# A Randomised Controlled Trial of Bright Light Therapy and Physical Activity for Delayed Sleep-Wake Phase Disorder in Adolescents

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# Table of Contents

# List of Tables

# List of Figures

## List of Appendices

## Summary

There is a widespread and consistent tendency for sleep timing to delay during adolescence. This places adolescents at risk of curtailed sleep duration and a plethora of negative daytime consequences. A more pronounced delay in sleep and circadian timing can lead to Delayed Sleep-Wake Phase Disorder (DSWPD). Given that adolescents' susceptibility to DSWPD was first reported over 20 years ago, it is surprising only three randomised controlled trials (RCTs) have investigated treatment for adolescent DSWPD, to date. This thesis aimed to address this research priority.

An overview of the empirical literature (Chapter 1) highlighted the need to further investigate the utility of bright light therapy for adolescent DSWPD. The recent development of portable short wavelength light emitting glasses (Re-Timer) allows for the inclusion of adjunct behavioural interventions, which may enhance treatment efficacy. A literature review suggested that scheduled physical activity may facilitate phase advances in circadian and sleep timing (Chapter 2). Therefore, a RCT evaluated the efficacy of bright light therapy and scheduled morning activity for the treatment of adolescent DSWPD. In particular, adolescents received either short wavelength (i.e., green, ~500nm) bright light therapy or long wavelength (i.e., red, ~640nm, placebo) light therapy and physical activity (i.e., interactive video gaming) or sedentary activity (i.e., sitting watching TV, control).

Results from the RCT added to existing literature supporting the use of bright light therapy for DSWPD, with adolescents reporting earlier sleep timing (i.e., sleep onset time, wake-up time), reduced sleep onset latency, increased total sleep time and improved daytime functioning (i.e., daytime sleepiness, fatigue, functional impairment) across treatment and follow-up (Chapter 4). However, treatment outcomes did not differ based on the wavelength of light or morning activity administered. Posteriori analyses revealed the manipulation

attempted in the RCT did not result in an objective increase in physical activity. Therefore, it is unclear from this thesis whether physical activity can supplement bright light therapy for adolescents with DSWPD.

DSWPD has been linked with adverse academic outcomes, which may be driven by impaired cognitive performance. Results from the RCT highlighted significant improvements in processing speed and some measures of working memory across treatment and follow-up (Chapter 5). However, it is unclear how clinically meaningful these improvements were, given a comparison with good-sleeping adolescents revealed a lack of statistically significant between-group differences in cognitive performance pre-treatment.

Researchers have called for further research investigating the aetiology of DSWPD and a review of the literature summarised emerging evidence for the role that cognitive "insomnia" processes may play in the development and maintenance of DSWPD (Chapter 3). Chapter 6 provided the first systematic evidence of such processes. Adolescents with DSWPD reported more repetitive negative thinking, physiological arousal, selective attention, sleep-onset misperception and safety behaviours, compared to good-sleeping adolescents. Most cognitive "insomnia" processes reduced alongside chronobiological treatment. However, it is possible that residual "insomnia" symptoms increased the risk of long-term relapse and therefore, further investigation in warranted.

The theoretical and clinical implications of these findings are discussed in Chapter 7, alongside avenues for future research.

## Declaration

I certify that this thesis does not incorporate without acknowledgement any material previously submitted for a degree or diploma in any university; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in text.

Signed: (Signature removed for security reasons)

Cele Richardson, B. Psych. (Hons.) Date: 06/10/2017

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## Glossary of Abbreviations

AASM= American Academy of Sleep Medicine

AAT= Alpha Attentuation Test

Act= Actigraphy

ANCOVA= Analysis of Covariance

ANOVA= Analysis of Variance

ASD= Autism Spectrum Disorder

BLT= Bright Light Therapy

BT= Bedtime

CBT= Cognitive Behavioural Therapy

CI= Catastrophising Interview

CID= Chronic Insomnia Disorder

CSHI= Clinical Sleep History Interview

DLMO= dim light melatonin onset

DSS= Digit-Symbol Substitution

DSWPD= Delayed Sleep-Wake Phase Disorder

EEG= electroencephalography

EMG= electromyography

EOG= electrooculography

ESS= Epworth Sleepiness Scale

FFS= Flinders Fatigue Scale

FIRST= Ford Insomnia Response to Stress Test

GP= General Practitioner

GS= Good Sleepers

ICSD-3= International Classification of Sleep Disorders- 3rd Edition

ISI= Insomnia Severity Index

KSS= Karolinska Sleepiness Scale

LED= Light Emitting Diode

LMM= Linear Mixed Model

LNS= Letter Number Sequencing

LOT= Lights Out Time

OOBT= Out of Bedtime

Op Span= Operational Span

PA= Physical Activity

PDSS= Pediatric Daytime Sleepiness Scale

PI= Primary Insomnia

PRC= Phase Response Curve

PSAS-C= Pre-Sleep Arousal Scale-Cognitive Subscale

PSAS-S= Pre-Sleep Arousal Scale-Somatic Subscale

PSG= Polysomnography

RCT= randomised controlled trial

REM= rapid eye movement sleep

SA= Sedentary Activity

SAAQ= Sleep Anticipatory Anxiety Questionnaire

SAMI= Sleep Associated Monitoring Index

SCN= suprachiasmatic nucleus

SD= Sleep Diary

SDS= Sheehan Disability Scale

SOL= Sleep Onset Latency

SOT= Sleep Onset Time

τ/ *tau*, the time taken to complete one cycle of the circadian rhythm

*Tmin*: core body temperature minimum

TST= Total Sleep Time

$Vo_2$: maximum volume of oxygen

WASO= Wake after Sleep Onset

WUT= Wake-up Time

ZT= zeitgeber time

## List of Manuscripts and Publications in Thesis

**Richardson, C.,** Gradisar, M., Short, M. & Lang, C. (2017) Can exercise regulate the circadian system of adolescents? Novel implications for the treatment of delayed sleep-wake phase disorder. *Sleep Medicine Reviews, 34,* 122-129.

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# Chapter 1

## Introduction to Sleep and Delayed Sleep-Wake Phase Disorder in Adolescence

## Sleep in Adolescence

Adolescence is a period of rapid developmental change. During adolescence, a number of "firsts" are experienced (e.g., first romantic relationship, first part-time job) and they develop several new skills (e.g., autonomy, how to drive, etc.; Carskadon, 2011). Although the term 'adolescents' is synonymous with 'teenagers', adolescents are not necessarily defined as being aged in their teen years (i.e., 13-19yrs). Instead, there are a number of markers (i.e., physiological, cognitive, psychosocial) that can be used to estimate the timing of adolescence. In relation to sleep, major changes (i.e., in sleep timing) occur from 10 years of age, until 21 years of age; hence, adolescence is thought to occur between 10-21 years of age (Roenneberg et al., 2004). Importantly, the changes that occur during adolescence often culminate in later bedtimes and restricted sleep duration, despite little change in sleep need (Carskadon, 2011; Gradisar, Gardner, & Dohnt, 2011; Sadeh, Dahl, Shahar, & Rosenblat-Stein, 2009). A number of biological, behavioural and environmental factors are thought to underlie later bedtimes in adolescence, and ultimately sleep restriction in this young population.

### Biological Contributing Factors

Pubertal development appears to trigger a delay in adolescents' sleep timing (i.e. evening chronotype; Andrade, Benedito-Silva, Domenice, Arnhold, & Menna-Barreto, 1993; Carskadon, Vieira, & Acebo, 1993; Randler, Bilger, & Díaz-Morales, 2009; Roenneberg et al., 2004). Evening chronotype is likely driven, and maintained, by a delay in the circadian system (i.e., body clock: ~24-hr rhythms of core body temperature, exogenous melatonin, behaviour, etc.), which is one of the primary determinants of sleep timing (Duffy, Rimmer, & Czeisler, 2001; Taillard, Philip, Coste, Sagaspe, & Bioulac, 2003). Indeed, the timing of endogenous melatonin (i.e., sleep promoting hormone) secretion becomes later across

adolescence (Carskadon, Acebo, Richardson, Tate, & Seifer, 1997; Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998) and later melatonin onset has been associated with later bedtimes in adolescents (Crowley, Acebo, Fallone, & Carskadon, 2006; Saxvig et al., 2013). Alternatively, there is some, albeit limited, evidence that evening preference during adolescence could be driven by a lengthening of the body clock (i.e., time taken for endogenous rhythms to complete one revolution; Carskadon & Acebo, 2002; Carskadon, Labyak, Acebo, & Seifer, 1999). However, further research is needed to test this hypothesis.

In addition to changes in the circadian system, sleep homeostatic pressure (i.e., sleepiness) is thought to accumulate slower in adolescents, delaying the onset of sleep (i.e., until adolescents feel sufficiently sleepy; Jenni, Achermann, & Carskadon, 2005). Sleep pressure has been operationalised by measuring the latency to sleep onset (cross-sectionally; Taylor, Jenni, Acebo, & Carskadon, 2005) or modelling changes in the amount of deep, slow wave, sleep (longitudinally; Campbell et al., 2011). Sleep latency tends to decrease alongside increases in the number of hours spent awake (Carskadon & Dement, 1982) and sleepiness is primarily dissipated by slow wave sleep (Dijk, Hayes, & Czeisler, 1993), hence the use of these measures as markers of sleep pressure. In a multiple sleep latency test protocol, pre-pubertal children (~11yrs) fell asleep significantly quicker than pubertal children (~14yrs) between the hours of 22:30 and 02:30 (Taylor et al., 2005). Additionally, there is a meaningful decrease in slow wave sleep from childhood to older adolescence (Baker, Turlington, & Colrain, 2012; Campbell et al., 2011; Campbell & Feinberg, 2009; Tarokh, Van Reen, LeBourgeois, Seifer, & Carskadon, 2011). Importantly, circadian and sleep homeostatic processes are thought to interact, with evening type individuals showing a slower accumulation of sleep pressure, compared to morning types (Taillard et al., 2003). As adolescents tend to build up sleep pressure more slowly, they are likely to feel more alert in the evening, and delay their bedtimes, until they feel sufficiently prepared for sleep.

**Behavioural Contributing Factors**

Biological contributing factors for delayed adolescent bedtimes are likely to remain consistent across cohorts (Gradisar, Gardner, et al., 2011). However, the number of adolescents reporting insufficient sleep has risen in recent decades, which suggests that other behavioural and environmental factors are important to consider (Keyes, Maslowsky, Hamilton, & Schulenberg, 2015). Adolescents have an increased number of academic, vocational, social and extra-curricular demands placed upon them, which may reinforce and/or worsen delayed bedtimes (Gau & Soong, 2003; Hansen, Janssen, Schiff, Zee, & Dubocovich, 2005; Owens & Group, 2014). In particular, adolescents typically have increased autonomy over their bedtime as they age (Gradisar, Gardner, et al., 2011). They inadvertently sacrifice their sleep duration on school nights, by squeezing part time work, homework, extra-curricular activities (e.g., sport) and socialisation in the evening- thus delaying their bedtimes (Carskadon, 2011). It is no wonder that parent-set bedtimes have been found to protect adolescents' sleep (Gangwisch et al., 2010; Short, Gradisar, Lack, Wright, Dewald, et al., 2013; Short et al., 2011), Furthermore, a positive family environment has been identified as a protective factor for adolescents' bedtimes (Bartel, Gradisar, & Williamson, 2015).

Importantly, advances in the capability and availability of technology may also be contributing to the rise in insufficient sleep. Interactive technology use (i.e., video game, phone, computer and internet use) appears to be a risk factor for later adolescent bedtimes (Bartel et al., 2015). It has been proposed that electronic media use may i) directly displace sleep (i.e., adolescents disengage from electronic devices later than they intended), ii) cause pre-sleep physiological hyperarousal, inhibiting sleepiness and sleep onset, and/or iii) delay the circadian rhythm and hence, sleep timing (Bartel & Gradisar, 2017; Cain & Gradisar, 2010). In particular, as light exposure suppresses evening melatonin secretion (Zeitzer, Dijk,

Kronauer, Brown, & Czeisler, 2000), it has been proposed that evening screenlight exposure partially mediates the relationship between technology use and later bedtimes (Bartel et al., 2015; Peixoto, da Silva, Carskadon, & Louzada, 2009). Pre- to mid-pubertal adolescents may be particularly susceptible to evening light, with melatonin suppression occurring to a greater extent within this developmental period, relative to post-pubertal adolescents (Crowley, Cain, Burns, Acebo, & Carskadon, 2015). However, the evidence to date does not support the hypothesis that bright screenlight affects young people's sleep (Bartel & Gradisar, 2017). Negative family environment, computer use and (non-screen) evening light have also been identified as risk factors for reduced total sleep time, alongside stimulant use (e.g., tobacco and caffeine use; Bartel et al., 2015).

**Environmental Contributing Factors**

There are a number of factors at the beginning of the sleep period which contribute to delayed sleep onset and subsequent sleep restriction in adolescents. However, it is also important to consider factors at the end of the sleep period (i.e., when adolescents finally wake for the day), which may play a role in truncating sleep duration. One of the most important *morning* factors influencing the adolescent sleep period is school start time. When given greater freedom over their sleep patterns (i.e., weekends, during school holidays) adolescents' go to bed later, attain more sleep and wake later (Bei et al., 2014; Olds, Maher, Blunden, & Matricciani, 2010; Warner, Murray, & Meyer, 2008). However, during the school term, school start times (i.e., 8:30am in Australia) curtail sleep duration, leading to the development of chronic sleep debt, which may compound across the school week and academic term (Dinges et al., 1997; Lo et al., 2017; Short, Gradisar, Lack, Wright, Dewald, et al., 2013; Warner et al., 2008). Consequently, adolescents tend to "catch up" on lost sleep on non-school nights (i.e., Fri, Sat) by sleeping in (Short, Gradisar, Lack, Wright, & Dohnt, 2013; Warner et al., 2008). Having a later weekend wake-up time can further delay circadian

timing (Crowley & Carskadon, 2010; Taylor, Wright, & Lack, 2008), and also prevents sufficient accumulation of sleep pressure prior to the required bedtime on Sunday night (i.e., first school night), resulting in prolonged onset to sleep and reduced total sleep time. This pattern of "social jet lag" (i.e., difficulty initiating sleep and curtailment of sleep on school nights, recovery sleep on weekends) is common in teenagers (Dahl, 2008; Short, Gradisar, Lack, Wright, & Dohnt, 2013) and such irregularity in sleep is likely driven by adolescents' delayed circadian and sleep timing (Phillips et al., 2017). In summary, an interaction between biological, behavioural and environmental factors unique to adolescence, place teens at risk of late bedtimes and restricted sleep duration, particularly during the school term.

**Delayed Sleep-Wake Phase Disorder in Adolescence**

Given the plethora of unique risk factors compromising adolescent sleep, it is unsurprising that sleep problems are common in this population (Gradisar, Gardner, et al., 2011). One of the most common sleep disorders in adolescence is Delayed Sleep-Wake Phase Disorder (DSWPD; American Academy of Sleep Medicine [AASM], 2014), which is thought to affect between 1-16% of adolescents (AASM, 2014; Danielsson, Markström, Broman, von Knorring, & Jansson-Fröjmark, 2016; Lovato, Gradisar, Short, Dohnt, & Micic, 2013; Saxvig, Pallesen, Wilhelmsen-Langeland, Molde, & Bjorvatn, 2012; Sivertsen et al., 2013). DSWPD often presents as an inability to initiate sleep at a desired/ required clock time and difficulty waking to fulfil morning requirements, such as school, university or work (AASM, 2014; Sack, Auckley, Carskadon, et al., 2007). Many adolescents with DSWPD who wake to attend school or work, have curtailed sleep duration throughout the school week, causing significant daytime disruption (Saxvig et al., 2012). However, when adolescents with DSWPD are allowed to choose their sleep timing (i.e., weekends, school holidays), their sleep is generally of improved quality and duration, albeit later (AASM, 2014; Saxvig et al., 2013).

DSWPD was first conceptualised over 35 years ago (Weitzman et al., 1981) and adolescents' susceptibility to DSWPD was reported over two decades ago (Carskadon et al., 1993). Therefore, it is surprising how little research has focused upon the treatment of adolescent sleep problems, such as DSWPD (Gradisar & Richardson, 2015). There are over one billion adolescents in the world, with two thirds residing in western and eastern civilisations (UNICEF, 2016). Thus, even one percent of this population represents ~6.5 million adolescents worldwide who could meet criteria for DSWPD. Given increased industrialisation occurring in developing nations, it is likely this figure will rise. Developing and evaluating effective treatments for adolescent DSWPD should be a priority now, and for the future. The current thesis aims to address this research priority.

**Assessment of Adolescent Sleep and DSWPD**

Clinicians and researchers commonly use The International Classification of Sleep Disorders- Third Edition (ICSD-3) to diagnose DSWPD (AASM, 2014). For diagnosis, the major sleep period must be significantly delayed, relative to desired/ required sleep-wake timing (i.e., reports of difficulty initiating sleep and awakening at a required time) (Criterion A). Additionally, symptoms must be present for at least three months (Criterion B). Typically, individuals with DSWPD report improved sleep quality and duration, when allowed to sleep *ad-libitum* (Criterion C). Importantly, the sleep disturbance must not be better accounted for by another sleep, psychological or medical disorder, or medication or substance use (Criteria E, AASM, 2014).

In researching this clinical population, it is important to quantify sleep disturbance and daytime impairment. Adolescent sleep patterns are commonly measured using parent or adolescent self-report sleep surveys (i.e., Sleep Habits Survey, Wolfson et al., 2003; Morningness-Eveningness Questionnaire, Horne & Ostberg, 1976; Short, Gradisar, Lack,

Wright, & Chatburn, 2013). However, parents have shown to be inaccurate reporters of adolescent sleep (i.e., indicating earlier bedtimes and more total sleep, Short, Gradisar, Lack, Wright, & Chatburn, 2013). There has been good agreement between adolescent self-report survey and objective sleep data for school nights. However, on weekends, large discrepancies for estimates of sleep period (52min) and wake-up time (56min) occur; which calls into question the accuracy of self-report sleep survey data (Short, Gradisar, Lack, Wright, & Chatburn, 2013).

Daily sleep diaries are likely to be more sensitive to the school night vs weekend night irregularities in sleep, which are common during adolescent development and in DSWPD (Gradisar, Smits, & Bjorvatn, 2014; Moore & Meltzer, 2008). Sleep diaries provide more detailed information about sleep; including bed times, wake times, latency to sleep, total sleep time, sleep quality (i.e., amount of wake after sleep onset, sleep efficiency) and napping (Moore & Meltzer, 2008). Relevant to adolescents, bedtime is not synonymous with the intention to sleep (Exelmans & Van den Bulck, 2017). For example, teenagers may go to bed and use electronic media prior to attempting sleep (Tavernier & Willoughby, 2014). Consequently, the importance of measuring sleep intention and behaviour (i.e., lights out time, sleep onset time) has been highlighted (Exelmans & Van den Bulck, 2017). Importantly, 7-14 days of sleep diary recordings are considered essential for the diagnosis of DSWPD (Criterion D, AASM, 2014).

In terms of objective measures of sleep, polysomnography (PSG) is indicated for the assessment of a number of sleep disorders (Kushida et al., 2005). PSG typically involves the application of electrodes to measure brain activity (electroencephalography, EEG), eye movement (electrooculography, EOG) and muscle tone (electromyography EMG) and allows for mapping of sleep architecture (i.e., cycling between wake, light [stages 1-2 sleep] to deep sleep [stages 3-4] and rapid eye movement sleep [REM; dreaming sleep]; Chesson et al.,

1997). However, as adolescents and individuals with DSWPD are said to have "normal" sleep architecture when allowed to sleep *ad libitum* (Saxvig et al., 2013) and PSG is financially expensive to administer (i.e., a trained sleep technician is required to apply PSG and score sleep data), PSG is not a recommended component of assessment (Kushida et al., 2005).

As abnormal sleep *timing* is more common in adolescents and individuals with DSWPD, actigraphy is considered a more appropriate objective measure of sleep in the home environment (Acebo et al., 1999; Sadeh, Sharkey, & Carskadon, 1994). Actigraphic recording of sleep is less invasive than polysomnography and involves the wearing of a wristwatch-like device which measures rest and activity patterns across the 24-hour day via an accelerometer (Acebo et al., 1999). Software validated for use with adolescents can then be used to score objective sleep onset and offset time (i.e., sleep onset time and wake-up time; Acebo et al., 1999; Sadeh et al., 1994). Wearable devices, such as *Fitbit* and *Jawbone*, are also commonly used to measure sleep; however, these devices are inaccurate relative to PSG, and therefore inadequate for scientific enquiry (Evenson, Goto, & Furberg, 2015). Relative to PSG, actigraphy provides a more naturalistic measure of sleep patterns over multiple nights and is not susceptible to "first night effects"[1] (Acebo et al., 1999; Arora et al., 2016; Scholle et al., 2003). Additionally, actigraphy is considered essential for the diagnosis of DSWPD (Criterion D, AASM, 2014).

As circadian delays occur in adolescence and are thought to underlie DSWPD, measures of circadian timing, such as plasma or salivary dim light melatonin onset (DLMO), can also be taken (AASM, 2014). A recent comparison showed the DLMO for good-sleeping adolescents and young adults to be at 21:08±73min, relative to 00:15am±133min for those

[1] "first night effect" refers to the reduction in sleep quality on the first night of sleep recording, caused by equipment and changes in the sleeping environment (Arora, Omar, & Taheri, 2016).

with DSWPD (Saxvig et al., 2013). However, measurement of circadian timing is labour and time intensive, expensive, difficult to measure in clinical settings and not considered essential for the diagnosis of DSWPD (AASM, 2014; Gradisar et al., 2014). Consequently, the vast majority of sleep clinics around the world that treat DSWPD do not assess circadian timing (Gradisar et al., 2014).

Although sleep diaries and actigraphy are important components of the assessment of adolescent sleep and of DSWPD, these measures are not sensitive to an individual's appraisal of their sleep. Importantly, the perception of one's sleep can be more predictive of impairment than the abovementioned ways of defining sleep (Lichstein, 2017). Consequently, it is important to assess the perception of sleep disturbance alongside subjective and objective measures of sleep (Lichstein, 2017). Such measures include, but are not limited to, the Insomnia Severity Index (ISI; Morin, 1993) and the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) and can focus on particular symptoms of sleep disturbance, including perceptions of difficulty initiating sleep (i.e., the ISI, Morin, 1993), pre sleep arousal (i.e., the pre-sleep arousal scale, PSAS, Nicassio, Mendlowitz, Fussell, & Petras, 1985) and sleep-related monitoring (i.e., the sleep associated monitoring index, SAMI, Neitzert Semler & Harvey, 2004b), to name a few. Additionally, clinically significant impairment needs to be confirmed prior to DSWPD diagnosis. Consequently, it is important to measure adolescents' functioning (e.g., functional impairment, Sheehan Disability Scale, Sheehan, Harnett-Sheehan, & Raj, 1996) and common consequences of sleep disturbance, such as daytime sleepiness (e.g., Pediatric Daytime Sleepiness Scale, Drake et al., 2003) and fatigue (e.g., Flinders Fatigue Scale, Gradisar et al., 2007). In summary, a comprehensive assessment of adolescent sleep and DSWPD during adolescence should include measures of subjective sleep (i.e., sleep diary), objective sleep (i.e., actigraphy) and perceptions of sleep disturbance (i.e., ISI, PSAS, SAMI) and daytime impairment (i.e., SDS, PDSS, FFS).

**Consequences of Inadequate Sleep in Adolescence**

Adolescence is a critical period of development which may influence longer term prospects; and sleep disturbance can have a significant impact upon adolescents' emotional, behavioural, cognitive, social and physical wellbeing (Gregory & Sadeh, 2012; Owens & Group, 2014; Shochat, Cohen-Zion, & Tzischinsky, 2014). The most immediate effects of insufficient sleep for adolescents include excessive daytime sleepiness and fatigue (Beebe, 2011; Findlay, 2008; Moore & Meltzer, 2008). Similarly, adolescents with DSWPD report daytime sleepiness and fatigue as their primary daytime impairment (Danielsson, Markström, et al., 2016; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2012; Sivertsen et al., 2013; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013).

It has also been suggested that sleep loss can affect adolescents' ability to regulate their emotions (Brand, Kirov, et al., 2016; Dahl, 1999). Importantly, findings from longitudinal and clinical research studies suggest that sleep disturbance may precede the development of depression and anxiety in adolescents (Alfano, Zakem, Costa, Taylor, & Weems, 2009; Asarnow, McGlinchey, & Harvey, 2014; Fredriksen, Rhodes, Reddy, & Way, 2004; Goetz, Wolk, Coplan, Ryan, & Weissman, 2001; Gregory & O'Connor, 2002; Lovato & Gradisar, 2014; Nielsen et al., 2000; Reddy, Palmer, Jackson, Farris, & Alfano, 2017; Roane & Taylor, 2008; Roberts & Duong, 2013; Shain, Naylor, Shipley, & Alessi, 1990). More specifically, evening chronotype, later bedtimes and reduced sleep duration emerge as risk factors for emotional problems, such as anxiety, depression, bipolar disorder, internalising problems and suicidality (Asarnow et al., 2014; Chelminski, Ferraro, Petros, & Plaud, 1999; Gangwisch et al., 2010; Gaspar-Barba et al., 2009; Gau et al., 2007; Kitamura et al., 2010; Staton, 2008). Given these findings, it is unsurprising that adolescents with DSWPD report significant psychological comorbidity (i.e., depression and anxiety) and

reduced resilience (Danielsson, Markström, et al., 2016; Kripke et al., 2008; Reid et al., 2012; Saxvig et al., 2012; Sivertsen, Harvey, Pallesen, & Hysing, 2015; Thorpy, Korman, Spielman, & Glovinsky, 1988), with recent evidence suggesting circadian misalignment drives the increased risk of depression (Murray et al., 2017).

In addition to internalising problems, sleep disturbance is also a predictor of externalising behaviour, such as attention problems and aggression in adolescence (Gregory & O'Connor, 2002; O'Callaghan et al., 2010; Pasch, Laska, Lytle, & Moe, 2010; Wong, Brower, Fitzgerald, & Zucker, 2004). Additionally, sleep problems predict substance use (i.e., alcohol, tobacco, illicit drugs, Pasch, Latimer, Cance, Moe, & Lytle, 2012; Wong et al., 2004; Wong, Brower, & Zucker, 2009), and there is some evidence that adolescents are using such substances to aid sleep, or to overcome tiredness (Noland, Price, Dake, & Telljohann, 2009; Tynjälä, Kannas, & Levälahti, 1997). More specifically, short sleep duration, later bedtimes and evening chronotype have been associated with attention problems, aggression and violence towards others, truancy, substance use, unprotected sex and theft (Adan, Natale, Caci, & Prat, 2010; Caci, Bouchez, & Baylé, 2009; Gau et al., 2007; Goldstein, Hahn, Hasher, Wiprzycka, & Zelazo, 2007; Hennig, Krkovic, & Lincoln, 2017; McGlinchey & Harvey, 2015; Susman et al., 2007; Yen, King, & Tang, 2010). Unsurprisingly, symptoms of ADHD (i.e., inattention, hyperactivity) and substance use (i.e., alcohol, marijuana, nicotine, caffeine) are also commonly reported by adolescents with DSWPD (Lovato, Gradisar, et al., 2013; Reid et al., 2012; Saxvig et al., 2012; Sivertsen et al., 2015). Taken together, adolescent sleep is globally related with risk-taking behaviour (McGlinchey & Harvey, 2015).

Insufficient sleep during adolescence has also been associated with adverse academic outcomes. A meta-analysis revealed small effects between sleep quality ($r$=0.096), sleep duration ($r$=0.069), sleepiness ($r$=0.133) and school performance in children and adolescents

(Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010). More relevant to DSWPD, evening chronotype, late bedtimes and restricted sleep duration have been associated with worse academic performance (Asarnow et al., 2014; Clarisse, Le Floc'h, Kindelberger, & Feunteun, 2010; Fredriksen et al., 2004; Giannotti, Cortesi, Sebastiani, & Ottaviano, 2002; Kim et al., 2011; Merikanto, Lahti, Puusniekka, & Partonen, 2013; Noland et al., 2009; Randler & Frech, 2009; Short, Gradisar, Lack, & Wright, 2013; Wolfwon & Carskadon, 1998). Impaired cognitive performance may explain the link between adolescent sleep and poor academic functioning. Reduced sleep duration and evening chronotype have been associated with deficits in working memory, sustained attention and executive functioning in adolescents (Agostini, Carskadon, Dorrian, Coussens, & Short, 2017; Goldstein et al., 2007; Gradisar, Terrill, Johnston, & Douglas, 2008; Hahn et al., 2012; Lo, Ong, Leong, Gooley, & Chee, 2016). Importantly, there is some suggestion that adolescents' cognitive performance may become increasingly susceptible to sleep loss following repeated "bouts" of sleep deprivation across the school term (Dinges et al., 1997; Lo et al., 2017). Individuals with DSWPD commonly self-report school lateness, absenteeism, dropout and poor academic performance (Danielsson, Markström, et al., 2016; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lack, 1986; Pallesen et al., 2011; Saxvig et al., 2012; Sivertsen et al., 2013). Although one randomised controlled trial has shown improvements in cognitive performance following treatment for DSWPD (Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), this finding has not been replicated and it is not yet known whether the cognitive performance of adolescents with DSWPD is impaired relative to good sleepers. Consequently, this gap in knowledge will be addressed in **Chapter 5**.

Although social influences may contribute to poor sleep during adolescence, sleep disturbance itself also appears to significantly impact adolescents' ability to maintain relationships with parents, peers and wider social supports (Noland et al., 2009; Roberts,

Roberts, & Chen, 2002; Roberts, Roberts, & Duong, 2009). In particular, social problems are more prevalent within evening-type adolescents (Gau et al., 2007; Goldstein et al., 2007) and clinical samples of adolescents with DSWPD attribute difficulty socialising to their sleep problem (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011).

Finally, poor sleep during adolescence has also been identified as a risk factor for adverse health outcomes (Roberts et al., 2002). For example, evening chronotype and reduced sleep duration have been linked with reduced physical activity, increased food intake and increased risk of obesity (Garaulet et al., 2011; Gupta, Mueller, Chan, & Meininger, 2002; Olds, Maher, & Matricciani, 2011; Weiss et al., 2010). Whereas, adequate sleep duration has been linked with better health and health promoting behaviours (i.e., healthy diet, stress management, regular exercise, Chen, Wang, & Jeng, 2006). One study has shown that later bed times may be causally linked with increases in body mass index, through unhealthy food intake (Asarnow, McGlinchey, & Harvey, 2015). These adverse outcomes may place adolescents at risk of developing cardiovascular and metabolic disease (Gonnissen et al., 2012; Shochat et al., 2014). However, limited research has focused on the physical health consequences of DSWPD. There is conflicting evidence as to whether body mass index differs between individuals with DSWPD and controls (Kripke et al., 2008; Saxvig et al., 2012). However, as an indication of general health, adults and adolescents with DSWPD have reported greater medication use and less involvement in sports than good sleepers (Kripke et al., 2008; Lovato, Gradisar, et al., 2013). Consequently, the potential health consequences of DSWPD warrant further investigation.

