175	**.510****	.375							
TIB	-.523**	-.311	.242	-.356	-.252	.044						
TST	.309	-.176	.058	-.215	-.209	.206	**.828****					
SlpEff	.232	.148	-.259	.181	.035	**.411***	.017	**.556****				
#Wake	-.249	.126	-.081	-.186	-.135	-.118	.271	.066	.241			
Frag	.031	-.092	-.161	-.073	-.118	-.190	-.259	**-.381***	-.300	**.469****		-

Note. PSQI = Pittsburgh Sleep Quality Index; MSF*sc* = Midsleep on free days corrected for sleep debt; SJL = Social Jetlag; GHQ = Global Health Questionnaire; CFQ = Cognitive Failures Questionnaire; DBAS = Dysfunctional Beliefs and Attitudes towards Sleep; TIB = Time in bed; TST = Total Sleep Time; SlpEff = Sleep Efficiency; #Wake = Number of wake bouts during sleep. **p*<.05; ***p*<.01; ****p*<.001

6.3.1 Association between subjective perception of LAN and objective LAN intensity

There was a significant association between objectively measured window LAN intensity and objectively measured bedside LAN intensity x^2 (1, 25) = 4.81, p = .028, phi = .439. Those with higher bedroom LAN intensity reported higher objectively measured window LAN intensity (76.9%). Those exposed to lower bedside LAN intensity more frequently experienced lower window LAN intensity (66.7%). However, there was no significant association between the subjective perception of external LAN and objectively measured window LAN intensity x^2 (1, 25) = 3.38, p = .066, phi = .368. Similarly, there was no association between perceived room brightness and window LAN intensity x^2 (1, 25) = 6.98, p = .404, phi = .439. No association was observed between objectively measured bedside LAN intensity and perceived room brightness x^2 (1, 30) = 2.40, p = .624, phi = .089.

6.3.2 Association between objectively measured LAN intensity and circadian rhythm of activity

Descriptive statistics on the measures of key circadian rhythm of activity measures derived from actigraphy can be observed in Table 6.5.

Table 6.5

Descriptive statistics indicating the central tendency and dispersion of scores for the circadian rhythm of activity across the sample

	Mean(SD)	CI[95%]	Median	Range
L5 Average	1000.50(728.81)	[252.45-3247.33)	742.94	252.45-3247.33
L5 Start Hour	1.8(.99)	[1.47-2.20]	2.00	0.00-4.00
M10 Average	20748(112080	[16563-24933]	15005	7554-47699
M10 Start Hour	10.90(1.47)	[10.335-11.56]	11.00	7.00-13.00
Relative Amplitude	.91(.05)	[.89-.92]	.92	.79-.96
Inter-daily Stability	.47(.08)	[.44-.50]	.47	.29-.62
Intra-daily Variability	.99(.20)	[.91-1.06]	.98	.67-1.45

Mann-Whitney U tests were carried out to examine whether there was an effect of bedside light intensity (median split high and low) on the circadian rhythm of activity. Analysis found that those experiencing low level LAN intensity displayed a significantly earlier timing of L5 (*Md* = 1.00, *n* = 15) compared to those experiencing higher level of LAN intensity (*Md* = 2, *n* = 15, *U* = 165.50, *z* = 2.33, *p* = .026. The magnitude of the difference in the medians was moderate (*r* = .43). These findings suggest that higher levels of LAN intensity at bedside are associated with a delay in L5. As can be seen from Table 6.6 there were no significant differences between bedside light intensity and the remaining parameters of the circadian rhythm of activity.

Table 6.6

Mann-Whitney U test depicting group differences in bedside light intensity on actigraphy derived estimates of rest-activity rhythms.

	Low LAN n=15	High LAN n=15				
	Median	Median	U	z	*p*	r
L5 Average	999.78	730.97	90	-.933	.367	
L5 Start Hour	1.00	2.00	165	2.31	**.026**	.43
M10 Average	17564	13558	90	-.933	.367	
M10 Start Hour	11.00	11.00	141	1.241	.233	
Relative Amplitude	.92	.92	118	.228	.838	
Inter-daily Stability	.50	.47	87	-1.06	.305	
Intra-daily Variability	.98	.99	107	-.228	.838	

Separate analysis was conducted to examine whether there was an effect of window LAN intensity on the circadian rhythm of activity. Analysis found that individuals exposed to higher window LAN intensity reported statistically later onset of L5 (*Md* = 2, *n* = 14) compared those exposed to low LAN intensity (*Md* = 1, *n* = 11), *U* = 128, *z* = 2.94, *p* =.004. The magnitude of the difference in the medians was large (*r* = .588). As can be seen from Table 6.7 analysis found no statistically significant difference between window LAN intensity groups on timing of M10 (*p* = .851), scores of relative amplitude (*p* = .609), inter-daily stability (*p* = .851), inter-daily variability (*p* = .687).

Table 6.7

Mann-Whitney U test depicting group differences in window light intensity on actigraphy derived estimates of rest-activity rhythms.

	Low LAN n=11	High LAN n=14				
	Median	Median	U	z	*p*	r
L5 Average	740.92	700.23	80	.164	.893	
L5 Start Hour	1.00	2.00	128	2.94	**.004**	.588
M10 Average	14013.39	13928.30	81	.219	.851	
M10 Start Hour	11.00	11.50	110	1.86	.075	
Relative Amplitude	.92	.91	67	-.547	.609	
Inter-daily Stability	.47	.47	73	-.219	.851	
Intra-daily Variability	.98	.98	69	-.438	.687	

6.3.3 Association between the subjective perception of bedroom LAN and circadian rhythm of activity

The subjective perception of external LAN is associated with statistically significant differences in timing of L5. Individuals who perceive external LAN report later L5 (*Md* = 2, *n* = 14) compared to those that do not perceive external LAN (*Md* = 1, *n* = 16), *U* = 56.5, *z* = -2.45, *p* = .019. The magnitude of the difference in the medians was moderate (*r* = .45). As can be seen from Table 6.8 the timing of L5 is similar for both objective window LAN intensity and the subjective perception of external LAN. The perception of external LAN is not associated with differences in the other measures of circadian rhythms of activity.

Table 6.8

Mean timings of L5 for subjective perception of external LAN and objectively measured window LAN.

	Perception of External LAN	High LAN intensity	No Perception of External LAN	Low LAN intensity
	M(SD)	M(SD)	M(SD)	M(SD)
L5	2.21(.89)	2.42(.94)	1.50(.97)	1.27(.65)

Statistically significant differences were found between perception of internal LAN trespassing into the sleeping environment and later timing of L5. Given a

disproportionate sample size an independent sample t-test with equal variances not assumed was carried out to examine between group differences on the perception of internal LAN and on circadian rhythm of activity. Analysis reported that those that perceive internal LAN report statistically later timing in L5 (*Md* = 2, *n* = 24) compared to those that do not perceive external LAN (*Md* = 1, *n* = 6), *U* = 123, *z* = 2.80, *p* = .006. The magnitude in the differences of the medians was large (*r* = .511). However, these results must be taken with caution given the small number (*n* = 6) of respondents that perceive internal LAN.

6.3.4 Association between window LAN intensity and subjective measures of psychological wellbeing, sleep quality and circadian parameters

A series of Mann-Whitney U-tests were carried out to investigate the effect of window LAN intensity on each of the subjective measures. Analysis found that those that experience higher levels of window LAN intensity experience a later chronotype (*Md* = 5.54, *n* = 14) compared to those who experience lower levels of window LAN intensity (*Md* = 4.36, *n* = 11), *U* = 115, *z* = 2.08, *p* = .037, with a medium effect size (*r* = .42). As can be seen from Table 6.9 analysis reported no statistically significant differences between LAN intensity and scores on the remainder of the subjective scales.

Table 6.9

Mann-Whitney U tests examining the effects of window LAN intensity on each of the subjective measures (n = 25)

	Low LAN (n=11)	**High LAN (n=14)**	**Boot**	**U**	**z-score**	***p***	**R**
PSQI	6	5		71	.334	.739	
DBAS	4.13	4.31		80.50	.192	.848	
MSFsc	4.36	5.54		115	2.08	**.037**	.42
SDweek	7.66	7.75		90.5	.740	.460	
Social Jetlag	1.25	1.45		99	1.20	.228	
GHQ	24	27		85.50	.466	.641	
CFQ	35	45		112	1.92	.055	

6.3.5 Association between bedside LAN intensity and subjective measures of psychological wellbeing, sleep quality and circadian parameters.

A series of Mann-Whitney U-tests were carried out to investigate the effect of bedside LAN intensity on each of the subjective measures. As can be seen from Table 6.10 there no statistically significant difference between bedside LAN intensity and subjective measures.

Table 6.10

Mann-Whitney U tests examining the effects of bedside LAN intensity on each of the subjective measures (n=30)

	Low LAN (n=15)	**High LAN (n=15)**	**Boot**	**U**	**z-score**	***p***	**r**
PSQI	6	5		97	.654	.539	
DBAS	4.25	4.34		94	.146	.905	
MSFsc	4.02	4.88		159.50	1.95	.050	
SDweek	7.54	7.71		119.50	.771	.775	
Social Jetlag	1.40	1.59		131.50	.789	.436	
GHQ	26	18		93	.810	.436	
CFQ	45	41		123	.436	.683	

6.3.6 Association between bedside LAN intensity and sleep parameters

Overview of the average estimates derived from actigraphy for sleep quality, duration and timing for the whole sample can be observed in Table 6.11.

Table 6.11

Sleep quality, sleep duration and timing derived from actigraphy.

	Mean(SD)	CI[95%]	Median	Range
Sleep Onset (hh.mm)	00.25	[23.87-00.62]	00.46	21.94-26.35
Sleep End (hh.mm)	8.54(.91)	[8.19-8.88]	8.46	6.49-10.62
Average Assumed Sleep (hh.mm)	8.17(.70)	[7.91-8.44]	8.23	6.77-9.15
Average Actual Sleep (hh.mm)	7.13	[6.84-7.41]	7.14	5.16-8.32
Average Sleep Efficiency (%)	85.53(5.72)	[83.40-87.67]	86.99	69.76-92.93
Average Fragmentation (%)	27.72(7.99)	[24.73-30.70]	26.94	14.77-52-25
Average Wake Bouts (N)	45.58(12.00)	[41.02-50.15]	42	30.29-75.69
Sleep Variability				
Sleep Onset	.33(31)	[.21-.45]	.26	.11-1.47
Sleep End	.29(.27)	[.25-.33]	.37	.09-.59
Actual Sleep	.25(.08)	[.22-.28]	.25	.12-.42
Sleep Efficiency	1.05(.54)	[.84-1.24]	.92	.39-2.82

Mann-Whitney U tests were used to examine the effect of bedside light intensity on average sleep timing, actual sleep and sleep efficiency as measured by actigraphy. Higher bedside light intensity was associated with statistically later sleep onset (*Md* = 00.65, *n* = 15) compared to those who experience lower levels of bedside LAN (*Md* = 23.51), *U* = 183, *z* = 2.92, *p* = .003. The magnitude in the difference of the median was large (*r* = .53). Higher bedside LAN intensity led to statistically higher sleep offset (*Md* = 8.86) compared to those that experience lower bedside LAN intensity (*Md* = 8.18), *U* = 178, *z* = 2.71, *p* = .007. However, the magnitude in the difference of medians was moderate (*r* = .49). As can be seen from Table 6.12 no other statistically significant differences on the sleep-rest activity measure derived from actigraphy.

Table 6.12

Mann-Whitney U tests examining the effects of bedside LAN intensity on measures of actigraphy (n=30)

	Low LAN (n=15)	High LAN (n=15)	U	z-score	*p*	r
SO	23.51	00.65	183	2.92	**.003**	**.53**
SE	8.18	8.86	178	2.72	**.006**	**.49**
Assumed	8.35	8.18	108.50	-.166	.870	
Actual	6.92	7.17	126	.560	.595	
Slp Eff	84.58	89.94	149	1.51	.137	
Frag	26.53	27.19	126	.560	.595	
NWake	42	40	100	-.519	.624	

6.3.7 Association between window LAN intensity and sleep parameters

Mann-Whitney U tests examined the effect of window LAN intensity on actigraphy derived sleep parameters. Higher levels of window LAN intensity was associated with statistically later sleep onset (*Md* = 00.76, *n* = 11) compared to lower levels of LAN intensity (*Md* = 00.08, *n* = 14), *U* = 124, *z* = 2.57, *p* = .009. The magnitude in the difference of the medians was large (*r* = .51). As can be seen from Table 6.13 there was no statistically significant differences between window LAN intensity and the remaining rest-activity parameters as measured by actigraphy.

Table 6.13

Mann-Whitney U tests examining the effects of window LAN intensity on measures of actigraphy (n=25)

	Low LAN (n=11)	High LAN (n=14)	U	z-score	*p*	*r*
SO	00.08	00.76	124	2.57	**.009**	.51
SE	8.28	8.66	93	.876	.403	
Assumed	8.66	8.17	55	-1.205	.244	
Actual	7.54	7.14	65.50	-.630	.536	
Sleep Eff	86.17	89.70	92	.821	.434	
Frag	27.09	27.25	67	-.547	.609	
NWake	40.71	42.35	82	.274	.809	

6.3.8 Effect of window LAN intensity on sleep timing on work and free days

Table 6.14 provides an overview of the average of actigraphy derived estimates of sleep quality, duration and timing for free- and work-days across the whole sample.

Table 6.14

Overview of the average estimates derived from actigraphy for sleep quality, duration, and timing for the whole sample.

		Mean(SD)	CI[95%]	Median	Range
Mid-Sleep	Work	4.01(.95)	[3.66-4.37]	3.90	2.28-6.31
	Free	5.04(.94)	[4.69-5.40]	5.11	3.43-6.40
Sleep Onset	Work	00.06(.83)	[23.75-00.37]	23.97	22.93-26.09
	Free	00.91(4.98)	[24.52-01.29	00.77	23.19-03.00
Sleep End	Work	8.11(.99)	[7.74-8.48]	7.98	6.27-10.76
	Free	9.45(1.05)	[9.05-9.84]	9.74	7.05-10.90
Assumed Sleep	Work	8.00(.71)	[7.42-8.27]	7.97	6.88-9.20
	Free	8.55(.82)	[8.24-8.86]	8.49	7.13-10.31
Actual Sleep	Work	6.99(.79)	[6.68-7.29]	6.97	4.96-8.28
	Free	7.46(.82)	[7.16-7.77]	7.40	5.67-8.97
Sleep Efficiency	Work	85.72(5.68)	[83.60-87.95]	86.47	70.57-93.44
	Free	85.54(5.64)	[83.43-87.79]	87.18	67.75-92.92
Fragmentation	Work	27.47(8.91)	[24.11-30.83]	25.12	14.16-53.64
	Free	29.42(8.39)	[16.43-49.74]	28.57	16.43-49.74

On workdays, higher window LAN intensity was associated with statistically later sleep onset (*Md* = 00.34, *n* = 14) compared to lower those with lower window LAN intensity (*Md* = 23.64, *n* = 11), *U* = 123, *z* = 2.51 *p* =.012. A median effect size was observed between the medians (*r* = .50). Similarly, on free-days sleep onset was later in those that experienced higher levels of window LAN intensity (*Md* = 01.59, *n* = 14) compared to those who experience lower levels of window LAN intensity (*Md* = 00.08, *n* =11), *U* = 120, *z* = 2.35 *p* = .018. The moderate magnitude in the difference of the medians (*r* = .43). As can be seen from Table 6.15 there was no effect of window LAN intensity on sleep timings and quality on work- or free-days.