In summary, late bedtimes and reduced sleep duration place adolescents at increased risk of negative emotional, behavioural, cognitive, social and physical outcomes. Although there has been less research focusing on clinical samples of adolescents, current research suggests adolescents with DSWPD are likely to experience impaired daytime functioning as a

result of their sleep problem, and are at increased risk of developing comorbid psychological and physical conditions.

**Aetiology of DSWPD**

In order to improve the sleep and daytime functioning of adolescents with DSWPD, efficacious treatments are needed. Treatment strategies are informed by theories pertaining to aetiology; however, the cause of DSWPD is still not precise (Micic, Lovato, Gradisar, Ferguson, et al., 2016). DSWPD is most commonly attributed to a mistiming (i.e., delay) of the circadian rhythm. Rhythms of sleepiness/alertness fluctuate across the 24-hr day alongside the circadian system (i.e., core body temperature, melatonin, cortisol; Carrier & Monk, 2000), and strongly influence the timing of our sleep. The propensity for sleep is maximal at the lowest point of the circadian rhythm (i.e., core body temperature minimum, *Tmin*), with a rise in core body temperature associated with a shift towards wakefulness (Dijk & Czeisler, 1994). The endogenous "sleep hormone", melatonin, also promotes and maintains sleep and is secreted by the pineal gland approximately two hours prior to habitual bedtime (Revell, Burgess, et al., 2006; Saxvig et al., 2014). Light supresses melatonin secretion via a pathway between photoreceptors in the eye, the suprachiasmatic nucleus (SCN; central clock within our brain) and the pineal gland (Moore, 2007; discussed later in the Introduction). Therefore, melatonin is virtually undetectable throughout the day and rises in dim light, during the evening (i.e., dim light melatonin onset, DLMO; Lewy, Wehr, Goodwin, Newsome, & Markey, 1980). Consequently, the most common measures of circadian timing are core body temperature and endogenous melatonin (Benloucif et al., 2005).

In support of the theory that DSWPD is driven by delays in the body clock, research has shown significant delays in the dim light melatonin onset, sleep onset and core body

temperature timing of individuals with DSWPD, compared to good sleepers (Micic et al., 2013; Micic et al., 2015; Micic, Lovato, Gradisar, Burgess, et al., 2016; Oren, Turner, & Wehr, 1995; Saxvig et al., 2013; Shibui, Uchiyama, & Okawa, 1999; Wyatt, Stepanski, & Kirkby, 2006; see Figure 1.1). However, as treatments for DSWPD aiming to advance circadian and sleep timing (discussed later in the Introduction) often fail to produce robust, long-term improvements in sleep and daytime functioning (Abu-Salah & Auger, 2013; Sack, Auckley, Auger, et al., 2007; Saxvig et al., 2014), Micic and colleagues (2016) have suggested a range of other aetiological factors should be considered.

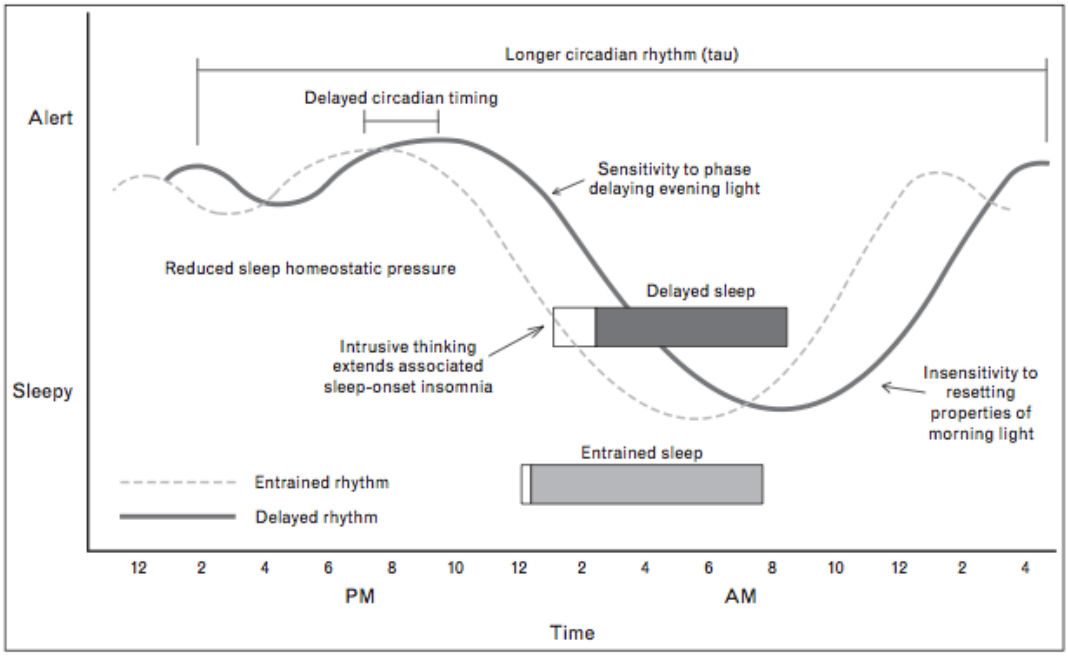


*Figure 1.1* Contributing factors to Delayed Sleep-Wake Phase Disorder. Figure taken from Gradisar and Crowley (2013).

An alternative theory postulates that individuals with DSWPD may have a longer circadian rhythm period length (*tau*; e.g., core body temperature and melatonin rhythms) than good sleepers, placing them at greater risk of a delayed sleeping pattern (Campbell & Murphy, 2007; Micic et al., 2013). The circadian rhythm of normal sleepers oscillates slightly longer than 24-hrs (Czeisler et al., 1999); however environmental time cues (i.e., morning bright light, activity patterns) facilitate circadian adjustment to the 24-hr day. In contrast,

having a significantly longer *tau* makes it arguably more difficult to adapt sleeping patterns to the 24-hr social world, resulting in delayed sleep timing. Case studies (Campbell & Murphy, 2007; Micic et al., 2013) and more recently, a well-designed 80hr lab-based study, have demonstrated that adults with DSWPD had significantly longer melatonin (24hr 34min) and core body temperature (24hr 34min) *tau*s, relative to good sleepers (24hr 22min, 24hr 13min, respectively, Micic, Lovato, Gradisar, Burgess, et al., 2016). Individuals with DSWPD have also shown larger circadian rhythm amplitudes (i.e., the difference between the peak and trough of core body temperature rhythm) and longer sleep duration, relative to controls (Ozaki, Uchiyama, Shirakawa, & Okawa, 1996; Shibui et al., 1999; Uchiyama et al., 2000). Therefore, increased circadian amplitude may also contribute to slower oscillation of the circadian system, and thus, delayed sleep timing (Carskadon & Acebo, 2002).

Another circadian abnormality in DSWPD relates to the timing of sleep, relative to circadian timing (i.e., phase angle of entrainment). DSWPD individuals have shown a tendency to sleep at a later circadian phase, in relation to core body temperature minimum and melatonin rhythm (Ozaki et al., 1996; Shibui et al., 1999; Uchiyama et al., 2000; Watanabe et al., 2003). That is, good sleepers wake approximately 3hr after the circadian trough (*Tmin*), whereas the DSWPD group woke closer to 5hr following the circadian low point (Uchiyama et al., 2000). Exposure to morning bright light facilitates circadian adjustment to the 24-hr day (i.e., through suppression of melatonin secretion), with the largest phase advances resulting from light administered directly following core body temperature minimum (Minors, Waterhouse, & Wirz-Justice, 1991). As DSWPD individuals wake at a later circadian time, it is possible that the phase resetting properties of bright light are less potent at this time, making it arguably harder for DSWPD individuals to adjust to the 24-hr day (Shibui et al., 1999; Uchiyama et al., 2000). However, as other studies have shown no difference in the phase angle of entrainment between DSWPD individuals and matched good

sleepers, further research is needed to confirm this hypothesis (Chang, Reid, Gourineni, & Zee, 2009; Wilhelmsen-Langeland et al., 2012).

In a similar vein, it has been suggested that individuals with DSWPD may have altered sensitivity to light and as such, are more prone to delaying their sleep period. Namely, it has been suggested that DSWPD individuals have reduced melatonin suppression from morning light and heightened melatonin suppression from evening light. As evidence for this hypothesis, Aoki, Ozeki, and Yamada (2001) found that evening bright light (1000 lux, 2hrs duration) suppressed melatonin excretion to a larger extent in DSWPD individuals, compared to good sleepers, which may place them at greater risk of delayed sleep onset. Cain and colleagues (2013) replicated these findings more recently and demonstrated that lower intensities of light (i.e., 200lux, 3hr duration) exert the same effect. More specifically, the DSWPD group showed significantly greater evening melatonin suppression and an enhanced alerting response, relative to controls (Cain et al., 2013). However, with only two studies investigating light sensitivity in DSWPD to date, further replication is needed, particularly focusing on DSWPD individuals' sensitivity to morning light.

In addition to circadian rhythm factors, it is also important to consider sleep homeostasis (Crowley, 2016). Gradisar and Crowley (2013) have suggested that DSWPD may be caused by reduced homeostatic sleep pressure, which delays sleep onset at night (Figure 1.1). In extrapolating findings from the adolescent literature, this theory seems plausible (Baker et al., 2012; Campbell et al., 2011; Campbell & Feinberg, 2009; Tarokh et al., 2011; Taylor et al., 2005). Initial data suggest that adults with DSWPD may accumulate sleep pressure more slowly (Uchiyama et al., 1999). However, replication is needed. As DSWPD has been associated with longer total sleep time, relative to controls, Micic and colleagues (2016) theorised that the dissipation of sleep pressure during sleep may also occur more slowly, thus extending sleep duration on free days, and exacerbating the tendency

towards delayed sleep timing. Indeed, individuals with DSWPD have shown longer total sleep time and altered patterns of deep, slow wave sleep (Saxvig et al., 2013; Watanabe et al., 2003) and less recovery sleep following sleep deprivation (Uchiyama et al., 1999). Again, given the paucity of research in the area, more studies investigating this theory are needed.

It is possible that many of the aforementioned aetiological factors could be driven by genetic expression, with individuals with DSWPD commonly reporting a family history of delayed sleep (Ancoli-Israel, Schnierow, Kelsoe, & Fink, 2001; Takahashi, Hohjoh, & Matsuura, 2000). Whereas *Per1* and *Per2* genes have been linked with morningness (Carpen, Archer, Skene, Smits, & Schantz, 2005; Carpen, von Schantz, Smits, Skene, & Archer, 2006), polymorphisms[2] of the *Per3* gene have been linked with DSWPD (Archer et al., 2003; Ebisawa et al., 2001; Pereira et al., 2005). The role of the *Per3* allele appears to be most influential in young people (18-29yrs), with the strength of the association reducing with age (Jones et al., 2007). Interestingly, *Per3* has also been linked with alterations in sleep homeostasis and the ability to cope with sleep deprivation (Viola et al., 2007). The *CLOCK 3111c* allele has also been associated with eveningness and therefore warrants further enquiry (Iwase et al., 2002; Katzenberg et al., 1998). Further research is needed to fully elucidate how genetic expression may contribute beyond "eveningness" and thus, towards DSWPD.

Although much of the current explanation focuses upon physiological causes, it is also important to acknowledge cognitive and behavioural factors. Behavioural patterns appear altered in DSWPD, with individuals showing significantly more activity late at night and significant less activity in the morning (i.e., 8-11am; Joo et al., 2017). Unsurprisingly then, individuals with DSWPD have shown increased exposed to light between 22:00pm-02:00am and reduced light exposure between 08:00-09:00am and 10:00am-12:00pm (Auger, Burgess, Dierkhising, Sharma, & Slocumb, 2011). The unpleasant experience of awakening

[2] "polymorphism" refers to multiple variations (forms) of a single gene.

early may lead to conditioned avoidant behaviours (Lack & Wright, 2007). Following forced early morning awakening, it is likely that DSWPD individuals experience sleepiness, irritability, low mood and reduced motivation (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). In contrast, individuals with DSWPD report feeling most alert in the evening and thus, may inadvertently delay bedtimes (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Therefore, Lack and Wright (2007) have suggested that morning resistance, and evening preference, may serve to maintain a delayed sleeping pattern. As such, protective avoidant cognitions (e.g., "*To function well today, I need to "catch up" on lost sleep*") and behaviours (e.g., sleeping in) may serve to maintain DSWPD.

Additionally, researchers have identified several personality factors, which may be common to individuals with DSWPD. Two research groups have independently identified a link between DSWPD and lower conscientiousness and extraversion and higher neuroticism in adults (Micic, Lovato, Gradisar, & Lack, 2016; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Longitudinal research has shown that sleep-onset problems during adolescence predict later neuroticism (Danielsson, Jansson-Fröjmark, Linton, Jutengren, & Stattin, 2010).There is some consensus that lower conscientiousness may predispose individuals to DSWPD, through a lack of self-discipline and thus, later and more variable sleep times (Digdon & Howell, 2008; Micic, Lovato, Gradisar, & Lack, 2016; Sivertsen et al., 2015; Tonetti, Fabbri, & Natale, 2009; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). However, as research in this area is correlational, causation cannot be determined and further enquiry has been called for (Micic, Lovato, Gradisar, Ferguson, et al., 2016).

Psychosocial factors may also contribute to the development of DSWPD, with 57% of one clinical sample identifying factors such as interpersonal conflict, job dissatisfaction and occupational change as precipitating their sleep disorder (Takahashi et al., 2000).

Psychosocial stress has shown to predict longer sleep latency (Lee, Crain, McHale, Almeida, & Buxton, 2017) and shorter total sleep time in adolescents (Bauducco, Flink, Jansson-Fröjmark, & Linton, 2016), therefore it is possible that delayed sleep timing becomes conditioned in times of high stress and is difficult to rectify. Conversely, adolescents who possess greater mental toughness report better sleep quality (Brand, Kalak, et al., 2016). Adolescents and those with DSWPD also face a number of psychosocial challenges as a result of their sleep problem, for example, conflict with family/ friends, difficulty balancing responsibilities, self-blame, inability to change and associated feelings of failure (Lemola, Schwarz, & Siffert, 2012; Wilhelmsen-Langeland et al., 2012). Therefore, it is important to develop an appreciation of the psychosocial context of sleep disturbance in the assessment of DSWPD.

Finally, it is possible that sleep disordered cognitive processes also contribute to the development and maintenance of DSWPD. Although individuals with DSWPD are said to have normal sleep quality when allowed to sleep *ad libitum* (AASM, 2014), many individuals are not afforded this luxury, instead needing to wake early to meet educational or work commitments. Individuals with DSWPD may attempt to initiate sleep at an earlier time in order to gain sufficient sleep prior to awakening. Self-selected earlier weekday bedtimes are likely to align with the "wake maintenance zone", the major peak in the circadian rhythm which promotes wakefulness, and consequently, individuals are likely to experience prolonged sleep onset (Strogatz, Kronauer, & Czeisler, 1987). Indeed, DSWPD was initially thought of as a circadian rhythm sleep disorder, with associated sleep onset insomnia (Weitzman et al., 1981) and more recent research has highlighted the overlap in phenomenology between the two disorders (Espie, Marchetti, MacMahon, Minto, & Biello, 2006; Sivertsen et al., 2013).

Within the insomnia literature, wakefulness in bed is theorised to give rise to repetitive negative thinking (e.g., rumination, worry; Mansell, Harvey, Watkins, & Shafran, 2008, 2009), which can be worsened by maladaptive sleep-related beliefs (Harvey, 2002). Negatively-toned cognitive activity (i.e., repetitive negative thinking) promotes physiological hyperarousal and distress, which may lead to changes in information processing, such as an attentional bias towards sleep-related stimuli (Espie, 2002; Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006; Harvey, 2002; Lundh & Broman, 2000). These cognitive "insomnia" processes can lead the individual to overestimate the extent of their sleep problem and the impact of sleep on daytime functioning, consequently worsening repetitive negative thinking (Harvey, 2002). Additionally, individuals may engage in safety behaviours in an effort to promote sleep (e.g., alcohol use, sleeping aids, etc.), or to avoid the negative daytime consequences of inadequate sleep (e.g., excessive consumption of caffeine; Harvey, 2002). However, safety behaviours may inadvertently maintain sleep disturbance (e.g., alcohol reduces sleep quality; poor sleep quality leads to poor daytime functioning; poor daytime functioning leads to excessive caffeine use; etc.; Ree & Harvey, 2004). There is a robust evidence base to support the aetiological role of cognitive processes in insomnia (Harvey, 2000; Harvey & Greenall, 2003; Harvey & Payne, 2002; Harvey & Tang, 2012; Jones, Macphee, Broomfield, Jones, & Espie, 2005; Morin, Rodrigue, & Ivers, 2003; Neitzert Semler & Harvey, 2004a, 2004b, 2005, 2006, 2007; Ree & Harvey, 2004; Tang & Harvey, 2004, 2006; Wicklow & Espie, 2000; Woodley & Smith, 2006). Given the distinct overlap in phenomenology between DSWPD and insomnia (i.e., sleep-onset difficulties), it is possible that cognitive insomnia processes are also involved in the aetiology of DSWPD. Evidence for the existence of cognitive "insomnia" processes in DSWPD will be reviewed in **Chapter 3,** and the possible role of these processes in adolescents with DSWPD is investigated in **Chapter 6**.

In summary, the aetiology of DSWPD is still relatively unknown. However, there are a large number of possible theories which pertain to i) circadian abnormalities, ii) altered sleep homeostatic drive, iii) genetic expression, and more recently, iv) psychosocial factors. As treatment techniques are informed by our understanding of DSWPD, it should be a priority of future research to better understand why DSWPD develops and how the disorder is maintained.

## Treatment for DSWPD in Adolescence

Without treatment, adolescent sleep problems are unlikely to spontaneously resolve (Roberts, Roberts, & Chan, 2008). Treatments addressing DSWPD typically aim to advance circadian timing, in an effort to advance sleep timing, improve total sleep time and ameliorate daytime dysfunction. The human circadian rhythm is sensitive to light, melatonin and behaviour and consequently these chronobiological agents are most commonly used in the treatment for DSWPD.

### Bright Light Therapy

Light entrains the human circadian rhythm to the 24-hr "social" world. Photic information is detected by melanopsin receptors within ganglion cells in the eye, and transmitted to the SCN (i.e., Suprachiasmatic Nucleus) via the retinohypothalamic tract (Freedman et al., 1999; Morin, 1994; Morin & Allen, 2006). Melatonin, a sleep inducing hormone, is produced and released by the pineal gland in dim light, following input from the SCN (Arendt, 2003). Melatonin onset is associated with heat loss and decreases in core body temperature, which promote sleep onset (Cajochen, Kräuchi, & Wirz-Justice, 2003; Krauchi, Cajochen, Werth, & Wirz-Justice, 2000). Indeed, closing the eyes initiates rapid changes in thermoregulation and preparedness for sleep (Gradisar, Lack, & Wright, 2005). Upon awakening, exposure of the eye to light results in suppression of melatonin and increases in

core body temperature and alertness (Cajochen, Chellappa, & Schmidt, 2014; Strassman, Qualls, Lisansky, & Peake, 1991; Van Someren, 2006). Consequently, appropriately timed exposure to light around the end of the sleep period can be used to phase advance timing of the circadian rhythm, and thus, sleep timing.

For circadian phase shifts to occur, the timing, duration, illumination and wavelength of light are all important factors to consider. The propensity for light to alter the human circadian rhythm is phase dependent, with circadian phase delays (i.e. of core body temperature and melatonin) observed when light is administered prior to core body temperature rhythm minimum and phase advances observed when light is administered after the circadian trough, near the end of the sleep period (Duffy & Wright, 2005; Khalsa, Jewett, Cajochen, & Czeisler, 2003; Minors et al., 1991). Similarly, in adolescents, phase advances occur following the midpoint of sleep (Crowley & Eastman, 2017; see Figure 1.2). Importantly, the magnitude of phase shift is greatest when light is administered proximal to *Tmin* (Minors et al., 1991). In terms of the duration of bright light exposure, there is limited understanding of a potential phase response curve (Wyatt, 2004). However, there is some consensus that adolescents need a minimum of 30mins per day to phase advance their circadian timing (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Gradisar et al., 2014; Saxvig et al., 2014).

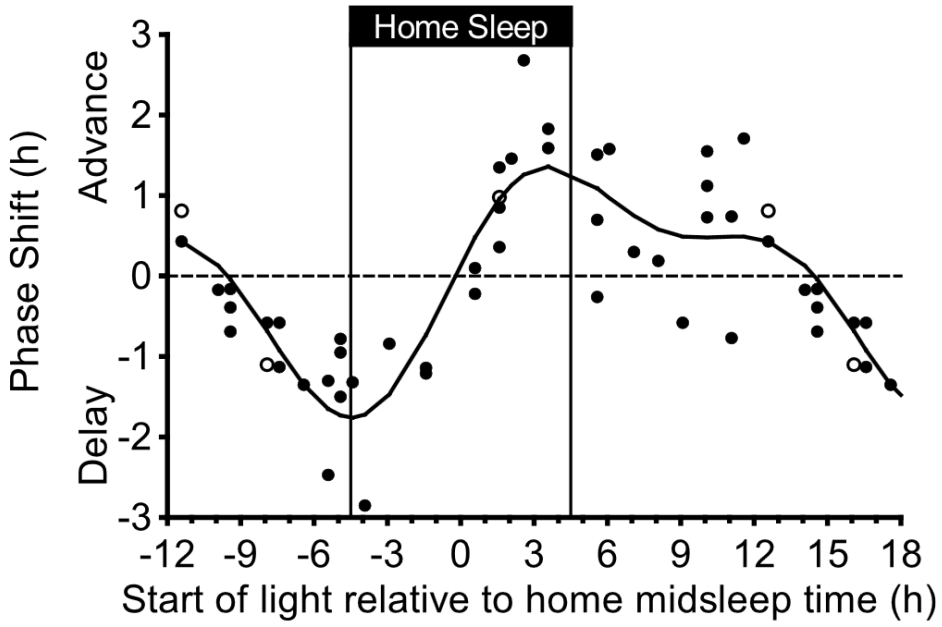


*Figure 1.2* Adolescent phase response curve to ~5,000lux bright broad spectrum light. Figure taken from Crowley and Eastman (2017).

In terms of illuminance, empirical research shows that humans are comparatively responsive to light. Historically, it was believed that approximately 1500 lux of white light was required to suppress melatonin secretion (Lewy et al., 1980). However, more recently, low intensity (117lux) blue light (460nm) has shown to suppress melatonin secretion in good sleepers (Cajochen et al., 2005). Broadly speaking, light intensity is assumed to have an exponential relationship with phase shifts, in that greater intensity light is associated with better treatment outcomes. However, this relationship may be greatly influenced by the wavelength of light and also, the method of bright light administration (e.g., light box, light lamp or portable bright light devices; Cajochen et al., 2005).

In relation to the impact of the wavelength of light, the human circadian rhythm appears to be most sensitive to shorter wavelengths of light. When considered alone (e.g., without altered sleep schedules), shorter wavelengths of light (e.g., blue-green, 470-525nm) have produced greater phase shifts in circadian rhythm timing, when compared to longer wavelengths (e.g., red, 660nm; Wright, Lack, & Kennaway, 2004) and broad-spectrum

(white) light (Warman, Dijk, Warman, Arendt, & Skene, 2003). However, these findings have not been replicated clinically (i.e., clinical samples undergoing treatment in their home environment) and as such, priority should be placed upon replicating experimental findings in less tightly controlled settings (Auger et al., 2015).

Bright light therapy (BLT) for adolescent DSWPD typically involves allowing the teenager to sleep-in on the first day of treatment (Gradisar et al., 2014). Adolescents are then advised to obtain 30-60 minutes of bright light immediately after awakening (Bjorvatn & Pallesen, 2009; Gradisar et al., 2014). Empirical literature provides an evidence base for the administration of short wavelength (e.g., 500nm) bright (10,000 lux) light therapy, for improvements in both sleep timing and daytime functioning (Bjorvatn & Pallesen, 2009). Rise times and bright light therapy times are then advanced by 30mins each day, until the desired wake time is reached (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Gradisar et al., 2014; see Figure 1.3). As evening light, close to bedtime, can phase delay the circadian rhythm (Crowley & Eastman, 2017), evening light restriction is a common component of bright light therapy (Rosenthal, Joseph-Vanderpool, Levendosky, & Johnston, 1990). More specifically, adolescents are instructed to minimise light exposure in the 2-3hrs prior to bedtime (Appleman, Figueiro, & Rea, 2013; see Figure 1.3). This thesis will evaluate bright light therapy for the treatment of adolescent DSWPD (primarily **Chapter 4**) and current evidence for the use of BLT, and other treatments, will be reviewed later in this introduction.

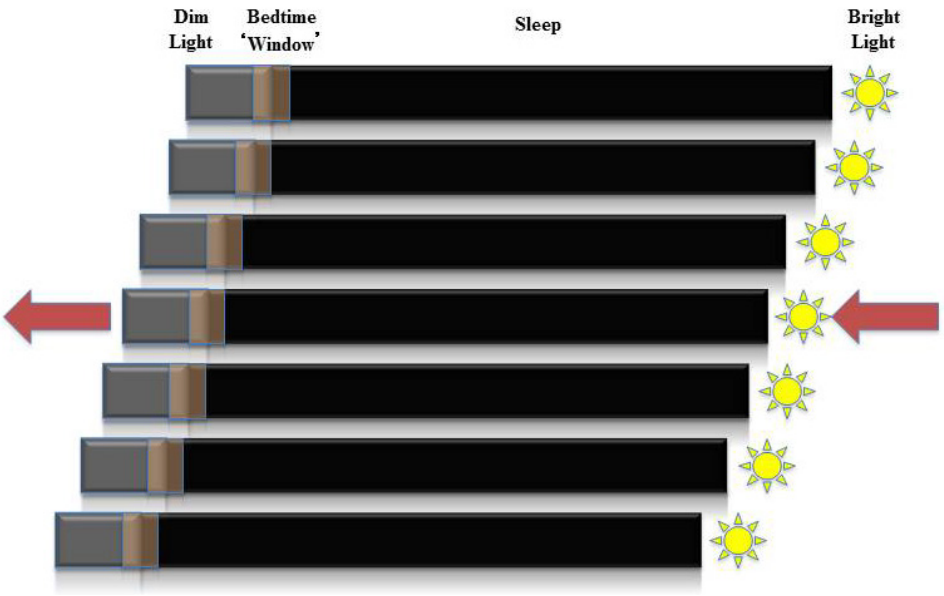


*Figure 1.3* Schematic illustrating the protocol for bright light therapy.

**Evening Exogenous Melatonin**

As previously mentioned, endogenous melatonin is secreted by the pineal gland in dim light, approximately 2hrs prior to habitual bedtime (Revell, Burgess, et al., 2006; Saxvig et al., 2014). Exogenous melatonin can be administered to facilitate phase shifts of the human circadian rhythm and consequently sleep timing (Lewy et al., 1998), with the magnitude of phase advances peaking 5hrs before dim light melatonin onset (Burgess, Revell, & Eastman, 2008). Consequently, it has been recommended that 1mg+ exogenous melatonin be administered close to this time and advanced by 30-60min each subsequent night (Gradisar et al., 2014). Similar to bright light therapy, melatonin treatment typically ceases when ideal sleep timing is reached (Gradisar et al., 2014).

**Chronotherapy**

Historically, chronotherapy has also been used to treat DSWPD. Chronotherapy involves administering evening bright light and morning light restriction to progressively *delay* circadian timing (i.e. by 2-3hrs a day), until preferred sleep timing is attained (Czeisler

et al., 1981; Gradisar et al., 2014). However, this treatment is likely to cause significant disruption, particularly for adolescents who attend school. More specifically, this treatment can take 2-3 weeks to implement, and in most cases requires total absence from school (as their sleep period may overlap school hours), unlike bright light therapy, which may only involve partial morning absence from school (due to only sleeping-in). Additionally, chronotherapy has triggered more irregular sleep-wake patterns post-treatment (Oren & Wehr, 1992) and there is a paucity of research supporting its use (Ito et al., 1993; Wyatt, 2004). In particular, there is a lack of evidence as to whether rhythms of core body temperature a melatonin delay at the same rate as the delay in sleep timing (i.e., 2-3hrs daily). Consequently, it is unclear whether circadian timing is normally aligned, in relation to sleep timing, at the cessation of treatment. As such, chronotherapy will not be discussed further as a viable treatment for adolescent DSWPD.

## Current Evidence for the Treatment of Adolescent DSWPD

Recent clinical guidelines suggest there is weak evidence to support the use of either combined bright light therapy and behavioural therapy (i.e., sleep scheduling), or strategically timed melatonin in adolescents (Auger et al., 2015).

### Bright Light Therapy

To date, three randomised controlled trials (RCT) have investigated the efficacy of bright light therapy for the treatment of adolescent DSWPD (Danielsson, Jansson-Fröjmark, Broman, & Markström, 2016; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). In the first study, the treatment consisted of bright light therapy for advancing circadian timing and cognitive behavioural therapy (CBT) for the associated sleep-onset insomnia (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Adolescents who received

bright light therapy and CBT showed moderate-to-large improvements in sleep timing, sleep latency, total sleep time and daytime functioning (i.e., sleepiness and fatigue), relative to a waitlist control (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). In terms of treatment outcome, 87% of adolescents who received treatment no longer fulfilled the criteria for a DSWPD diagnosis, compared to 18% in the waitlist condition. Importantly, these benefits were maintained 6-months post-treatment. The second RCT extended upon these initial findings, by comparing the efficacy of bright broad spectrum white light (10,000 lux, 50cm distance) with dim red light (400 lux, 50cm distance, placebo; Saxvig et al., 2013; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Unexpectedly, both the bright white and dim red light treatments resulted in advancement of sleep and circadian timing and improvement in daytime functioning over two weeks of treatment, with no between group differences. However, only those who continued treatment maintained benefits at the 3-month follow-up, with those ceasing treatment returning to baseline levels (Saxvig et al., 2013). These results could be due to the exclusion of relapse prevention instructions from treatment. In the most recent study, Danielsson and colleagues (2015) aimed to elucidate whether (white) light therapy (10,000lux, 50cm distance) supplemented with CBT showed improvements in sleep above and beyond chronobiological treatment (i.e., bright light therapy) alone. Light therapy significantly improved sleep timing and sleep duration, and although the addition of CBT did not further improve sleep outcomes, it did result in better outcomes for depression and anxiety (Danielsson et al., 2015). This is particularly important given the high rate of psychological comorbidity within DSWPD (Reid et al., 2012; Sivertsen et al., 2015). Although, the use of bright light therapy in the treatment of adolescent DSWPD appears promising, to date there have only been three controlled trials. Thus, this thesis will provide further data relating to the efficacy of bright light therapy, as a standalone treatment for adolescent DSWPD.

### Melatonin

Fewer well designed studies have evaluated the efficacy of exogenous melatonin for the treatment of DSWPD in adolescents. One retrospective study showed improvements in sleep timing and sleep duration following approximately six months of treatment (Szeinberg, Borodkin, & Dagan, 2006). More recently, a randomised controlled trial compared the efficacy of 3mg evening melatonin to a placebo (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Again, both groups improved their sleep and circadian timing across two weeks of treatment, however sleep reverted back to baseline once treatment was ceased (Saxvig et al., 2014). To date, there is limited evidence supporting the efficacy of melatonin for adolescent DSWPD. In addition, there is some concern about the safety of melatonin when used in paediatric samples (Kennaway, 2015), with adverse outcomes including excessive sedation, increased risk of type II diabetes and influence on reproductive development (Burgess & Emens, 2016; Kennaway, 2015). Consequently, compared to melatonin, light therapy may be a preferable first line treatment for adolescent DSWPD.

### Administration of Bright Light Therapy

At present, all randomised controlled trials of light therapy in adolescent DSWPD have delivered artificial light via light boxes or lamps (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). These methods of bright light administration (i.e., light boxes and lamps) may be seen as burdensome, in terms of the duration of time participants are required to be seated in front of these devices to receive light therapy. Such a burden may result in reduced compliance and poorer treatment outcomes (Burgess & Emens, 2016). Therefore, it is anticipated that newly developed portable light

emitting diode (LED) devices (e.g., Re-Timer light glasses; Re-Time Pty Ltd, Adelaide, Australia) may improve compliance and treatment outcomes (Burgess & Emens, 2016), as they allow adolescents the freedom to move around whilst undergoing light therapy. Furthermore, teenagers are virtually guaranteed to receive consistent bright light regardless of whether they turn their head or avert their gaze when wearing LED devices, unlike lamps (see Figure 1.4). To date, there is insufficient evidence supporting the use of portable light devices in the treatment of DSWPD (Auger et al., 2015). However, the Re-Timer glasses have shown to phase delay circadian timing (Lovato & Lack, 2015), and improvements in sleep latency and total sleep time were observed in adolescents with delayed sleep timing (DST), in a school-based sleep intervention (Bonnar et al., 2015).

Another wearable device is the Luminette® (Lucimed S.A., Belgium), which is worn like a visor, and emits blue-enhanced white light (468nm peak). This portable device has been tested in a small number (*N*=10) of adolescents with DSWPD (Langevin, Laurent, & Sauvé, 2014). However, the trial involved post-awakening bright light administration alone, without many of the traditional components of bright light therapy (i.e., psychoeducation about circadian rhythms, sleep scheduling, gradually advanced bed- and wake-up times). Adolescents receiving active treatment showed improvements in sleep onset time (1hr 59min), sleep duration (2hr 3min) and daytime sleepiness after three weeks of treatment (Langevin et al., 2014). Adolescents within the control condition (*N*=5), received orange light (wavelength not reported) and had little change in sleep onset time (9min advance), total sleep time (10min increase) or daytime sleepiness. Given the small number of participants, results should be interpreted cautiously (Ioannidis, 2005). It is possible that portable light devices may be effective for the treatment of adolescent DSWPD; however without larger-scale studies validating their use, it is unlikely that such devices will be recommended clinically, thus highlighting an important gap in knowledge that needs addressing.

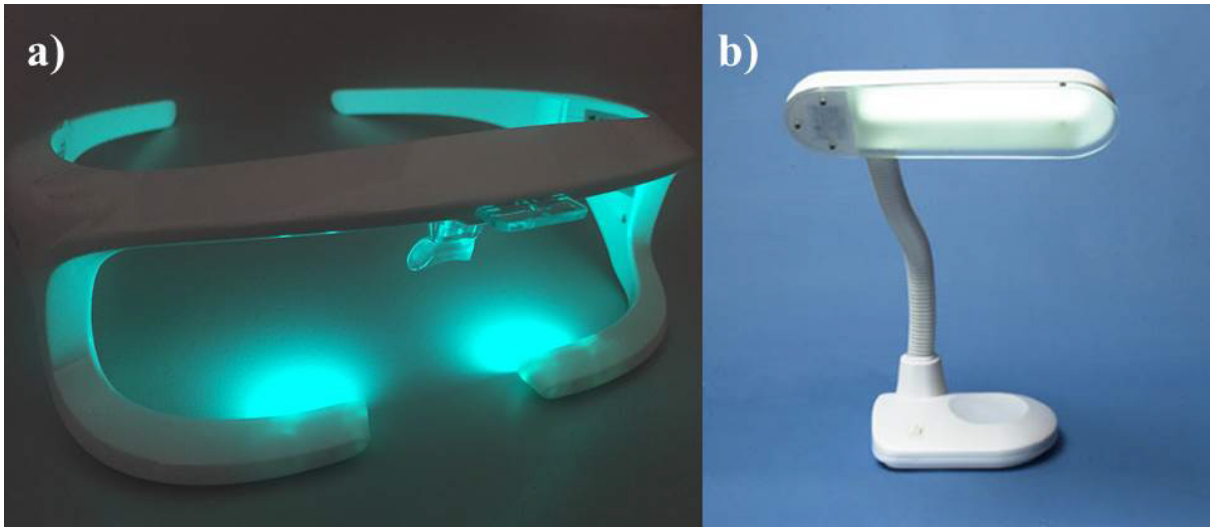


*Figure 1.4* Methods of bright light administration include, but are not limited to, a) portable LED devices (e.g., Re-Timer glasses) and b) light lamps.

Another gap in our knowledge pertains to the influence of wavelength of light therapy, when administered in clinical samples of adolescents with DSWPD. Each of the three large RCTs evaluating light therapy for adolescent DSWPD have used bright broad spectrum white, or ambient light as the active treatment (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014). As previously mentioned, only one RCT has directly compared the efficacy of two different 'wavelengths' of light; with both bright broad spectrum white and dim red light treatment groups improving similarly (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Interestingly, the small trial evaluating the Luminette® device found little change in sleep and sleepiness as a result of three weeks of post-awakening orange light. Whereas clinically meaningful changes in sleep onset time, total sleep time and sleepiness were seen following administration of blue-enriched white light therapy (Langevin et al., 2014). However, the small sample size limits the generalisability of these finding, therefore, more controlled trials are needed to better understand the optimal wavelength and method of bright light administration for the treatment of DSWPD.

There is a convincing evidence base which suggests the human circadian rhythm is optimally sensitive to short wavelength (blue-to-green) light (Warman et al., 2003; Wright et al., 2004). Importantly for adolescents with DSWPD, light has the propensity to positively influence aspects of "functioning" independently of the circadian system and a significant body of evidence suggests that short wavelengths of light are also most effective at increasing alertness and improving cognitive performance (Cajochen et al., 2005; Lehrl et al., 2007; Lockley et al., 2006; Revell, Arendt, Fogg, & Skene, 2006). However, short wavelength light therapy has not been evaluated for the treatment of adolescent DSWPD to date. As the Re-Timer light device emits short wavelength green (500nm) light, recent developments in technology also allow researchers to address this important gap in the literature. That is, elucidating whether short (e.g., green) wavelength light is superior to long (e.g., red, placebo) wavelength light, in the treatment of adolescent DSWPD. The comparison of (short) green vs (long) red wavelength light is a novel aim in the present thesis, and this will be combined with another novel treatment technique.

**Physical Activity as an Adjunct to Light Therapy**

It has long been acknowledged that behaviour, such as social interaction and physical activity, promotes circadian entrainment to the 24-hr social world (Mrosovsky, Reebs, Honrado & Salmon, 1989). However, behaviour can also elicit changes in circadian timing (i.e., phase advances and delays) (Mrosovsky, Reebs, Honrado & Salmon, 1989). Much of our current understanding of the effect of physical activity on circadian timing has stemmed from experimental research aiming to facilitate circadian adjustment to nightshift work. There is robust evidence that nocturnal exercise results in small-to-moderate delays in endogenous melatonin and core body temperature rhythms (Baehr, Eastman, Revelle, Olson, Wolfe & Zee, 2003, Barger, Wright, Hughes & Czeisler, 2004, Buxton et al., 1997; Eastman, Hoese, Youngstedt, & Liu, 1995; Van Reeth et al., 1994).

In terms of circadian phase advance, there is some evidence that physical activity at other times of the day may induce phase advances (Buxton, Lee, L'Hermite-Balériaux, Turek, & Van Cauter, 2003; Miyazaki, Hashimoto, Masubuchi, Honma, & Honma, 2001). A recent study mapping the human phase response curve to exercise reported robust circadian phase advances following 60mins of moderate intensity exercise completed in the morning (6-9am) or early afternoon (1-5pm) in good-sleeping individuals (Youngstedt, Kripke, & Elliott, 2017).

Individuals with DSWPD have shown increased activity and light exposure in the evening and decreased activity and light exposure in the morning, relative to controls (Joo et al., 2017). Therefore, evening activity and morning inactivity may inadvertently be maintaining or worsening DSWPD for adolescents. As behaviour exerts control over circadian timing (Aschoff et al., 1971; Klerman et al., 1998), it is possible that physical activity could be used as a chronobiological agent in the treatment of adolescent DSWPD when timed correctly. Evidence for this hypothesis will be reviewed next in **Chapter 2**.

Importantly, there is some evidence that physical activity can facilitate circadian phase advances, when gradually advanced each day, similarly to the protocol for bright light therapy (Miyazaki et al., 2001). The delivery of bright light via portable LED devices has inherent benefits, in terms of freedom of movement. Therefore, it is possible that physical activity could be completed at the same time as portable bright light therapy, to produce larger phase shifts. However, research has not yet combined bright light therapy and physical activity to phase advance the human circadian rhythm, highlighting another gap in our knowledge.

A number of studies also suggest that physical activity has a beneficial impact upon adolescent's sleep and functioning, including externalising problems ($d$=0.32), internalising

problems (*d*=0.32) and academic achievement (*d*=0.37; Brand, Beck, Gerber, Hatzinger, & Holsboer-Trachsler, 2010; Brand, Gerber, et al., 2010a, 2010b; Kredlow, Capozzoli, Hearon, Calkins, & Otto, 2015; Spruit, Assink, van Vugt, van der Put, & Stams, 2016). Relevant to the current thesis, brief (30min) morning exercise administered over three weeks has resulted in improvement in adolescents' sleep (i.e., reduced sleep onset latency, increase in slow wave sleep), mood, concentration and daytime sleepiness (Kalak et al., 2012). Although a majority of studies investigating exercise as an intervention for sleep and daytime functioning have used non-clinical samples, these findings potentially could extrapolate to the treatment of adolescent DSWPD. Therefore, one research question addressed in this thesis is "*Can exercise supplement bright light therapy and assist the sleep timing and daytime functioning of adolescents with Delayed Sleep-Wake Phase Disorder*?"

**Aims and Outline of Thesis**

Although, the use of bright light therapy in the treatment of adolescent DSWPD appears promising, there is need for further research to investigate its potential as a standalone treatment. To date, portable bright light administration and short wavelength light therapy have never been evaluated in adolescents with DSWPD. The development of short wavelength portable bright light therapy glasses opens up the potential for adjunct behavioural interventions, which may enhance the treatment of adolescent DSWPD. Physical activity may facilitate advances in the circadian rhythm and improve adolescents' daytime functioning. Evidence for the role that physical activity may play in the treatment of adolescent DSWPD is therefore reviewed in **Chapter 2**.

The effect of bright light and exercise on the human circadian rhythm have been investigated rather independently and morning physical activity has not been previously investigated as a method to enhance bright light therapy for the treatment of adolescent

DSWPD. This thesis aims to evaluate whether green (~500nm) portable bright light therapy is an effective treatment for adolescent Delayed Sleep-Wake Phase Disorder, compared to a control condition (~640nm red light). This thesis also aims to evaluate whether physical activity supplements portable bright light therapy to improve outcomes for adolescents with DSWPD. Primary outcome variables include sleep onset and offset time, and secondary variables include sleep onset latency, total sleep time and daytime functioning (i.e., morning alertness, daytime sleepiness, fatigue, functional impairment). Results from a randomised controlled trial investigating bright light therapy and morning activity for the treatment of adolescent DSWPD will be presented in **Chapter 4**.

Current evidence suggests that the cognitive performance of adolescents with DSWPD is impaired relative to their good-sleeping counterparts. However, a comparison between adolescents with DSWPD and good sleepers has not been undertaken to date. Additionally, there is some evidence that the cognitive performance of adolescents with DSWPD improves following chronobiological treatment (Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013); however this standalone finding needs replication. Consequently, a between group comparison of cognitive performance in adolescents with DSWPD and good sleepers is presented in **Chapter 5**. Changes in cognitive performance from the RCT will also be presented in **Chapter 5**.

The aetiology of DSWPD is still somewhat unclear and researchers have called for further research elucidating factors that may contribute to the development and maintenance of DSWPD. As an under-researched area, psychological contributing factors warrant further investigation. Therefore, evidence for the role that cognitive "insomnia" processes may play in the development and maintenance of DSWPD is reviewed in **Chapter 3**. As cognitive "insomnia" processes have not been systematically investigated in DSWPD to date (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), a comparison of these processes

between adolescents with DSWPD and good sleepers is outlined in **Chapter 6**. It is also unclear whether standard chronobiological treatments for DSWPD improve cognitive "insomnia" processes. Consequently, insomnia severity outcomes from the randomised controlled trial of bright light therapy and morning activity are also presented in **Chapter 6**. The thesis concludes with a final Discussion section (**Chapter 7**).

# Chapter 2

## Can Exercise Regulate the Circadian System of Adolescents? Novel Implications for the Treatment of Delayed Sleep-Wake Phase Disorder.

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**Author Contributions**

CR led review conceptualisation, literature search, results interpretation and manuscript preparation. MG contributed to review conceptualisation, literature search, results interpretation and manuscript preparation. MS provided data presented in Figure 2.2 and assisted with manuscript preparation. CL assisted with results interpretation and manuscript preparation.

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## Abstract

Adolescents are vulnerable to inadequate sleep due to a unique constellation of risk factors. In particular, the puberty-related phase delay in the timing of the circadian system postpones the onset of sleep. Resultantly, disordered sleep is common among teenagers and young adults, with the most common sleep problem being Delayed Sleep-Wake Phase Disorder (DSWPD). Although current treatments for DSWPD show promise, novel ways to improve our youth's sleep are needed. The purpose of this review is to critically evaluate the evidence for the role of exercise as a method to shift and/or regulate circadian timing, and thus improve sleep, in adolescents and young adults. A growing body of evidence suggests that nocturnal exercise can delay circadian timing. However, exercise administered at different times of the 24-hr day may result in phase advances, particularly when the timing of exercise is gradually advanced in small daily increments. The implications of these results for young people's sleep health are discussed and suggestions are provided for ways that exercise could be used clinically, to improve the treatment of Delayed Sleep-Wake Phase Disorder.

## Introduction

Adolescence and young adulthood is a developmental period of rapid physical and psychological change. Many of these changes place youth at heightened risk for inadequate sleep (Carskadon, 2011). Indeed, there is a ubiquitous, worldwide trend for later bedtimes and less sleep across adolescence, despite sleep need remaining unchanged (Carskadon, Harvey, & Dement, 1981; Crowley, Acebo, & Carskadon, 2007; Gradisar, Gardner, et al., 2011). Later bedtimes, coupled with the requirement to wake early for educational or vocational commitments, mean the opportunity for sleep might be insufficient, possibly leading to daytime sleepiness, fatigue, and impaired attention, concentration, memory and mood (American Academy of Sleep Medicine [AASM], 2014).

The trend for later bedtimes during adolescence and young adulthood is thought to be at least partly attributable to puberty-related changes in the adolescent circadian system, with circadian timing becoming gradually later from childhood until early adulthood (Carskadon et al., 1997; Carskadon et al., 1993; Roenneberg et al., 2004). Circadian rhythms exist in many plants and animals, and result in fluctuations of alertness and sleepiness over the solar day (Dijk, Duffy, & Czeisler, 1992). During adolescence, the circadian system both delays and lengthens (slightly over 24 hours), causing peaks of alertness (e.g., 7:00-8:00pm) and sleepiness (e.g., 6:00am) across the 24 hour day, and consequently sleep timing, to become later (Carskadon et al., 1999; Crowley et al., 2007; Dijk, Duffy, Riel, Shanahan, & Czeisler, 1999; Micic et al., 2013), as shown in Figure 2.1. Additionally, educational (e.g., study), occupational (e.g., part-time work), extra-curricular (e.g., after school sport/music, etc.), and recreational (e.g., technology use, socialising, etc.) obligations, increase the number of tasks that adolescents need to complete in the evening (Carskadon, 2011). Adolescents undergo a series of "firsts" (e.g., first job, first relationship etc.) and thus may also be more prone to pre-

sleep worry which may further delay sleep onset (Hiller, Lovato, Gradisar, Oliver, & Slater, 2014).

A more pronounced delay to circadian timing can lead to a circadian rhythm disorder called Delayed Sleep-Wake Phase Disorder (DSWPD; AASM, 2014). DSWPD occurs when the biological rhythms governing sleep-wake timing (e.g., endogenous melatonin secretion[3], core body temperature rhythm) are timed significantly later than the 24-hr social and physical environment of the individual (AASM, 2014). This delay results in difficulty falling asleep early enough to ensure sufficient sleep and difficulty waking up at the socially desired time. Consequently, the major sleep period is frequently delayed and truncated, as shown in Figure 2.1.

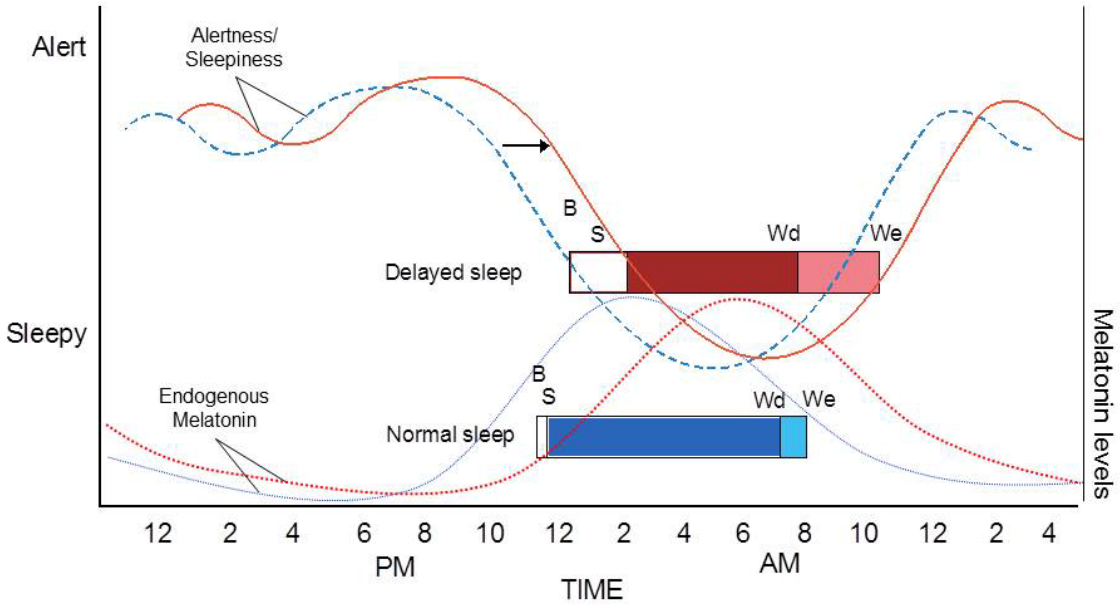


*Figure 2.1* The delayed circadian rhythm of alertness, endogenous melatonin and sleep (red) compared to normal rhythms and sleep (blue) (Gradisar et al., 2014). Note: Wd= end of weekday sleep; We= end of weekend sleep; B= bedtimes; S=sleep onset. White rectangle indicates sleep latency, which is prolonged in the delayed sleeper.

Delays in circadian and sleep timing are especially problematic for individuals who are required to wake early in the morning, such as adolescents needing to attend school

[3] For some patients DLMO (Dim Light Melatonin Onset) may not reliably predict sleep or circadian timing (Keijzer, Smits, Duffy, & Curfs, 2014).

(AASM, 2014; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2012). Individuals with DSWPD exhibit markedly reduced daytime functioning (e.g., excessive daytime sleepiness, fatigue, low mood) and impaired cognitive performance (e.g., concentration, memory and attention), particularly during the morning. Morning deficits occur due to the delayed circadian nadir and chronic sleep restriction across week days (AASM, 2014; Lovato, Gradisar, et al., 2013; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Unsurprisingly, DSWPD is most common during adolescence, affecting 1-16% of teenagers (AASM, 2014; Lovato, Gradisar, et al., 2013). It is associated with school lateness, absenteeism and dropout (Dewald et al., 2010; Giannotti et al., 2002; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Meijer, Habekothé, & Van Den, 2000; Saxvig et al., 2012). The lower threshold of prevalence estimates likely do not capture adolescents who have dropped out of school, thus the prevalence is likely to be somewhat higher (Lovato, Gradisar, et al., 2013). For adolescents both with and without DSWPD, the combination of a delayed circadian rhythm and accumulated sleep debt results in sleeping-in on weekends (Crowley et al., 2007; Crowley & Carskadon, 2010; Gradisar, Gardner, et al., 2011) or late afternoon naps (Gradisar, Wright, Robinson, Paine, & Gamble, 2008). Both of these compensatory behaviours can delay circadian rhythms even further (Crowley et al., 2007).

Although DSWPD is most common in adolescents and young adults (relative to middle and older adulthood), it can persist into adulthood if not treated. Current recommendations to shift circadian timing of adolescents earlier include controlled light exposure and the administration of exogenous melatonin. Light is the predominant circadian timekeeper helping to stabilise human's sleep timing (Boivin, Duffy, Kronauer, & Czeisler, 1996). Light suppresses the secretion of endogenous melatonin (Lewy et al., 1980; Zeitzer et al., 2000). Melatonin concentrations increase in the evening in dim light conditions, to

encourage sleep onset (Pandi-Perumal et al., 2007). As such, the administration and gradual advancement of morning bright light (following the nadir in core body temperature rhythm) can advance both circadian rhythms and sleep timing (Bjorvatn & Pallesen, 2009; Wyatt, 2004). However, ambient bright light is not always available. For example, there is reduced number of daylight hours in some latitudes (Friborg, Rosenvinge, Wynn, & Gradisar, 2014). Additionally, adolescents may be resistant to using an external device for morning bright light therapy (e.g., light lamps) and may also be reluctant to reduce use of bright light emitting devices in the evening (e.g., smart phone and tablet), particularly if they have sleep-onset difficulties (Tavernier & Willoughby, 2014). A recent longitudinal study found that sleep problems predicted television and online social media use, but not the reverse, suggesting that young adults may use technology as a way to cope with their sleep problem (Tavernier & Willoughby, 2014). Exogenous melatonin, and the gradual advancement of bedtimes (Dewald-Kaufmann, Oort, & Meijer, 2013), can also be administered in the evening to shift circadian rhythms earlier. However, currently, there is a lack of research investigating the long-term safety of melatonin in paediatric samples (Kennaway, 2015). Therefore, given the challenges associated with advancing circadian rhythms in adolescents and young adults with DSWPD, together with the plethora of negative sequelae resulting from a delayed circadian phase, researchers and clinicians alike seek novel ways to improve treatment modalities, which may in turn improve long term outcomes for both clinical and non-clinical groups.

One alternate chronotherapy may be scheduled exercise, with appropriately timed exercise potentially advancing circadian rhythms earlier. This occurs because the activity-rest schedule exerts influence over circadian timing independently of light (Aschoff et al., 1971; Klerman et al., 1998; Skene, Lockley, James, & Arendt, 1999). As such, young people may benefit from alterations to the timing of their activity-rest schedules in order to advance or

better entrain their circadian rhythms. Regular physical exercise is inexpensive, safe and has concurrent benefits to sleep latencies, sleep quality and mood. In particular, morning exercise has also shown to improve the sleep, mood and daytime functioning (i.e., attention/ concentration) of adolescents (Kalak et al., 2012), whilst curtailing late night activities may also curtail phase delay. This is a particularly salient issue given that a significant proportion of teenagers report completing active tasks (e.g., part-time work, exercise, tasks of daily living, socialising) as their primary activity within the last hour before bed (see Figure 2.2; Short, 2011). However, further research is required to determine whether a causal relationship exists between evening activities and circadian/ sleep timing.

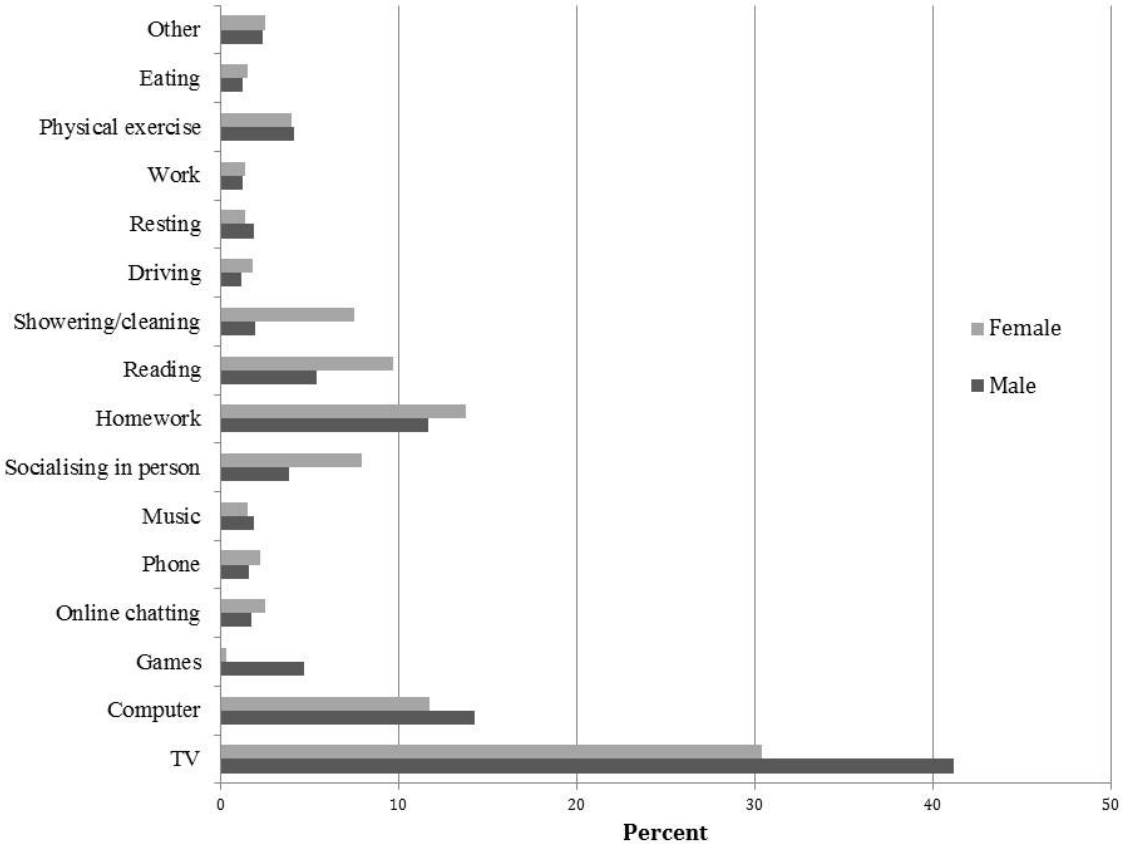


*Figure 2.2* Proportion of males and females engaging in each activity in the hour before bedtime. Note: some electronic media activities (e.g., computer, phone) could be used for other tasks (e.g., homework).

The primary aim of this review is to examine extant scientific literature to determine the effect that the timing of exercise may have on adolescent circadian rhythms. Specifically, the review will summarise and critically evaluate the literature to answer the following questions; i) "Can exercise alter circadian timing?", and, ii) "Can the treatment of Delayed Sleep-Wake Phase Disorder in adolescents and young adults benefit from appropriately timed exercise?"

## Can Exercise Alter Circadian Timing?

### Can Exercise Delay Circadian Timing?

Seven well controlled experimental studies have conceptualised exercise as a method to phase delay the human circadian rhythm (Baehr et al., 2003; Baehr, Fogg, & Eastman, 1999; Barger, Wright, Hughes, & Czeisler, 2004; Buxton et al., 1997; Eastman et al., 1995; Van Reeth et al., 1994; Youngstedt, Kripke, & Elliott, 2002). Early studies investigated whether a single bout (or pulse) of exercise was sufficient to produce phase shifts in the human circadian rhythm. In investigating the effect of a single 3hr pulse of moderate intensity exercise (arm and cycling exerciser alternating at 40% and 60% of peak $VO_2$ [maximum volume of oxygen]) at various times across the night (3hrs before core body temperature minimum [*Tmin*], *Tmin*, or 2hrs after *Tmin*) using a between-subjects design, Van Reeth and colleagues (1994) found that nocturnal exercise and moderate light (300 lux) resulted in a significant phase delay of endogenous plasma melatonin levels (55min±38), relative to a baseline night (9min±32, *Cohen's d*=1.31)[4]. Buxton and colleagues (1997) replicated these findings in dim light conditions, whilst extending upon our understanding of the impact of nocturnal exercise intensity and duration on circadian timing, using a within-subjects design. A 3hr bout of moderate intensity exercise (arm and leg exerciser alternating

[4] Data reported is for all conditions. Between group findings will be discussed in the "A Phase Response Curve to Exercise" section.

at 40% and 60% of peak $VO_2$) (63min±8, *d*=4.42) and a 1hr bout of high intensity exercise (stairclimber with 10min warm up and cool down at 25% of peak $VO_2$ and 40min at 75% of peak $VO_2$) (55min±15, *d*=2.51) both resulted in phase delays of dim light plasma melatonin onset , relative to a baseline night (23min±10) under controlled light condition (70-80 lux; Buxton et al., 1997). Interestingly, a single 3hr bout of moderate intensity exercise (cycling exerciser alternating at 40% and 60% of peak $VO_2$) appears to cause similar delays in plasma melatonin rhythms for both younger (27.1y±4.3, 45min±27) and older adults (64.5y±6.8, 30min±46.8, *d*=0.39; Baehr et al., 2003). However, the timing of the core body temperature rhythm minimum (*Tmin*), which is another phase marker of circadian timing, did not significantly change for either age group (Baehr et al., 2003). Although these studies provide evidence that exercise can delay melatonin rhythms, the measurement of the change in circadian timing only occurred at one time point (e.g., the night after nocturnal exercise). As such, they do not provide insight into whether these effects are durable (i.e. over days or weeks), whether longer term exercise intervention is required for sustainable circadian phase delays, or whether melatonin secretion is a reliable marker of psychophysiological processes.