Table 6.15

Mann-Whitney U test illustrating the effect of window LAN intensity on actigraphy derived estimates of sleep quality, duration and timing.

		Low n=11	High n=14				
		Median	Median	U	z	*p*	r
Mid-Sleep	Work	3.80	4.24	99	1.20	.224	
	Free	4.86	5.59	108	1.69	.095	
Sleep Onset	Work	23.64	00.34	123	2.52	**.011**	.50
	Free	00.52	01.69	114	2.02	**.044**	.40
Sleep End	Work	7.96	8.21	101	1.31	.202	
	Free	9.05	9.90	92	.821	.434	
Assumed Sleep	Work	7.97	8.08	68	-.493	.647	
	Free	8.50	8.26	58	-1.040	.317	
Actual Sleep	Work	6.95	7.15	77	.000	1.00	
	Free	7.41	7.19	63	-.766	.467	
Sleep Efficiency	Work	86	89.61	98	1.150	.267	
	Free	86.40	89.03	87	.547	.609	
Fragmentation	Work	25.30	26.28	67	-.547	.609	
	Free	29.60	27.94	64	-712	.501	

6.3.9 Effect of bedside LAN intensity on sleep timing, quality, and duration on work and free days

All analysis of actigraphy derived estimates of sleep timing, sleep duration and sleep quality in LAN group can be observed in Table 6.16.

Table 6.16

Mann-Whitney U test illustrating the effect of bedside LAN intensity on actigraphy derived estimates of sleep quality, duration, and timing.

		Low n=15	High n=15				
		Median	Median	U	z	*p*	r
Mid-Sleep	Work	3.63	4.31	162	2.05	**.041**	.37
	Free	4.83	5.29	170	2.41	**.016**	.4
Sleep Onset	Work	23.59	00.32	179	2.76	**.005**	.50
	Free	00.17	01.66	178	2.97	**.006**	.54
Sleep End	Work	7.85	8.31	165	2.18	**.029**	.40
	Free	8.91	10.11	173	2.51	**.011**	.46
Assumed Sleep	Work	7.89	7.97	108	-.187	.870	
	Free	8.50	8.47	111	-.062	.967	
Actual Sleep	Work	6.66	6.99	127	.601	.567	
	Free	7.25	7.44	121	.353	.744	
Sleep Efficiency	Work	85.10	90.01	155	1.76	.081	
	Free	85.65	89.03	137	1.02	.325	
Fragmentation	Work	25.30	24.89	110	-.104	.935	
	Free	24.54	29.85	143	1.27	.217	

Our findings report that higher levels of bedside light intensity are associated with a later L5, delayed sleep onset and later sleep end. However, our calculation of LAN intensity was computed from the average photic lux between 0.00 and 04.00. In order to examine whether these effects were due individual behaviour rather than LAN exposure, the intensity of bedside light was calculated again and only included LAN intensity between 02.00 and 04.00. After the average score was calculated a median split was performed where light intensity was characterised into high bedside LAN and low bedside LAN (see Table 6.17).

Table 6.17

Bedtime intensity between midnight and 4 am, and also between 2 am and 4 am.

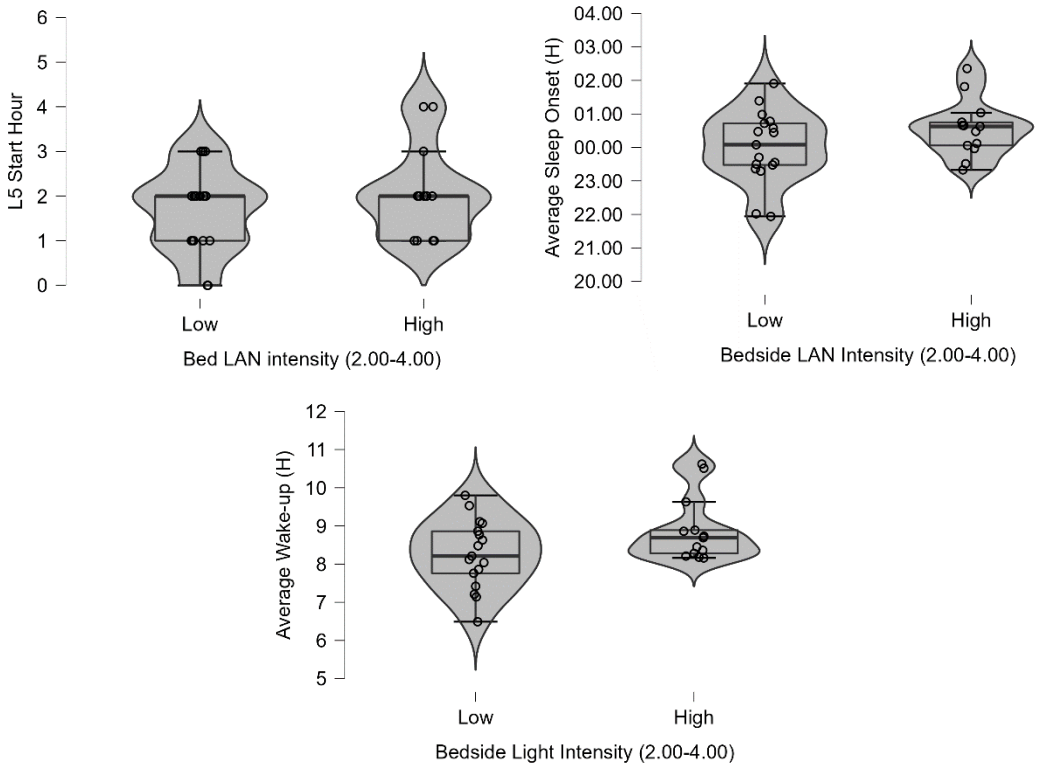
	M(SD)	Median	Range
Bedside Intensity 00.00-04.00	2.01(5.49)	.075	.00-29.54
Bedside Intensity 02.00-04.00	.47(2.26)	0.00	.00-12.41

6.3.10 Descriptive report outlining the descriptive scores of bedside LAN intensity at 00.00-.4.00 and 02.00-04.00

Mann-Whitney U-tests were carried out on sleep and circadian parameters that were previously reported to be significant when light intensity was computed between 00.00 and 04.00 (L5, sleep onset and sleep end). Our results found that when LAN intensity between 02.00 and 4.00 did not have an effect on L5 (p = .408), sleep onset (p = .198) or sleep end (p = .113). See Figure 6.4.

Figure 6.4

L5 (top left), sleep onset (top right) and sleep end (bottom middle) in high and low intensity groups between 2 and 4 am.



6.3.11 Day-to-day mood variation

In order to examine whether mood and subjective sleepiness varied across the 4 timepoints (10.00, 14.00, 18.00, 22.00) the mean score for each of the four timepoints across the duration of the study were calculated. As previously outlined subjective mood was rated on a 5-point Likert scale with 1 indicating very happy and 5 indicating very sad. Subjective sleepiness was rated on a 9-point Likert scale with 1 indicating being extremely alert and 9 indicating being extremely sleepy. Table 6.18 provides the mean score for both mood and sleepiness across the four time points and Figure 6.5 illustrates the variance in change of score across participants.

Table 6.18

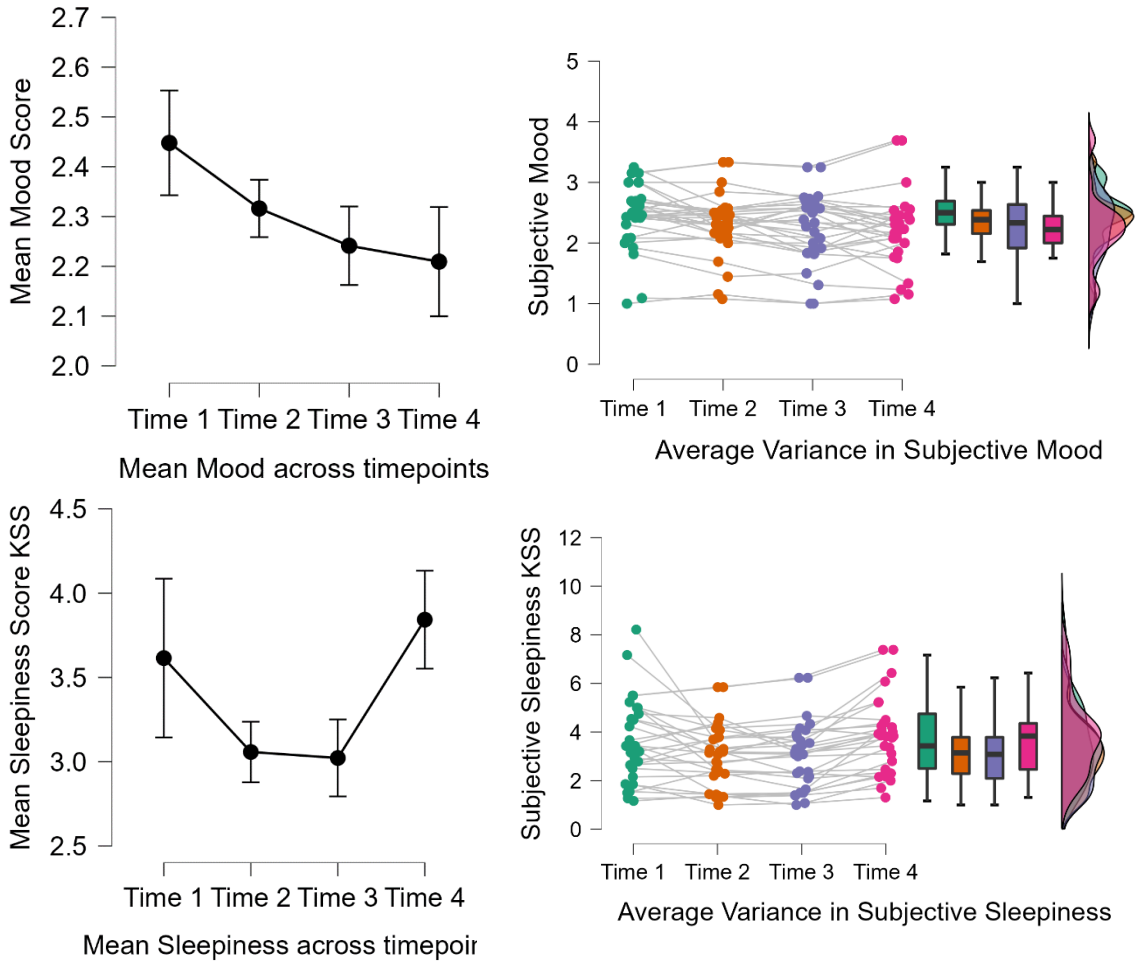
Mood and sleepiness across the four timepoints.

	Timepoint 1	Timepoint 2	Timepoint 3	Timepoint 4	Total Average
	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)	Mean(SD)
Mood	2.45(0.53)	2.32(.52)	2.24(.57)	2.21(.61)	2.30(.52)
Sleepiness	3.62(1.73)	3.06(1.28)	3.02(1.36)	3.84(1.59)	3.38(1.32)

Note. Average and standard deviation scores for each time point and overall average for sleep and mood.

Figure 6.5

Mood across the four timepoints (top panel). Timepoint 1 was significantly higher than timepoint 3 (suggesting lower mood). No other significant differences noted. Sleepiness across the four timepoints (bottom panel). Sleepiness was higher at timepoint 1 than timepoint 3. No other statistically significant differences noted.



The Friedman Test reported a statistically significant difference in average scores of mood across the 4 timepoints (time 1, time2, time3 and time4), X^2 (3, n = 29) = 9.21, p = .027. Inspection of the median values showed that mood was lower at time point 1 (*Md* = 2.50), increased at timepoint 2 (*Md* = 2.38), timepoint 3 (*Md* = 2.33) and timepoint 4 (*Md* = 2.22). Post-hoc comparisons using the Wilcoxon Signed Rank tests (using a Bonferroni adjusted alpha value = .008) indicated that the median score for timepoint 1 mood was statistically lower (*Md* = 2.50) compared to timepoint 3 mood (*Md* = 2.33) with a large effect size (r = .54). No statistically significant differences was observed between timepoint 1 and 2 (p = .029), timepoint

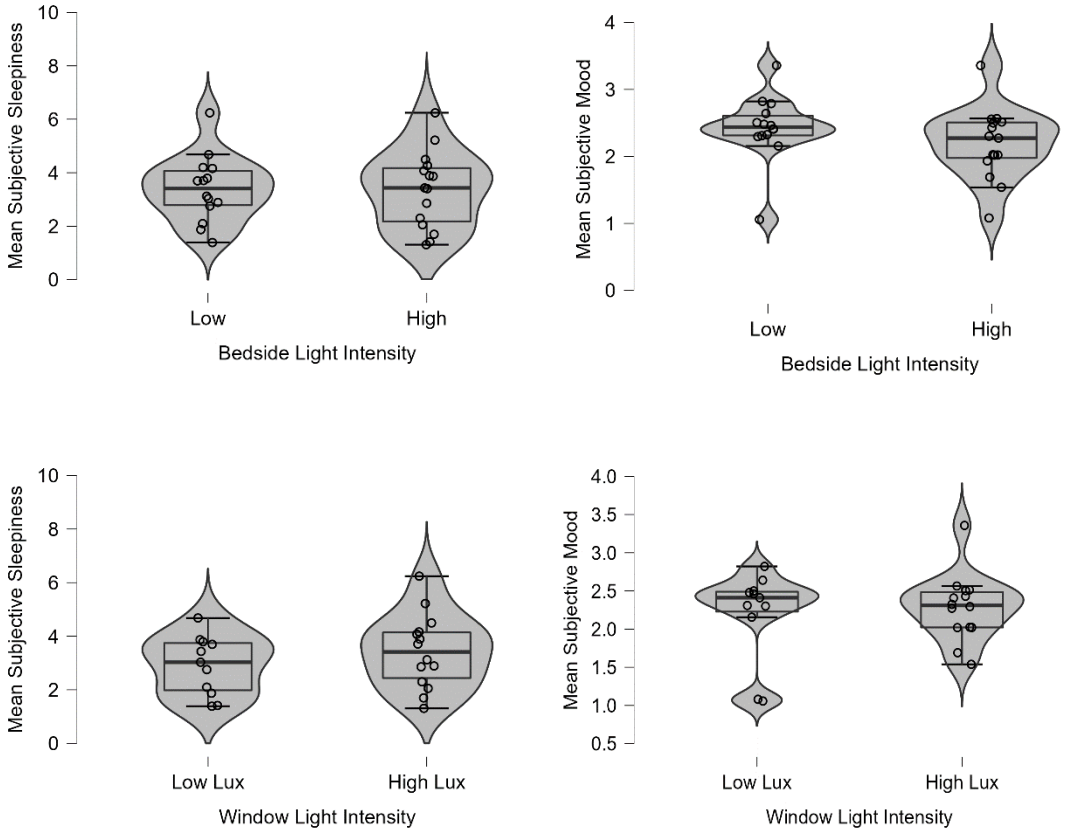
1 and 4 (p = .018), timepoint 2 and 3 (p = .090), timepoint 2 and 4 (p = .057) and timepoint 3 and 4 (p = .733).