To address these limitations, Barger and colleagues (2004) investigated the propensity for daily exercise to phase delay the human endogenous melatonin rhythm, whilst controlling for light exposure (<5 lux). The authors were able to replicate and extend upon previous findings, by demonstrating that the dim light melatonin onset of young adult males completing three 45min bouts of moderate-to-high intensity evening exercise (cycling exerciser at 65-75% of maximal heart rate), per night for seven nights, adapted more quickly to a 9hr delay in their sleep period (3.17h±0.49), compared to their inactive counterparts (1.67h±0.45, *d*=3.19). The three bouts of exercise were completed one hour apart, beginning 8.75hrs after waking, during participant's biological night, following the 9hr delay of their sleep period, to simulate adjustment to nightshift work.

Whereas most research within this field has used melatonin to measure circadian timing, Eastman and colleagues (1995) examined the effect of nocturnal exercise on rectal core body temperature rhythms. Using a similar protocol to Barger and colleagues (2004), participants experienced a 9hr delay in their sleep period to simulate nightshift. However, participants only completed the moderate intensity nocturnal exercise (riding cycle exerciser at 50-60% of maximum heart rate for 15mins each hour e.g., eight 15min bouts) during the first three of eight consecutive simulated nightshifts (e.g., during the participant's biological night), in bright light conditions <500 lux. After controlling for baseline morning-eveningness, participants who completed exercise had significantly larger phase delays in average core body temperature minimum in the last four days of the night shift (6.6h±2.5), compared to a control (sedentary) condition (4.2h±3.4, $d$=0.80). In addition to a larger phase delay, participants within the exercise condition were able to gain more sleep and reported superior vigour and mood, as well as less fatigue (Eastman et al., 1995). These findings suggest that exercise, as a chronotherapy, may have multiple additional benefits to sleep, mood and daytime functioning. However, specific improvements to sleep were not reported, therefore it is unclear whether delayed circadian timing resulted in later wake-up times (i.e. rather than earlier sleep onset times) and thus, more sleep.

While previous studies examined the effect of exercise in isolation, Baehr and colleagues (1999) evaluated whether nocturnal exercise had an additive effect by enhancing the phase shifting effects of evening bright light, the latter of which already had robust empirical support (Eastman & Martin, 1999). Participants were subjected to a 9hr delay in their sleep wake timing and were exposed to either very bright (5,000 lux) or bright (500 lux) evening light and either intermittent moderate intensity evening exercise (six, hourly, 15min bouts on cycle exerciser at 50-60% maximum heart rate) or inactivity, for the first three of eight simulated nightshifts (e.g., during the participant's biological night). Unsurprisingly,

participants who received very bright evening light had larger phase delays in their rectal core body temperature rhythm (7.8h±2.0), compared to those in the bright light conditions (5.3h±3.0, *d*=0.98). There was a non-significant trend (and small effect) for larger phase delays for participants completing exercise in bright evening light (5.7h±3.2), compared to their inactive counterparts (4.8h±2.9, *d*=0.29). The authors suggested that perhaps the intensity and duration of nocturnal exercise needed to be increased to enhance the therapeutic effect on circadian timing (Baehr et al., 1999).

In addressing the limitation of exercise intensity, Youngstedt, Kripke and Elliot (2002) hypothesised that a single 3hr pulse of bright light (3,000 lux) combined with 3hr of high intensity exercise (cycling at 70% of heart rate reserve) would result in longer phase delays of circadian timing compared to a 3hr pulse of bright light alone. Using a within-subjects design, Youngstedt and colleagues (2002) found that the combined bright light and exercise condition resulted in a significant delay in urinary melatonin acrophase (peak) the night after the intervention, compared to the baseline night (68min±10, *d*=0.57), whereas the phase delay caused by the bright light alone was not statistically significant compared to the baseline night (20min±19, *d*=0.20). Interestingly, the opposite pattern of results was evident for phase shifts in rectal core body temperature nadir. The bright light alone resulted in a significant delay in temperature nadir the night after the intervention compared to the baseline night (70min±38, *d*=0.56). While the phase delay for the bright light and exercise condition was of a similar magnitude of change (62min±38, *d*=0.49), this did not reach statistical significance. The two conditions did not display statistically or clinically significant differences in change to the core body temperature nadir. It is also important to note that core body temperature data were lost for the four out of 18 participants who showed the largest melatonin delay in the bright light and exercise condition (Youngstedt et al., 2002). Regardless, markers of melatonin phase have been found to be more stable and correlate

more highly with sleep timing, compared to estimates of core body temperature minimum (Benloucif et al., 2005).

To date, only three studies have investigated the effect of nocturnal exercise on core body temperature rhythms; two of which combined exercise and bright light exposure (Baehr et al., 2003; Eastman et al., 1995; Youngstedt et al., 2002). Overall, the studies reviewed provide support for the hypothesis that nocturnal exercise has small-to-moderate effects on delaying core body temperature rhythms. It may be the case that exercise has a greater influence on melatonin rhythms, than core body temperature rhythms (Baehr et al., 2003; Youngstedt et al., 2002). However, due to a lack of studies measuring both phase markers, more studies are needed to provide confidence in these findings.

**Can Exercise Advance Circadian Timing?**

Only two well controlled experimental studies have explored whether morning exercise phase *advances* the human circadian rhythm (Miyazaki et al., 2001; Yamanaka, Honma, et al., 2010). Miyazaki and colleagues (2001) used a protocol to gradually advance the sleep episode of good-sleeping individuals, using a protocol similar to that used in the treatment of Delayed Sleep-Wake Phase Disorder (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lack & Wright, 2007), but with exercise as the chronobiologic agent rather than bright light. Participants complied with a forced sleep/wake episode length of 23hrs and 40mins under dim light (10 lux) conditions within a sleep laboratory, so that their sleep episode was advanced by 20mins daily over 12 days (e.g., four hours in total). Participants who completed two hours of physical exercise (15min intervals of alternating rowing and cycling, at a heart rate of 140 beats/min, with 15min rest intervals in between) in the morning (three hours after awakening), and again in the afternoon (seven hours after awakening), were significantly phase advanced in their plasma melatonin peak (1.60h±0.42), compared to the control group who did not exercise (see Figure 2.3). In fact, the control

group who did not exercise, were phase delayed in their plasma melatonin peak (-.80h±0.71). This indicates that the melatonin rhythm of participants in the exercise condition adapted to match their advanced sleep schedule better than those in the control group. A similar pattern of results was apparent for rectal core body temperature, however descriptive and inferential statistics were not reported. In terms of limitations, measures of sleep (e.g., sleep onset latency) were not reported and it is unclear whether the dose, timing, duration, intensity or modality (e.g., rowing/ cycling) of the exercise was responsible for phase shifts, as participants completed two separate bouts of daytime exercise.

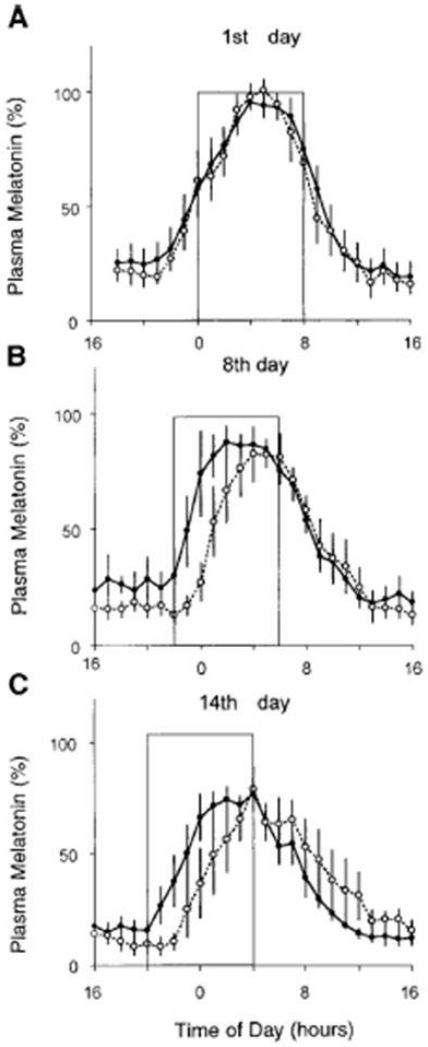


*Figure 2.3* Plasma melatonin rhythms under forced schedule with (closed circles) and without physical exercise (open circles) (Miyazaki et al., 2001). Rectangle indicates rest period. Panel A= 1st day, Panel B= 8th day, Panel C= 14th day.

More recently, Yamanaka and colleagues (2010) used a similar design to investigate whether exercise can hasten adjustment to an immediate 8hr advance in sleep-wake timing (e.g., similar to shift work). Participants in the exercise condition completed two hours of high intensity physical exercise (15min cycling at 65-75% of maximal heart rate, followed by 15min rest) in the subjective morning and afternoon for four days. Following four days of the advanced sleep pattern and daytime exercise, participants were allowed to choose their own sleep timing for a period of six days (free-run phase). On the first night following the 4-day schedule, participants in the exercise condition had a significantly advanced sleep onset time (ZT[5]11.6±3.3h) compared to baseline (ZT16.2±0.1h, *d*=1.97) and the control group (ZT14.9±3.4h, *d*=0.98). However, both groups displayed a significant delay in their plasma melatonin rhythm, signalling internal de-synchronisation. Throughout the free-run phase of the experiment, all participants adjusted their sleep-wake pattern to match their circadian (melatonin) timing, by either phase advancing or phase delaying their sleep timing (e.g., depending on the phase angle between sleep and circadian timing). A similar pattern of results was observed when implementing an 8hr advance in sleep-wake timing, without exercise during the subjective day (Hashimoto, Nakamura, Honma, & Honma, 2004). Therefore, it appears as if exercise, or at least changes in rest-activity patterns, can influence sleep timing. However, to facilitate sustainable phase advances of both sleep patterns and circadian markers, it is hypothesised that exercise should be advanced in small daily increments (e.g., 20-30mins), similar to what is performed during bright light therapy for adolescents with DSWPD (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). As exercise may augment the phase shifting effects of light (Baehr et al., 1999; Youngstedt et al., 2002), it is plausible that a combination of bright light and exercise that gradually

[5] ZT= Zeitgeber Time. Zeitgeber Time is a standardised unit of measurement whereby ZT20 equates to the melatonin peak. This standardised unit of measurement was used, as the clock time of melatonin peak varied between each participant.

advances each successive day, may result in advances in circadian phase timing. However, this is yet to be tested.

**A Phase Response Curve to Exercise**

Researchers have begun to develop a phase response curve (PRC) to exercise (see Figure 2.4; Buxton et al., 2003; Miyazaki et al., 2001); however, there are surprisingly few studies to provide conclusive support for this PRC. Much of our early understanding was informed by studies investigating the effect of nocturnal exercise. For example, Barger and colleagues (2004) found that the timing of nocturnal exercise was important, in that a phase delay was more pronounced for participants completing evening exercise closer to their dim light melatonin onset (e.g. ~20:30 for adolescents aged 13-16 years, Crowley et al., 2006). Similarly, Van Reeth and colleagues (1994) showed a trend towards larger phase delays when exercise was completed 3hrs before *Tmin* (as early as 2230h) and smaller phase delays 2hrs after *Tmin* (as late as 1030h) and as such, proposed that phase advances in response to exercise may be possible later in the subjective day (~1200h). In contrast, Eastman and colleagues (1995) found that individuals responded similarly to nocturnal exercise, regardless of baseline circadian preference (e.g., morningness/eveningness) and associated core body temperature timing. However, preliminary evidence indicates that the circadian response to exercise may vary systematically, depending upon the relative timing of administration.

Two studies measured the impact of exercise at different times of the day on phase timing (Buxton et al., 2003; Miyazaki et al., 2001). Miyazaki and colleagues (2001) measured changes in the timing of melatonin rhythms in response to a single 2hr bout of moderate intensity exercise (15min periods of alternating rowing and cycling, at heart rate of 140 beats/min, with 15min rest periods in between) in the morning (~0900h), afternoon (~1500) and night-time (~0000h). There was no significant phase shift of plasma melatonin rhythms

following morning exercise (e.g., melatonin onset change:-0.05±0.22, peak: -0.02±0.12, offset: -0.15±0.39). However, afternoon exercise elicited a significant phase delay in the peak plasma melatonin (-0.35h±0.13, *d*= 0.44), but not plasma melatonin onset or offset, and exercise at night resulted in significant phase delays in the onset (-0.45h±0.15, *d*= 0.67), peak (-0.45h±0.12, *d*= 0.77) and offset (-0.62h±0.21, *d*= 0.91) of plasma melatonin. These findings provide further support for the role that nocturnal exercise plays in delaying circadian timing, yet no support for morning exercise.

More recently, Buxton and colleagues (2003) measured phase shifts in dim light plasma melatonin onset in response to a single 1hr pulse of high intensity exercise (stairclimber exercise with 10min warm up and cool down at 25% of $VO_2$ max and 40min at 75% of $VO_2$ max) performed in the morning (~0930h), afternoon (~1300h), evening (~1830h) and night-time (~0030h). Interestingly, participants experienced a significant decrease in melatonin levels *during* morning exercise (-21.8pg/ml ± 15.3), compared to smaller decreases at the same circadian time in the no-exercise group (-5.2pg/ml ± 7.4). The implications of this finding will be discussed later.

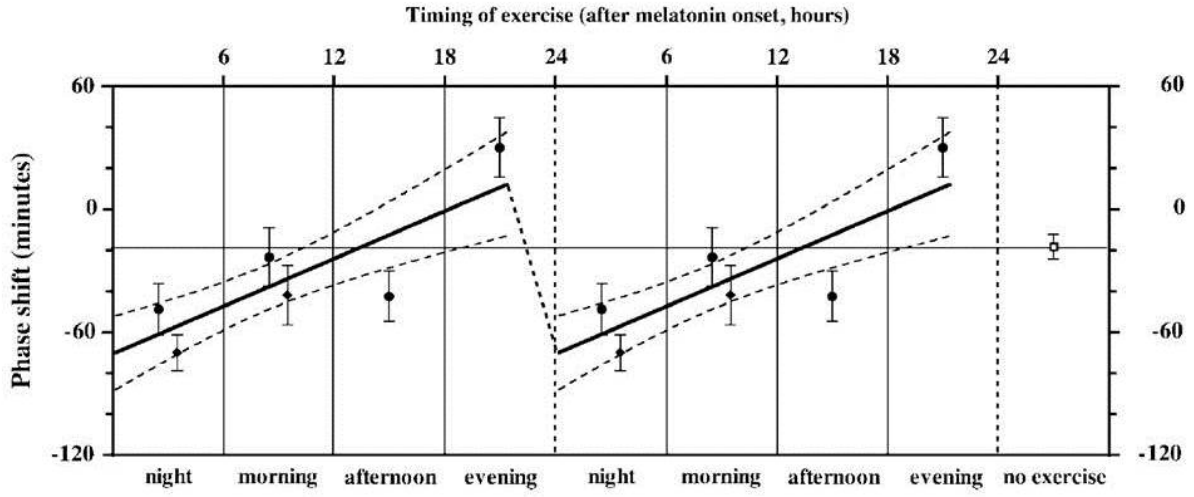


*Figure 2.4* Possible Phase Response Curve (PRC) to exercise, of varying intensities, at different times across the 24 hour period. Positive numbers indicate phase advance and negative numbers indicate phase delay(Buxton et al., 2003).

In an attempt to map a potential PRC to exercise, Buxton and colleagues (2003) combined data from a series of studies (Buxton et al., 1997) and found that early evening exercise (~18:30h) resulted in a significant phase advance (30min ± 15) in melatonin onset compared to the no-exercise group (-25min ±14, *d*= 3.79) one day post-exercise. However, this effect was attenuated and no longer significant two days post-exercise. Regardless, when looking at the combined results, it was hypothesised that exercise has the propensity to phase advance circadian timing ~three hours prior to melatonin onset and tendency to phase delay circadian timing during the nocturnal sleep period (Buxton et al., 2003; Lavie, 1986). Given the findings that exercise completed until 1500h can result in a phase delays in peak melatonin (Miyazaki et al., 2001) and that exercise completed until 1930h can result in phase advances of melatonin onset (Buxton et al., 2003), it is possible that there is a short time frame whereby exercise elicits phase advances in circadian timing in the late afternoon/ early evening. However, these findings contradict the earlier prediction made by Van Reeth and colleagues (1994) that exercise may induce phase advances to the circadian rhythm when completed earlier in the day (~1200), and are consistent with the phase advancements found by Miyazaki and colleagues (2001). Therefore, future research is needed to resolve these controversies.

**Potential Mechanisms of Action**

While extant experimental literature supports the phase-altering properties of timed exercise, little is understood about the mechanisms of action. There is some consensus in the literature that nocturnal exercise completed on several consecutive days does have a cumulative influence on delaying the timing of melatonin rhythms (Barger et al., 2004; Eastman et al., 1995). Importantly, Monteleone and colleagues (1990) found that 20mins of nocturnal exercise of moderate to high intensity (cycling for 10mins at both 50% and 80% of maximal working capacity), *acutely* inhibits night-time increases in plasma melatonin levels.

The findings of Buxton and colleagues (2003) suggest that perhaps the same is true in the morning (e.g., exercise acutely suppresses melatonin production). If true, this may have significant implications for individuals with DSWPD, as morning exercise could be used to suppress melatonin and improve functioning (e.g., morning sleepiness) and thus, treatment compliance (e.g., early improvements to daytime functioning may enhance subsequent engagement with therapy). However, the effect of exercise *acutely* inhibiting plasma melatonin levels has not been consistently replicated, likely due to methodological inconsistencies (for a more comprehensive discussion see Atkinson, Edwards, Reilly, & Waterhouse, 2007). As such, future research should continue to investigate the *acute* impact of exercise on markers of circadian timing (e.g., endogenous plasma melatonin and core body temperature) under controlled conditions.

In terms of the mechanisms for the *chronic* impact of exercise on melatonin rhythms (e.g., in the days subsequent to exercise), it has been hypothesised that exercise suppresses and either delays or advances the excretion of melatonin, depending on the relative timing of the exercise (e.g., nocturnal exercise or gradual advance of morning exercise, respectively; Van Reeth et al., 1994). However, exercise may also lead to altered melatonin degradation and thus altered plasma melatonin concentrations (Monteleone et al., 1990). Alternatively, exercise may simply reduce melatonin production at the time of the exercise stimulus. There has been little speculation as to how exercise may have a chronic impact on core body temperature rhythms. At this stage, too little is known about the potential mechanisms for the phase delay and advance properties of exercise on human melatonin rhythms and core body temperature rhythms, thus, this is an area deserving much needed attention in the future.

Miyazaki and colleagues (2001) have proposed alternate mechanisms by which exercise may phase shift the human circadian rhythm. One proposal is that daytime exercise increases the amplitude of daytime circadian and homeostatic rhythms (e.g., core body

temperature and arousal, sleep propensity); causing individuals to more rapidly accumulate sleep pressure and subsequently, initiate sleep earlier (Buman & King, 2010; Miyazaki et al., 2001; Yamanaka, Hashimoto, et al., 2010). In line with this hypothesis, it is also possible that nocturnal exercise may delay sleep propensity, due to increases in core body temperature and arousal (Buxton et al., 2003). In addition to increasing the amplitude of circadian rhythms, emerging evidence supports the role of exercise in shortening circadian *tau* (τ; i.e. the time taken to complete one cycle of the circadian rhythm), which is particularly relevant to this review given the recent proposal that DSWPD could be caused by having a longer τ (Micic et al., 2013). Using a forced desynchrony protocol, Beersma and Hiddinga (1998) investigated the role that physical activity may play in determining τ. Participants completed either sedentary (e.g., studying, watching videos, reading), low intensity (e.g., cycling for 30mins every 2hrs, so that heart rate increased by no more than 10 beats/min) or moderate intensity (e.g., cycling for 30mins every 2hrs, so that heart rate was between 140-150 beats/min) activities during a 13.5 hr wake period. Although findings did not reach statistical significance (likely due to the small sample size, $N$= 11), there was a small-to-medium trend for shortened tau length in the low (τ= 24.17h, $d$= 0.32) and moderate intensity conditions (τ=23.98h, $d$= 0.44) compared to the inactive condition (τ= 24.30h). However, the clinical significance of this effect would make it arguably easier for individuals with DSWPD to adjust and maintain their sleep timing to an advanced sleep schedule.

## Future Research Recommendations

Further research is needed to develop a sound understanding of the impact that exercise may have on sleep and circadian phase timing, and whether these effects are *timing-, duration-, dose-*, *intensity-* or *modality- dependent*. In particular, further studies are needed to confirm that exercise ~3hrs before dim light melatonin onset (e.g., 1700-1930) and/or after midday result in phase advances of the human circadian rhythm (e.g., melatonin rhythm).

Additionally, it is important to continue investigating whether exercise can induce more meaningful shifts in circadian timing, when used in combination with light.

The empirical literature is sparse and differences in study methodology (e.g., type, intensity, dose, duration, modality of exercise) have made it difficult to assimilate current research findings to plot a PRC to exercise. As such, future research should aim to map a comprehensive PRC to exercise, using a consistent methodological approach. More specifically, it would be worthwhile to investigate the effect of exercise of a practical dose, (e.g., once daily), duration (e.g., one hour) and intensity (e.g., moderate or vigorous) across the 24-hr day, in both sleep laboratory and community settings to improve generalisability. These findings are likely to have the most clinical and community significance for the treatment of circadian rhythm sleep disorders like DSWPD (National Sleep Foundation, 2014; Raising Children Network, 2013; Sleep Health Foundation, 2011).

It would also be important for future research to continue to investigate the impact of regular exercise (e.g., daily for 1-2 weeks), to explore whether alterations to activity-rest schedules can influence circadian timing in a cumulative and sustainable way and to what extent such physiological alterations might have an impact on behavioural and cognitive-emotional levels. Importantly for the focus of this review, it should also be a priority to replicate the findings of Miyazaki and colleagues (2001). That is, investigating whether small advances (e.g., 20-30mins) in the timing of exercise, advances circadian and sleep timing. Finally, it is important to note that much of the current research in this field has been conducted in populations of young adults (e.g., 20-30yrs; Baehr et al., 2003; Baehr et al., 1999; Barger et al., 2004; Buxton et al., 1997; Buxton et al., 2003; Miyazaki et al., 2001; Van Reeth et al., 1994; Yamanaka, Hashimoto, et al., 2010). However, the implications of these findings are perhaps most pertinent to adolescent populations. As such, future research should

aim to map a phase response curve and investigate the phase advance properties of exercise in adolescent populations.

## Clinical and Practical Implications

In terms of clinical implications, the current review provides promising evidence for nocturnal exercise causing delays to the human circadian rhythm (e.g., melatonin rhythm; Baehr et al., 2003; Baehr et al., 1999; Barger et al., 2004; Buxton et al., 1997; Eastman et al., 1995; Van Reeth et al., 1994; Youngstedt et al., 2002).These findings lend support for current recommendations that schools and other regulatory bodies should limit late-evening activities, such as exercise, close to adolescent bedtimes (e.g., after dim light melatonin onset; Carskadon, 2011; Raising Children Network, 2013; Sleep Health Foundation, 2011), to give young people at risk of DSWPD the best opportunity for sufficient sleep, at least from a circadian standpoint. At assessment, practitioners should be asking their clients about the types of activities they perform prior to bedtime and shortly after awakening. If necessary, practitioners should encourage adolescents and young adults reporting sleep-onset difficulties, particularly those with DSWPD, to limit moderate to vigorous physical activity, close to bedtime. Instead, these adolescents and young adults should seek bright light and exercise in the morning (Carskadon, 2011; Kalak et al., 2012). Given the finding that many teenagers report completing active tasks in the hour before bed (Short, 2011), this recommendation alone could have a significant impact upon the quality and timing of sleep in our youth, and perhaps flow on effects for their daytime functioning and performance.

Currently, the field lacks consensus about when exercise should be completed for the purposes of phase advancing the circadian rhythm (Buxton et al., 2003; Miyazaki et al., 2001; Van Reeth et al., 1994). However, preliminary evidence suggests that appropriately timed exercise may be a feasible treatment option to advance circadian phase in adolescents with

DSWPD. For individuals with DSWPD, this may involve completing one hour of moderate intensity exercise in the subjective "morning" (e.g., soon after awakening) and advancing wake-up time and exercise timing 20-30mins earlier each day, until desired sleep timing is achieved. This treatment protocol allows for the combination of exercise and morning bright light therapy (e.g., possibly performed outside in natural light (weather permitting), or via portable light devices, e.g., LED Re-Timer glasses; Bonnar et al., 2015; Lovato & Lack, 2015). As such, using a combined approach to treatment of DSWPD (e.g., compound melatonin, bright light therapy, exercise) may be more effective than the traditional evidence-based methods of morning bright light therapy or compound melatonin alone (Gradisar et al., 2014).

## Summary

To date, relatively little is known about the impact of exercise on circadian and sleep timing across the 24-hr day. There is a convincing body of literature that suggests nocturnal exercise causes delays in the human circadian rhythm (Baehr et al., 2003; Baehr et al., 1999; Barger et al., 2004; Buxton et al., 1997; Eastman et al., 1995; Van Reeth et al., 1994; Youngstedt et al., 2002). This finding may have particular relevance to the many adolescents and young adults who complete physically-alerting activities (e.g., exercise) prior to bed, as doing so may place them at a greater risk of developing Delayed Sleep-Wake Phase Disorder. On the other hand, the field currently lacks consensus on the time that exercise needs to be administered to induce phase advances of circadian timing. There is some evidence to suggest that the gradual advancement (e.g., 20-30mins) of the timing of exercise may help to facilitate phase advances in circadian timing (e.g., melatonin rhythms; Miyazaki et al., 2001). Therefore, combining phase advances of activity- rest schedules, in line with current gold standard treatments for DSWPD (e.g., exogenous melatonin and morning bright light therapy) could improve the efficacy of treatment for DSWPD, particularly for those at risk of

withdrawal or relapse. In conclusion, this review suggests that exercise can alter circadian timing, at least in terms of circadian phase delays and finally, that the treatment of Delayed Sleep-Wake Phase Disorder in adolescents and young adults may benefit by providing guidance regarding the timing of exercise.

# Chapter 3

## Are Cognitive "Insomnia" Processes Involved in the Development and Maintenance of Delayed Sleep-Wake Phase Disorder?

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### Author Contributions

CR led review conceptualisation, literature search, results interpretation and manuscript preparation. MG contributed to review conceptualisation, literature search, results interpretation and manuscript preparation. SB provided data presented in Figure 3.1 and 3.2, and assisted with manuscript preparation.

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## Abstract

Although individuals with Delayed Sleep-Wake Phase Disorder (DSWPD) and Chronic Insomnia Disorder (CID) share many of the same phenomenological experiences, theories relating to the development and maintenance of these disorders are distinct in focus. Unlike CID, theory relating to DSWPD is primarily physiologically based and assumes almost no cognitive pathway. However, recent research findings suggest that individuals with DSWPD also display many of the sleep-disordered cognitive processes that were previously assumed to be unique to the insomnia experience. As such, this review aims to summarise current research findings to address the question "Could cognitive processes be involved in the development and maintenance of DSWPD?" In particular, the presence of cognitive and physiological pre-sleep arousal, sleep-related attentional bias, distorted perception of sleep and daytime functioning, dysfunctional beliefs and safety behaviours will be investigated. As this emerging area of research requires a stronger evidence base, we highlight suggestions for future investigation and provide preliminary practice points for clinicians assessing and treating "insomnia" in clients with DSWPD.

## Introduction

Historically speaking, Delayed Sleep-Wake Phase Disorder (DSWPD) has been poorly distinguished from Chronic Insomnia Disorder (CID[1]) (Weitzman et al., 1981). DSWPD arises when the circadian rhythm and sleep-wake pattern of individuals is significantly later than the 24-hr social world (American Academy of Sleep Medicine [AASM], 2014). Individuals with DSWPD typically report an inability to initiate sleep at a desired clock time and difficulty awakening to fulfil morning requirements such as school, university or work (Sack, Auckley, Auger, et al., 2007). As a consequence of delayed sleep timing, individuals with DSWPD often report gaining inadequate sleep (e.g., reduced total sleep time due to early morning sleep onset). In an effort to compensate for poor sleep and counteract daytime impairments, individuals may attempt to go to bed at a much earlier clock time, which can then further exacerbate symptoms of sleep-onset insomnia (AASM, 2014). Due to this overlap between sleep-onset insomnia and the delayed sleep onset of DSWPD, there is emerging evidence that cognitive "insomnia" processes may be implicated in the nocturnal, and to a lesser extent daytime, functioning of DSWPD individuals.

Whereas individuals with DSWPD commonly report symptoms of severe sleep onset insomnia, those with chronic insomnia can experience difficulty initiating *and/or* maintaining sleep, or gaining sleep which is non-restorative (AASM, 2014). However, individuals with CID or DSWPD report many of the same phenomenological experiences, including prolonged sleep onset, impaired sleep quality and significant daytime sleepiness (although morning sleepiness may be more severe in DSWPD individuals; AASM, 2014; Campbell & Murphy, 2007; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; MacMahon, Broomfield, & Espie, 2006; Saxvig et al., 2013; Wyatt, 2004).

[1] Insomnia nomenclature includes the terminology chronic insomnia disorder, chronic insomnia and primary insomnia.

Given the similarities in phenomenology, it is somewhat unsurprising that individuals with DSWPD have improved significantly on measures of insomnia following adjustment of circadian timing (Danielsson, Jansson-Fröjmark, Broman, & Markström, 2013). Additionally, similar treatment techniques have shown to be successful for both disorders (e.g., cognitive behaviour therapy [CBT] and light therapy; Danielsson et al., 2013; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lack, Wright, Kemp, & Gibbon, 2005; Lack, Wright, & Paynter, 2007). Considering the similarities in client presentation and treatment approach, it seems counterintuitive that modern day theory relating to the two disorders is so distinct. The aim of this paper is to review the evidence for the existence of cognitive insomnia processes in those diagnosed with DSWPD. We begin by summarising current conceptualisations of insomnia. Subsequently, we explore how these models may apply to DSWPD and review the evidence for each cognitive insomnia process. We end by providing suggestions for clinical practice and future research studies.

## Conceptualisations of Insomnia

Leading theories relating to the development and/or maintenance of insomnia were established in the early 21st century (Espie, 2002; Harvey, 2002; Lundh & Broman, 2000). Each of these theories expand upon earlier conceptualisations (Spielman, Caruso, & Glovinsky, 1987) by proposing a series of relationships between cognitive and physiological processes in the development and/or maintenance of sleep disturbance. Although the mechanisms behind each of these models are somewhat distinct, there are a number of commonalities. Firstly, each of the three prevailing models acknowledge the role that cognitive arousal (e.g., rumination and worry) plays in preventing sleep. This is unsurprising, given that pre-sleep cognitive arousal is the most commonly cited cause of sleep disturbance, by those suffering from insomnia (Lichstein & Rosenthal, 1980) and has been consistently

associated with prolonged sleep onset (Gross & Borkovec, 1982; Harvey, 2000; Nicassio et al., 1985; Wicklow & Espie, 2000).

Heightened physiological arousal (e.g., heart rate, core body temperature, metabolic rate; Bonnet & Arand, 1998a, 2010; Riemann et al., 2010) is common for individuals with insomnia, and experimental research has confirmed a causal relationship between pre-sleep physiological arousal and prolonged sleep onset (Bonnet & Arand, 1998b, 2005). Theories of insomnia propose a bidirectional causal relationship between cognitive (e.g., rumination, mental imagery) and physiological arousal (Espie, 2002; Lundh & Broman, 2000), which may further prevent normal sleep engagement. However to date, empirical evidence supporting this relationship is limited (Hall et al., 2007; Wuyts et al., 2012).

In their conceptualisations of insomnia, Harvey (2002) and Espie (2002; 2006) both suggest that emotional distress and concern about sleep can lead to the development of an attentional bias towards sleep-threat stimuli (e.g., allocating attention toward the internal (e.g., hunger) or external (e.g., outside noises) cues that signal sleeplessness). To date, individuals with Primary Insomnia have reliably shown an attentional bias towards sleep-related stimuli (MacMahon et al., 2006; Marchetti, Biello, Broomfield, MacMahon, & Espie, 2006; Neitzert Semler & Harvey, 2004b; Taylor, Espie, & White, 2003; Woods, Marchetti, Biello, & Espie, 2009). However, any direct relationship between attentional bias and sleep is still unclear (Neitzert Semler & Harvey, 2004b; Spiegelhalder et al., 2010). Regardless, changes in attentional processing may maintain disturbed sleep, in that sleep-related attentional bias increases the likelihood that sleep inhibiting signals (e.g., physiological arousal) will be detected.

Sleep-related attentional bias, as well as pre-sleep cognitive and physiological arousal, may also fuel distorted perceptions of sleep and daytime functioning (Bonnet & Arand, 1997;

Espie, 2002; Harvey, 2002; Harvey & Tang, 2012; Lundh & Broman, 2000; Tang & Harvey, 2004). Individuals with insomnia are said to often underestimate their total sleep time (e.g., by overestimating sleep onset; Wicklow & Espie, 2000) and misattribute daytime consequences (e.g., memory lapses) as being caused by poor sleep (AASM, 2014; Carskadon et al., 1976). Negative evaluation of sleep and daytime functioning has been associated with more negative sleep-related thoughts, daytime sleepiness, sleep-threat monitoring and the use of safety behaviours during the day (Neitzert Semler & Harvey, 2005, 2006).

Dysfunctional beliefs and safety behaviours are thought to further contribute to the development and maintenance of sleep disturbance (Espie, 2002; Harvey, 2002; Lundh & Broman, 2000). Individuals with insomnia commonly hold erroneous views about the nature of "good" sleep (e.g., how much sleep is required) and overestimate the effect that poor sleep has upon functioning (e.g., health). This type of thinking may prolong sleep onset (Wicklow & Espie, 2000). Although this review primarily focuses upon cognitive processes, elements of behaviour also come into play. Safety behaviours are often employed by individuals in an effort to avoid feared night time and/or daytime outcomes (e.g., lengthy sleep onset and impaired school performance, respectively; Hood, Carney, & Harris, 2011; Woodley & Smith, 2006). However, in many cases safety behaviours (e.g., thought suppression, going to bed earlier) prevent the disconfirmation of dysfunctional beliefs and can paradoxically, worsen sleep disturbance (e.g., by increasing pre-sleep cognitive arousal; Espie, 2002; Harvey, 2002; Lundh & Broman, 2000). Interestingly, there appears to be a positive relationship between sleep-related monitoring, negative thinking, daytime sleepiness and the use of safety behaviours for individuals with insomnia (Neitzert Semler & Harvey, 2004a, 2007), further highlighting the complex role that cognitive processes may play in the development and maintenance of sleep disturbance.

In summary, there is strong research evidence of the role of cognitive factors in the development and maintenance of insomnia, however, it has been suggested that similar processes may also apply to other sleep disorders (Harvey, 2002). Given the similarities in phenomenology with CID, we propose it is likely that these processes are also involved in the development and maintenance of DSWPD.

## Conceptualisations of DSWPD

The underlying mechanisms involved in the development and maintenance of DSWPD are still somewhat unclear. Until relatively recently, DSWPD has been conceptualised primarily from a chronobiological standpoint. Therefore, current explanations are physiological in nature (Wyatt, 2004). DSWPD is most commonly explained as a mistiming of the circadian rhythm. In support of this supposition, research has shown significant delays in the dim light melatonin onset, sleep onset and core body temperature of individuals with DSWPD, compared to good sleepers (Saxvig et al., 2013). Individuals with DSWPD may have a longer circadian rhythm length (*tau*; e.g., core body temperature rhythm) than good sleepers, placing them at greater risk of adapting sleeping patterns to the 24-hr day (Campbell & Murphy, 2007; Micic et al., 2013). Altered light sensitivity (i.e., greater evening melatonin suppression from bright light) may delay the sleep timing of those with DSWPD (Aoki et al., 2001); however, confirmation from replication studies is needed. Although much of the current explanation of DSWPD focuses solely on physiologically-driven changes in circadian timing, there are few studies supporting physiological contributing factors (Aoki et al., 2001; Micic et al., 2013; Saxvig et al., 2013).

In line with advances in the conceptualisation of insomnia, researchers have begun to investigate DSWPD using a cognitive and behavioural framework. As such, support for the role that psychological factors may play in the development and maintenance of delayed

sleep timing has increased over recent years (Gradisar & Crowley, 2013; Lack & Wright, 2007; Takahashi et al., 2000). The delayed sleep onset associated with DSWPD can occur gradually (e.g., throughout puberty), or via psychological stressors (e.g., interpersonal difficulties, change of occupation, Takahashi et al., 2000). A combination of an evening chronotype (e.g., later circadian preference, Roenneberg et al., 2004) and choosing a "socially acceptable" bedtime (Lack & Wright, 2007) may maintain a delayed sleep onset. Significantly prolonged sleep onset (>45mins), without the necessary cognitive and physiological readiness for sleep (Lack & Wright, 2007), provides the opportunity for excessive unhelpful thinking, which can act to further delay the onset of sleep and lead to the development of other symptoms of conditioned insomnia (e.g., negatively toned rumination, physiological pre-sleep arousal, selective attention, distorted perception, dysfunctional beliefs and safety behaviours). Therefore, in this review, we argue that cognitive processes may contribute to the development and maintenance of DSWPD, and likely need addressing in treatment with psychological techniques.

## Cognitive Processes in DSWPD

### Cognitive Pre-sleep Arousal

In terms of client presentation, individuals with DSWPD commonly report a difficulty initiating sleep (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; van Maanen, Dewald-Kaufmann, Smits, Oort, & Meijer, 2013) and experience prolonged sleep onset latencies when compared to good sleepers ($d$=0.52, Saxvig, Pallesen, Wilhelmsen-Langeland, Molde & Bjorvatn, 2012; $d$=0.59, Saxvig et al., 2013). Cognitive arousal and prolonged sleep onset may go 'hand-in hand", thus it is unsurprising that 89% of adolescents with DSWPD experience "racing thoughts" in bed (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Although this phenomenology is likely to be universal to

the DSWPD experience (Takahashi et al., 2000), teenagers may be at particular risk given adolescence is a series of 'firsts' (i.e., first job, first relationship, etc.; Dahl & Lewin, 2002). It is plausible that a relationship between emotional regulation and adolescents' sleep is mediated by pre-sleep cognitive arousal. Emotional competence, particularly the ability to understand and regulate one's own emotions, has indeed been linked with sleep disturbance in adolescents (Brand, Kirov, et al., 2016). However, at this stage, it is unclear whether impaired cognitive-emotional processing increases adolescent's vulnerability to pre-sleep cognitive arousal as thus, the development of DSWPD.

To better understand the nature of pre-sleep cognitive arousal in adolescents with DSWPD, Hiller and colleagues (2014) assessed sleep-related cognitions using a catastrophising interview (Harvey & Greenall, 2003). Similar to previous findings (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), 87% of the sample reported catastrophic thoughts, with a majority of these concerns about sleep-related effects on school performance (48%, academically) or interpersonal functioning at school (18%, teacher and peer interactions) – findings not dissimilar to adults with insomnia (Wicklow & Espie, 2000). No direct link was found between catastrophising (number of catastrophic thoughts) and sleep latency ($\beta$=0.08), however, catastrophising was associated with sleep anticipatory anxiety (e.g., rehearsal ['*what happened today*'] and planning ['*what to do tomorrow*']), which then predicted an extended latency to sleep ($\beta$=3.76; Hiller et al., 2014). As such, it is likely that the affect of pre-sleep cognitive activity has a greater influence on sleep onset, than the number of catastrophic thoughts, per se. These findings are supported with data from non-clinical young adults, whereby later bed times (and reduced sleep duration) were associated with repetitive negative thinking (Nota & Coles, 2014, $d$=0.62). However, due to these cross-sectional research designs, it is not known whether pre-sleep cognitive arousal

causes the chronic delayed sleep onset of those with DSWPD, or if these negative thinking styles 'fill the void' whilst waiting for their biologically-driven delayed sleep onset to occur.

Recent treatment studies provide further insight for the role of cognitive arousal in DSWPD. Advancement of circadian timing, using bright light therapy alone, has been associated with improvements in sleep onset latency (*d*=0.80) *and* cognitive sleep anticipatory anxiety (*d*=1.01) in those experiencing sleep-onset difficulties[2] (Lack et al., 2007). More recently, these findings have been replicated in adolescents with DSWPD, who experienced significantly improved sleep onset latency (e.g., 40mins, *d*=0.69), total sleep time (61mins, *d*=0.66), insomnia severity (*d*=1.28; see Figure 3.1), and pre-sleep worry (e.g., rehearsal and planning; Figure 3.2, *d*=1.04), following treatment with bright light therapy (Barbero & Gradisar, 2013, unpublished thesis). Unfortunately, no control group was used; yet, these findings suggest that improving the sleep of adolescents with DSWPD has associated improvements in pre-sleep cognitive arousal, even in the absence of cognitive therapy. We do not yet know if the reverse is true, that is, whether increasing cognitive arousal delays sleep timing, or whether delaying sleep timing increases pre-sleep cognitive arousal. For example, a recent attempt by Richardson et al. (2015) at exposing good sleepers to an analogue stressor delayed their sleep timing that night. However, this did not appear to be due to a change in cognitive arousal, and the delayed sleep was not sustained. As such, future research should aim to investigate the development of delayed sleep timing and cognitive pre-sleep arousal prospectively.

[2] N.B. eligibility criteria for sleep-onset insomnia closely aligned with DSWPD diagnostic criteria

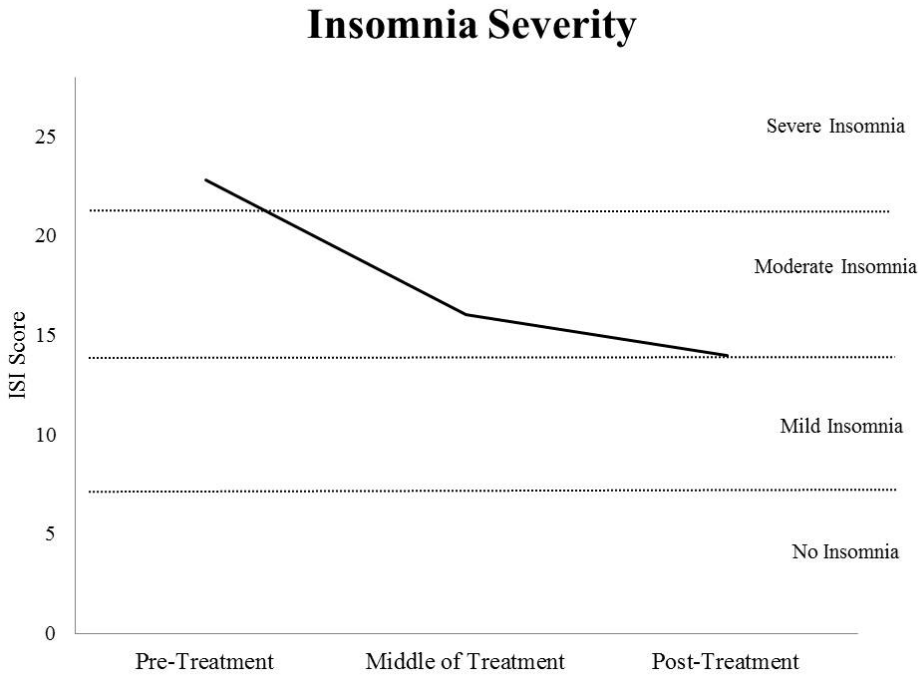


*Figure 3.1* Mean insomnia severity index score at pre-treatment, middle of treatment and post-treatment. ISI= insomnia severity index.

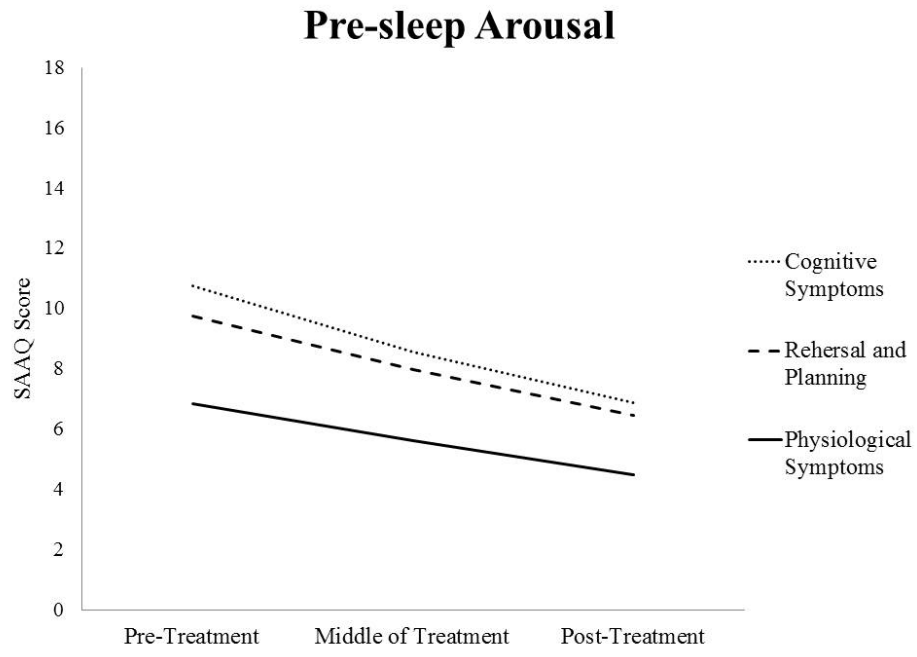


*Figure 3.2* Mean sleep anticipatory anxiety questionnaire subscale scores at pre-treatment, middle of treatment and post-treatment. SAAQ= sleep anticipatory anxiety questionnaire.

**Physiological Pre-sleep Arousal**

Individuals with DSWPD have displayed significantly delayed dim light melatonin onset (Saxvig et al., 2013, *d*=1.74), and core body temperature rhythms (Micic et al., 2013, *d*=1.91) compared to healthy good sleepers. Therefore, peak physiological alertness occurs later in individuals with DSWPD (Gradisar & Crowley, 2013). However, those with DSWPD may attempt to initiate sleep at a much earlier "socially acceptable" time, in an effort to compensate for sleep loss and impairments in daytime functioning (AASM, 2014). If individuals with DSWPD attempt sleep without the physiological signs of sleep preparedness, it is likely they will experience heightened pre-sleep physiological arousal. Detection of physiological arousal is likely to contribute to and/or worsen negatively-toned thinking in bed (Harvey, 2002), especially if the individual is unable to engage in sleep quickly, thus prolonging sleep onset. Unfortunately, we could not find studies which have empirically linked peak physiological alertness with cognitive arousal in individuals with DSWPD. As a result, future research should aim to measure pre-sleep cognitive and physiological arousal in individuals with DSWPD, possibly prior, during and following treatment.

**Sleep-related Attentional Bias**

Given the role attentional bias plays in maintaining many psychological disorders (e.g., depression, Peckham, McHugh, & Otto, 2010; anxiety, Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & Van Ijzendoorn, 2007; eating disorders, Dobson & Dozois, 2004), it is unsurprising individuals experiencing insomnia disorders reliably show attentional biases towards sleep-related stimuli (MacMahon et al., 2006; Marchetti et al., 2006; Taylor et al., 2003). Using a flicker paradigm task and rigorous diagnostic screening (e.g., sleep diary, actigraphic measurement and diagnostic clinical interview), Marchetti and colleagues (2006) found that sleep-related attentional bias existed within both Primary Insomnia (PI) and

DSWPD populations. Both groups took significantly longer to fall asleep than good sleepers, and detected a sleep-related change to a visual scene significantly quicker (PI: *d*=2.96, DSWPD: *d*=0.83). However, attentional bias was significantly stronger for the PI group. These findings were supported by MacMahon and colleagues (2006), who found the PI (*d*=0.67) and DSWPD (*d*=0.40) groups showed greater attentional biases towards sleep-related stimuli compared to good sleepers when using a dot-probe task.

In terms of limitations, studies have used a sample of convenience, mostly comprising university students and staff. This may affect generalisability, as university students have greater ability to sleep "in sync" with their circadian timing, with relatively little consequence (e.g., without being late to class), especially compared to adolescents. Thus, individuals are less likely to narrow attention towards sleep stimuli, if they do not perceive sleep loss as a threat, or alternatively, are able to sleep in, thus reducing daytime sleep craving. Therefore, future research should aim to measure sleep-related attentional bias in individuals with DSWPD, within clinical settings. Regardless, current research findings suggest that sleep-related attentional bias may be implicated in the development and/or maintenance of DSWPD, and as such, attentional processing may be assessed and targeted in treatment, if appropriate.

**Distorted Perception of Sleep and Daytime Functioning**

Individuals experiencing sleep disturbance tend to hold a distorted perception of their sleep and daytime functioning. Where some studies have reported agreement between objective and subjective sleep measures in samples of DSWPD (Ancoli-Israel et al., 2003), this is not always the case. For example, DSWPD adolescents (*N*=49) misperceived the time that they fell asleep by 14mins (Gradisar, Dohnt, Gardner, Paine, Starkey, Billows, et al., 2011, *d*=0.10). However, this was more pronounced for females, who misperceived their

sleep onset time by 45mins (*d*= 0.83; Billows & Gradisar, 2009, unpublished thesis). Although sleep misperception was not directly assessed, a recent randomised-control trial for the treatment of DSWPD found participants overestimated the time it took them to fall asleep by 23mins (*d*=0.84; Saxvig et al., 2014). Following two weeks of circadian treatment, differences in objective vs. subjective sleep onset latency occurred to a lesser extent (10.8mins, *d*=0.65). Although these studies provide preliminary evidence for the presence of sleep-onset misperception, it is unclear whether this phenomenon is a unique feature of DSWPD, given comparison control groups (e.g., good sleepers) were lacking.

Misperception of daytime functioning and performance has received little research attention. This may be, in part, due to the innate difficulty in measuring misperception of daytime deficits, especially given that DSWPD has actually been associated with significant impairments (e.g., academic performance, daytime sleepiness, fatigue, inattention, mood, energy; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2012). However, one study has measured both subjective and objective daytime sleepiness (Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Although measures of subjective (e.g., measured by the Karolinska sleepiness scale [KSS] and the Epworth sleepiness scale [ESS]) and objective sleepiness (e.g., measured by the alpha attenuation test [AAT]), are not directly comparable, a discrepancy was observed over time. Measures of subjective sleepiness significantly improved (KSS: *d*=1.14, ESS: *d*=1.02), whereas there was no significant change in objective sleepiness (*d*=0.15). This may suggest individuals with DSWPD overestimate the effect that their sleep is having on daytime functioning; a trademark of the insomnia experience (Harvey, 2002). As only one study has been performed in this area, it is crucial that future research replicate these findings using multiple sensitive measures of both subjective and objective daytime functioning (e.g., multiple sleep latency test).

## Dysfunctional Beliefs

Given the paucity of research into insomnia processes present in those with DSWPD, it is not surprising that there has been only a single study investigating dysfunctional sleep-related beliefs. This recent study provided insight into experiences held by individuals with DSWPD using a qualitative design (Wilhelmsen-Langeland et al., 2012). Participants expressed a belief that having DSWPD required them to choose between sleep or their daytime commitments, and expressed worries about the impact sleep was having on their mood and psychological health (e.g., "*if I go to bed now then I cannot sleep…that results in problems the rest of the day and then, I maybe become grumpy*"; "*it affects me a lot psychologically*"; Wilhelmsen-Langeland et al., 2012). Some DSWPD participants also reported losing control over their ability to sleep (e.g., "*I know that it won't work and then, I sort of just give up*"). Individuals with insomnia report similar dysfunctional beliefs and attitudes about sleep (Morin, 1993). Whilst novel, it is unclear whether these qualitative findings generalise to the wider DSWPD population, over and above what is observed within the general population. As such, future research should aim to quantitatively measure and compare dysfunctional beliefs and attitudes held about sleep (e.g., using the dysfunctional beliefs and attitudes about sleep scale), in clinical samples of DSWPD, chronic insomnia patients, and good sleepers. Changes in dysfunctional and erroneous beliefs should also be measured throughout assessment and treatment in future research and clinical practice.

## Safety Behaviours

Although the focus of this review has been on cognitive processes, cognitions are not mutually exclusive from behaviours. Individuals with DSWPD may use overt and covert coping strategies in an effort to prevent sleep-onset difficulties and daytime impairment (AASM, 2014; Harvey, 2002). Alcohol, sedatives and hypnotics may be used to prevent

feared night time consequences and associated cognitions (e.g., prolonged sleep onset; "*it's not normal to take this long to fall asleep*"; AASM, 2014; Hiller et al., 2014; Saxvig et al., 2012). Delayed sleep timing by adolescents on weekends (O'Brien & Mindell, 2005) and oversleeping on weekdays has been linked with greater likelihood of alcohol and marijuana use (Pasch et al., 2010). More recent studies investigating the prevalence of DSWPD in both Australian and Norwegian school-based samples have partially supported these findings, with significantly greater alcohol consumption in delayed compared to non-delayed sleepers (Lovato, Gradisar, et al., 2013, d=0.62; Saxvig et al., 2012, d=1.22). In the Norwegian sample, alcohol consumption was above the clinical cut-off, suggesting harmful levels of drinking (Saxvig et al., 2012). Differences in drug use (e.g., marijuana) were not found, however this may be due to the small number of participants meeting full diagnostic criteria for DSWPD, or underreporting (Lovato, Gradisar, et al., 2013). Although limited, there is some evidence that individuals with delayed sleep timing may use these overt safety behaviours (i.e., central nervous system [CNS] depressants) for the purpose of promoting sleep onset (Lund, Reider, Whiting, & Prichard, 2010; Weitzman et al., 1981). However, adolescents with DSWPD report caffeine use (e.g., coffee, tea, chocolate, Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011) and a higher prevalence of smoking (61%), compared to non-delayed sleepers (28.3%, Saxvig et al., 2012). Therefore, CNS stimulants may be used to prevent feared daytime outcomes and associated cognitions (e.g., impaired mood, "*I hate feeling grumpy*"; school performance, "*I'll miss important information at school*" and interpersonal relationships, "*I don't want to mum to worry*"; Hiller et al., 2014).

It is also likely individuals with DSWPD use covert coping strategies (e.g., thought and imagery control) to prevent excessive pre-sleep cognitive arousal and prolonged sleep onset. Adolescents with later weekday bedtimes have more commonly reported using music (*rs*=0.174), television (*rs*=0.118) and computer games (*rs*=0.091) as a sleep aid (Eggermont

& Van den Bulck, 2006), which may help suppress unwanted thoughts. Television has been used by school-aged children as a way to help regulate emotions (Chen & Kennedy, 2005). Most recently a longitudinal study has shown that young adults may use electronic media (e.g., television and social networking) as a method to cope with their sleep problems (Tavernier & Willoughby, 2014). These findings go against previous assumptions, that media use displaces sleep and/or prolongs sleep onset. However, given the recency of these findings, it is not yet known whether individuals with DSWPD also use certain technological devices as a way to avoid pre-sleep cognitive arousal during their prolonged sleep onset (Harvey, 2002).

Safety behaviours may also be used by individuals with DSWPD to overcome diurnal rhythm preference and associated daytime symptoms. Upon "early morning awakening", individuals with DSWPD are likely to experience excessive sleepiness, irritability, low mood and reduced motivation and therefore, resume sleep (Lack & Wright, 2007) (e.g., "*when I have to get up at six, seven, eight o'clock…it is really just a hassle…it demands that you are awake and clear when things start*"; Wilhelmsen-Langeland et al., 2012). This may help to explain why students with DSWPD are more prone to oversleeping, school lateness, absences and drop out (Gradisar & Crowley, 2013; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2012). Unfortunately, oversleeping prevents exposure to the circadian re-setting properties of bright light (Sack, Auckley, Auger, et al., 2007). If not oversleeping, individuals with delayed sleep timing or DSWPD have shown to use daytime naps as a way of coping with inadequate sleep (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). This may produce a vicious cycle whereby napping reduces sleep homeostatic pressure, creating insufficient sleep, and the need to nap the next day. Sleeping-in, napping and daytime stimulant use can further maintain DSWPD by delaying exposure to

morning bright light, impeding the build-up of sleep pressure and preventing pre-sleep physiological de-arousal, respectively.

In summary, it may be important for clinicians and researchers to assess for overt and covert strategies that individuals with DSWPD may employ to promote sleep onset and to prevent deficits in daytime functioning. The use of a detailed sleep diary (e.g., incorporating caffeine use), a sleep history interview and questionnaire measures (e.g., sleep-related behaviours questionnaire; Ree & Harvey, 2004) would be particularly useful. Future qualitative research should aim to link specific safety behaviours (e.g., morning caffeine use) with feared outcomes (e.g., impaired performance at school). Safety behaviours may ultimately worsen sleep disturbance via various means (e.g., preventing disconfirmation of dysfunctional beliefs), and thus may need addressing during treatment.

## Clinical Implications

Several of the abovementioned findings highlight differences in insomnia processes between those with DSWPD and good sleepers (Espie, Marchetti, et al., 2006; MacMahon et al., 2006; Marchetti et al., 2006), stressing the importance of undertaking a thorough assessment of sleep disturbance phenomenology when DSWPD is suspected. Clinicians could administer self-report questionnaires assessing pre-sleep cognitive and physiological arousal (e.g., pre-sleep arousal scale, Nicassio, Mendlowitz, Fussell & Petras, 1985; sleep anticipatory anxiety questionnaire, Bootzin, Shoham, & Kuo, 1994), sleep-related attentional bias (e.g., sleep associated monitoring index, Neitzert Semler & Harvey, 2004b), sleep misperception (e.g., sleep diary and actigraphy), dysfunctional beliefs (e.g., dysfunctional beliefs and attitudes about sleep scale, Morin, 1993), and safety behaviours (e.g., sleep-related behaviours questionnaire, Ree & Harvey, 2004). For clinicians who have access to the

resources, objective measures could also be administered (e.g., computerised attentional bias task).

Despite a small evidence base for insomnia processes occurring in those experiencing DSWPD, some clinicians may ask – *is there a need to treat insomnia processes in patients with DSWPD*? Lack and Wright (2007) highlighted the importance of including treatment techniques targeting non-circadian aetiological factors, and recent data support using adjunct treatments to traditional behavioural therapies (Danielsson et al., 2013; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). In a randomised control trial of cognitive and behavioural (light) therapy for adolescent DSWPD, many sleep improvements occurred (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Unfortunately, systematic measures of insomnia (e.g., insomnia severity index) were not used, thus the relative contribution from cognitive therapy to improving insomnia processes is unknown. However, Danielsson and colleagues (2013) recently compared the effectiveness of cognitive behaviour (light) therapy, with behaviour (light) therapy alone. Both groups experienced improvements in sleep timing and insomnia symptoms, yet those receiving adjunct cognitive therapy appeared to better maintain these improvements in the long term (e.g., six months). Given the high rate of relapse within DSWPD populations, this new study suggests long-term treatment outcomes may be improved through the use of cognitive therapy techniques (Alvarez, Dahlitz, Vignau, & Parkes, 1992).

So what can clinicians do to reduce insomnia processes occurring in their DSWPD patients? Cognitive restructuring (e.g., challenging negative automatic thoughts and generating alternative thoughts), mental imagery and relaxation training may be particularly useful in breaking the cycle of pre-sleep cognitive and physiological arousal, which prevents sleep onset. A thorough assessment of the content of individual's pre-sleep thoughts and

imagery may improve the effectiveness of cognitive strategies in the treatment of DSWPD (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), especially given the type of pre-sleep mental activity most detrimental to sleep may be unrelated to sleep (e.g., thinking about the day's events, Hiller et al., 2014). Sleep-related psycho-education and behavioural experiments may assist clinicians to challenge dysfunctional and erroneous beliefs about the nature and consequences of sleep (e.g., teaching about individual sleep needs rather than generic need for 8-9hours sleep per night to function; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). The use of similar treatment techniques has resulted in more durable treatment effects in individuals with insomnia (Morin, Blais, & Savard, 2002). Cognitive therapy techniques may also be used to challenge safety behaviours (e.g., testing the belief that alcohol is required to be able to fall asleep and psycho-education around the effects of alcohol on sleep quality; Bennett-Levy et al., 2004). Similar to the treatment of insomnia, comparisons of objective and subjective assessment measures (e.g., sleep diary and actigraphy) may also help to train client's perception of their sleep onset (Harvey, Sharpley, Ree, Stinson, & Clark, 2007; Tang & Harvey, 2006). By targeting sleep misperception in therapy, individuals with DSWPD may feel less anxious about their sleep (e.g., as they are gaining more sleep), which may encourage cognitive and physiological de-arousal processes, and as such, automatic sleep engagement (Espie, 2002; Tang & Harvey, 2006). Finally, although not widely used in the treatment of sleep disturbance, the potential to use attentional bias modification tasks to reallocate individual's attention away from sleep-threat stimuli (MacLeod & Mathews, 2012) would further promote automatic sleep engagement (Espie, 2002; MacLeod & Mathews, 2012) for those with DSWPD. In summary, cognitive therapy can be used in the treatment of DSWPD, to target cognitive "insomnia" processes and as such, promote more durable treatment outcomes.

## Summary

At the beginning of this review, we posed the question "Could cognitive processes be involved in the development and maintenance of DSWPD?" Based on the findings from 12 studies performed on DSWPD and delayed sleep timing samples (Barbero, 2013; Billows, 2009; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Hiller et al., 2014; Lack et al., 2007; Lovato, Gradisar, et al., 2013; MacMahon et al., 2006; Marchetti et al., 2006; Nota & Coles, 2014; Saxvig et al., 2012; Takahashi et al., 2000; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), our preliminary answer is 'yes'. Worth noting is that many of these studies did not possess an *a priori* focus on cognitive insomnia processes in DSWPD. Three further studies have found interrelationships between cognitive and behavioural therapies and insomnia processes in DSWPD patients (Barbero, 2013; Danielsson et al., 2013; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), with the primary benefit being potential long-term gains. This field is thus open to significant scientific exploration. Innumerable adolescents and young adults with DSWPD are being assessed and treated worldwide, yet the empirical literature is suffering itself from a lack of data, which has the potential to give researchers and clinicians a 'cognitive view' of this sleep disorder. We urge the field to begin including cognitive measures of insomnia at pre- and post-treatment, and where possible, design trials geared towards understanding whether cognitive therapy benefits the thousands of individuals experiencing DSWPD.