The Friedman Test reported a statistically significant difference in average scores of sleepiness across the 4 timepoints (time 1, time2, time3 and time4), X^2 (3, n = 29) = 24.01, p < .001). Inspection of the median values showed that higher levels of sleepiness at time point 1 (*Md* = 3.43), reducing at both timepoint 2 (*Md* = 3.14), and timepoint 3 (*Md* = 3.08) and increasing again at timepoint 4 (*Md* = 3.83). Post-hoc comparisons using the Wilcoxon Signed Rank tests (using a Bonferroni adjusted alpha value = .008) indicated that the median sleepiness score for timepoint 1 was statistically higher (*Md* = 3.43) compared to timepoint 3 (*Md* = 2.22) with a large effect size (r = .76). This indicates that on average most report having higher levels of sleepiness in the morning compared to the evening. There was no reported statistical significance reported between timepoint 1 and 2 (p = .086), 1 and 4 (p = .018), 2 and 3 (p = .090), 2 and 4 (p = .057) and 3 and 4 (p = .733).

Mann-Whitney U tests were carried out to separately investigate the effect of bedroom and window LAN intensity on the overall average score for sleepiness and mood (Figure 6.6). Analysis found that that there was no effect of bedside LAN intensity on mood (p = .146) or mood (p = .983). Similarly, there was no effect of window LAN intensity on average mood (p = .727) or average sleepiness scores (p = .344).

Figure 6.6

Mean subjective sleepiness and subjective mood between low and high light intensity (bedside – top panel; window – bottom panel)



6.3.12 Effect of window LAN intensity on variance of actigraphy derived sleep-rest activity and variance in daily ratings of mood and sleepiness

Mann-Whitney U tests reported no effect of window LAN intensity effecting variance in actigraphy derived sleep parameters across the study duration or on the variance of subjective sleepiness or subjective daily mood (Table 6.19).

Table 6.19

Mann-Whitney U tests examining the effects of window LAN intensity on variance of actigraphy measured rest-activity and daily subjective ratings of mood and sleepiness (n=25)

	Low LAN (n=11)	High LAN (n=14)	U	z-score	*p*
SO	.21	.28	101	1.31	.202
SE	.27	.24	70	-.383	.727
Actual	.25	.23	68	-.493	.647
Sleep Eff	1.00	.77	56	-1.15	.267
Sleepiness	.93	.91	74	-.164	.893
Mood	1.85	2.07	71	-.329	.767

Note. SO = Sleep onset SEM, SE = Sleep end SEM, Actual = Actual sleep duration SEM, Sleep Eff = Sleep efficiency SEM.

6.3.13 Effect of beside LAN intensity on variance of actigraphy derived sleep-rest activity and variance in daily ratings of mood and sleepiness

As can be seen from Table 6.20 analysis reported no statistically significant difference between bedside LAN intensity and variance in rest-activity as measured by actigraphy across the study period or in the variance of daily mood and subjective sleepiness ratings.

Table 6.20

Mann-Whitney U tests examining the effects of bedroom LAN intensity on variance of actigraphy estimates of rest-activity rhythms and daily subjective ratings of mood and sleepiness (n=30)

	Low LAN (n=15)	High LAN (n=15)	U	z-score	*p*
SO	.21	.29	146	1.39	.174
SE	.25	.29	140	1.41	.267
Actual	.25	.28	134	.892	.389
Slp Eff	.96	.89	94	-.767	.461
Sleepiness	1.85	1.87	120.50	.332	.744
Mood	.88	1.05	135	.934	.367

Note. SO = Sleep onset SEM, SE = Sleep end SEM, Actual = Actual sleep duration SEM, Sleep Eff = Sleep efficiency SEM

6.4 Discussion

Our findings report that window LAN intensity is associated with delayed timing of L5, later sleep onset and later MCTQ derived midsleep. However, objective measures of bedside LAN intensity did not have an effect on the remaining rest-activity circadian parameters derived from actigraphy. Additionally, higher LAN intensity was not associated with differences in sleep duration, sleep end or indices of sleep quality and disturbance. This is in contrast with previous observational studies which have reported that home-setting LAN is associated with increased prevalence of insomnia (Obayashi et al., 2014) and an experimental study which found that exposure to 1lux elicited poor sleep quality as indexed by increased fragmentation (Stebelova et al., 2020). As previously noted, observational studies examining light intensity are limited. However, in the current study the average median light intensity at both the bedside and window were lower than previous studies which recorded LAN exposure and found that average median light intensity reported ranged between .7lux and .8lux in older adults (Obayashi et al., 2013; Obayashi et al., 2015) and the median in a LAN intensity of younger adults with bipolar was 1.9lux (Esaki et al., 2019). Specifically, while Obayashi et al. (2014) provided evidence that higher level bedroom environment LAN was associated with insomnia this was based on LAN measurements being split into quartiles. In their study the highest quartile group was categorised by light levels above 3.4lux while the lowest quartile was based on measurements of .1lux and below. In our study the overall median for bedside lux was .08. Additionally, some of the other studies have used cut off points of >5 lux to categorise high LAN intensity (Obayashi et al., 2013; Esaki et al., 2019) This may suggest that the levels of light recorded in our study may not be at sufficient levels to elicit biological responses to light.

Our study reported no significant association between the subjective perception of external LAN and objective measures of bedside and window LAN. This contrasts with Harrison and colleagues (2019) who reported that those that subjectively report that their bedroom is bright after the lights are turned off or perceive LAN have higher levels of objectively measured LAN intensity. However, the findings from Harrison and colleague's (2019) study may not be generalizable as the results were based on a small sample size comprising of shift-workers. This also contrasts with Ohayon et al. (2016) who reported that those that perceive their bedroom to be bright at night live in areas with high outdoor LAN. As indicated in

chapter 1, subjective measures which access the subjective perception of LAN in the sleeping environment are quite limited and have not fully examined the sources of LAN perception (Johns et al., 2017; Park et al., 2019; Ohayon et al., 2016). Our current findings may suggest that measures of subjective perception of LAN may not be fully reliable given our lack of association between self-rated perception of LAN and objective LAN intensity. This may be due numerous reasons such as those who are not sensitive to light (Chellappa et al., 2021) or who have good quality sleep, may not consciously perceive LAN in the sleeping environment. Our findings did report that those that are exposed to higher levels of window LAN intensity were also exposed to higher levels of bedside LAN intensity. This may suggest that outdoor LAN can affect indoor LAN through insufficient light blockage from curtains or shades. This would suggest that external LAN not only leads to greater LAN exposure when outside but also to greater light exposure while inside in the sleep environment. However, the current study cannot confirm this argument as distance from the window to the bedside was not measured.

Although higher levels of window intensity were associated with later sleep onset and later timing of L5 no other between group differences were observed in total sleep timing, sleep efficiency, fragmentation, number of awakenings or other rest-activity estimates derived from actigraphy. These findings are in contrast with observational and experimental studies which found that higher levels of sleeping environment light or exposure to LAN intensity greater than 5 or 10lux was associated lower total sleep time, lower sleep efficiency and increased wake time after sleep onset compared to total darkness (Cho et al., 2018; Harrison et al., 2019). A possible reason for the differences in findings is that in the aforementioned studies the categorization of LAN intensity was based on a higher lux threshold. However, exposure to LAN at 1lux during sleep has been associated with poor quality sleep as indexed by increases in sleep fragmentation (Stebelova et al., 2020). Our findings are consistent with previous studies which reported no between groups differences in home setting LAN intensity on either latency to sleep onset and mean wake time after sleep onset (Harrison et al., 2019). Furthermore, in Chomorro et al. (2021) experimental study it was found that exposure to 3lux LAN during the in-bed period resulted in no differences in sleep duration, sleep efficiency, or proportion of wake stages compared to sleepers who slept in darkness. Additionally other observational studies have indicated that exposure to LAN does not lead to differences in sleep

duration (Beale et al., 2017; Wright et al., 2013; Stothard et al., 2017). However, it must be noted that in Chomorro and colleagues (2021) study while 3lux did not elicit differences in actigraphy derived sleep parameters it was found that dLAN exposure resulted in alterations to the organisation and architecture of sleep which is similar to observations found in other studies where dLAN exposure lead to increases in the latency to NREM sleep stage 3, reduction in time spent in stage N2 sleep, increased REM sleep along with reductions to slow waves and delta bands (Chamorro et al., 2021; Cho et al., 2016; Cho et al., 2018). This indicates that exposure to low level LAN can impact on sleep stage consolidation throughout the night and these impacts can happen interpedently of sleep timing. This is consistent from studies from animal studies which have found that dLAN exposure impacts on sleep organization in mice (Stenvers et al., 2016; Panagiotou & Deboer, 2020). However, the possible differences in the proxies used to measure sleep disruption induced by light exposure calls into question the metrics employed. For instance, although actigraphy provides an estimate measure of sleep timing which derived from rest-activity counts it must be viewed as proxy which reflects a highly systematic clock output (Broussard et al., 2017). Actigraphy cannot fully inform about the possible impacts that home setting LAN has on endogenous clock function nor does it provide an understanding of organismal or molecular interplay of clock-related processes (Vetter et al., 2020). Support for this argument comes from animal studies which has observed that while dLAN exposure of 5lux does not disrupt locomotor activity it does impose alterations to the molecular rhythmicity of core clock genes (Bedrosian et al., 2013; Fonken et al., 2013).

Our findings report that higher levels of window LAN intensity were associated with earlier sleep onset and earlier L5. These results are in part consistent with other studies that have reported that LAN exposure is associated with later bedtimes and later timing in inactivity (Beale et al., 2017; Quante et al., 2019). One study of particular interest is from Beale et al. (2017) who reported that those with no access to electricity had earlier sleep onset and earlier onset of L5. Although Beale and colleagues (2017) investigated between group differences between those who had access to electricity and those who did not, the findings can be in part comparable to the current study as in the electrified group light levels were comparable to the current study as levels of light were low and at low brightness which is significantly different to the high intensity illumination devices that are

present in houses in the hours preceding bedtime. The later onset of L5 in those experiencing higher intensity of LAN could be due to phase delaying effects on the circadian clock (Czeisler et al., 1989), or through acute alerting action (Cajochen et al., 2007). A wide number of studies have indicated that phase delaying effects may be mediated through suppression of melatonin however, a number of studies investigating low level LAN in the home environments have showcased no effects of melatonin concentration levels. However, our findings conflict with Beale et al. (2017) who found that no access to electricity had phase advancing effects with an earlier sleep end and earlier onset of M10. Additionally, our findings also align with other studies which report that higher levels of LAN intensity are not associated with fragmented rest-activity patterns as indicated by IS or IV (Beale et al., 2017; Quante et al., 2019). Associations between more fragmented and less stable 24-hour rhythms has been found to be associated cognitive deficits, psychiatric and neurogenerative illness (Luik et al., 2015; Zuurbier et al., 2015). This may indicate that IS and IV is not influenced by the timing of zeitgebers but by factors such as ill health.

Our findings reported that when bedside LAN exposure is measured between 00.00 and 4.00 it was associated with delayed L5, sleep onset and sleep end. These findings support Obayashi et al. (2014) which found that higher levels of evening light exposure was associated with delayed sleep initiation. However, when bedside LAN intensity was measured between 02.00 and 04.00 there is no between group differences of light intensity any of the parameters which were previously significant. This suggest that it may not be the light that keeps individuals awake but the behaviours of individuals who select their sleep schedules.

Our findings reporting no effect of window LAN intensity on sleep duration or sleep quality call into question light pollution studies which have used satellite data as a proxy of light exposure. These studies have regularly reported that higher levels of outdoor LAN is associated with reduced sleep duration and increases incidences of poor sleep quality, insomnia, and use of hypnotic drugs (Koo et al., 2016; Min & Min, 2018; Ohayon et al., 2016; Paksarian et al., 2020; Xiao et al., 2020). It may be that higher levels of LAN and their association with sleep timing and sleep quality is as a result of being a third variable as high levels of light pollution may afford individuals to work, socialize and engage in leisure time activities at night which are not directly related to external LAN entering into the sleeping environment (Lunn et al., 2017). Our findings reported a positive

association between window LAN intensity and MCTQ derived midsleep but not midsleep derived from actigraphy. Previous studies have reported that high levels of outdoor light as measured by satellite data is correlated with eveningness in adolescents (Koo et al., 2016; Vollmer et al., 2012). Our finding reporting delayed midsleep in those exposed to higher levels of window LAN may suggest that outdoor LAN may be a contributing factor to delaying midsleep. Our findings did report that high bedside LAN intensity is associated with later timing in midsleep. This may suggest that LAN exposure closer to the bedside is a contributing factor to delaying the timing of midsleep. This aligns with observational and mathematical modelling studies which have reported that higher LAN exposure during the biological night is a predictor of a later midsleep (Papatsipa et al., 2021; Philips et al., 2010; Wright et al., 2013).

Research has indicated that variability in sleep parameters alongside the average values of sleep parameters may adversely impact health and quality of life outcomes (Chaput et al., 2020; Duncan et al., 2016). Our study separately reported that bedside and window LAN intensity was not associated with variance in sleep parameters or on daily variance of mood and sleepiness. Secondly our results found that variability in total sleep time, sleep end and sleep onset was not associated with variance in daily mood and daily levels of sleepiness. This is in contrast with Fang et al. (2021) who reported that greater variability in total sleep time and wake time were associated with increased depression. Slavish et al. (2019) also report that a two-fold increase in the odd of depression with every hour increase in the standard deviation of total sleep time. However, Chaput and colleagues (2020) argue that the quality of studies reporting associations between sleep timing/consistency and depression are rated low to very low due to bias risk. Our exploratory analysis does not reveal significant differences in the degree of sleep timing variability between those who experience high and low LAN intensity. There are number of possible reasons for this. Firstly, for the overall sample the median sleep duration was high. Secondly, window LAN intensity only impacted on average sleep onset but did not impact on average sleep timings and as a result the LAN intensity may not have been at high levels to elicit impacts on sleep timing variability. Overall, the majority of sample were categorised as good quality sleepers with those poor sleepers reporting only modest levels of sleep efficiency rather than a population of clinical sleep disorders. It is plausible that with examination of a larger sample who are exposed to

a greater range of sleeping environment LAN may elicit greater variance in sleep parameters and subjective mood and sleepiness variance.