# Chapter 4

## A Randomised Controlled Trial of Bright Light Therapy and Morning Activity for Adolescents with Delayed Sleep-Wake Phase Disorder.

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**Author Contributions**

CR led study conceptualisation, design, recruitment, data collection, management and analysis, results interpretation and manuscript preparation. NC and KB assisted with recruitment, data collection and manuscript preparation. GM assisted with data collection and manuscript preparation. BM assisted with data management and analysis. MG contributed to study conceptualisation, design, recruitment, data collection, results interpretation and manuscript preparation.

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**Abstract**

A randomised controlled trial evaluated bright light therapy and morning activity for the treatment of Delayed Sleep-Wake Phase Disorder (DSWPD) in adolescents. 60 adolescents (mean= 15.9±2.2 y, 63% f) diagnosed with DSWPD were randomised to receive Green Bright Light Therapy and sedentary activity, Green Bright Light Therapy and morning activity, Red Light Therapy and sedentary activity or Red Light Therapy and morning activity. Sleep and daytime functioning was measured pre-treatment, post-treatment and at 1- and 3-month follow-up. Contrary to predictions, interaction effects between treatment group and time for all outcome variables were not statistically significant. However, adolescents in the morning activity conditions did not meaningfully increase their objective activity. Overall, adolescents reported significantly improved sleep timing (*d*=0.53-0.61), sleep onset latency (*d*=0.57), total sleep time (*d*=0.51) and daytime functioning *(d*=0.52-1.02) across treatment and follow-up. However, relapse was common, with 38% of adolescents returning for additional treatment. Although there is convincing evidence for the short-term efficacy of chronobiological treatments for DSWPD, it is clear that treatment for adolescent sleep disorders can be improved.

## Introduction

Delayed Sleep-Wake Phase Disorder (DSWPD) arises when the endogenous circadian rhythm and sleep timing of individuals is significantly later than the 24-hr world (American Academy of Sleep Medicine [AASM], 2014). DSWPD presents as the inability to initiate sleep at a desired clock time and difficulty waking to fulfil morning requirements. DSWPD is estimated to affect between 1-16% of adolescents (AASM, 2014; Lovato, Gradisar, et al., 2013).

Changes in sleep occur throughout puberty, placing adolescents at particular risk of DSWPD. There is a widespread and consistent tendency for later bedtimes (Gradisar, Gardner, et al., 2011), driven by reduced homeostatic sleep pressure, delayed circadian timing, longer circadian period length (tau) and/or altered sensitivity to light (Gradisar & Crowley, 2013). Compounding obligations (e.g., study, part-time work, extra-curricular pastimes) increase the number of tasks adolescents need to complete before sleep (Carskadon, 2011; Gradisar & Crowley, 2013) and lessening of parent-set bedtimes may reinforce later bedtimes (Short et al., 2011). When waking for school, this leads to sleep restriction and impaired daytime functioning (Lovato, Gradisar, et al., 2013; Sivertsen et al., 2015; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Due to the prevalence and impact of adolescent DSWPD, it is important to ensure efficacious treatments are available.

Current treatments include behaviourally-based methods to manipulate circadian timing (e.g., bright light therapy (BLT), exogenous melatonin; Gradisar et al., 2014). However, clinical trials evaluating the efficacy of treatments for DSWPD are rare. There is weak evidence supporting the use of strategically timed melatonin (Saxvig et al., 2014; van Maanen et al., 2013) or combined post-awakening light (via light box) and prescribed sleep-wake scheduling (Auger et al., 2015; Danielsson et al., 2015; Gradisar, Dohnt, Gardner,

Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014). However, the degree of phase advancement can be influenced by light duration (e.g., ≥30 mins; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014), illuminance (e.g., ≥117 lux; Cajochen et al., 2005) and wavelength (e.g., shorter>longer; Warman et al., 2003; Wright et al., 2004). Portable light devices have been recently developed and may be more practical for adolescents preparing for school. However, there is insufficient evidence to support use in treatment (Auger et al., 2015). Additionally, although short wavelength light is thought to be superior in the treatment of circadian rhythm sleep disorders, there is a lack of clinical research to support this hypothesis (Auger et al., 2015).

There is a paucity of research relating to many new areas of enquiry, for example, scheduled physical activity (Auger et al., 2015). Individuals with DSWPD are active late at night and relatively inactive in the morning (i.e., 8-11am, Joo et al., 2017), which may perpetuate delayed sleep-wake timing. Nocturnal exercise delays circadian timing (Baehr et al., 2003; Baehr et al., 1999; Buxton et al., 1997; Van Reeth et al., 1994; Youngstedt et al., 2002), whereas exercise administered at other times of the day may result in phase advances (Richardson, Gradisar, Short, & Lang, 2017), particularly when advanced in small daily increments (i.e., 20 mins, Miyazaki et al., 2001). Therefore, it is possible that post-awakening morning activity, when combined with gradually earlier wake-up times, may help to phase advance the circadian and sleep timing of adolescents with DSWPD. This protocol overlaps that of BLT for DSWPD (Gradisar et al., 2014). Given that BLT can be delivered by portable, wearable devices, it is possible that adding scheduled exercise could improve outcomes.

Clinical guidelines place importance on replicating laboratory-based research within less tightly controlled field settings (Auger et al., 2015). Therefore, the present randomised-controlled study aimed to evaluate i) the efficacy of BLT delivered by portable short-wavelength light devices and ii) whether supplementing BLT with morning activity enhances

treatment outcomes for adolescents[6] with DSWPD. To extend upon a recent trial comparing bright broad-spectrum white light (active treatment) with dim red light (control condition), we compared the efficacy of green (short) wavelength light, to red (long) wavelength light (Saxvig et al., 2014). Within each of these conditions, adolescents were randomly allocated to complete physical activity (upright, motion-sensing videogame) or sedentary activity (sitting, watching TV) during LT. It was predicted that green LT groups, and morning activity groups, would have better outcomes at post-treatment.

## Method

### Participants

455 families made contact with the Child & Adolescent Sleep Clinic following referral from GPs, or in response to community advertisements (Figure 4.1). 164 adolescents and young adults met age criteria for inclusion and 83 were assessed. Of these, 63 met ICSD-3 (AASM, 2014) criteria for DSWPD, and 60 were randomised into the study (mean= 15.9±2.2 y, 63% f). The trial was registered with the Australian and New Zealand Trials Registry (ACTRN12614000308695) and approved by the Southern Adelaide Clinical Human Research Ethics Committee (Application Number 165.14).

Inclusion criteria for the study were i) age 13-24 years, and ii) primary diagnosis of DSWPD. Participants were not excluded for psychological comorbidity, given its prevalence (Reid et al., 2012; Sivertsen et al., 2015), which enhances generalisability of findings (Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). However, severe suicidal ideation led to exclusion.

[6] Participants were 13-24 years, with 90% of the sample aged 13-19.

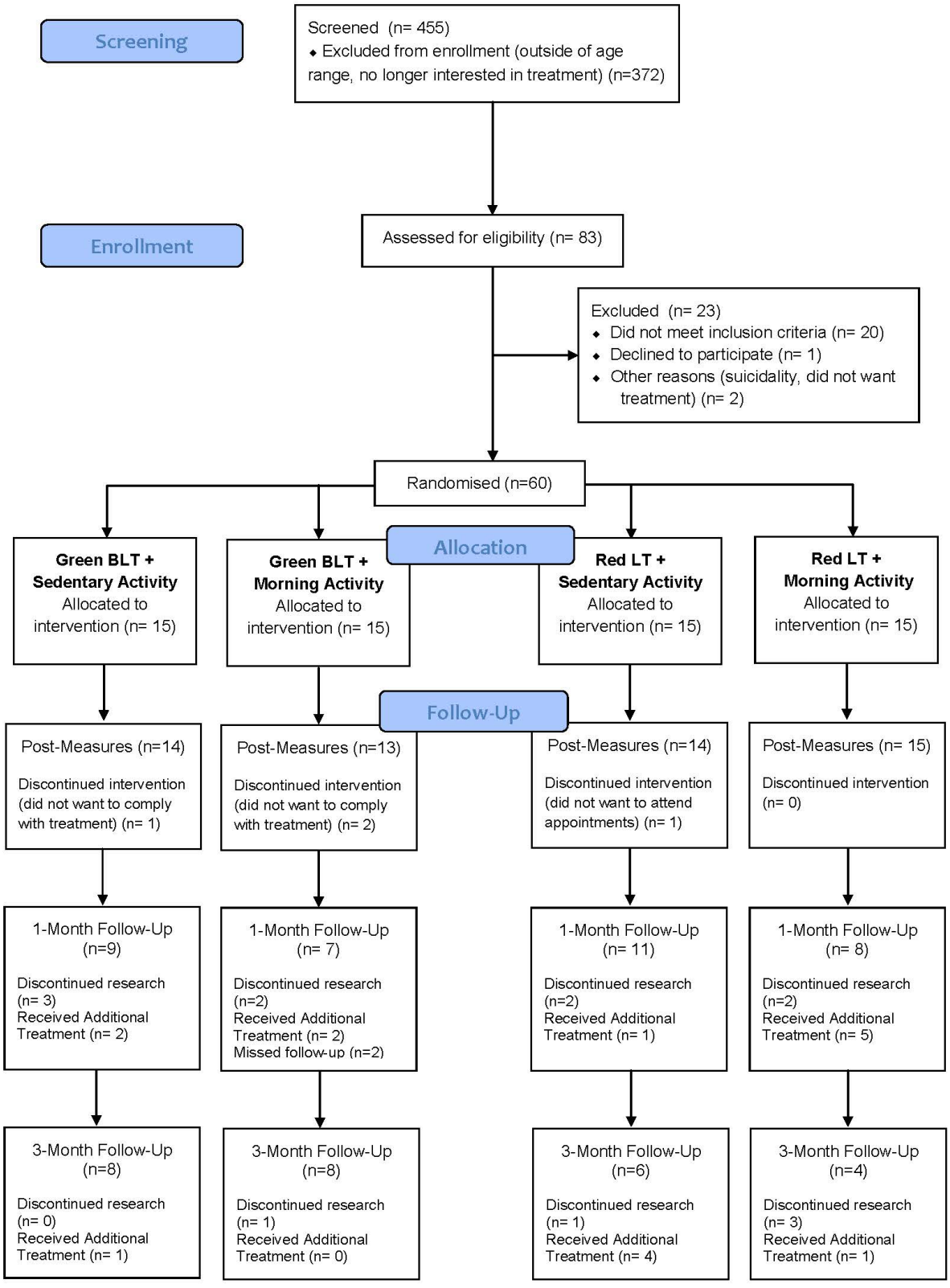


*Figure 4.1* Participant flow.

Adolescents were randomly allocated to receive Green BLT and sedentary activity (SA) (*N*=15, mean age= 15.9±1.8 y, 60% f), Green BLT and morning activity (MA) (*N*=15, mean= 16.2±2.8 y, 67% f), Red LT and SA (*N*=15, mean= 15.5±1.6 y, 67% f) or Red LT and MA (*N*=15, mean= 15.7±2.8 y, 60% f). Groups did not differ in terms of age, $F(3,56) = 0.22$, $p=.88$, or gender distribution, $\chi^2(1, N = 60) = 0.29$, $p= 0.96$. Four adolescents who withdrew prior to treatment completion reported significantly lower baseline total sleep time, $t(57) = -3.00$, $p=.004$, $d= 1.75$, and sleep efficiency, $t(57) = -2.45$, $p=.017$, $d= 1.08$, on school nights, and greater functional impairment, $t(9.07) = -3.68$, $p=.005$, $d= 1.00$.

Table 4.1

*Clinical Presentation of Adolescents with DSWPD.*

| **Primary Sleep Problem** | **Daytime Impairments*** |
|---|---|
| Difficulty falling asleep (68%) | Tired/ fatigued (98%) |
| Difficulty waking (15%) | Daytime sleepiness (93%) |
| Unrefreshing sleep (7%) | Poor attention/ concentration/ memory (93%) |
| Night time awakenings (5%) | Low energy/ motivation (85%) |
| Daytime consequences (5%) | Moody/ irritable (82%) |
| **Secondary Sleep Problem** | Worries about sleep (68%) |
| Difficulty waking (50%) | Poor school performance (65%) |
| Night time awakenings (23%) | Somatic complaints (55%) |
| Difficulty falling asleep (20%) | Problems socialising (48%) |
| Daytime consequences (7%) | Accident prone (40%) |
| **Past Sleep Problem/s** | Behavioural issues (i.e., hyperactivity) (37%) |
| Incidence of past sleep problem (e.g., chronic insomnia disorder, bedtime resistance) (28%) | Repeatedly woken in morning (68%) |
| Parental presence required to sleep in primary schooling (25%) | Frequent lateness to school, university or work (68%) |

* Adolescents could select more than one daytime impairment.

Most adolescents reported their sleep problem occurred gradually (88%), with mean chronicity of 3yrs 5mo. See Table 4.1 for clinical presentation. 66% of participants reported a diagnosed psychological condition, with anxiety (45%) and depression (43%) most common. Distribution of psychological comorbidity between groups was similar, $\chi^2(1, N = 59) = 0.32$, $p= 0.10$. Participants were taking medication to treat mood (*n*=15, Endep, Seroquel, Lexapro, etc.), sleep (*n*=7, melatonin, Circadin) and asthma (*n*=7, Ventolin). Medication timing and

dosage remained consistent, except melatonin/Circadin, which were ceased prior to treatment. Adolescents receiving psychological intervention ceased therapy during the sleep trial.

## Materials and Apparatus

### Clinical Sleep History Interview (CSHI)

A revised semi-structured interview (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011) was used to diagnose DSWPD, as per the ICSD-3 (AASM, 2014). The CSHI elicited information pertaining to Criterion A (chronic difficulty initiating sleep and awakening); Criterion B (present for ≥three months) and Criterion E (differential diagnosis).

### Sleep Diary

The daily online sleep diary measured sleep timing, sleep onset latency (SOL), wake after sleep onset (WASO) and total sleep time (TST). Sleep parameters were averaged for school nights (Sun-Thurs) and weekend nights (Fri-Sat) separately. Sleep diaries are essential for DSWPD diagnosis (Criterion D; AASM, 2014).

### Wrist Actigraphy

Participants wore a MicroMini Motionlogger wrist actigraphy monitor (Ambulatory Monitoring Inc., Ardsley, NY) throughout treatment. Data was collected using zero-crossing mode. Adolescent's sleep onset time and offset time were scored using an algorithm validated for adolescents (AMI-Sadeh, Action 4, Ambulatory Monitoring Inc, Ardsley, NY; Meltzer, Walsh, Traylor, & Westin, 2012), to confirm delayed sleep timing at baseline (Criterion D), and to monitor changes across treatment. Motor activity (i.e., activity frequency) for the hour after awakening was also scored to confirm compliance with morning activity.

### Sleepiness and Fatigue

Sleepiness and fatigue are distinct daytime consequences of inadequate adolescent sleep (Short, Gradisar, Lack, & Wright, 2013). The Pediatric Daytime Sleepiness Scale (PDSS) is an 8-item self-report scale of daytime sleepiness (e.g., "*How often do you fall asleep or feel drowsy in class*?"; 0="*Never*", 4= "*Always*"; Drake et al., 2003). Total scores range from 0-32, with higher scores indicating higher sleepiness. Drake and colleagues (2003) reported good internal consistency (Cronbach $\alpha = 0.80$); however, reliability was lower in the current study (Cronbach $\alpha = 0.57$).

The Flinders Fatigue Scale (FFS) is a seven item self-report measure of global fatigue (e.g., "*Was fatigue a problem for you?*"; 0="*Not at all*", 4="*Extremely*"; Gradisar et al., 2007). Total scores range from 0-31, with higher scores indicating greater fatigue. The FFS had good reliability (Cronbach $\alpha = 0.86$).

Adolescents provided a daily rating of morning alertness, using a 5-point Likert scale with pictorial stimuli (e.g., *"which face best describes how you are feeling one hour after waking up in the morning"*; Maldonado, Bentley, & Mitchell, 2004). Scores ranged from 1-5, with higher scores indicating higher alertness.

### Functional Impairment

Individuals with DSWPD report lifestyle impairment (Rajaratnam, Licamele, & Birznieks, 2015). The Sheehan Disability Scale (SDS) is a 5-item measure of functional impairment (e.g., "*To what extent has your sleep pattern disrupted your school work*"; 0="*Not at all*", 10="*Extremely*"; Sheehan et al., 1996). Participants also reported the number of missed and under-productive days/week (0-7). Total scores range from 0-47, with higher scores indicating more impairment. The SDS had adequate reliability (Cronbach $\alpha = 0.71$).

### Portable Light Devices

Re-Timers (i.e., glasses with two LED lights per eye; Re-time Pty Ltd., Adelaide, Australia) were the light source for LT. Adolescents received either commercially available green light (~507nm) glasses on the highest setting (~112 lux per diode at 2cm distance), or Re-Timer frames, altered to emit red light (~643nm), on the lowest setting (~54 lux per diode at 2cm distance; spectrometer: Ocean Optics, Florida, USA; lux meter: A.P.C.S., NSW, Australia). As the human circadian rhythm is least sensitive to long wavelengths of light (Wright et al., 2004), red light was used as a placebo (Saxvig et al., 2014). All participants were asked to draw curtains and blinds in the home during LT, reducing ambient light and isolating light emitted from the devices.

### Morning Activity

Adolescents in the morning activity conditions were asked to complete 30-60mins of mild physical activity during LT. Physical activity involved playing a motion-sensing videogame (e.g., XBox Kinect, Nintendo Wii, Playstation Move), whilst standing upright. This modality was chosen, as videogames are played indoors (i.e., not weather-affected) and adolescents were anticipated to enjoy this activity. Active videogames increase heart rate, oxygen consumption and energy expenditure in adolescents, relative to sedentary videogames (Graves, Stratton, Ridgers, & Cable, 2008; Peng, Lin, & Crouse, 2011) and are more enjoyable relative to walking/ jogging (Graves et al., 2010). Adolescents in the sedentary conditions remained seated, watching TV for 30-60mins. It was anticipated that adolescents received the same amount of light emitted from the television and thus, screen light was controlled across conditions.

## Procedure

Recruitment occurred from July 2014 to December 2016. 83 adolescents underwent an assessment with a clinical, registered or provisional psychologist, consisting of the CSHI, 7-day sleep diary and questionnaires. Adolescents wore an actigraphy wristwatch and completed a sleep diary and questionnaires between the assessment and first treatment session (Gradisar et al., 2014). DSWPD diagnosis was confirmed at a consensus meeting between therapists and the clinic supervisor (MG). Participants deemed eligible were randomly allocated into one of four treatment conditions, using a block randomisation schedule. Sleep diaries and questionnaires were completed across treatment and at 1- and 3-month follow-up. An abridged version of the CSHI was administered at 3-month follow-up to re-assess DSWPD diagnosis.

Treatment involved three, weekly 50-minute sessions. The first session consisted of psychoeducation about circadian rhythms, instructions to reduce evening light 2hr prior to bed and plans for morning LT. Adolescents were instructed to sleep-in until their natural wake time to begin treatment (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014). Adolescents wore the portable light glasses for 30-60mins immediately after rising from bed each day and commenced LT 30mins earlier each subsequent day, until they reached a wake-up time of 06:00 (Gradisar et al., 2014). If adolescents reached this target time, they ceased LT and sleep patterns were stabilised, with a wake-up time no later than 07:30am. Adolescents were instructed to complete their allocated morning activity for the same duration and at the same time as LT. To prevent relapse following cessation of LT, participants were instructed to continue to minimise evening light, avoid sleeping in and seek ambient light after awakening (Gradisar et al., 2014).

## Statistical Analyses

To minimise the potential impact of missing data (~25% from pre-treatment to 3-month follow-up), Linear Mixed Modelling (LMM) was used to analyse data. Effect sizes (Cohen's *d*) were calculated to establish the magnitude of within- and between-subjects differences. Cohen's *d* was calculated as $d=M_1-M_2/(SD_{pooled})$. Treatment effects did not differ based on medication use or psychological co-morbidity and therefore were not controlled for.

# Results

## Treatment Compliance

There were no significant differences in self-reported duration of daily LT between groups ($F(3,50)= 0.146$, $p=0.93$). However, duration of use reduced between treatment week one (M=32min, SE=2) and two (M=31min, SE=2) to week three (M=15min, SE=2, $F(2,49)= 20.14$, $p<0.001$). This is likely due to adolescents reaching a 6am wake-up time and ceasing use of the light glasses. The duration of LT was in accordance with previous randomised controlled trials and clinical guidelines (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014).

There were no between group differences for self-reported daily activity duration ($F(3,52)= 0.969$, $p=0.41$). Activity duration also reduced from treatment week one (M=27min, SE=2) and two (M=27min, SE=2) to week three (M=16min, SE=2, $F(2,49)= 8.247$, $p=0.001$). Motor activity for the hour after awakening showed a small decrease in activity from baseline across the first two weeks of treatment, for the Green BLT + Sedentary group ($d=-0.31$). However, changes in activity frequency for the Red LT + Sedentary ($d=-0.13$), Green BLT + Activity ($d=-0.06$) and Red LT + Activity groups ($d=0.12$) were negligible, and there were no significant between-group differences, $F(3,29)= 1.62$, $p=0.206$.

These findings suggest the experimental manipulation did not lead to a meaningful increase in physical activity.

**Treatment**

Figures 4.2 to 4.4 show changes in sleep diary sleep, actigraphy sleep timing and daytime functioning, respectively. Descriptive statistics are presented in full in Appendix A. Contrary to predictions, interaction effects (group*time) for all outcome variables were not statistically significant ($p$>0.05). However, main effects of time were found for all variables, except bedtime and WASO.

From pre- to post-treatment, across all groups, adolescents demonstrated significant improvements for SOL, SOT and WUT, and a 47min improvement in TST (Figure 4.2). Actigraphy defined SOT and WUT also showed significant improvement (Figure 4.3; i.e., 39mins, 43mins, respectively). In terms of daytime functioning, morning alertness, daytime sleepiness, fatigue and functional impairment improved from pre- to post-treatment (Figure 4.4).

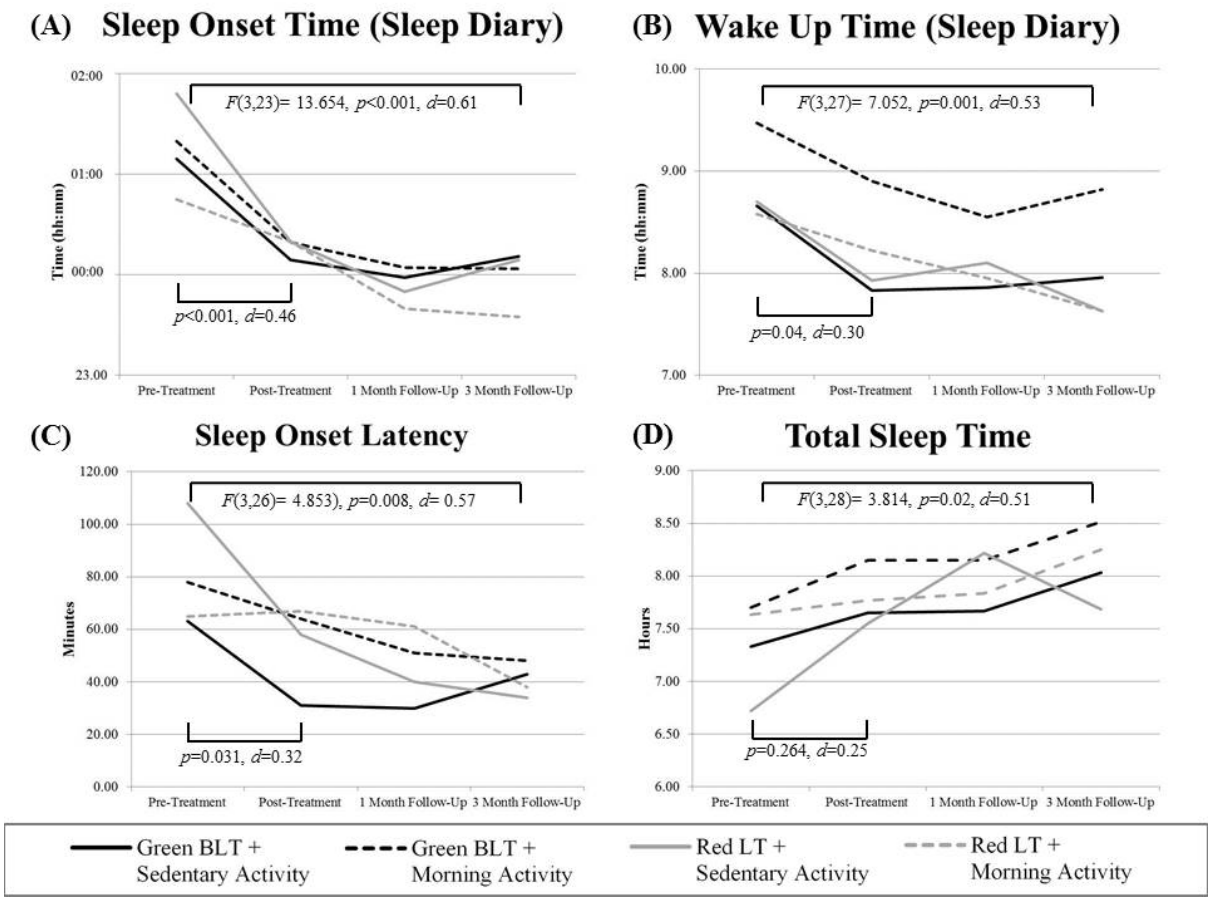


*Figure 4.2* Mean school night sleep diary (A) sleep onset time, (B) wake-up time, (C) sleep onset latency and (D) total sleep time, across treatment and follow-up. Error bars were excluded as there were no between-group differences and inclusion led to visual overcrowding.

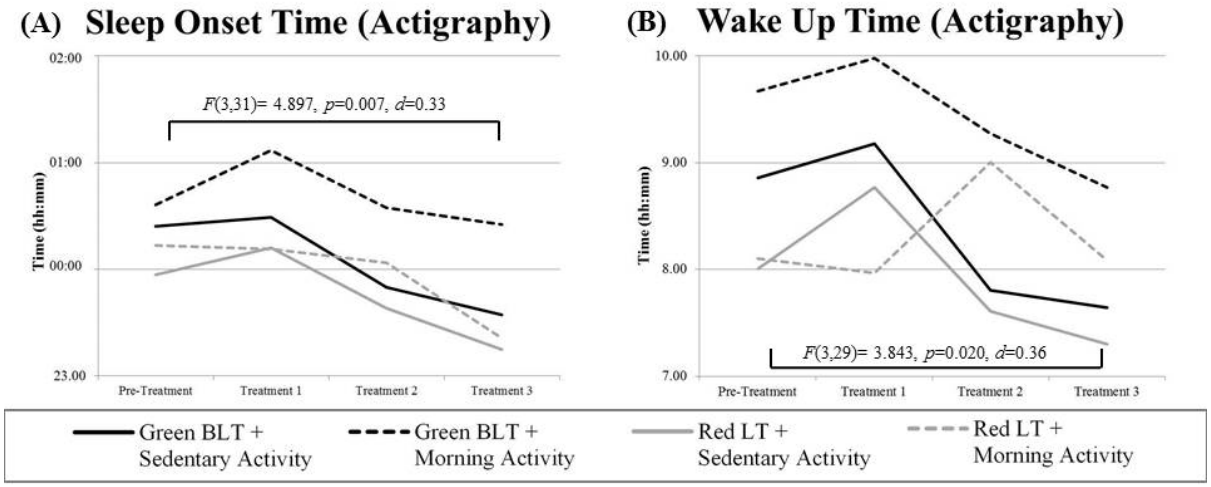


*Figure 4.3* Mean actigraphy school night (A) sleep onset time and (B) wake-up time for treatment groups across treatment. Error bars were excluded as there were no between-group differences and inclusion led to visual overcrowding.

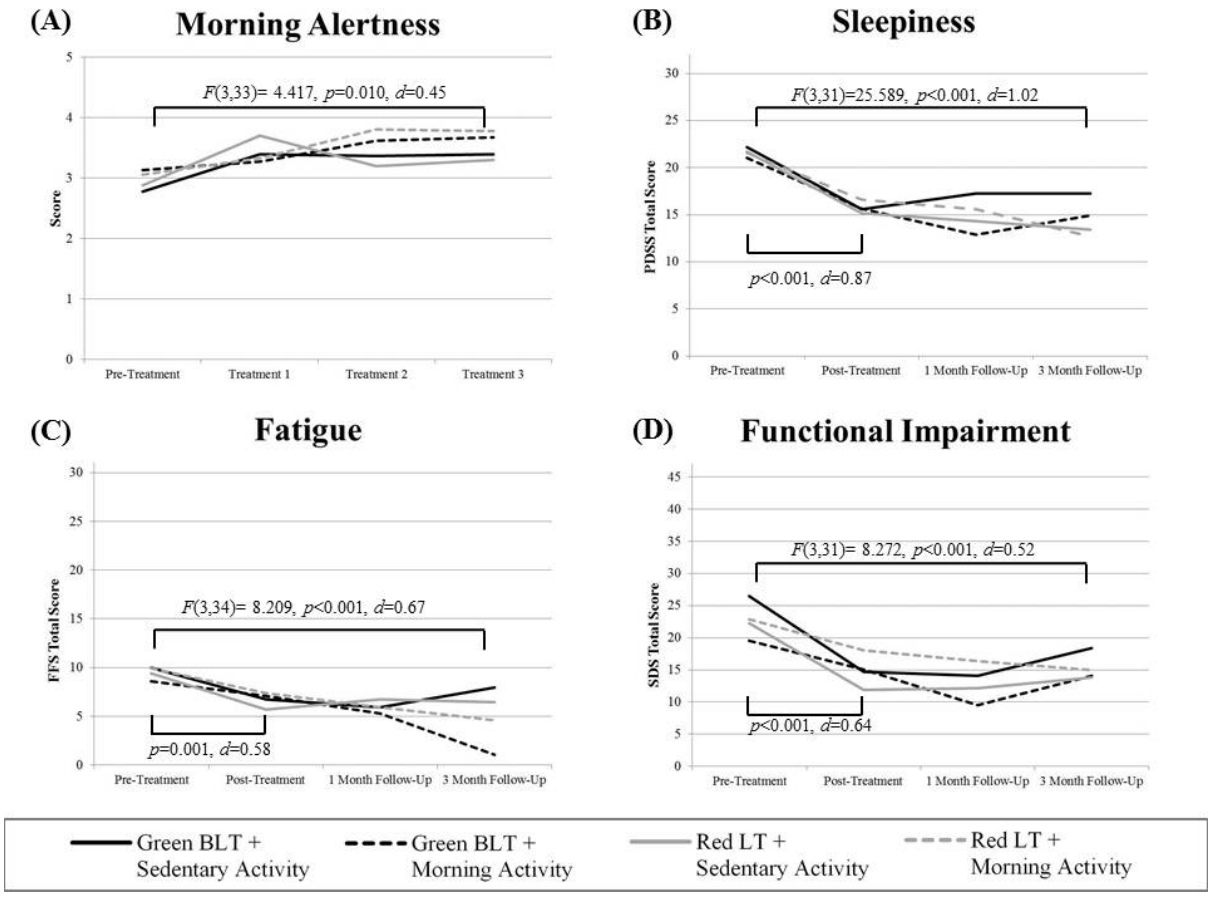


*Figure 4.4* Mean (A) morning alertness across treatment and mean (B) daytime sleepiness, (C) fatigue and (D) functional impairment across treatment and follow-up. Error bars were excluded as there were no between-group differences and inclusion led to visual overcrowding.

**Long-Term Outcomes**

Adolescents maintained improvements for SOL, SOT, WUT, TST, sleepiness, fatigue and impairment at follow-up[7]. Forty percent of participants (*N*=24) completed a sleep diary, questionnaires and abridged clinical interview at the 3-month follow-up[8]. There was no significant difference in proportion of DSWPD diagnosis between the groups (Table 4.2). However, partial relapse was common, with many adolescents reporting DSWPD symptomology at follow-up.

[7] Effect sizes were calculated between pre-treatment and 3-month follow-up scores.
[8] Adolescents who had received additional treatment were not included in follow-up.

Table 4.2

*Proportion of Adolescents who Reported Symptoms of DSWPD at Follow-up.*

| | **Green BLT + SA (*n*=8)** | **Green BLT + MA (*n*=8)** | **Red BLT + SA (*n*=5)** | **Red BLT + MA (*n*=3)** | ***p*** |
|---|---|---|---|---|---|
| **DSWPD Criteria Met** | 25% | 25% | 0% | 0% | 0.399 |
| **Sleep-Onset Difficulties** | 38% | 38% | 40% | 33% | 1.00 |
| **Difficulty Awakening** | 50% | 88% | 60% | 33% | 0.288 |
| **Sleep Problem** | 43% | 38% | 40% | 0% | 0.596 |

38% of the sample required additional treatment (Table 4.3), with no between-group differences. Additional treatment consisted of further BLT (*n*=4), exogenous melatonin (*n*=3) or both (*n*=9), motivational interviewing (*n*=2), cognitive therapy (*n*=2), sleep restriction therapy (*n*=1), sleep hygiene (*n*=1) and behavioural modification for evening technology use (*n*=1).

Table 4.3

*Summary of Adolescents Requiring Additional Treatment.*

| **Received Additional Treatment Following:** | **Green BLT + SA (*n*=14)** | **Green BLT + MA (*n*=13)** | **Red BLT + SA (*n*=14)** | **Red BLT + MA (*n*=15)** |
|---|---|---|---|---|
| **Post-Treatment** | *n*=3 | *n*=2 | *n*=1 | *n*=5 |
| **1-Month Follow-Up** | *n*=1 | *n*=0 | *n*=4 | *n*=1 |
| **3-Month Follow-Up** | *n*=2 | *n*=1 | *n*=0 | *n*=1 |
| **Total** | 43%* | 23%* | 36%* | 47%* |

* $\chi^2(3, N = 56) = 0.18$, $p = 0.60$.

## Discussion

A randomised controlled design evaluated the efficacy of bright light therapy, delivered by portable LED light glasses, and morning activity for the treatment of adolescent DSWPD. Despite a lack of interaction effects, improvements were found across a range of night time and daytime variables (13 out of 15). Sleep timing was advanced and sleep quality and daytime functioning also improved, with these changes maintained at follow-up.

Given adolescents had experienced their sleep problem for ~3yrs, 5mo, these results suggest that brief chronobiological treatment can result in rapid improvements in adolescent's sleep and wellbeing.

**Light Therapy**

These findings add to evidence from previous RCTs of LT for adolescent DSWPD (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014). Experimental studies have provided convincing evidence for the superiority of short wavelengths of light (relative to long) in advancing the human circadian rhythm (Warman et al., 2003; Wright et al., 2004). However, clinical practice guidelines have called for the replication of experimental research findings in clinical samples, within less tightly controlled environments (Auger et al., 2015). Initial data (Saxvig et al., 2014) suggest the wavelength of LT, when administered in accordance with practice guidelines, may not significantly influence the outcome of treatment for adolescents with DSWPD. It is possible that changes in behavioural patterns alone (i.e., fixed earlier sleep-wake routine, evening light restriction) contributed to advance circadian timing (Dewald-Kaufmann, Oort, & Meijer, 2014; Saxvig et al., 2014; Sharkey, Carskadon, Figueiro, Zhu, & Rea, 2011) and/or that the three weekly sessions with a psychologist (including psychoeducation), in itself, provided benefit (Bowers & Clum, 1988). Saxvig and colleagues (2014) also suggested sleep pressure could drive phase advances; however, total sleep time increased across treatment in the present study, so evidence for this hypothesis is limited.

**Morning Activity**

This is the first study to investigate the efficacy of scheduled physical activity for the treatment of adolescent DSWPD. However, there was no objective evidence for increases in physical activity. Interactive motion-sensing video gaming has been considered more

enjoyable than traditional physical activity for young adults (i.e., walking, running, Graves et al., 2010). Although adolescents reported complying with morning activity, it is possible that physical activity was not of sufficient intensity or duration to have a therapeutic effect. Experimental studies have administered one 3hr pulse of moderate (Baehr et al., 2003; Buxton et al., 1997; Van Reeth et al., 1994) or high intensity (Youngstedt et al., 2002) exercise, which is impractical to implement in clinical practice. However, there is some evidence that shorter durations of high intensity exercise could be as effective (Buxton et al., 1997). Therefore, future studies could evaluate physical activity of a longer duration (e.g., 45-60min) and higher intensity (e.g., moderate-high intensity). Additionally, future clinical trials could evaluate more practical forms of physical activity (i.e., walking/ cycling to school).

**Treatment of DSWPD**

Although there is convincing evidence for the short-term efficacy of chronobiological treatments for DSWPD, partial treatment success and relapse are common (Abu-Salah & Auger, 2013; Saxvig et al., 2014). 38% of our sample required additional treatment by the 3-month follow-up. Therefore, although treatment produced meaningful improvements in sleep and functioning, it is clear more can be done.

**Limitations and Future Directions**

It is a limitation that measures of circadian timing were not taken (i.e., dim light melatonin onset, core body temperature nadir). Regardless, objective data from actigraphy suggest circadian timing may have advanced across treatment (Ancoli-Israel et al., 2003). Additionally, although participants were instructed to minimise natural light during LT, ambient light levels within participants' homes were not measured. Although this data might have elucidated why there were no differences between the green and red LT conditions, the clinical application of this data is questionable.

Future research could compare the relative efficacy of portable light devices with empirically established modes of bright light delivery (i.e., light boxes/ lamps; Auger et al., 2015). Alternatively, it would be worthwhile knowing whether the inclusion of an artificial light source (e.g., light box, lamp or glasses) in BLT is necessary. For example, dismantling research designs may help to elucidate what components of bright light therapy (e.g., evening light restriction, fixed earlier wake up times, post-awakening light therapy, wavelength of light therapy) drive improvements in sleep and daytime functioning for adolescents and young adults with DSWPD.

Researchers could also aim to identify 'risk factors' (e.g., DSWPD severity, low motivation to change, repetitive negative thinking) for poor treatment outcome. The addition of cognitive behavioural therapy (Danielsson et al., 2015) to LT shows promise. However, identifying "risk factors" for poor treatment outcome, might allow for more effective case-conceptualised treatment of DSWPD (i.e., treatment which focuses on the unique experience of the individual, Dudley, Kuyken, & Padesky, 2011). Further refinement of treatment components should be a priority, with particular importance placed upon measuring long term outcomes, including relapse.

**Summary**

In recent years, momentum is gaining for the evaluation of efficacious sleep treatments for adolescents. Findings from the present study add to existing literature supporting the use of light therapy for adolescent DSWPD (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). However, the wavelength of light administered did not influence treatment outcome. Therefore, it is unclear what components of light therapy are the "active ingredients" of treatment and dismantling studies may be needed. As adolescents did not meaningfully increase physical activity, we cannot yet draw

conclusions about the potential utility of morning physical activity. Importantly, results suggest that three sessions of light therapy alone may not be sufficient to produce long-term benefits for many adolescents. Consequently, a number of priorities for future research have been highlighted.

# Chapter 5

## Cognitive Performance in Adolescents with Delayed Sleep-Wake Phase Disorder: Treatment Effects and a Comparison with Good Sleepers.

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### Author Contributions

CR led study conceptualisation, design, recruitment, data collection, management and analysis, results interpretation and manuscript preparation. GM, NC and KB assisted with recruitment, data collection and manuscript preparation. BM assisted with data management and analysis. MG contributed to study conceptualisation, design, recruitment, data collection, results interpretation and manuscript preparation.

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Richardson, C., Micic, G., Cain, N., Bartel, K., Maddock, B. & Gradisar, M. (Under Review). Cognitive performance in adolescents with Delayed Sleep-Wake Phase Disorder: Treatment effects and a comparison with good sleepers. *Journal of Adolescence*.

## Abstract

The present study aimed to investigate whether adolescents with DSWPD have impaired cognitive performance and whether chronobiological treatment for DSWPD improves adolescents' sleep, daytime functioning and cognitive performance. Adolescents with DSWPD reported significantly later sleep timing ($d$=1.03-1.45), less total sleep time ($d$=0.82) and greater daytime sleepiness ($d$=2.66), fatigue ($d$=0.63) and impairment ($d$=2.41), compared to good-sleeping adolescents. However, there were no significant between-group differences (all $p$>0.05) in performance on the Operation Span ($\eta p^2$=0.043), Digit Span (forwards: $\eta p^2$=0.002, backwards: $\eta p^2$=0.003), Letter Number Sequencing ($\eta p^2$<0.001) (working memory) and Digit-Symbol Substitutions Tasks ($\eta p^2$=0.010) (processing speed). Adolescents with DSWPD went on to receive three weeks of light therapy. At three months post-treatment, adolescents with DSWPD reported significantly advanced sleep timing ($d$=0.56-0.65), more total sleep time ($d$=0.52) and improved daytime sleepiness ($d$=1.33), fatigue ($d$=0.84) and impairment ($d$=0.78). Performance on the Operation Span ($d$=0.46), Letter Number Sequencing ($d$=0.45) and Digit-Symbol Substitution tasks ($d$=0.57) also significantly improved.

## Introduction

Adolescence is the developmental period when sleep timing tends to become later (Crowley et al., 2007; Gradisar, Gardner, et al., 2011; Roenneberg et al., 2004). Adolescents are uniquely at risk of delayed sleep timing due to physiological (i.e., the delay and/or lengthening of the circadian rhythm, slower build-up of sleepiness; Gradisar & Crowley, 2013; Micic et al., 2015; Micic, Lovato, Gradisar, Burgess, et al., 2016) and psychosocial influences (i.e., increased number of school, work, extra-curricular and recreational activities to complete before bed, Carskadon, 2011). Additionally, adolescents typically strive for independence, with reduced parental influence over bedtimes contributing to later bed- and sleep-times and impaired sleep quality and daytime functioning (Gangwisch et al., 2010; Short et al., 2011).

It is unsurprising then, that some teenagers (~1-16%) develop a more severe form of this sleep problem, Delayed Sleep-Wake Phase Disorder (DSWPD; American Academy of Sleep Medicine [AASM], 2014; Lovato, Gradisar, et al., 2013; Saxvig et al., 2012). DSWPD is a circadian rhythm disorder, whereby the body clock of an individual (i.e., melatonin and core body temperature rhythms) is timed significantly later than the 24-hr social world (Micic, Lovato, Gradisar, Burgess, et al., 2016). Due to the severity of delayed circadian timing, DSWPD typically presents as difficulty initiating sleep and an inability to wake early in the morning, to fulfil daytime commitments (AASM, 2014). Many adolescents with DSWPD attempt to wake at a socially conventional time to attend school, during their subjective night (i.e., point of maximal sleepiness; Carskadon et al., 1998), which is associated with reduced alertness, slower reaction time, poorer working memory and impaired grip strength (Johnson et al., 1992; Wright, Hull, & Czeisler, 2002). Subsequently, DSWPD has been associated with a plethora of negative daytime consequences (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lovato, Gradisar, et al., 2013), and

ultimately, school lateness, absenteeism, dropout and poor academic performance (Dewald et al., 2010; Giannotti et al., 2002; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lack, 1986; Meijer et al., 2000; Saxvig et al., 2012). Therefore, sleep disturbance is likely a risk factor for poor school performance in adolescence (Beebe, 2011).

Impaired cognitive performance may explain the link between DSWPD and poor academic functioning. Although there is a convincing relationship between evening chronotype (i.e., late to sleep, late to rise) and impaired academic achievement (Preckel, Lipnevich, Schneider, & Roberts, 2011; Randler & Frech, 2009; van der Vinne et al., 2015), evidence for the relationship between an evening chronotype and cognitive performance is more mixed (e.g., Ritchie et al., 2017; Roberts & Kyllonen, 1999). However, the chronotype-performance relationship is likely influenced by the time of testing, with evening types most impaired in the morning (Goldstein et al., 2007; Hahn et al., 2012; Lara, Madrid, & Correa, 2014; Matchock & Mordkoff, 2009). Importantly, clinical samples are commonly excluded from such investigation (Preckel et al., 2011) and adolescents with DSWPD possibly require an evidence base to help their plight; thus highlighting an important gap in knowledge.

Another way in which clinically delayed sleep timing might impact upon adolescent's cognition is through reduced total sleep time (TST). Adolescents with DSWPD often experience restricted sleep due to a very late time of falling asleep and the need to rise early for school (Saxvig et al., 2012). Reduced sleep duration in adolescents (e.g., TST<8hr) has been associated with impaired working memory performance (Gradisar, Terrill, et al., 2008). More extreme experimental sleep restriction (time in bed=5hr) results in significant impairments in sustained attention, working memory and executive function (Lo et al., 2016), particularly in the morning (Agostini et al., 2017). Pertinent to adolescents with DSWPD, recovery sleep was not sufficient to rectify these impairments (Agostini et al., 2017; Lo et al., 2016). On the other hand, small advances in sleep timing and total sleep time, over two

weeks, resulted in improvements in some aspects of adolescent cognitive performance (i.e., visuospatial processing, Dewald-Kaufmann et al., 2013).

Although evening chronotype and reduced sleep duration have been associated with impairments in cognition, a deficit in cognitive performance between adolescents with DSWPD and good sleepers has not been confirmed to date. As such, the first aim of the present study was to compare the cognitive performance (i.e., processing speed, working memory) of adolescents with DSWPD with their good-sleeping counterparts.

Due to the significant impact that impaired cognition may have on adolescents with DSWPD (i.e., poor grades and attendance, school dropout), and that sleep has been identified as a modifiable risk factor for many conditions (e.g., depression; Clarke & Harvey, 2012), it is also important to know whether interventions for sleep improve cognitive performance. Randomised controlled trials evaluating light therapy + cognitive behaviour therapy, or light therapy + exogenous melatonin have shown clinically meaningful improvements in the sleep and daytime functioning of adolescents with DSWPD (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014). However, only one trial to date has measured changes in cognitive performance as a result of treatment (Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Following two weeks of treatment, adolescents improved their performance on measures of working memory, processing speed and executive functioning, maintaining sleep and daytime benefits at the 3-month follow-up (i.e., working memory, processing speed, sustained attention and verbal fluency).

Importantly, with only one study investigating whether cognitive performance improves alongside chronobiological treatment for DSWPD in adolescents, more trials are needed to confirm this single finding (Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus,

Vedaa, et al., 2013). Therefore, the second aim of the present study was to measure changes in adolescents' cognitive performance across treatment for DSWPD. Evidence for the efficacy of light therapy is building (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011) and as advancements in the delivery of light therapy occur (i.e., development of portable light glasses), this opens the door for novel treatments. For example, timed physical activity may be used as an adjunct to light therapy, to assist in phase advancing the human circadian rhythm (Richardson et al., 2017). We hypothesised that light therapy and timed morning activity will advance sleep timing and increase total sleep time, and consequently, improve adolescents' daytime functioning and cognitive performance across treatment and follow-up. If improvements in cognition occurred, we also aimed to shed light on whether advancement of sleep timing, extension of total sleep time, or improvements in daytime functioning (i.e., sleepiness) contributed to these improvements. Processing speed and working memory have been linked with academic attainment (Alloway & Alloway, 2010; Fry & Hale, 1996; Rindermann & Neubauer, 2004) and were previously found to be sensitive to improvements in sleep (Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). As such, processing speed and working memory were chosen as measures of cognitive performance in the present study.

## Method

### Participants

DSWPD participants were referred via GP referrals, advertisements in school newsletters, community notice boards or social media. 83 adolescents were assessed by a trained psychologist, and 63 met International Classification of Sleep Disorders-3rd Edition (ICSD-3; AASM, 2014) criteria for DSWPD. Pre-treatment cognitive performance measures were completed by 56 adolescents diagnosed with DSWPD and 54 entered the randomised

controlled trial. However, as Autism Spectrum Disorder (ASD) has been linked with altered neuropsychological functioning (Minshew, Goldstein, & Siegel, 1997), three adolescents diagnosed with ASD were excluded from final analyses. Therefore pre-treatment data from 53 adolescents were included in between-group comparisons (mean= 15.68±2.1 y, 62% f) and 51 adolescents in treatment analyses (mean= 15.7±2.1 y, 61% f). Adolescents were randomly allocated to receive Green Bright Light Therapy (BLT) and Sedentary Activity (SA), Green BLT and Physical Activity (PA), Red LT and SA or Red LT and PA. However, grouped data were collapsed as there were no significant interaction effects across time (Richardson, Cain, et al., Under Review).

370 adolescents, recruited from the general public via advertisements in school newsletters, community notice boards and social media, completed an online screening questionnaire to assess good sleeper criteria. Inclusion criteria were: self-reported mean sleep onset latency (SOL) ≤30 mins, wake after sleep onset (WASO) ≤20 mins, total sleep time (TST) ≥eight hours, weekday-to-weekend wake-up time discrepancy ≤two hours, no difficulty falling asleep, staying asleep or unrefreshing sleep and no co-morbid psychiatric, physical or sleep condition. Forty adolescents, aged 13-24, met good sleeper criteria and were included in data analysis (mean= 15.9±2.4 y, 75% f). Participant characteristics are summarised in Table 5.1 (see Results).

The trial was registered with the Australian and New Zealand Trials Registry (ACTRN12614000308695) and was approved by the Southern Adelaide Clinical Human Research Ethics Committee and the Social and Behavioural Research Ethics Committee.

## Materials and Apparatus

### Clinical Sleep History Interview (CSHI).

A revised semi-structured interview (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011) was used to diagnose Delayed Sleep-Wake Phase Disorder (AASM, 2014) as per ICSD-3 criteria. The CSHI elicited information pertaining to: i) difficulty initiating sleep and awakening at a desired time (Criterion A), ii) symptom duration (i.e., ≥three months, Criterion B) and iii) other sleep, psychological and medical conditions (i.e., differential diagnosis, Criterion E).

### Sleep Diary.

Participants completed a daily sleep diary, measuring sleep timing (sleep onset time [SOT] and wake-up time [WUT]) and total sleep time (TST), via a password protected website. As adolescents with DSWPD are likely to have delayed and truncated sleep on school nights (Saxvig et al., 2012), sleep parameters were averaged weekly, for school nights (Sun-Thurs) and weekend nights (Fri-Sat) separately.

### Wrist Actigraphy.

Participants wore a MicroMini Motionlogger wrist actigraphy monitor (Ambulatory Monitoring Inc., Ardsley, NY) following their initial assessment and throughout treatment, to confirm and monitor changes in sleep timing. The actigraphy watch collected movement data in one minute epochs using zero-crossing mode. Sleep onset and offset time were scored using the Sadeh algorithm (AMI, Action 4, Ambulatory Monitoring Inc, Ardsley, NY), which has been validated for adolescents (Meltzer et al., 2012; Sadeh et al., 1994). Actigraphy data were also averaged separately for school and weekend nights.

**Daytime Functioning.**

Adolescents with DSWPD report daytime sleepiness, fatigue and functional impairment (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Micic et al., 2015) and these are said to be related to young people's school performance (Dewald et al., 2010). The Paediatric Daytime Sleepiness Scale (PDSS) is an 8-item self-report scale of daytime sleepiness (e.g., "*How often do you fall asleep or feel drowsy in class*?"; Drake et al., 2003), with total scores ranging from 0-32 (higher scores indicate higher sleepiness). The Flinders Fatigue Scale (FFS) is a 7-item self-report measure of global fatigue (e.g., "*Was fatigue a problem for you*?"; Gradisar et al., 2007), with total scores ranging from 0-31 (higher scores indicate greater fatigue). The Sheehan Disability Scale (SDS) is a 5-item measure of the disruption to one's work/school, social and family/home lives caused by symptoms of their sleeping pattern (e.g., "*To what extent has your sleep pattern disrupted your work/school work*"; Sheehan et al., 1996), with total scores ranging from 0-47 (higher scores indicate more lifestyle disruption and impairment). The PDSS (Cronbach $\alpha = 0.87$), FFS (Cronbach $\alpha = 0.90$) and SDS (Cronbach $\alpha = 0.88$) had good reliability in the current study and were completed by the DSWPD and good sleeper groups at baseline. Sleepiness, fatigue and impairment were also monitored across treatment.

**Cognitive Performance.**

***Working Memory.***

*Operation Span Task (Op Span).* In the computerised Op Span, adolescents were required to remember a sequence of words, whilst alternately solving simple arithmetic operations (i.e., dual-task). A single, one syllable word (e.g., seal, joke) was presented on the screen for one second, before being replaced by a simple mathematical equation (e.g., 40+50=80, 14-7=7). Once participants had indicated whether the mathematical equation was

correct or incorrect, another word was presented, and so on. At the end of the trial, adolescents were asked to type all words into a text box in the order they were presented. The first trial consisted of two words and two equations, with each subsequent trial including another word and equation. There were five trials in total. Adolescents were awarded one point for every word recalled in the correct order. The Op Span score was obtained by dividing the number of words correctly recalled, by the total number of words presented (i.e., 20). In this task, memory processing (i.e., equation solving) could be neglected to optimise memory storage (i.e., word remembering), although adolescents were not informed of this. Therefore, Op Span scores were only included in data analysis if adolescents scored ≥85% on maths accuracy. The Op Span Task was revised by Gradisar and colleagues (2008), so that it was appropriate for use with Australian adolescents and the same stimuli were used in the current study.

*Digit Span.* The computerised Digit Span task, was based on the Digit Span subtest of the Wechsler Intelligence Scale for Children- 4$^{th}$ Edition (WISC-IV; Wechsler, 2003). The test required adolescents to remember a series of numbers, which were presented on the screen one at a time, for one second each. At the end of each "forward" trial, participants were asked to type the sequence of numbers into a textbox, in the order they were presented. The initial sequence contained three digits, with subsequent trials increasing in difficulty (i.e., sequence length). The "backward" subtest required adolescents to remember a sequence of numbers, instead, typing the digit sequence into the text box in the reverse (backwards) order. For both the forward and backwards subtest, the longest digit span correctly recalled was calculated (max=10).

*Letter Number Sequencing (LNS).* The computerised LNS task required adolescents to hold an unordered sequence of letters and number (i.e., 4-B-3) in their conscious awareness, before performing a mental operation (i.e., ordering the numbers numerically, followed by

the letters alphabetically, i.e., 3-4-B; Wechsler, 2003). Letter and number stimuli were presented on the screen, one at a time, for one sec. Sequences initially consisted of three letters/ numbers and gradually increased in difficulty until eight letters/ numbers were presented. Participants earned one point for each correctly ordered sequence. Incorrect responses were scored zero. As there were 24 trials, total scores ranged from 0-24.

***Processing Speed.***

*Digit-Symbol Substitution.* The computerised Digit-Symbol Substitution Task was based on the coding subtest of the WISC-IV (Wechsler, 2003). In this computerised version, participants were presented with a key denoting matched symbols and numbers. Adolescents had two minutes in which they matched a grid of symbols with their corresponding number, as quickly as possible. Total scores were calculated by subtracting the number of incorrectly matched digit-symbol pairs from the number of correctly matched pairs (max=135).

*Perception of Cognitive Performance***.** Following each cognitive performance task, participants were asked to rate their *performance relative to other adolescents their age*, on a 5-point Likert scale (i.e., 1= *Far below average*, 5= *Far above average*). Higher scores indicate perception of better performance, irrespective of objective performance.

**Treatment Materials.**

Adolescents received light therapy via portable light glasses (Re-Time Pty Ltd., Adelaide, Australia). The Re-Timer device is worn like a pair of spectacles and has two LED lights per eye, built into the frame. Adolescents received either green (~507nm, ~112 lux per diode at 2cm distance, commercially available) or red (~643nm, ~54 lux per diode at 2cm distance, placebo) light therapy (spectrometer: Ocean Optics, Florida, USA; lux meter: A.P.C.S., NSW, Australia; Wright et al., 2004). However, as wavelength of light did not

impact treatment outcome, light therapy groups were collapsed for the present study (Richardson, Cain, et al., Under Review).

It has been suggested that exercise could supplement light therapy to enhance therapeutic effects (Richardson et al., 2017). Therefore, adolescents were also randomised to complete either sedentary activity (i.e., sitting watching TV) or mild physical activity (i.e., standing, playing motion-sensing video games) during light therapy. However, as there was no meaningful difference in physical activity between the two conditions, or any differential effect on treatment outcome, activity condition was also collapsed for the present study (Richardson, Cain, et al., Under Review).

**Procedure**

Adolescents with DSWPD attended an initial assessment, conducted by a psychologist, which included the semi-structured interview (CSHI), sleep diary and self-report measures of daytime functioning (PDSS, FFS, SDS). At the time of assessment, adolescents completed a battery of cognitive performance tasks. All cognitive performance tasks were computerised to ensure standardisation (Presentation, Neurobehavioral Systems, Inc., California, USA), and were administered using a 21.5" iMac computer. As adolescents with delayed circadian timing are most likely to be cognitively impaired in the morning (Goldstein et al., 2007; Hahn et al., 2012; Lara et al., 2014; Matchock & Mordkoff, 2009; Ritchie et al., 2017; Thorpy et al., 1988), the cognitive performance battery was completed in the morning (i.e., prior to 1pm). Participants wore an actigraphy watch and completed an online 7-day sleep diary between the assessment and first treatment session. DSWPD diagnosis was confirmed with other sleep psychologists and the clinic supervisor at a consensus meeting following each assessment.

Adolescents and young adults from the general public completed an online screening questionnaire (i.e., modified CSHI, self-reported estimates of sleep) to determine their eligibility for the study as a good sleeper. After confirmation of eligibility, adolescents attended a meeting, where they collected an actigraphy watch and log-in credentials to access the online 7-day sleep diary. After recording their sleep at home for one week, adolescents returned to the sleep laboratory to complete daytime functioning measures (i.e., PDSS, FFS, SDS) and the battery of computerised cognitive performance measures. Forty good-sleeping adolescents were included in the final analyses.

After completion of the baseline measures, adolescents with DSWPD commenced sleep treatment. Sleep treatment involved psychoeducation about circadian rhythms, instructions to minimise evening light in the two hour before bed and plans to implement post-awakening light therapy. Adolescents slept in on the first day of therapy and were instructed to wear the portable light glasses for 30-60 mins, immediately after rising from bed. Participants were instructed to complete their allocated morning activity (i.e., TV watching, motion sensing video gaming) for the same duration as light therapy. Participants rose from bed and commenced light therapy and activity 30min earlier each subsequent day. Once a 6:00am wake-up time was reached, participants ceased light therapy and maintained a wake-up time no later than 7:30am. Participants completed sleep diaries and wore an actigraphy watch throughout treatment and completed measures of daytime functioning and cognitive performance again at post-treatment. To determine the long term impact of treatment, adolescent's sleep, daytime functioning and cognitive performance were measured again at 3-months post-treatment.

**Statistical Analyses**

Independent-samples t-tests and Analysis of Covariance (ANCOVA) were used to compare the sleep, daytime functioning and cognitive performance of adolescents with DSWPD, with good sleepers. Effect sizes were calculated to establish the magnitude of between-subjects differences. Cohen's *d* was calculated as *d*=M1-M2/(SDpooled). To minimise the potential impact of missing data (~21% from pre-treatment to 3-month follow-up), Linear Mixed Modelling (LMM) was used to analyse treatment results. Cohen's *d* was also calculated to establish the magnitude of within-subjects differences. Change variables for sleep, daytime functioning and cognitive performance were calculated as the difference between pre- and post-treatment values, so that positive values reflected improvement. Relationships between the change in sleep, daytime functioning and cognitive performance, were investigated using Pearson product-moment correlations.

## Results

As an indication of when cognitive testing was completed in relation to natural circadian timing, good sleepers woke on weekends at 7:47am±57min, whereas, the DSWPD group woke at 9:35am±117min. The first cognitive testing occurred at 10:45am±83min for the good sleepers and at 11:25am±107min for the DSWPD group. The difference in testing time approached significance, $t(91)=-1.93$, $p=0.06$, $d=0.41$. Although not statistically significant, there were small relationships between time of cognitive testing in relation to circadian timing (i.e., discrepancy between weekend wake-up time and time of cognitive testing) and cognitive performance for the DSWPD group ($r=0.10$ to $0.20$, $p>0.05$). Consequently, ANCOVAs were used to compare cognitive performance between the DSWPD and good sleeper groups, whilst controlling for the discrepancy between weekend wake-up time and time of cognitive testing. DSWPD participants who entered the treatment

study completed cognitive testing at a similar time at pre- (M= 11.25am, SE= 15min) and post-treatment (M=11.27am, SE= 17min). However, the 3-month follow-up was completed significantly earlier (M=10.29am, SE=14min), $F(2,25)= 7.82$, $p=0.002$, $d=0.54$. Therefore, time of cognitive testing, in relation to circadian timing (i.e., weekend wake-up time) was controlled for in final analyses.

**Do adolescents with DSWPD have impaired cognitive performance?**

Adolescents with DSWPD had significantly later sleep timing, worse sleep quality and impaired daytime functioning, relative to the good sleepers (see Table 5.1). However, cognitive performance on all measures of working memory and processing speed did not differ significantly between the groups (see Table 5.1). There was a small-to moderate between-group difference in Op Span (i.e., the task which placed a higher cognitive load on adolescents). However, this difference did not reach statistical significance. Adolescents with DSWPD rated their performance on three of the four cognitive tasks significantly worse than the good sleepers (see Table 5.1), which may indicate a negative expectations bias towards the consequences of poor sleep (Courtauld, Notebaert, Milkins, Kyle, & Clarke, 2017).