The current study reported no effect of bedside or window LAN intensity on GHQ scores. This is in contrast with several studies which have indicated that higher levels of bedroom light intensity are associated with increased risk of depression (Obayashi et al., 2018; Obayashi et al., 2013). However, it must be noted that in Obayashi et al. (2018) analyses they found that splitting in-bed light intensity into quartiles was not associated with between groups differences on incidence of depression. Additionally, LAN between 5lux and 9lux was not associated with increased risk? of depression. Obayashi et al.'s (2018) significant association findings were also based on analysis which calculated the amount of time spent in LAN greater than 5lux and 10lux. These levels are significantly higher than the levels of light experienced in the current study. Our findings are also in contrast with other studies which found that higher levels of outdoor LAN are associated with increased risk of mood disorders (Min & Min, 2018; Paksarian et al., 2020). However, as discussed previously there are many limitations to using satellite data as a proxy of LAN exposure. Additionally, a study by Helbich et al. (2020) reported that when confounding variables such as demographic variables and air pollution are controlled for then higher levels outdoor LAN exposure are not associated with increased risk of depression. In Paksarian and colleagues (2020) study the odds ratio risk of ALAN being a predictor of depression was significant the odds were small. This highlights that the research associating outdoor LAN and with depression may not be fully reliable. Although accumulating evidence from animal studies have provided causal evidence that dLAN exposure provokes the onset of depressive-like phenotype (Bedrosian et al., 2013; Fonken et al., 2013; Walker et al., 2013) it is difficult to ascertain whether these effects translate to humans due to differences in photosensitivity and eye anatomy (Walbeek et al., 2021). It may also be plausible that the thresholds of light in the current study were far too low to elicit any between group differences on mood.

The current study reported no effect of LAN intensity on variation on mood and subjective sleepiness. To the researcher's knowledge no study to date has examined whether LAN intensity is associated with variances in mood. Studies to date have predominantly investigated individuals with bipolar disorder to showcases greater mood variation is associated with objective variability in diurnal physiology

(Carr et al., 2018; Kane-Gartiser, 2016; McGowen et al., 2020). To the researcher's knowledge only Palmer et al. (2019) examined in adolescents whether sleep variability was associated with emotional regulation. They reported that greater variability in sleep timing was associated with less avoidance of negative situations when watching video clips and that higher variability in both sleep timing and duration was associated with greater variability in sleep timing and sleep duration. Other studies have reported no associated between emotion dysregulation and sleep duration (George et al., 2019). Gillett et al. (2021) argue that the relationship between mood instability and actigraphic measures appear to be more consistent in those with psychiatric conditions than health controls.

The current study has a number of limitations. Firstly, the sample size was very small which limits the generalisability of these findings. The small sample size is due to the impact of Covid-19 restrictions. This study commenced before the onset of COVID-19 in March 2020. During most of the Covid-19 pandemic public health guidelines advised against visits to other individuals dwelling. Given the complexity of setting up the study's materials in the participants sleeping environment and ensuring the materials were returned to the researcher remote data collection was not feasible. Secondly, objective measurement of LAN intensity was confined to the average LAN intensity between 00.00h and 04:00. However, this timing and calculation of average LAN intensity was guided by other studies (Huss et al., 2011; & Rea et al., 2019). Additionally, the times used to measure external LAN intensity in the current study provide a more reliable measurement of artificial light which does not include natural solar daylight. Previous observational studies examining LAN exposure in home settings have measured LAN intensity between sleep onset and wake-up time (Esaki et al., 2019; Obayashi et al., 2013; Obayashi et al., 2015; Obayashi et al., 2018). This limits the generalisability of these study's findings which found associations between increased levels of LAN intensity and impacts on sleep. These findings may have been in part influenced by natural light exposure. This argument is supported by Obayashi and colleagues (2019) who found that independent of post bedtime light exposure pre-awake light exposure which occurs mainly through morning sunlight trespassing into the sleeping environment was associated with higher frequency of sleep as indexed by self-report and actigraphy. Thirdly, the light meters were not ambulatory devices. As a result, measures of light intensity may have been underestimated as the light meters only recorded light

exposure in the sleeping environment and at specific times. Future work should employ ambulatory eye-level light meters such as the wearable spectrophotometer employed by Cain and colleagues (2020). This will provide more accurate representation of light exposure in the hours before sleep outside of the sleeping environment and the intensity of LAN individuals are exposed to in the sleeping environment. The study measured LAN intensity in photic lux which is the illuminance which reflects the spectral sensitivity of long and middle wavelength light (Lucas et al., 2014) and not short wavelength light to which the circadian system is most sensitive (Brainard et al., 2001; Thapan et al., 2001). Although, the current study did not measure light intensity in terms of melanopic lux it did provide a measurement of light exposure. While spectral wavelength does elicit strong effects on non-visual behaviours other factors such as the timing, and duration play a significant role which the current study considered (Chang et al., 2012; Khasla et al., 2003). The metric used to calculate LAN intensity per participant was based on the average between 00:00 and 04:00. However, in the current study intensity can include LAN with high intensity and short duration. It has been found that high intensity LAN in short period can elicit stronger effects on the circadian system than the exposure to the same intensity light over a prolonged period of time (Chang et al., 2012).

This study has several strengths. Firstly, it builds upon previous field studies which have examined the association between home-setting LAN exposure and its impacts on both sleep and psychological health to include examination of younger individuals. These type of field studies provide an insight into how home-setting LAN can impact on sleep under more naturalistic conditions. As discussed previously the findings from these studies may not be fully representative to the true effects of LAN exposure as they were based in older adults to which effects of LAN on non-image forming behaviours is less responsive or based on clinical populations whom are more sensitive to LAN exposure. Secondly, sleep was objectively measured with the use of actigraphy over a two-week period. Actigraphy devices provide individual data under ecologically valid conditions which may not be fully possible in studies conducted in laboratories. In addition, the duration of investigation provides a more reliable investigation of sleep behaviour. Thirdly, LAN was measured objectively using light meters. This improves upon previous studies which have used the light sensors from actigraphy which may not provide an

accurate measurement of light reaching the eye or underestimates LAN exposure due to covering of the actigraph (Okudaira et al., 1983; Figueiro et al., 2013). Particular attention was provided to ensure that the bedside meter was located at retina level beside the bed to capture LAN intensity experienced where the participant slept. Secondly, external LAN was measured from the window area where external LAN trespasses into the sleeping environment. This improves upon previous studies which have used satellite data as a proxy of light exposure which does not consider light exposure at the individual level and takes into consideration previous studies which have reported no association between bedroom LAN intensity and satellite data. Additionally, the current study findings are more reliable as oftentimes the satellite data measurements are not concurrent with the year participants were recruited. The current study cannot infer whether this external LAN reaches the retina however, the current findings did report that there was an association between bedside and window LAN intensity. Mood and subjective sleepiness was measured at multiple timepoints daily. This method has advantages as subjective ratings were measured in real time and not at one point during the study where state may not be reflective of how the participant feels on average but instead how they feel at that one time point. This provides a better understanding of the variability of these states and is less prone to recall bias (Proudfoot et al., 2010).

Due to studies which would provide a causal understanding of whether low level sleeping environment LAN exerts an effect on both sleep and psychological health are unethical to conduct future studies should employ similar methodologies to the current study. However, future studies should not focus on the amount and timing of light exposure throughout the day. Several classic studies have showcased how exposure to high level natural light during the day can act as a protective factor towards the phase shifting effects of LAN exposure on both phase markers, sleep timing and alertness (Cajochen et al., 2019; Chang et al., 2011; Chang et al., 2013; Hebert et al., 2002; Smolensky et al., 2015). Additionally, a growing number of studies have showcased that exposure to bright light during the day leads to earlier sleep timing and improved sleep quality and reduced risk of stress and depression (Burns et al., 2021; Figeuoro et al., 2021). The current study used the stability of rest-activity rhythms as a proxy for circadian rhythms however, future studies should examine other objective phase markers to examine whether home setting LAN during sleep impacts on the circadian phase. However, future work should take into

consideration whether DLMO is an appropriate phase marker to understand the effects of LAN on the endogenous rhythm. Several studies have reported that light-sensitive melatonin is not significantly inhibited by dim light (Obayashi et al., 2013; Obayashi et al., 2014; Stebelova et al, 2020). The procedure to collect DLMO states that individuals should be kept in 10lux light or less for DLMO to be collected. This would suggest that dim light at lower levels does not have a significant effect on the secretion of melatonin (Cho et al., 2018).

In conclusion, our proof-of-concept study suggests that the intensity of light experienced in the bedroom environment may not be at levels of intensity which could adversely impact on sleep and health. There is a need to examine the specific threshold of light which predicts adverse consequences on sleep and health. Field studies in this area are quite limited and the analyses used to calculate LAN exposure varies from paper to paper.

Chapter 7

General Discussion

The aim of this research was to examine what the perceived sources of LAN in the sleeping environment were and whether the perception and intensity of these sources were associated with adverse consequences for sleep and psychological health. This research is warranted given the increased prevalence of LAN in our external environment, with global satellite observable light emissions having increased by at least 42% between 1992 to 2017 (de Miguel et al., 2021). There is also an increased prevalence of LAN in our private dwellings due to luminaires and the use of electronic devices which emit short-wavelength light (Gringras et al., 2015). Cain and colleagues (2020) reported that individuals are exposed to continuous levels of LAN from early in the biological evening right up right up to bedtime. Crucially, due to technological innovations individuals are increasingly being exposed to LED or fluorescent lighting via luminaires, light emitting technologies and outdoor lighting (Cain et al., 2020; Gringras et al., 2015). While this lighting has been found to be beneficial in terms of light quality and energy efficiency (Pattison et al., 2018; Tsoa & Wailde, 2010) this lighting contains blue wavelength which the circadian system is most sensitive to (Brainard et al., 2001). While research has demonstrated that exposure to LAN at levels experienced in the home-setting can impact on both sleep and the circadian system (Cajochen et al., 2011; Gooley 2011; Philips et al., 2018; Zeitzer et al., 2000), the speed of understanding the biological and health impacts of LAN exposure has been outpaced by the availability and exposure to light sources and technologies. As a result, this research aimed to examine the associations between LAN routinely experienced in the home-setting and its association with sleep and psychological health.

The objective of this research was achieved by firstly examining the perceived sources of LAN and assessing individuals' attitudes towards these light sources as disruptive to sleep. The research then examined the associations between the perception of LAN with sleep quality, circadian misalignment, chronotype and psychological health. Consequently, the research investigated whether the perception of LAN is driven by an attention bias to sleep-related information. This research examined whether objectively measured window LAN intensity trespassing into the sleeping environment and bedroom LAN intensity during the in-bed period was associated with disruption to objectively measured sleep and circadian rhythms derived from actigraphy. Finally, this thesis examined whether higher levels of LAN intensity were associated with day-to-day variance in sleep timing and quality along

with variance in daily mood and sleepiness. A summary of the main findings from this thesis can be found in Table 7.1.

Table 7.1: Summary of main finding arising from this research

Study	Research Questions	Study Design	Methods	Main Findings
Chapter 2	Examine: 1. What are the perceived sources of LAN in the sleeping environment. 2. Perceived attitudes towards the various sources of LAN on being disruptive to sleep/sleep quality. 3. What are the strategies individuals can employ to reduce LAN exposure in the sleeping environment.	N=552 Age 18-74 (M=37; SD=13) 66% Female Cross-sectional Design Exploratory descriptive analysis	Development of a detailed questionnaire which concurrently assess the various sources of LAN rather than focusing on one single item question to determine individual level LAN exposure in the sleeping environment	1. The majority perceive LAN to be disruptive to sleep and negatively impacts sleep after falling asleep. 2. Perception of LAN comes from sources outside the bedroom environment (External 42%; Internal 25%). 3. Perception of external LAN is more frequently reported in cites and less frequently in the countryside. However, suburbs more frequently endorse not perceiving external LAN. 4. Perception of trespassing LAN is associated with negative attitudes about the impacts of LAN on sleep and sleep quality. 5. Technology use of frequent before sleep in bed. 6. Individuals who do use devices more frequently report that these sources are either not disruptive to sleep or they do not know.
Chapter 3	1. What are the associations between the perception of LAN in the sleeping environment with psychological distress, sleep quality, circadian misalignment and chronotype. 2. Is there are association between subjective perception of external LAN in the sleeping environment and objective measures of external light which is measured at a house specific level. 3. Are objective measures of external LAN associated with psychological distress,	Same participants as in chapter 2	<u>LAN Exposure Questionnaire</u> As used in chapter 2 <u>Sleep Measures</u> PSQI MCTQ <u>Psychological Health</u> CFQ GHQ <u>Objective Measures of LAN</u> Measurement of public street lighting individualised to the participant's dwelling	1. The subjective perception of external LAN is associated with poorer sleep quality, higher psychological distress and higher everyday errors in cognitive function. 2. Perceived impacts of external LAN being disruptive to sleep is associated with poorer cognitive function and higher levels of psychological distress. 3. Perception of internal LAN is not associated with adverse impacts on sleep and psychological health. 4. No association between the perception of external LAN and objective measures of external LAN. 5. No association between objective measures of external LAN and sleep or psychological health.

		sleep quality, circadian misalignment and chronotype.		
Chapter 4	1. Do individuals who perceive LAN from various sources in the sleeping environment display an attention bias towards sleep-related word stimuli.	N=177 Age 18-70 (M=25;SD=8.98) 71% Female Quasi-experimental	<u>LAN Exposure Questionnaire</u> As used in chapter 2 <u>Sleep Measures</u> PSQI MCTQ DBAS <u>Psychological Health</u> CFQ GHQ <u>Attention Bias</u> EST	1. The perception of LAN from various sources in the sleeping environment was not associated with differences in response latencies to sleep-related stimuli or attention bias scores.
Chapter 5	1. Do individuals who perceive LAN from various sources in the sleeping environment display vigilance towards images depicting LAN sleeping environments. 2. Do individuals who perceive LAN in the sleeping environment display facilitated attention towards LAN stimuli, difficulty disengaging from LAN stimuli.	N=149 Age 18-51 (M=26; SD=8.10) 68% Female Quasi-experimental	<u>LAN Exposure Questionnaire</u> As used in chapter 2 <u>Sleep Measures</u> PSQI MCTQ DBAS <u>Psychological Health</u> CFQ GHQ <u>Attention Bias</u> DPT	1. The perception of LAN from various sources in the sleeping environment was not associated with differences in response latencies, orientation towards or disengagement from LAN related stimuli

Chapter 6	1. Do increased levels of LAN intensity (at bedside and window) in the sleeping environment impact on sleep and rest-activity patterns. 2. Is there an association between LAN intensity in the sleeping environment and subjective measures of psychological health. 3. Do higher levels of LAN intensity lead to greater variance in sleep timing and quality along with increased variance in daily subjective mood and sleepiness.	N=30 Age 24-73 (M=32.33; SD=9.57) 53% Male Observational Ecological-study design	Objective measurement of bedside and window LAN intensity Objective measurement of sleep and rest-activity patterns derived from actigraphy Daily monitoring of sleepiness and mood at 4 timepoints Subjective questionnaires which have been outline above.	1. Higher levels of window LAN intensity are associated with delayed timing of L5, MCTQ derived chronotype and sleep onset. 2. Higher levels of bedside LAN intensity (measured between 00.00-04.00) is associated with delayed timing of L5, timing of sleep onset and sleep end. 3. These effects are not observed when bedside LAN intensity is measured between 02.00-04.00. 4. LAN intensity level is not associated with increased day-to-day variance in sleep timing, duration or quality. 5. LAN intensity level is not associated with increased day-to-day variance in daily subjective mood and sleepiness.

7.1 Contribution of research findings

The first contribution of this project is that it developed a detailed questionnaire which for the first time comprehensively assessed the perceived sources of LAN in the sleeping environment, the perceived impacts of these light sources on sleep and assessment of strategies which may minimise LAN in the sleeping environment. There was a significant need for this assessment questionnaire as most epidemiological studies to date have used satellite data to assess associations between sleep and health (Koo et al., 2016; Min & Min, 2018; Ohayon et al., 2016; Paksarian et al., 2020), with only a small number of satellite imaging studies addressing individual-level bedroom environment LAN exposure by assessing room brightness (Ohayon et al., 2016). While no fully established questionnaire has been developed to assess sources of LAN exposure, the questionnaire was guided by previous research studies and their limitations.

Our findings provide a novel examination of which specific source of LAN in the sleeping environment is associated with adverse sleep and psychological health outcomes. Most research to date has examined the biological impacts of LAN however, research studies examining the psychological impacts of home-setting LAN exposure are limited to just a small number of studies (Obayashi et al., 2018; 2014). However, it is important to examine the psychological impacts of LAN exposure as it can exert adverse impacts on psychological wellbeing independent of sleep and circadian rhythmicity (LeGates et al., 2012; LeGates et al., 2014; Fernandez et al., 2018). Our findings indicate that the perception of external LAN is adversely associated with poor sleep quality and psychological health. Additionally, our findings from chapter 6 indicate that higher levels of outdoor LAN intensity are associated with delayed L5, sleep onset and delayed chronotype derived from the MCTQ. These findings are difficult to explain as our findings from chapter 2 and 6 indicate that levels of objective measured LAN are not associated with the subjective perception of external LAN. Research findings from chapter 4 and 5 found no evidence of whether those that perceive LAN display an attention bias to sleep-related information or more specifically to sleeping environments which display LAN environments.

We report important findings regarding the individual-level perception of external LAN and the possible unreliability of studies employing satellite imaging as a proxy measure of light exposure (Koo et al., 2016; Min & Min, 2018; Ohayon et al., 2016; Paksarian et al., 2020). In chapter 2 we report that although residing in a city was associated with increased perception of external LAN, residing in higher densely

populated areas such as urbanised environments – like suburbs or urban towns – was associated with not perceiving external LAN. This conflicts with the conceptual underpinnings of use of satellite studies as these areas would typically be categorised as areas of high outdoor ALAN exposure (Kumar et al., 2019). This indicates that satellite images may not be accurate in accounting for individual level exposures. While two studies have previously shown evidence of no association between satellite image measurements of outdoor external LAN and LAN intensity measured at the bedroom window (Huss et al., 2019; Rea et al., 2011) our findings from chapter 3 and 6 report no association between subjective perception and objective measurement. Additionally, our findings call into question the reliability of risk associations found between satellite measured data on sleep and health. Our studies reported no evidence between objective measures of LAN on psychological health. Previous studies which have used satellite data have also reported small effect sizes (Paksarian et al., 2020; Patel, 2018; Zhong et al., 2021) or when controlling for other confounding environmental factors the association between outdoor LAN and health outcomes no longer exists (Helbich et al., 2020). This indicates that satellite data may not be an appropriate proxy due its association being confounded by other factors, not providing an accurate measurement of LAN intensity at an individual level and associations observed of low effect size.

What is important about the questions employed in the survey is that it examined factors which individuals can employ to minimise exposure to external LAN. For example, in chapter 1 we found that individuals who rated their blinds/curtains to be either effective or very effective more strongly endorsed not perceiving external LAN, while those that rated their blind/curtains to be moderately effective or ineffective more likely endorsed perceiving external LAN. These findings are important as it identifies that the perception of external LAN in the sleeping environment occurs frequently and that citizens should be educated and informed of inexpensive ways to minimise external LAN by using a good quality light blocking curtain. As highlighted by Zerbini et al. (2018) strategies to minimise LAN exposure can positively impact sleep timing and phase advance DLMO. Our findings also report that individuals need to be made aware and educated about the adverse impacts of light emitting technologies in bed before sleep. In chapter 2, 57% reported that either they do not know or do not perceive that light emitting technology adversely impacts their sleep. As outlined, most of these devices have been found to adversely impact on sleep, sleepiness and circadian rhythmicity (Cajochen et al., 2011; Chang et al., 2015). Our findings may be of importance to building developers and policy makers when deciding the proximity and

frequency of public and commercial lighting in relation to dwellings. Strategies to minimise outdoor LAN trespassing into private dwellings could be to use shielding on streetlamps to prevent the dispersion of LAN into residences from street and commercial lighting.

The current thesis provides a better measurement of outdoor LAN. While numerous studies have provided evidence of associations between higher levels of outdoor light and adverse impacts on sleep and health (Koo et al., 2016; Ohayon et al., 2016; Paksarian et al., 2020; Xiao, 2020), these studies have significant methodological limits which reduces the reliability of the findings. These studies employed satellite imaging which acts as a surrogate for measuring circadian effective light. However, satellite images cannot fully capture personal exposure. For instance, higher elevated outdoor light may not translate into higher levels of sleeping environment light, with previous research reporting no association between photometric measurements of LAN intensity measured at the bedroom window and satellite measured light (Huss et al., 2019; Rea et al., 2011). There are numerous reasons for this, such as individuals taking measures to minimize external LAN trespassing into the sleeping environment such as blinds and curtains or the location of the sleeping environment's window facing the opposite side of the street lighting. Our survey ensured to specifically assessed the perception of external LAN and the effectiveness of curtains. Other limitations of previous studies employing satellite imaging is that these studies only measure level of light directed upwards in the outdoor environment and not at ground level. This may impact the reliability of light intensity levels as findings from Katz and Levin (2016) reported that measurement of light levels were darkest when light is measured in the downwards direction with only a low to moderate correlation between LAN measured at ground level and satellite measured LAN. Our metric of outdoor light is based upon measurement of light intensity measured horizontally at ground level. This is a superior approach as horizontal light is more reflective of the light that will enter the house. Garcia-Saenz et al. (2018) also report that measurements of upwards light is weakly correlated with horizontal measured light. The satellite images also reach saturation, meaning that two areas may have different levels of high light intensity but cannot be differentiated between (Tuttle et al., 2014). Furthermore, the spatial resolution (e.g., 2.5km) of the devices can be low resulting in the accuracy of LAN intensity in the accuracy at ground level being quite poor. This may lead to some areas being categorized as having low outdoor lighting due to shielding of lighting by buildings and vegetation. To overcome this limitation, our measurement of objective LAN using MSI

estimated outdoor lighting based at the level of individual residences using a database of public lighting in Ireland. Not only does this provide a more accurate measurement of external LAN by individualizing LAN intensity to the house location, but the resolution of data is superior as LAN illuminance is weighted based upon the distance between the lantern and the residence. In addition, the light measured by satellite may not comprise of light levels which are experienced at ground level at different directions (Beannie et al., 2014). Finally, satellite data is measured to closely match the spectral sensitivity of the cones and not that expressed in short wavelength light. This is problematic given that existing public lighting is being updated to LED to enhance the photic quality of light and LED being more economical (Pattison et al., 2018). This means that satellite data may be unable to capture the increased level of LAN experienced and may not capture the lights at wavelengths which exert a stronger influence on the circadian clock (Brainard, et al., 2001; Thapan et al., 2001). The use of MSI as a measure of light exposure is superior to the collection of light measurements in luminance by considering the amount of blue wavelength light most likely to exert biological effects. As such, we believe that the approach taken to measure LAN is superior to approaches based on satellite data due to measurements being individualised and at spectrums which are biologically impactful to sleep and circadian rhythms and health (Brainard et al., 2001; Cahochen et al., 2011; Chang et al., 2015; Lazzerini et al., 2017; LeGates et al., 2012).

7.2 Reflection on the challenges faced in examining the effects of LAN exposure and the reliability of findings

7.2.1 Experimental vs Field Research

This research mainly comprised of cross-sectional and ecological study design. However, our findings are in contrast with experimental studies which have demonstrated that exposure to low-level LAN or comparing groups exposed to modern living environments to those that have no electricity have delayed sleep onset, shorter sleep duration, delayed timing of phase markers, poor sleep quality, higher levels of sleep disturbance and alterations to sleep architecture (Cho et al., 2016; 2018; Wright et al., 2013; Pilz et al., 2018; Stebalova et al., 2020). While these experimental studies provide a mechanistic understanding of the impacts of LAN exposure, their findings may not be as reliable. Additionally, the findings derived from these studies may not generalise to the real-world. This is due to experimental studies being carried out in highly controlled environments or comparing conditions which are part of two extremes

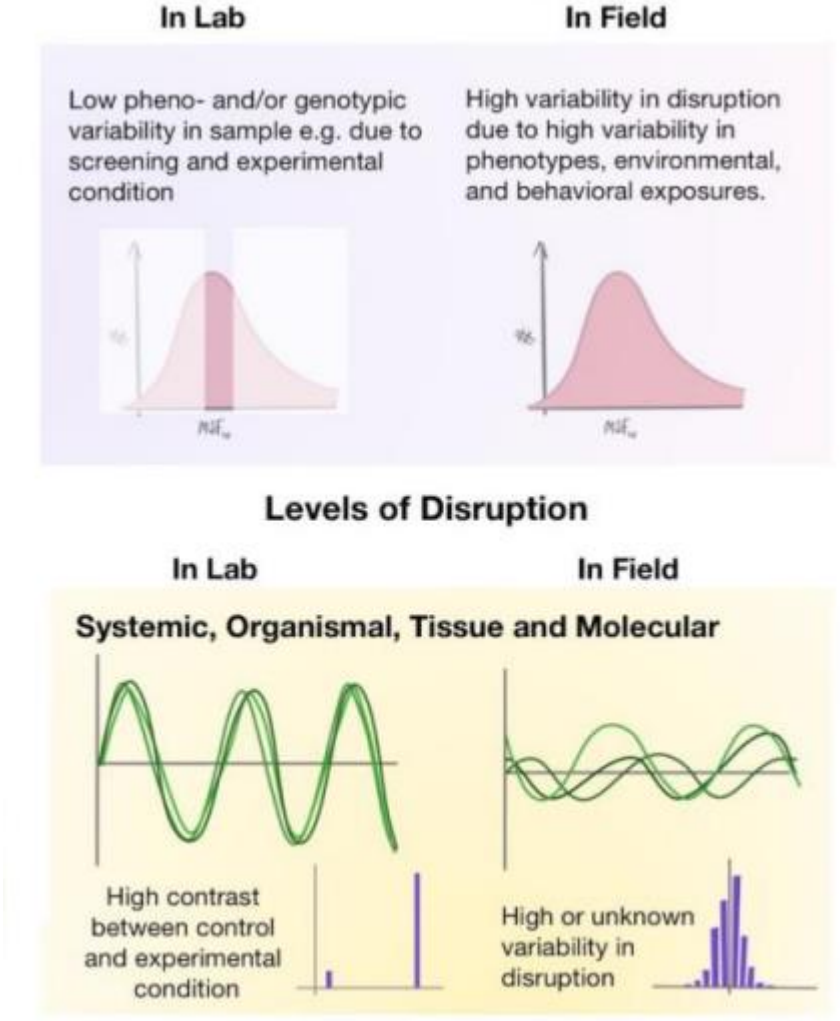
(de la Iglesia et al., 2015; Pilz et al., 2018; Peixoto et al., 2009; Stothard et al., 2017; Wright et al., 2013). Laboratory studies are conducted in highly controlled environments which allows for a contrast between the control and experimental condition. In experimental LAN studies the manipulation of conditions (e.g., presence of LAN vs no presence) enables an understanding of the magnitude of circadian disruption that is induced by light exposure. Although these studies provide a mechanistic understanding of the effects of LAN exposure, the magnitude of difference between conditions may not be as extreme in real life conditions. This is due to methodological procedures such as exposing to dim light in the hours proceeding LAN exposure. Exposing individuals to light boxes which are placed closely to the participants visual field while controlling pupil dilation and gaze direction. Using only one type of light source which may be at brightness levels higher than are typically experienced in home settings or being exposed to LAN at schedules which may not be ordinarily experienced in the real world. In addition, to the experimental conditions being very fixed the participants included in these studies are highly screened. Inclusion criteria may be based upon a selected range of sleep and circadian phenotypes. This approach provides an opportunity to understand the basic mechanistic impacts of LAN exposure on sleep and circadian regulation along with increasing statistical power by reducing inter-individual variability. Using this approach, the core assumption is that the level of disruption induced by the administration of a light is equal among all participants in that condition (Vetter et al., 2020). However, given that there are individual differences with respect to light sensitivity (Chellappa et al., 2021), the findings from these experimental studies may only be reflective of the screened group/particular conditions and are not generalisable to the population/real world. This can lead to divergence in findings between experimental and observational studies (Vetter, 2021) and may possibly account for some of the findings arising from this research.

As can be seen in figure 7.1, n field studies the variability between participants differs widely due to individual differences in in geno- and phenotypes within the population (Wulff et al., 2010). As a result of this in field studies, the magnitude of circadian disruption due to LAN exposure may not be as severe as observed in experimental studies. This may in part be due to the methodological environment not being as heavily controlled in the real world (e.g., participants can turn their gaze from a light source or have the intensity of their light source reduced). Additionally, due to the wide variance of differing pheno and genotypes within the population exposure to LAN

in the sleeping environment may modify, increase and weaken the level of circadian disruption. For example, Philips and colleagues (2018) highlighted within a sample of 61 participants 4 individuals experienced greater than 50% suppression of melatonin in response to exposure to light levels of 10lux while one person only experienced this level of suppression when exposed to 400lux light. That is a 50-fold difference in evening light sensitivity in melatonin suppression in response to light. This indicates that individual differences in light sensitivity can impact on the level of circadian disruption experienced due to light exposure.

Figure 7.1

Schematic image illustrating how the effect of variability in the expression of genotype and phenotype can impact on the generalisaiblity of findings.



Note ***Schematic image illustrating that the findings observed in laboratory studies may not translate to what is observed in field studies. For example, in laboratory studies, participants are screened to include either a healthy profile of participant or a specific clinical sample. This results in the profile of the sample including a narrow window of variability in terms of genotype/phenotype. The strength of the association or the magnitude of the effect of LAN exposure on sleep and psychological health may be more pronounced in laboratory settings. However, in field studies there is a wide range of inter-individual variability with regards to the expression of genotypes and phenotypes. For example, recruiting participants from a diverse age range and examining the effect of LAN on melatonin profiles will yield different results within the sample. This is due to interindividual differences (which can be due to an individual's geno-/phenotype) in how an individual responds to light and their photic history. Additionally, in laboratory studies, the timing, intensity, duration and spectral wavelength of light is highly controlled this allows for the level of circadian disruption at a systemic level. However, this is difficult to achieve in field studies, as the timing and levels of light an individual is exposed to differs from person to person. This leads to difficulties in ascertaining the level to which LAN exposure can impact on circadian disruption at a systemic level.*** *Image taken from Vetter (2020).*

A number of animal studies have provided evidence of significant effects of dLAN on eliciting alterations to sleep, circadian rhythmicity, cognitive performance and depressive-like phenotypes (Bedrosian et al., 2013; Fonken et al., 2013; LeGates et al., 2014; Walker et al., 2020). Animals are employed due to similarities with humans in the molecular underpinnings of the circadian clock along with similarities in the anatomical structure of both the SCN and RHT (Walbeek et al., 2021). Animals also express similar responses to light in the same way humans do on a behavioural, cellular, and molecular level. The only difference is that diurnal animals and humans' response to light are in the opposite manner to what is experienced by nocturnal animals. As a result of this animals have been used to investigate the effects of low-level LAN on sleep, circadian rhythmicity, and health. However, it is unclear whether the results from these studies can reliably translate over to humans or are findings from animals oversensitive to the effects of LAN exposure. Issues about translation of findings is due to anatomical differences between humans and rodent model. As stated earlier each of the photoreceptors have been found to be associated with NIF. However, anatomically, the mouse retina does not have the three types of cones which are found in human retina. This leads mice being unable able to differentiate between red and green light along with being less sensitive to red light (Calderone & Jacobs, 1995). The geometry of the eye differs between humans and mice with 99% of the cones found in the fovea in humans resulting in high acuity vision in a small area of the visual field (Perry & Cowey, 1985). Mice do not have a fovea and as a result the whole retina is similar to the peripheral retina in humans. The total number of photoreceptors also differ between the mice and humans (Walbeek et al., 2021). Finally, animals appear to be more sensitive to light as indicated with NIF occurring at lower melanopic lux light intensities in hamsters compared to humans (Walbeek et al., 2021). These anatomical differences indicates that although animal models are advantageous to understanding both the molecular and mechanistic impacts of LAN exposure on both circadian rhythmicity and health, these effects may not be fully reflective of the impacts experienced in humans.

7.2.2 Measurement of individual-level light

Measuring light in relation to effects it exerts on the circadian system is quite complex and while standardised practices to measure light in relation to the effects it exerts on NIF has been found, the validity of these approaches are still not fully understood. As indicated in chapters one and six, photic lux only describes the luminous sensation of light source under photopic conditions which is weighted by the sum of the rods and cones (Schlangen & Price, 2021; Spitchan et al., 2019). It is now known that

NIF responses are mediated intrinsically by the melanopsin ipRGCs and extrinsically by the rods and cones (Berson et al., 2002; Dacey et al., 2005; Hattar et al., 2002). As a result, two commonly used metrics have been proposed to measure the biological potency to light which are based upon the spectral response of each of the photopigments. These include Lucas and colleagues (2014) equivalent melanopic lux (EML) and the CIE's (2018) international of units (SI) compliant Melanopic Equivalent Daylight illuminance (mel-EDI).

While both approaches attempt to quantify the biological potency of light, they both have significant limitations. Both method's quantification of biological potency to light is based upon the spectral response of the photopigments however, each of the photoreceptors have different spectral sensitivities and the context of the light exposure (i.e., duration, dose and timing) impacts on whether either extrinsic, intrinsic signaling or a combination of both signaling processes elicits an NIF means that no action spectrum can describe all NIF responses to light (Schlangen & Price, 2021). As a result, it is unclear how the intrinsic and extrinsic signals are combined by the photoreceptors and processed within the brain (Houser & Esposito, 2021). This is supported by the CIE's (2018) acknowledgement that the SI compliant computation may not represent the precise extent to which light exposure elicits NIF responses in real world settings. The CIE's metric does not take into consideration how individual differences such as pupil size, (Brown, 2020; Spitschan, 2019), age, (Watson & Yellott, 2012) and photic history (Chang et al., 2011) influences the NIF responses to light.

In addition to the conceptual limitations the measurement of these of these metrics are difficult to achieve especially in real-world settings. Spitschan and colleagues (2019) have proposed a minimum standard of reporting guidelines that should be employed when reporting light exposure in chronobiology and sleep studies. However, these requirements can be costly and complex (Houser & Esposito, 2021). From the outset, the devices employed to measure LAN intensity can be limited due to poor sensor quality and poor resolution (Figueiro et al., 2013). In order to measure light, the light source's spectral power distribution is required and the photic illuminance at the plane of the eye. This involves measuring the spectral irradiance received at the cornea which incorporates a measurement of light from the entire visual field in the plane of the cornea and measuring the spectral radiance emitted from the emitting surface of a given light source in a given direction (Spitschan et al., 2019). This adds complexity as spectral irradiance are dependent upon the size of the light source, the distance of the observer from the source and the distribution of the emitted light. In field

studies this is difficult to achieve as light exposure and its intensity are constantly changing due to day-to-day variance in self-selected lighting patterns and sources of LAN. For example, there may be differences in the use of light emitting technologies in bed during sleep from night to night. Secondly, measurement of LAN at the plane of the eye is difficult and while the levels and sources of LAN are sufficient to elicit a physiological response the changes in the orientation of the head as well as opening and closing of the eyes leads to various photic exposure patterns. This limits the ability to measure light reaching the cornea. The importance of directionality of the light source is significant with Lasko and colleagues (1999) reporting that light exposure in the upper visual field resulted in a significant reduction in melatonin compared to exposure of the lower visual field. These findings are supported by Glickam and colleagues (2003) who reported that exposure of light to both the full retina and directed at the upper field resulting is significantly greater suppression of melatonin compared to exposure to the lower visual field. Other studies have reported that light presented at the nasal side of the eye results in greater suppression compared to when presented at the temporal side (Ruger et al., 2005; Visser et al., 1999). Other factors were LAN exposure calculated at one time point may not be representative is due to changes in light due to solar cycles, aging of the light source which results in reduction of lumen and changes to the spectral wavelength (Houser & Esposito, 2021).

7.2.3 Utililising appropriate proxies of circadian disruption

Throughout this research, metrics derived from proxies of circadian disruption were employed to examine whether either the perception of LAN or intensity of LAN experienced in the sleeping environment was associated with circadian disruption. As outlined in chapter 1, circadian rhythms are endogenously sustained oscillations which generate rhythms to a near 24h period. The rhythms which are generated under entrained conditions are in part influenced and driven by behavioural and environmental rhythms and are not circadian (Broussard et al., 2017; Vetter, 2020). Chronotype, and social jetlag are conceptualized based upon behaviour and environmental information and can only be viewed as proxies for circadian disruption. For example, one of the variables derived from the MCTQ is social jetlag. Social jetlag only captures system level outside (sleep timing) and does not provide an understanding into how social jetlag relates to internal phase relations or molecular level rhythms (Vetter, 2020). Additionally, as mentioned in chapter 6 actigraphy provides a proxy of circadian function which reflects highly systematic clock output but does not provide insight into

endogenous clock function or the molecular interplay of clock-regulated processes (Broussard et al., 2017).

While this thesis did utilise different metrics to measure circadian disruption, it did not assess the multiple levels where the impacts of circadian disruption can be observed. For example, Skene et al. (2018) found that circadian disruption induced by shift work did not impact on traditional markers of SCN phase however, several of the circulating plasma metabolites became misaligned from the SCN rhythm and instead became aligned to the new feeding/fasting and sleep/wake cycles. Separately, in studies examining the impact of exposure to 5lux dLAN found that while locomotor activity remained intact reflecting an intact rest/activity rhythm alteration in the expression of core clock genes were observed in the liver (Fonken et al., 2011).

Future work in this area will need to evolve from measurement of sleep timing and identify biomarkers to understand at what intensity does bedroom environment LAN result in circadian disruption. Most of the studies have used melatonin concentration, timing of DLMO and CBT minimum, changes in alertness levels, and alterations to core clock genes such as Per3 to investigate the effect of LAN on circadian disruption. However, there are limitations to this approach. Circadian biomarkers fluctuate throughout the day which results in higher costs and reduced capability to collect larger number of participants at a population. In addition, these classical biomarkers only provide information on the immediate acute effects of circadian disruption but provide no insight into the impacts of long-term circadian disruption which may be mediated by bedroom environment LAN. Future research should attempt to identify biomarkers which are independent of time sampling but may serve as universal biomarkers of circadian disruption. One such biomarker which is found in blood is CD36 which was found using a within groups design to be increased in mice exposed to various night shift models compared to when exposed to a 12:12LD cycle (Van Dycke et al., 2015). The increase in this gene was observed 14 days after exposure to the shift work model suggesting that this biomarker may be able to identify the long-term impacts on LAN exposure. Future studies should attempt to examine whether CD36 is increased in those with exposure to home setting LAN. This would allow for large scale epidemiology studies to investigate whether home-setting LAN can have chronic effects on the circadian system.

7.3 Strengths and limitations

From the outset, a key strength of this study is that it used both cross-sectional and ecological study design. Oftentimes cross-sectional research is seen as limited as cause and effect cannot be established. However, as highlighted above the findings from experimental work may not translate to the real-world. Additionally, few studies to date have examined the effects of home setting LAN exposure on sleep and health (Cain et al., 2020; Obayashi et al., 2013; 2014; 2018; Santhi et al., 2012) The overarching strength of this study is that it examined LAN exposure in the home environment. This is of importance given that LAN exposure in our private dwellings is now commonplace (Cain et al., 2020) with experimental studies suggesting that these levels may elicit adverse impacts on circadian rhythmicity (Philips et al., 2018). As highlighted above a major strong point of this research is that it utilized a detailed questionnaire to access the perceived sources of LAN in the sleeping environment. It employed validated measures of sleep, circadian misalignment and psychological health in order to examine for the first time whether individual-level LAN experienced in the sleep environment is associated with adverse impacts on both sleep and psychological health. The research also objectively measured LAN at the bedside and trespassing internal LAN. Although the intensity of LAN was measured in photic lux, this does not represent the spectral sensitivity of the ipRGCs photoreceptors which significantly contribute to NIF responses in response to light exposure. However, photic lux does provide a superior measure of LAN intensity compared to subjective measures of room brightness. Additionally, we measured the intensity of LAN at times where it could potentially have adverse impacts on sleep and circadian rhythmicity (Khasla et al., 2003). We calculated the average light intensity per night which is important as short pulses of LAN are more efficient in phase shifting the circadian clock than exposure to the same intensity of LAN over a long period (Chang et al., 2012). Additionally, position of the devices provided a measure of the intensity of external LAN trespassing into the sleeping environment. Our measurement of external LAN as indicated above is superior to satellite imaging data. This is due to light measurements being individualised to the participant's residence, being measured horizontally, and measuring the spectral wavelength which elicits the most influence on NIF responses to light (Brainard et al., 2001).

Sleep and rest-activity were measured using actigraphy. Measures derived from actigraphy has been found to be associated with polysomnography - the gold standard assessment for sleep measurement (Ancoli Israel et al., 2003). Measurement of sleep

and light intensity occurred over a two-week period rather than over a two-day period which occurred in previous studies (Obayashi et al., 2013; 2014; 2018). The longer duration of data collection provides a more accurate representation of sleep and true LAN exposure experienced as previous studies have indicated that two-day LAN intensity measurement are only moderately correlated with each other (Obayashi et al., 2014; 2018). This study also measured mood and sleepiness at multiple timepoints daily. This has been suggested to provide a more accurate representation of true state rather than measurement at one time point which may be influenced by other factors (Tsanas et al., 2016).

This research utilised the validated EST which has been previously employed to examine attention bias in poor sleepers (Barclay & Ellis, 2013). Additionally, the study modified the DPT to specifically examine whether the perception of LAN was associated with an attention bias towards LAN sleeping environments. This approach is novel as only a small number of studies have examined specific sources of the sleeping environment which elicit attention bias in other cognitive paradigms (Tang et al., 2006; Woods et al, 2009). Our findings also report that response time latency behaviour to stimuli may be dependent upon contextual factors unrelated specific content of the image.

Another strength of this research is that it examined whether low level-LAN intensity, which was recorded in chapter 6, recorded to be below 1lux was associated with adverse impacts for sleep and health. Oftentimes light at this level is employed as a control condition or in contrast to bright light condition (Chang et al., 2011; Gooley et al., 2011; St Hilaire et al., 2012; Philips et al., 2019; Zeitzer et al., 2000). However, several studies have showcased that LAN levels at this level (<1lux) has been observed to allow for entrainment in both humans and animals (Butler & Silver, 2011; Wright et al., 2001) along with being associated with increased fragmentation (Stebelova et al., 2020). As a result, this assumption of low-level lighting acting as a control group may potentially have two adverse outcomes. Firstly, if low level light intensity can elicit effects on the circadian system, then it is not a true control. In this case, if between groups differences exist between bright light and low-level light it can only be inferred that the intensity of LAN influences the circadian system and it cannot be inferred that the low LAN condition is similar to complete darkness. Employing low level LAN as a control group also limits the effect size in the differences of the means as the low-level LAN may elicit circadian responses to light.

In chapter 2 and 3 the age profile was diverse and in chapter 6 the field study focused primarily on a younger cohort. The investigation of the impacts of LAN on younger adults is important as previous field research work has only focused upon older adults, thus limiting the generalisability of findings (Obayashi et al., 2014;2014;2013;2018). As individuals age, circadian photosensitivity decreases. Aging results in a decrease in light transmission due to clouding of and yellowing of the crystalline lens (Pescosolido et al., 2016), reduced pupil size (Bitsios et al., 1996), less lens transmittance (Najjar et al., 2014), an increase in cataracts (Brondsted et al., (2015) and a reduction in the number of ipRGCs (Esquiva et al., 2017). Pupil size when pharmacologically dilated is associated with attenuation in the amount of melatonin suppression in response to light (Higuchi et al., 2008). Along with being less photosensitive, the loss of lens transmittance has been suggested to lead to less sensitivity to short wavelength light. This is supported by Najjar et al. (2014) who found that older adults display attenuated melatonin suppression to short wavelength light rather than long wavelength light (Najjar et al., 2014). However, while our study displays strength in examining the effects of LAN exposure in young adults, our findings are not generalizable to adolescents and children. It has been suggested that children may have a two-fold increased sensitivity to light compared to adults (Akacem et al., 2016; Turner & Mainster, 2008) with the magnitude of melatonin suppression in response to light exposure being higher in children compared to adults (Higuchi et al., 2014) and both low and high colour light eliciting similar melatonin suppressing effects (Higuchi et al., 2014). Future studies should incorporate all age groups and examine between group differences.

There are some limitations to this research, the first of which is that it employed self-report measures to assess psychological health. It is plausible that reporting the perception of LAN may not accurately reflect the true perception of LAN experienced as individuals may have completed the survey at a time when they were outside of their sleeping environment. As a result, our self-report measures may be influenced by recall bias. Secondly, although we previously outline the strengths in our approach to measure LAN intensity, we note that our measurements were based upon public street lighting. These measures are not reflective of private dwelling outdoor lighting or commercial lighting. As a result, our measurement may underestimate the true level of outdoor LAN.

A significant limitation of this thesis is that assessment of light exposure was confined to the sleeping environment. However, the photic history of an individual

throughout the day can impact on an individual's subsequent biological response to LAN, their sleep and mood. Several studies have highlighted that exposure to low level light throughout the day can lead to enhanced acute and circadian phase shifting responses when exposed to LAN later in the biological evening (Chang et al., 2011; Hebert et al., 2002; Smith et al., 2004). Conversely, exposure to light – which is of strong zeitgeber strength throughout the day – can positively impact the amplitude of the circadian rhythm along with improving sleep and mood (Bano-Otalora et al., 2021; Burns et al., 2021; Figueiro et al., 2021; Wright et al., 2015). Additionally, higher levels of LAN exposure 3h before habitual bedtime has been found to be a strong predictor of sleep disturbance when sleep ensues (Cain et al., 2020). These findings highlight the complexity in understanding the relationship between LAN exposure and its impacts on circadian rhythmicity and health and showcase that to examine these associations, assessment of light exposure of the whole day should be provided. Taking these findings into consideration, further research avenues should examine whether day to day variance in light exposure throughout a duration of study is a predictor of variance measures derived from actigraphy.

It is acknowledged that the results from chapter 6 must be taken with caution due to the limited sample size. As a result of the outbreak and sustained persistence of COVID-19 this study was unfortunately paused in March 2020 and later ceased. The study presented in chapter 6 reflects of proof-of-concept study. A significant effort was made to ensure that the methodology of this study was rigorous to provide reliable and valid understanding on the effects of bedroom environment LAN on sleep, circadian misalignment, and psychological health. There were significant challenges with this work. From the outset there were issues with the objective measure of LAN intensity with many recordings of bedroom intensity reporting values that no LAN was present. There were also issues with recording of daily monitoring of mood and sleepiness. As indicated in the methods of chapter 6, the methodology for measuring mood and sleepiness was changed. In addition, recruitment of participants was difficult for this study as individuals felt that the length of the study was too long and onerous on the participant. Finally, most of the studies included in this thesis were exploratory. This meant that we did not correct for multiple testing and as such the statistics presented should be interpreted with some caution. However, effect sizes and confidence intervals were presented.

7.4 Future research agenda

As mankind has modernised we are no longer exposed to the cyclical lighting pattern from the natural solar cycle. We each have individual differences in our exposure to both natural light during the day and artificial LAN during the night. These differences in the pattern of light exposure along with interindividual differences in response to light place significant barriers in trying to investigate the effect of home-setting LAN on sleep, circadian rhythmicity and health. As indicated throughout this thesis only a small number of studies have examined the effects of low-level LAN which is typically experienced in home settings. Some of these studies have been carried out in laboratories which lack ecological validity and only a small number of field studies have been conducted in older adults or those with bipolar disorder which limits the generalizability of their findings. Future work must endeavor to carry out more field research to identify the threshold of light most relevant for predicating adverse outcomes for sleep and psychological health. While studies from laboratories have showcased that 1lux LAN can increase sleep disturbance (Stebelova et al., 2020) and entrainment studies observing that exposure to 1lux can entrain the circadian clock (Butler & Silver, 2011; Wright et al., 2004), it is unclear the specific threshold of LAN intensity in the sleeping environment that predicts adverse impacts on sleep and health. This can be achieved by collecting LAN as a continuous variable rather than categorizing LAN exposure to identify the specific threshold. While the above-mentioned laboratory studies have provided an insight into the effect of specific levels of LAN on sleep, these studies findings may not fully translate into the real world. As discussed before, in laboratory studies many factors are controlled for (e.g., proximity to light source, previous light history) and as result the findings from laboratory environment may not fully complement the findings from field studies. However, the findings from basic science should provide a conceptual basis and understanding when examining the impacts of LAN in the home environment.

Future field studies from the outset must recruit a more diverse population sample with a large age profile due to the biological response to light varying as we age (Higuchi et al., 2008; Najjar et al., 2014) These studies should accurately record individual-level light exposure patterns across the full 24-h period. The placing of the light meter should be as close to the retina to reflect light perceived in the visual field (CIE, 2018). This is challenging however, as the meter would need to be placed on the forehead which may not be feasible during sleeping and would be challenging in ecological design studies to measure light during the day. Recordings should occur for

at least a one-week period to acquire an accurate representation of the typical level of light exposure experienced and to examine the degree variance across the duration of examination. While in laboratory studies individual photosensitivity can be measured by investigating melatonin suppression and phase shifts along with pupillometry measurements, this may not be feasible in field research. As a result, future field research should ensure to collect detailed information on individual's perception of light exposure in the sleeping environments along with other detailed information on factors known to predict individual photosensitivity such as age, gender, race, chronotype, medication usage (McGlashan et al., 2018), medical history and photic light history. Measures of sleep and rest-activity should be obtained objectively.

Future field research work will need to evolve from using measures of sleep timing and identify and utilise more appropriate biomarkers to understand both the acute and long-term impacts of circadian disruption induced by bedroom environment LAN. Most studies to date have used melatonin concentration, timing of DLMO, alertness levels, differences in the timing of CBT and alterations of core clock genes such as Per3, to investigate circadian disruption induced by light exposure. However, these measures are limited as circadian biomarkers fluctuate throughout the day, which results in higher costs and reduced capability to collect larger number of participants at a population. Future research should assess non circadian markers such as CR36 which was proposed by Van Dyke and colleagues (2015). This marker not only provides an understanding of the acute effects of LAN exposure but also the long-term effects. In addition, the collection of CR36 can be from a blood sample which may be more efficient for field research compared to collection of circadian biomarkers where collection is required at multiple time points. In addition, classical circadian biomarkers and metrics of circadian disruption only provide information on the immediate acute effects of circadian disruption but provide no insight into the impacts of long-term circadian disruption which may be mediated by bedroom environment LAN. However, one field study showcased using a longitudinal design that chronic exposure to increased levels of home setting LAN is associated with increased risk of developing depression (Obayashi et al., 2018).

While there is a need for more population level field studies, this brings its own limitations given the possible higher level of heterogeneity and variation between samples (Vetter et al., 2020). This may mean that in cross-sectional research that large and small increases in outcome variables may remain undetectable. To overcome these methodological limitations, future research should employ two strategies. First, if

collecting cross-sectional data at one time-period the duration of the study should be extended. Not only does a longer duration study allow for more accurate and reliable findings, it also enables a within group design to be employed so that variance within individual's data can be examined. As indicated increased variation in midsleep timing between workdays and free days (which may be mediated by increased LAN exposure) is associated with increased risk of adverse health outcomes (Henderson et al., 2019; Lavendovski et al., 2011). Future research should continue to be used within group designs to examine whether higher levels of LAN intensity are associated with increased levels of sleep variance. This is important to consider because if higher level LAN intensity is a predictor of increased day-to-day variance or of increased levels of social jetlag, this could potentially lead to adverse impacts on health. Additionally, these studies should further examine whether home-setting LAN is a predictor of delayed DLMO. Although some studies have provided evidence that LAN exposure is potential predictor of eveningness (Stolhard et al., 2017; Wright et al., 2013; Zerbini et al., 2018).

Future research needs identify the level of LAN most relevant for the prediction of adverse outcomes for circadian rhythmicity, sleep and health. When this level is identified, public health advice can educate individuals on what levels of light intensity in the sleeping environment should not be exceeded upon. However, most research identifies the minimum of light to elicit adverse effect by examining the half maximum response (ED_{50}) which is conventionally employed to investigate the photic dose required to elicit a half-maximum circadian response in comparison to a saturating light level that would create a maximum response. If low level light intensity can induce an equivalent ED_{50} response, then photic sensitivity can be inferred. While this approach operationally defines the lower limits of light sensitivity with respect to eliciting a NIF responses, a number of studies have indicated that light below these generally accepted thresholds of light sensitivity elicit NIF (Walbeek et al., 2021). For example, up until the late 2000s most research would suggest that 100-200lux was sufficient to induce phase shifting responses (Gooley et al., 2011; Zeitzer et al., 2000). More recently, the average light intensity of 24.60lux is sufficient to induce an ED_{50} of melatonin suppression. However, more recently, as previously discussed, light levels as low as 5lux can result in changes in sleep architecture, sleep duration (Cho et al., 2016; 2018) and sleep timing (Obayashi et al., 2014) with other studies reporting exposure to 1lux LAN can increase poor quality sleep (Stebelova et al., 2020). This illustrates that while the ED_{50} is important for providing a general approximation of thresholds of light in eliciting biological potency of light, this fails to recognize that lower levels of LAN

which are more commonly experienced in the sleeping environment may be adversely impacting on sleep. As a result of these findings future research may need to examine the influence of LAN intensity measured as a continuous variable to identify the specific threshold where LAN exposure is disruptive to sleep and impacts biological potency.

Our research reported that the perception of external LAN was associated with adverse impacts on sleep and psychological health. However, no association between objective measurements of external LAN and subjective perception of external LAN were found. In order to examine what is the contributing factor to the perception of external LAN, despite no measured presence, this research examined whether individuals who perceive LAN have an attention bias towards sleep stimuli/LAN. While our studies did not demonstrate evidence of attention bias, future studies should use similar methodologies employed by Beattie et al. (2017) which use eye tracking devices to examine where individuals orient their gaze. For example, studies could examine whether individuals who perceive LAN direct their gaze towards LAN sources when free-viewing sleeping environments.

7.5 Concluding remarks

The widespread use of artificial light has facilitated individuals in their home-settings to extend their biological day with LAN. Exposure to LAN continues to grow and evolve either through increases in urban light pollution or in the home environment through exposure to LED luminaires or through our use of ever increasing in size light emitting personal technologies. However, with this growth a more informed understanding of the biological impacts of these sources on circadian rhythmicity, general health and psychological health needs to be examined and investigated. As a result of LAN, individuals now have agency to control the times they sleep, socialise, eat and work. However, this agency may come with a price either through eliciting circadian disruption which leads to adverse health consequences or LAN exposure directly influencing general and psychological health. It is imperative that as LAN exposure increases research not only focuses on the quality and energy efficiency of light but also its impacts to health and sleep. While it not possible to eliminate LAN, further research is required on the biological and psychological impacts of LAN exposure to inform policy and educate citizens to minimize light. There is a need to create lighting technologies/solutions which do not impact on either biological rhythmicity or health.

An important room in everyone's residence is their sleeping environment. This is an area which should afford sleepiness, sleep maintenance and good sleep quality. However, through our lighting practices or trespassing of LAN these spaces may afford wakefulness and poor sleep quality. While this research was limited to LAN exposure in the sleeping environment, it is the space where the biological clock is most vulnerable to circadian disruption. The research derived from this thesis examined using a valid and comprehensive methodology how the perception and intensity of LAN in the sleeping environment is associated with impacts on sleep and psychological health. Given our findings, it is important that citizens are educated on strategies they can employ to minimise LAN in their sleep environment, which may have positive implications for their general psychological health.

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Appendix A

Light at Night Questionnaire

The following questionnaire is related to assessing your light at night exposure, lighting habits at night and your perception of these lighting habits. Your answers should indicate the most accurate reply for the majority of nights in the past month. Please answer all questions.

Where do you live?

- A city
- Suburb
- Urban Town
- Semi Rural Environment (village)
- Rural Environment (countryside)

What is your sex?

- Male
- Female

What age are you?

What is your Eircode/Address? Please use this address to locate your Eircode https://www.eircode.ie

What type of house do you live in?

- Detached House
- Semi detached house
- Terrace
- Apartment
- Bungalow

Where is your bedroom located?

- At the front of the premises
- At the back of the premises

Do you sleep with a light on (e.g. main light or bedside light)?

- Always
- Very often
- Sometimes
- Rarely

- Never

At what time do you usually turn the main light off in your bedroom before you go to sleep?

- 10 minutes before
- 20 minutes before
- 30 minutes before
- 40 minutes before
- 50 minutes before
- 60+ minutes before

When both your main bedroom light and bedside lights are off would you consider your bedroom to be bright?

- Yes
- No

With all lights turned off, would you consider your room to be

- Very Bright
- Bright
- Slightly Bright
- Dark
- Very Dark

If you awake from sleep during the night do you turn on the light?

- Yes
- No

If so, for how long?

- 0-5 mins
- 5-10 mins
- 10-20 mins
- 20+ mins
- N/A

In your bedroom do you have blinds/curtains on your windows

- Yes
- No

If so, how effective are these blinds/curtains in preventing light from trespassing into the bedroom

- Very effective
- Effective
- Moderately effective

- Slightly effective
- Not effective at all
- N/A

Does artificial light outside light (i.e. street lights, traffic lights, headlights etc.) enter the bedroom when you are sleeping?

- Yes
- No

Do you feel that these outdoor light sources at night effects your ability to fall asleep?

- Yes
- No

What is the colour of the dominant light (outdoor light) in the vicinity of your house?

- White Commercial
- White Streetlight
- Pink Streetlight (high pressure sodium or metal halide)
- Orange Streetlight (low pressure sodium)
- Other
- N/A

Do you feel that light at night interferes with your sleep quality after falling asleep?

- Yes
- No

In your bedroom, what are the most prevalent light emitting sources?

- Alarm Clock
- Computer/Tablet
- Street Lighting
- Car Headlights
- Mobile Phone
- Television
- Other
- N/A

Do you usually have lights on outside your bedroom door which illuminate your bedroom (i.e. bathroom or landing light)?

- Yes
- No

Before sleep, do you use electronic devices in bed (i.e.mobile phone, tablet, ebook, personal computer)?

- Yes

- No

How often do you use these devices

- Never
- Rarely
- Regularly
- Always

Do you check/use these electronic devices within an hour of attempting to sleep?

- Yes
- No

After using these electronic devices, how long does it usually take you to fall asleep?

- 0-10 mins
- 10-20 mins
- 20-30 mins
- 30-40 mins
- 40-50 mins
- 60+ mins

Do you feel that use of these devices affect your sleepiness/quality of sleep in a negative manner?

- Yes
- No

If you awake from sleep during the night, do you check an electronic device (i.e. phone, tablet, personal computer)?

- Yes
- No

If you awake from sleep during the night, how often do you check your device upon awaking?

- Never
- Rarely
- Regularly
- Always

To what degree do you think that light at night exposure before sleep effects the quality of your sleep?

- Not Disruptive
- Disruptive
- Very Disruptive
- Extremely Disruptive

Does any particular noise annoy you during the night?

- Yes
- No

At night, what are the major sources of noise pollution in your home?

- Transport (Cars, Trains & Planes)
- Neighbours
- Roommates/Family/Children
- Animals
- Religious Buildings

Do the above noise disturbances negatively impact on your ability to fall asleep?

- Yes
- No
- N/A

Do the above noise disturbances result in you awakening from sleep during the night?

- Yes
- No
- N/A

Do you think that the above noise disturbances negatively impacts on the quality of your sleep?

- Yes
- No
- N/A

In your bedroom, are you sensitive to environmental noise?

- Yes
- No

Pittsburgh Sleep Quality Index (PSQI)

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

1. During the past month, what time have you usually gone to bed at night? _____
2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night? _____
3. During the past month, what time have you usually gotten up in the morning? _____
4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.) _____

5. During the past month, how often have you had trouble sleeping because you…	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe:				
6. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
	No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem
8. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?				
	Very good	Fairly good	Fairly bad	Very bad
9. During the past month, how would you rate your sleep quality overall?				

	No bed partner or room mate	Partner/room mate in other room	Partner in same room but not same bed	Partner in same bed
10. Do you have a bed partner or room mate?				
	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
If you have a room mate or bed partner, ask him/her how often in the past month you have had:				
a. Loud snoring				
b. Long pauses between breaths while asleep				
c. Legs twitching or jerking while you sleep				
d. Episodes of disorientation or confusion during sleep				
e. Other restlessness while you sleep, please describe:				

Appendix C

Munich ChronoType Questionnaire (MCTQ)

In this questionnaire, you report on your typical sleep behaviour over the past 4 weeks. We ask about work days and work-free days separately. Please respond to the questions according to your perception of a standard week that includes your usual work days and work-free days.

I have a regular work schedule (this includes being, for example, a housewife or househusband):

Yes ☐ I work on 1☐ 2☐ 3☐ 4☐ 5☐ 6☐ 7☐ days per week.

No ☐

Is your answer "Yes, on 7 days" or "No", please consider if your sleep times may nonetheless differ between regular 'workdays' and 'weekend days' and fill out the MCTQ in this respect.

Please use 24-hour time scale (e.g. 23:00 instead of 11:00 pm)!

Workdays

Image 1: I go to bed at _____ o'clock.

Image 2: Note that some people stay awake for some time when in bed!

Image 3: I actually get ready to fall asleep at _____ o'clock.

Image 4: I need _____ minutes to fall asleep.

Image 5: I wake up at _____ o'clock.

Image 6: After _____ minutes I get up.

I use an alarm clock on workdays: Yes ☐ No ☐

If "Yes": I regularly wake up BEFORE the alarm rings: Yes ☐ No ☐

Free Days

Image 1: I go to bed at _____ o'clock.

Image 2: Note that some people stay awake for some time when in bed!

Image 3: I actually get ready to fall asleep at _____ o'clock.

Image 4: I need _____ minutes to fall asleep.

Image 5: I wake up at _____ o'clock.

Image 6: After _____ minutes I get up.

My wake-up time (Image 5) is due to the use of an alarm clock: Yes ☐ No ☐

There are particular reasons why I cannot freely choose my sleep times on free days:

Yes ☐ If "Yes": Child(ren)/pet(s) ☐ Hobbies ☐ Others ☐, for example:_____

No ☐

Appendix D

The Cognitive Failures Questionnaire (Broadbent, Cooper, FitzGerald & Parkes, 1982)

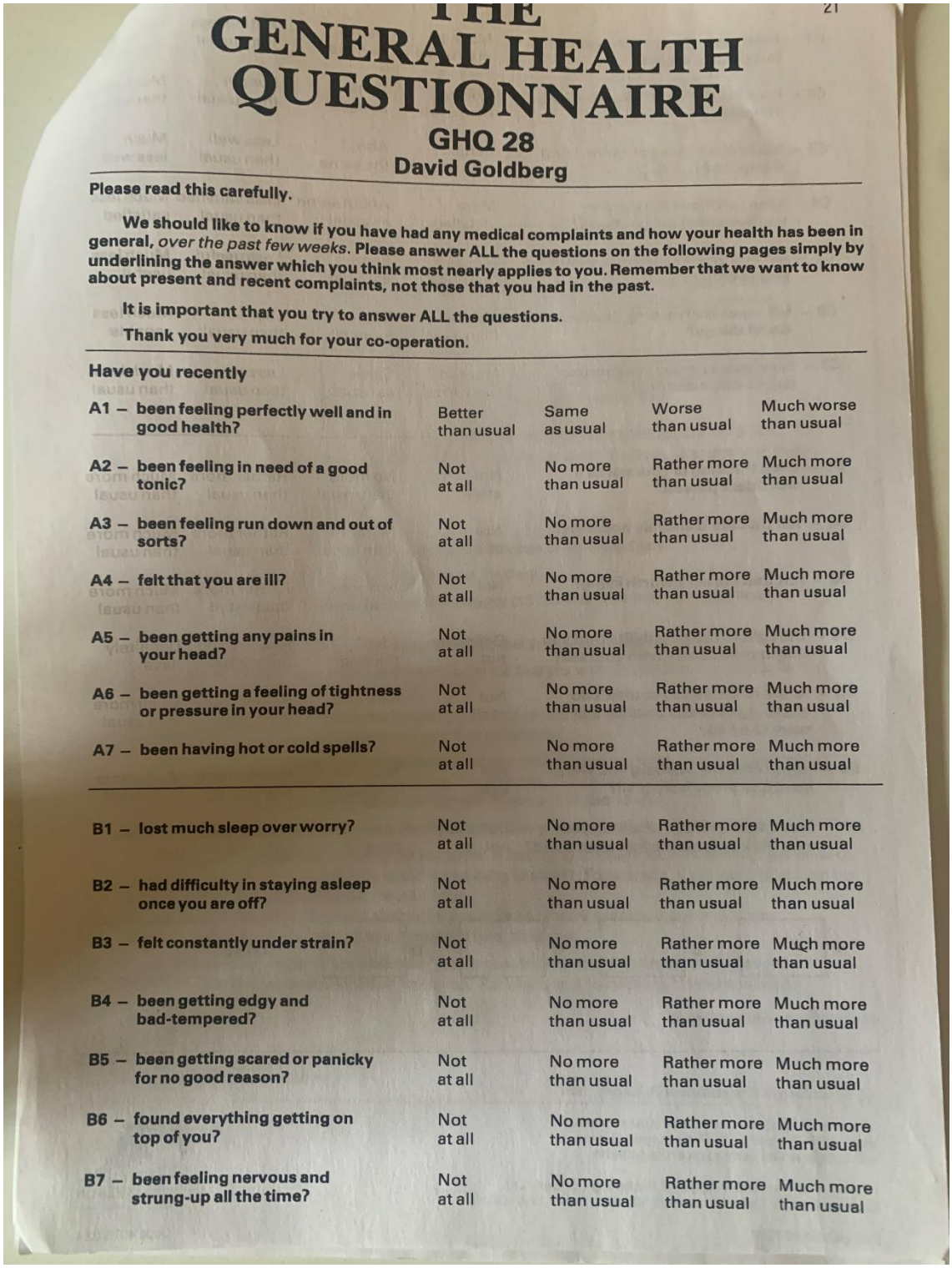
The following questions are about minor mistakes which everyone makes from time to time, but some of which happen more often than others. We want to know how often these things have happened to your in the past 6 months. Please circle the appropriate number.

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

Cont…

		Very often	Quite often	Occasion-ally	Very rarely	Never
15.	Do you have trouble making up your mind?	4	3	2	1	0
16.	Do you find you forget appointments?	4	3	2	1	0
17.	Do you forget where you put something like a newspaper or a book?	4	3	2	1	0
18.	Do you find you accidentally throw away the thing you want and keep what you meant to throw away – as in the example of throwing away the matchbox and putting the used match in your pocket?	4	3	2	1	0
19.	Do you daydream when you ought to be listening to something?	4	3	2	1	0
20.	Do you find you forget people's names?	4	3	2	1	0
21.	Do you start doing one thing at home and get distracted into doing something else (unintentionally)?	4	3	2	1	0
22.	Do you find you can't quite remember something although it's "on the tip of your tongue"?	4	3	2	1	0
23.	Do you find you forget what you came to the shops to buy?	4	3	2	1	0
24.	Do you drop things?	4	3	2	1	0
25.	Do you find you can't think of anything to say?	4	3	2	1	0

Appendix E



THE GENERAL HEALTH QUESTIONNAIRE

GHQ 28

David Goldberg

Please read this carefully.

We should like to know if you have had any medical complaints and how your health has been in general, *over the past few weeks*. Please answer ALL the questions on the following pages simply by underlining the answer which you think most nearly applies to you. Remember that we want to know about present and recent complaints, not those that you had in the past.

It is important that you try to answer ALL the questions.

Thank you very much for your co-operation.

Have you recently

A1 – been feeling perfectly well and in good health?	Better than usual	Same as usual	Worse than usual	Much worse than usual
A2 – been feeling in need of a good tonic?	Not at all	No more than usual	Rather more than usual	Much more than usual
A3 – been feeling run down and out of sorts?	Not at all	No more than usual	Rather more than usual	Much more than usual
A4 – felt that you are ill?	Not at all	No more than usual	Rather more than usual	Much more than usual
A5 – been getting any pains in your head?	Not at all	No more than usual	Rather more than usual	Much more than usual
A6 – been getting a feeling of tightness or pressure in your head?	Not at all	No more than usual	Rather more than usual	Much more than usual
A7 – been having hot or cold spells?	Not at all	No more than usual	Rather more than usual	Much more than usual
B1 – lost much sleep over worry?	Not at all	No more than usual	Rather more than usual	Much more than usual
B2 – had difficulty in staying asleep once you are off?	Not at all	No more than usual	Rather more than usual	Much more than usual
B3 – felt constantly under strain?	Not at all	No more than usual	Rather more than usual	Much more than usual
B4 – been getting edgy and bad-tempered?	Not at all	No more than usual	Rather more than usual	Much more than usual
B5 – been getting scared or panicky for no good reason?	Not at all	No more than usual	Rather more than usual	Much more than usual
B6 – found everything getting on top of you?	Not at all	No more than usual	Rather more than usual	Much more than usual
B7 – been feeling nervous and strung-up all the time?	Not at all	No more than usual	Rather more than usual	Much more than usual

C1 – been managing to keep yourself busy and occupied?	More so than usual	Same as usual	Rather less than usual	Much less than usual
C2 – been taking longer over the things you do?	Quicker than usual	Same as usual	Longer than usual	Much longer than usual
C3 – felt on the whole you were doing things well?	Better than usual	About the same	Less well than usual	Much less well
C4 – been satisfied with the way you've carried out your task?	More satisfied	About same as usual	Less satisfied than usual	Much less satisfied
C5 – felt that you are playing a useful part in things?	More so than usual	Same as usual	Less useful than usual	Much less useful
C6 – felt capable of making decisions about things?	More so than usual	Same as usual	Less so than usual	Much less capable
C7 – been able to enjoy your normal day-to-day activities?	More so than usual	Same as usual	Less so than usual	Much less than usual

D1 – been thinking of yourself as a worthless person?	Not at all	No more than usual	Rather more than usual	Much more than usual
D2 – felt that life is entirely hopeless?	Not at all	No more than usual	Rather more than usual	Much more than usual
D3 – felt that life isn't worth living?	Not at all	No more than usual	Rather more than usual	Much more than usual
D4 – thought of the possibility that you might make away with yourself?	Definitely not	I don't think so	Has crossed my mind	Definitely have
D5 – found at times you couldn't do anything because your nerves were too bad?	Not at all	No more than usual	Rather more than usual	Much more than usual
D6 – found yourself wishing you were dead and away from it all?	Not at all	No more than usual	Rather more than usual	Much more than usual
D7 – found that the idea of taking your own life kept coming into your mind?	Definitely not	I don't think so	Has crossed my mind	Definitely has

A ☐ **B** ☐ **C** ☐ **D** ☐ **TOTAL** ☐

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Appendix F

Questionnaire used for assessing experiences of ALAN, and P values for all comparisons with item responses for each question on each of the dependent variables analysed in the study.

	PSQI	MSFsc	SJL	SDW	GHQ	CFQ
What type of house do you live in?						
• Detached	.614	.056	.329	.078	.517	.409
• Semi-Detached						
• Terrace						
• Apartment						
• Bungalow						
Do you sleep with a light on (e.g. main light or bedside light)?						
• Always	**.001**	.401	.276	.463	.058	**.024**
• Very often						
• Sometimes						
• Rarely						
• Never						
When both your main bedroom light and bedside light are off would you consider your bedroom to be bright?						
• Yes	**.028**	.110	.103	.494	.763	.466
• No						
With all lights turned off would you consider your room to be…						
• Very Bright	.056	.743	.067	.739	**.008**	**.001**
• Bright						
• Slightly Bright						
• Dark						
• Very Dark						
If you awake from sleep during the night do your turn the light on?						
• Yes	**<.001**	.105	.317	.118	**<.001**	**.021**
• No						

Do you have blinds/curtains on your windows

• Yes	.752	.189	.089	.833	.211	.407
• No						

Does artificial outside light (i.e. street lights, headlights etc) enter the bedroom when you are sleeping?

• Yes	**.002**	.621	.467	.483	**< .001**	**<.001**
• No						

Do you feel that outdoor sources at night effects your ability to fall asleep?

• Yes	**.001**	.556	.632	.904	**.006**	.053
• No						

Do you feel that light at night interferes with your sleep quality after falling asleep?

• Yes	**.034**	.441	.230	.689	**.014**	**.002**
• No						

Do you have lights on outside your bedroom door which illuminate you bedroom (i.e. landing light)?

• Yes	**.037**	.877	.831	.524	.179	**.022**
• No						

Before sleep, do you use electronic devices in bed (i.e. mobile phone, tablet, ebook, personal computer?

• Yes	.190	.159	**.009**	.510	.118	.211
• No						

Do you check/use these electronic devices within an hour of attempting to sleep?

• Yes	.091	.323	.128	.527	**.005**	**.006**
• No						

Do you feel that these devices affect your sleepiness/quality of sleep in a negative manner?

• Yes	**<.001**	.126	**.001**	.914	**<.001**	**<.001**

- No
- Don’t know

If you awake from sleep during the night, do you check an electronic device?

• Yes	**.006**	**.014**	**.012**	.332	**<.001**	**<.001**
• No						

Do you think that light at night exposure before sleep effects the quality of your sleep?

• Not Disruptive	**.001**	**.009**	**.004**	.648	**<.001**	**<.001**
• Disruptive						

Appendix G

Dysfunctional Beliefs and Attitudes about Sleep (DBAS)

Name: _____ **Date:** _____

Several statements reflecting people's beliefs and attitudes about sleep are listed below. Please indicate to what extent you personally agree or disagree with each statement. There is no right or wrong answer. For each statement, circle the number that corresponds to your own personal belief. Please respond to all items even though some may not apply directly to your own situation.

Strongly Disagree — Strongly Agree

0 1 2 3 4 5 6 (7) 8 9 10

1. I need 8 hours of sleep to feel refreshed and function well during the day.

0 1 2 3 4 5 6 7 8 9 10

2. When I don't get proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer.

0 1 2 3 4 5 6 7 8 9 10

3. I am concerned that chronic insomnia may have serious consequences on my physical health.

0 1 2 3 4 5 6 7 8 9 10

4. I am worried that I may lose control over my abilities to sleep.

0 1 2 3 4 5 6 7 8 9 10

5. After a poor night's sleep, I know that it will interfere with my daily activities on the next day.

0 1 2 3 4 5 6 7 8 9 10

6. In order to be alert and function well during the day, I believe I would be better off taking a sleeping pill rather than having a poor night's sleep.

0 1 2 3 4 5 6 7 8 9 10

7. When I feel irritable, depressed, or anxious during the day, it is mostly because I did not sleep well the night before.

0 1 2 3 4 5 6 7 8 9 10