Table 5.1

*Sleep and Daytime Functioning of Good-Sleeping Adolescents and Adolescents with DSWPD.*

| | **Good Sleepers Mean±SD** | **DSWPD Mean±SD** | ***p*** | ***d*** |
|---|---|---|---|---|
| **Demographics** | | | | |
| Age | 15.9±2.4 | 15.7±2.1 | 0.64 | 0.08 |
| Socio-Economic Status∗ | 6.65±2.57 | 5.73±2.85 | 0.12 | 0.34 |
| **Sleep Diary (School Nights)** | | | | |
| SOT (hh:mm±mm) | 23:01±57 | 01:10±112 | **<0.001** | **1.45** |
| WUT (hh:mm±mm) | 07:26±48 | 08:48±102 | **<0.001** | **1.03** |
| SOL (mins) | 18±12 | 72±69 | **<0.001** | **1.09** |
| WASO (mins) | 6±6 | 17±37 | 0.06 | 0.42 |
| TST (hrs) | 8.32±0.66 | 7.37±1.50 | **<0.001** | **0.82** |
| **Actigraphy (School Days)** | | | | |
| SOT (hh:mm±mm) | 22:52±55 | 00:26±100 | **<0.001** | **1.17** |
| WUT (hh:mm±mm) | 07:24±48 | 08:46±109 | **<0.001** | **0.98** |
| **Daytime Functioning** | | | | |
| Daytime Sleepiness (PDSS) | 10.93±3.25 | 21.73±4.73 | **<0.001** | **2.66** |
| Fatigue (FFS) | 6.68±4.64 | 9.45±4.16 | **0.006** | **0.63** |
| Functional Impairment (SDS) | 5.00±5.97 | 23.50±9.09 | **<0.001** | **2.41** |
| **Academic Consequences of Sleep** | | | | ***phi*** |
| Poor Attention/ Concentration/ Memory | 10% | 94% | **<0.001** | **0.84** |
| Poor School Grades | 10% | 67% | **<0.001** | **0.57** |
| **Cognitive Performance**** | | | | |
| *Working Memory* | **Mean±SE** | **Mean±SE** | | $\eta p^2$ |
| Op Span | | | | |
| Score (0.00-1.00) | 0.78±0.03 | 0.71±0.03 | 0.11 | 0.043 |
| Digit Span | | | | |
| Forwards- Longest Span (0-10) | 6.67±0.15 | 6.58±0.15 | 0.69 | 0.002 |
| Backwards- Longest Span (0-10) | 5.87±0.17 | 5.74±0.18 | 0.62 | 0.003 |
| Letter Number Sequencing | | | | |
| Total Number Correct (0-24) | 14.30±0.40 | 14.35±0.41 | 0.94 | <0.001 |
| *Processing Speed* | | | | |
| Digit-Symbol Substitution | | | | |
| Score (0-135) | 56.84±2.48 | 53.72±2.52 | 0.39 | 0.010 |
| *Perception of Cognitive Performance* *(1=far below average, 5= far above average)* | **Mean±SD** | **Mean±SD** | | ***d*** |
| Op Span | 2.88±0.69 | 2.36±0.74 | **0.001** | **0.73** |
| Digit Span | 2.95±0.75 | 2.52±0.73 | **0.007** | **0.58** |
| Letter Number Sequencing | 2.64±0.63 | 2.53±0.72 | 0.44 | 0.16 |
| Digit-Symbol Substitution | 2.90±0.50 | 2.66±0.59 | **0.04** | **0.44** |

Note: ∗=Index of socio-economic advantage and disadvantage, decile (Pink, 2013).

**=Descriptive and inferential statistics for cognitive performance are adjusted, so that the effect of time of testing, in relation to circadian timing, and SES have been removed.

**Does cognitive performance improve following treatment?**

Figures 5.1-5.4 outline changes in actigraphy measured sleep timing across treatment and subjective sleep, daytime functioning and cognitive performance across, treatment and follow-up, respectively. Actigraphy defined SOT, $F(3,28)= 3.99$, $p=0.018$, $d= 0.29$, and WUT, $F(3,28)= 3.20$, $p=0.04$, $d= 0.25$, on school nights became significantly earlier across treatment (see Figure 5.1).

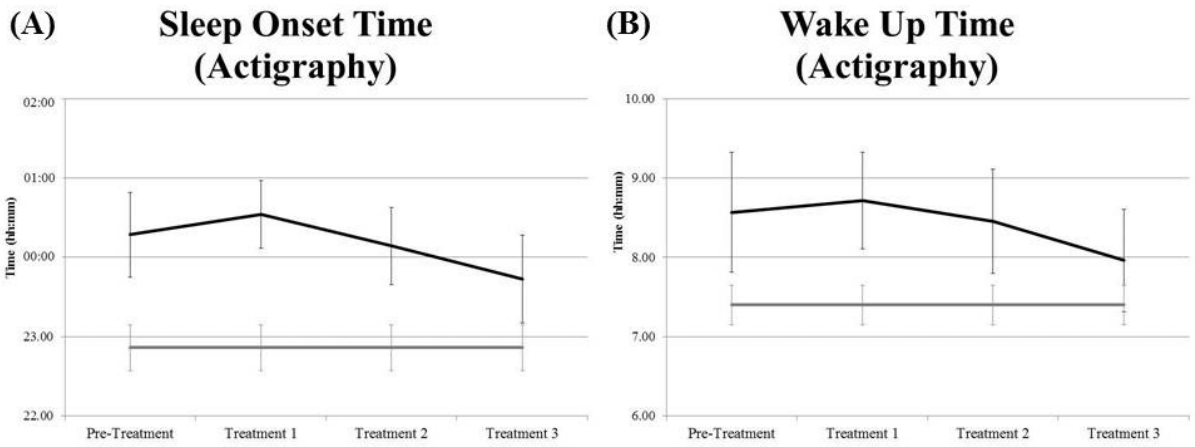


*Figure 5.1* Mean actigraphy defined school night (A) sleep onset time and (B) wake-up time for adolescents across treatment. Grey line indicates good sleeper adolescent mean at baseline. Error bars indicate 95% confidence interval.

Self-reported SOT, $F(2,26)=13.89$, $p<0.001$, $d=0.65$[9], and WUT, $F(2,26)= 8.42$, $p=0.001$, $d=0.56$, improved across treatment, and benefits were maintained at the 3-month follow-up. School night TST, $F(2,22)= 4.62$, $p=0.021$, $d=0.52$ also significantly improved across treatment and follow-up (see Figure 5.2). Daytime sleepiness, $F(2,32)= 37.03$, $p<0.001$, $d=1.33$, fatigue, $F(2,34)=11.70$, $p<0.001$, $d= 0.84$, and functional impairment, $F(2,30)=11.86$, $p<0.001$, $d=0.78$, showed similar patterns of improvement (see Figure 5.3).

[9] Effect sizes were calculated between pre-treatment and 3-month follow-up scores.

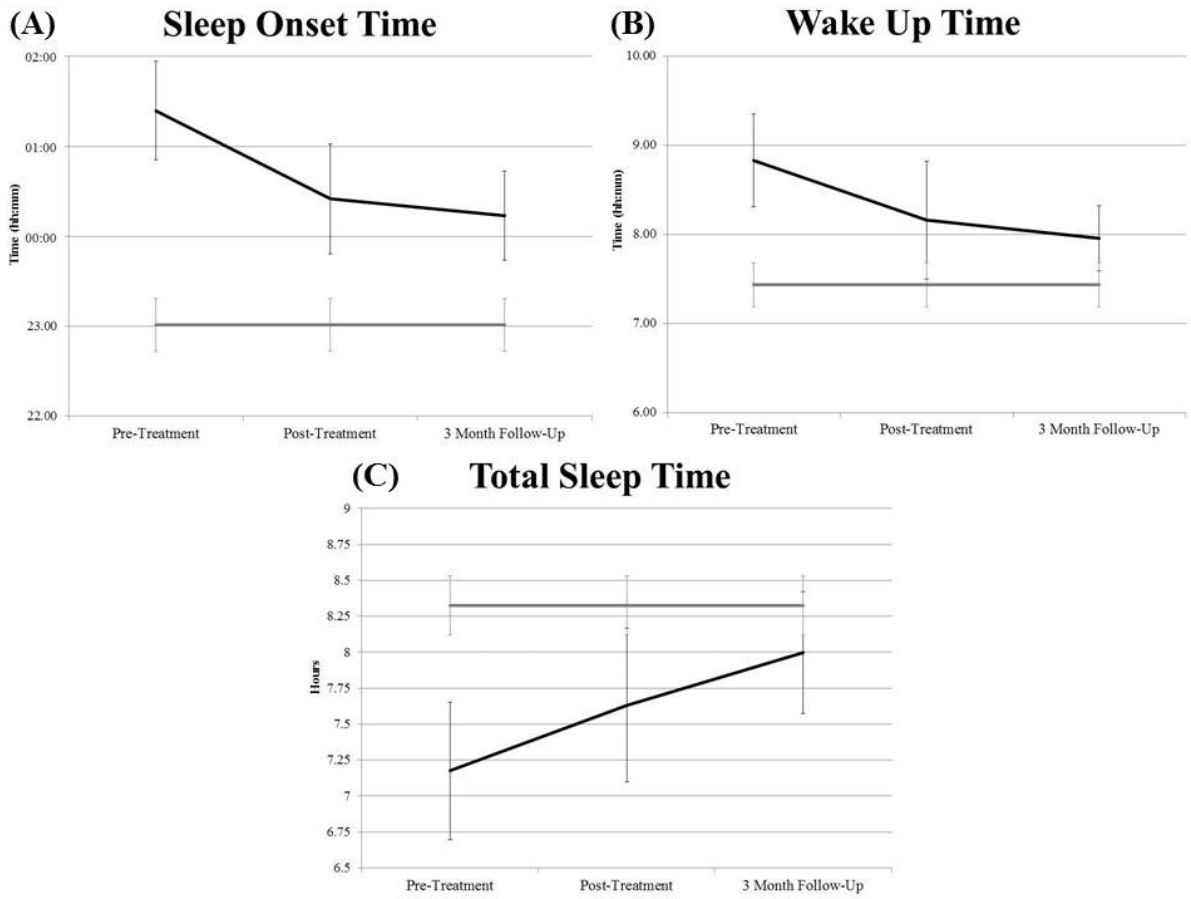


*Figure 5.2* Mean school night sleep diary (A) sleep onset time, (B) wake-up time and (C) total sleep time for adolescents across treatment and follow-up. Grey line indicates good sleeper adolescent mean at baseline. Error bars indicate 95% confidence interval.

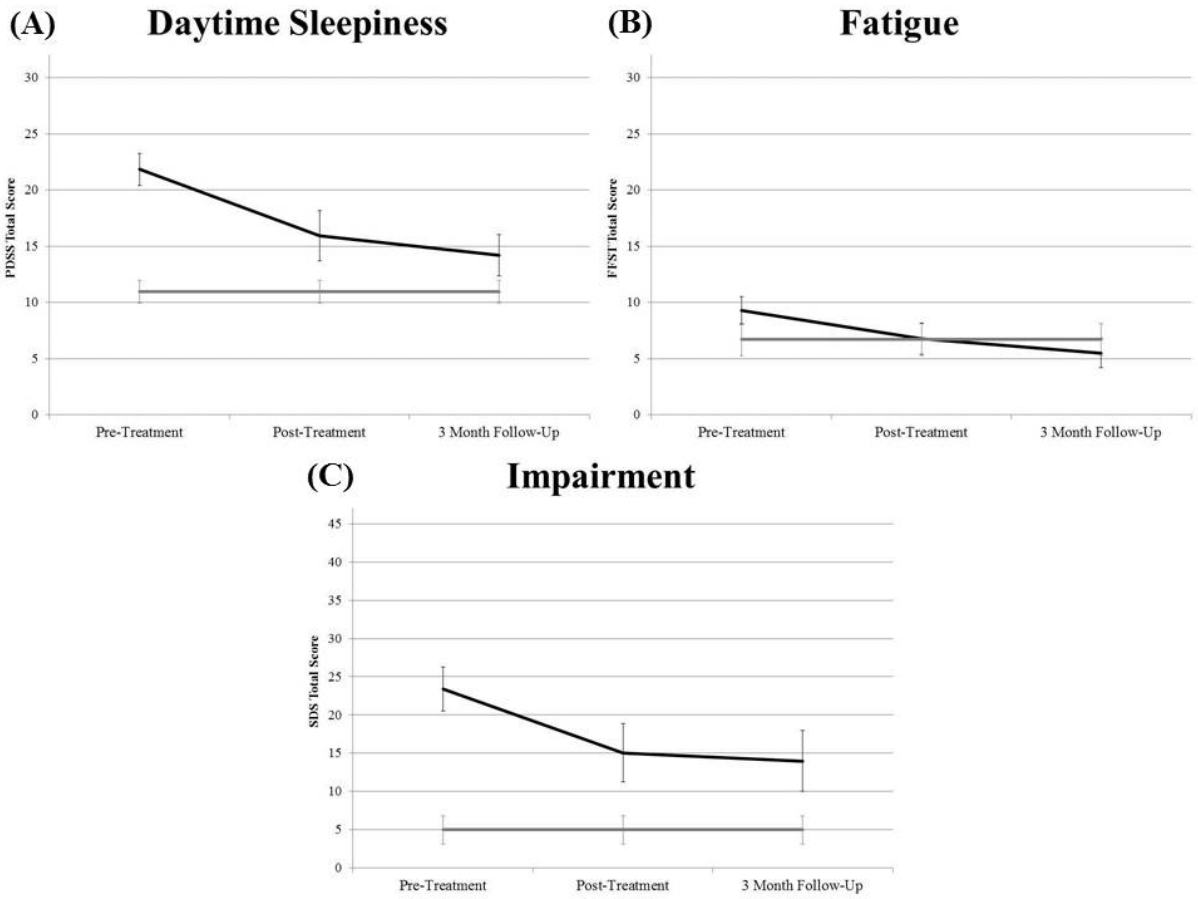


*Figure 5.3* Mean (A) daytime sleepiness, (B) fatigue and (C) functional impairment for adolescents across treatment and follow-up. Grey line indicates good sleeper adolescent mean at baseline. Error bars indicate 95% confidence interval.

Adolescents' working memory performance, as measured by Op Span, $F(2,23)=4.38$, $p=0.024$, $d=0.46$, and Letter Number Sequencing, $F(2,28)=5.69$, $p=0.008$, $d=0.45$, significantly improved across treatment and follow-up (see Figure 5.4). Improvement on Digit Span forwards approached significance, $F(2,23)=3.30$, $p=0.055$, $d=0.33$, whereas, performance on the backwards subtest did not significantly change over time, $F(2,28)=1.162$, $p=0.33$, $d=0.13$. Adolescents' processing speed, significantly improved across treatment and follow-up, $F(2,26)=11.23$, $p<0.001$, $d=0.57$.

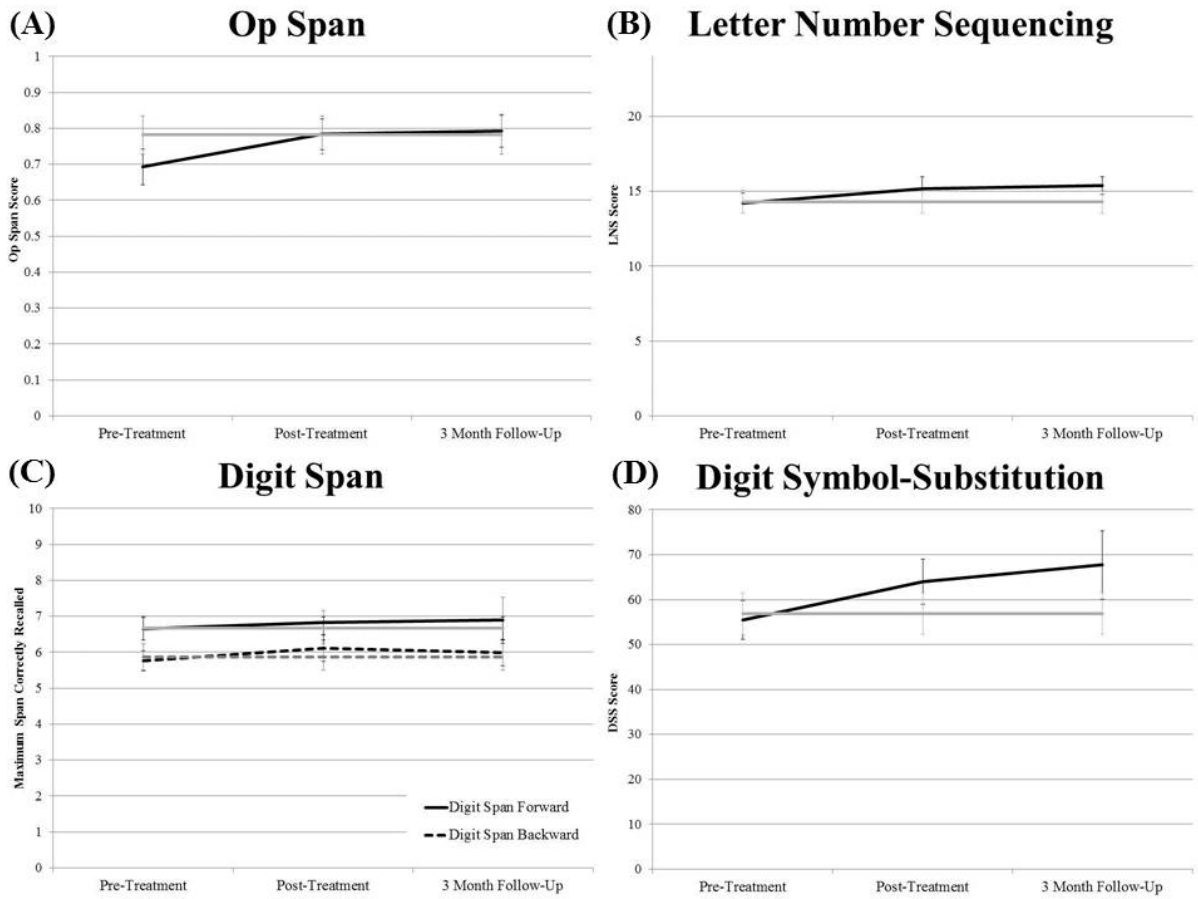


*Figure 5.4* Mean score on (A) Op Span, (B) Letter Number Sequencing, (C) Digit Span and (D) Digit-Symbol Substitution tasks for adolescents across treatment and follow-up. Grey line indicates good sleeper adolescent mean at baseline. Error bars indicate 95% confidence interval.

Adolescents' perception of their performance on Op Span, $F(2,30)= 2.13$, $p=0.136$, $d=0.06$, Digit Span, $F(2,34)=0.28$, $p=0.759$, $d=0.13$, Letter Number Sequencing, $F(2,33)=1.14$, $p=0.333$, $d=0.21$ and Digit-Symbol Substitution, $F(2,29)=2.75$, $p=0.081$, $d=0.29$, did not significantly change across treatment and follow-up.

**Relationship between sleep, daytime functioning and cognitive performance**

Relationships between the change in sleep, daytime functioning and cognitive performance are outlined in Table 5.2. There was limited evidence for a relationship between advancement in sleep timing (WUT) and improvements on the Op Span and Digit Span tasks (see Table 5.2). However, multiple moderate-to-strong relationships existed between changes in processing speed and changes in sleep onset time, total sleep time and daytime functioning.

Table 5.2

*Correlations between Changes in School Night Sleep, Daytime Functioning and Cognitive Performance, from Pre-treatment to Post-treatment.*

| | | | Working Memory | | | | Processing Speed |
|---|---|---|---|---|---|---|---|
| | | | Op Span | Digit Span Forward | Digit Span Backward | Letter Number Sequencing | Digit-Symbol Substitution |
| Sleep Timing | SOT (SD) | *r* | 0.11 | -0.14 | 0.33 | 0.16 | **0.44*** |
| | SOT (Act) | *r* | 0.05 | 0.33 | 0.09 | 0.17 | **0.65*** |
| | WUT (SD) | *r* | **0.43*** | -0.23 | 0.22 | 0.03 | 0.18 |
| | WUT (Act) | *r* | 0.19 | 0.02 | **0.50*** | 0.13 | 0.34 |
| | TST (SD) | *r* | -0.31 | 0.07 | 0.19 | 0.13 | **0.40*** |
| Daytime Functioning | Sleepiness | *r* | 0.06 | -0.06 | -0.23 | 0.04 | **0.38*** |
| | Fatigue | *r* | -0.12 | 0.14 | -0.23 | 0.12 | **0.43*** |
| | Impairment | *r* | 0.12 | -0.09 | -0.01 | 0.07 | **0.39*** |

Note: SD= Sleep Diary, Act= Actigraphy, *=$p<0.05$; **=$p<0.001$.

## Discussion

The present study is the first to investigate whether cognitive performance is impaired in adolescents with Delayed Sleep-Wake Phase Disorder (DSWPD), relative to their good-sleeping peers. Although between-group comparisons revealed adolescents with DSWPD had significantly later sleep timing, less sleep and worse daytime functioning, there were no significant differences between groups' working memory and processing speed performance. These findings contradict cross-sectional research which has shown that late chronotype (Goldstein et al., 2007; Hahn et al., 2012; Lara et al., 2014; Matchock & Mordkoff, 2009) and reduced sleep duration (Agostini et al., 2017; Gradisar, Terrill, et al., 2008; Lo et al., 2016) are associated with cognitive impairments.

DSWPD participants completed cognitive testing two hours later than their weekend wake-up time (which is a proxy indication of delayed circadian timing), therefore, it is possible that cognitive testing was conducted when circadian timing helped, rather than hindered performance (Goldstein et al., 2007). Posteriori analyses showed small correlations between the time of day of testing (in relation to sleep timing) and cognitive performance, therefore further investigation is warranted. Additionally, it is possible that performance on higher order or more ecologically valid tasks (i.e., tasks with increased cognitive complexity) could be impaired. As some preliminary evidence for this, the largest between-group effect size was observable on the Op Span Task (i.e., small-to-moderate effect), which required adolescents to complete two tasks simultaneously.

Another hypothesis used to explain a lack of cognitive impairment can be found in the field of adult insomnia, which is based on the hyperarousal model of insomnia (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012; Kyle et al., 2017; Riemann et al., 2010). That is, chronic hyperarousal may overcome daytime impairments of fatigue and sleepiness, allowing such individuals to perform similarly to good sleepers (Lovato, Lack, Wright, Cant, & Humphreys, 2013). If "insomnia" processes are also at play in DSWPD, as has been suggested (Richardson, Gradisar, & Barbero, 2016; Richardson, Micic, et al., Under Review), then hyperarousal may also explain the lack of evidence for acute cognitive impairment in adolescents with DSWPD. With such few studies within the field, replication is needed to confirm this hypothesis.

Despite a lack of objective evidence, adolescents with DSWPD subjectively rated their performance on the cognitive performance tasks significantly worse than the good sleepers, which may indicate a negative expectancy bias (Courtauld et al., 2017). In general, people tend to rate their performance as better-than-average (Alicke & Govorun, 2005). However, adolescents with DSWPD may hold strong beliefs about the consequences of poor

sleep (Harvey, 2002; Richardson et al., 2016), and thus, may anticipate impaired performance, despite a lack of confirmatory evidence.

Adolescents with DSWPD significantly advanced their sleep timing, increased their sleep duration on school nights, and improved their daytime functioning as a result of treatment. Performance on most working memory tasks (i.e., Op Span, LNS) and the Digit-Symbol Substitution task (processing speed) also improved across treatment and follow-up; however, perception of cognitive performance remained relatively stable. These findings lend further support to theory that cognitive "insomnia" processes (i.e., dysfunctional beliefs about the consequences or poor sleep, misperception of daytime performance) may also exist in DSWPD (Harvey, 2002; Richardson et al., 2016).

In terms of potential mechanisms underlying changes in cognitive performance, there was consistent evidence for moderate-to-strong relationships between improvements in sleep timing, total sleep time and daytime functioning and improvements in processing speed. These findings support empirical research which has shown processing speed is susceptible to sleep timing (Buckhalt, El-Sheikh, Keller, & Kelly, 2009), quality (Nader & Smith, 2015), duration (Cohen-Zion, Shabi, Levy, Glasner, & Wiener, 2016) and deprivation (Lo et al., 2016; Louca & Short, 2014), in paediatric samples. Importantly, these findings also add to evidence that sleep interventions can improve processing speed (Lim, Lo, & Chee, 2017; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), which may have important implications for educational psychologists looking for evidence based interventions to improve processing speed. In contrast, evidence for a relationship between sleep and working memory was modest. It is possible that bright light therapy itself had a direct alerting effect, which facilitated improvements in cognitive performance (i.e., rather than via changes in sleep; Chellappa et al., 2011; Lehrl et al., 2007).

Adolescent's performance on the Letter Number Sequencing and Digit-Symbol Substitution tasks in the present study showed similar improvements to those reported by Wilhelmsen-Langeland and colleagues (2013). In their trial, adolescents and young adults received either two weeks, or three months of treatment (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Despite improvement over the initial two weeks of treatment, participants who received no additional treatment had reverted to delayed sleep timing by the 3-month follow-up. However, improvements in working memory (i.e., LNS) and processing speed (i.e., DSS) remained ($d$=0.58, 0.70, respectively), albeit not to the same degree as the 3-month treatment group ($d$=0.83, 1.13). The magnitude of change on the Letter-Number Sequencing and Digit-Symbol Substitution tasks in the present study ($d$=0.53, 0.56, respectively) were most comparable to Wilhelmsen-Langeland's (2013) 2-week treatment group. As participants in the present study ceased bright light therapy after three weeks of treatment, this is unsurprising. It is possible that continuation of light therapy leads to more favourable outcomes for sleep and cognitive performance.

**Limitations and Future Directions**

Although there were no differences in cognitive performance between the DSWPD and good sleeper groups, when completed in the late morning, it is possible that between group differences could have been detected if measured earlier (e.g., 9:00am). This is particularly important as many adolescents with DSWPD are required to perform academically in the early morning. School start time in Australia is typically 8:30am (Short, Gradisar, Lack, Wright, & Dohnt, 2013; Warner et al., 2008), which poses the question, "*Are adolescents with DSWPD impaired in their cognitive functioning at the beginning of the school day*?" This research question could be answered by measuring cognitive performance at an earlier circadian and social time (i.e., 8:00-10:00am; Goldstein et al., 2007).

As academic performance relies upon the simultaneous use of many cognitive abilities (Beebe, 2011; Evans, Floyd, McGrew, & Leforgee, 2002; Fuchs et al., 2010), future research could also use more complex tasks, which draw upon many cognitive resources (i.e., fluid intelligence; Goldstein et al., 2007; McGrew, 2009). The present study is only the second to have measured changes in adolescent's cognitive performance as a result of chronobiological treatment (i.e., light therapy) for DSWPD. However, both studies lacked a waitlist control condition; therefore it is possible that changes in cognitive performance could be partly attributable to practice effects. Future randomised controlled trials could isolate treatment effects from practice effects; thus shedding further light on the relationship between sleep and cognition in this under-studied sample.

**Summary**

Empirical research investigating the relationship between evening chronotype (Goldstein et al., 2007; Hahn et al., 2012; Lara et al., 2014; Matchock & Mordkoff, 2009), sleep duration (Agostini et al., 2017; Gradisar, Terrill, et al., 2008; Lo et al., 2016) and cognitive performance suggests that adolescents with DSWPD would be impaired in their cognitive functioning, relative to good sleepers. Findings from the present study showed no between group differences. Nonetheless, bright light therapy was beneficial to the sleep and cognitive performance of such adolescents with DSWPD. As an under-researched area, replication is needed. Studies could manipulate the timing (i.e., early morning) and modality (i.e., fluid reasoning) of cognitive testing, to better understand whether adolescents with DSWPD are impaired in their cognitive functioning. Additionally, clinical trials using a waitlist control condition could shed more light on the relationship between changes in sleep and cognition.

# Chapter 6

## Cognitive "Insomnia" Processes in Delayed Sleep-Wake Phase Disorder: Do they exist and are they Responsive to Chronobiological Treatment?

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### Author Contributions

CR led study conceptualisation, design, recruitment, data collection, management and analysis, results interpretation and manuscript preparation. GM, NC and KB assisted with recruitment, data collection and manuscript preparation. BM assisted with data management and analysis. MG contributed to study conceptualisation, design, recruitment, data collection, results interpretation and manuscript preparation.

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Richardson, C., Micic, G., Cain, N., Bartel, K., Maddock, B. & Gradisar, M. (Under Review). Cognitive "insomnia" processes in Delayed Sleep-Wake Phase Disorder: Do they exist and are they responsive to chronobiological treatment? *Sleep.*

## Abstract

**Study Objectives:** To systematically investigate whether cognitive "insomnia" processes are implicated in adolescent Delayed Sleep-Wake Phase Disorder (DSWPD) and to examine whether these processes are responsive to chronobiological treatment.

**Methods:** 63 adolescents (mean= 15.8±2.2yrs, 63.5% f) diagnosed with DSWPD and 40 good-sleeping adolescents (mean= 15.9±2.4yrs, 75% f) completed baseline measures of sleep, daytime functioning and insomnia (i.e., repetitive negative thinking, physiological hyperarousal, distress, sleep-related attention and monitoring, sleep misperception). 60 DSWPD adolescents (mean= 15.9±2.2 y, 63% f) entered a randomised controlled trial evaluating bright light therapy and morning activity. Sleep, daytime functioning and insomnia were measured again post-treatment and at 3-month follow-up.

**Results:** Adolescents with DSWPD has significantly later sleep timing ($d$=0.99-1.50), longer sleep latency ($d$=1.14) and shorter total sleep time ($d$=0.85) on school nights, compared to the good-sleeping adolescents. There was evidence of cognitive "insomnia" symptoms, with the DSWPD group reporting more repetitive negative thinking ($d$=1.35-1.40), physiological pre-sleep arousal ($d$=1.12), distress ($d$=2.96), sleep associated monitoring ($d$=1.54) and sleep onset misperception ($d$=1.34). Across treatment and follow-up, adolescents with DSWPD reported advanced sleep timing ($d$=0.54-0.62), reduced sleep latency ($d$=0.53), increased total sleep time ($d$=0.49) and improved daytime functioning ($d$=0.46-1.00). Repetitive negative thinking ($d$=0.64-0.96), physiological arousal ($d$=0.69), distress ($d$=0.87), and sleep onset misperception ($d$=0.37) also showed improvement.

**Conclusions:** Cognitive "insomnia" processes may be implicated in the development and maintenance of DSWPD in adolescents. Many of these processes are amendable to chronobiological treatment; however residual symptoms may place adolescents at risk of poor treatment outcome or relapse.

## Introduction

Adolescence is a time of striving for independence, navigating new experiences (e.g., first job, first relationship) and taking on more responsibilities (e.g., study, part-time work, extra-curricular activities and recreational pastimes; Carskadon, 2011). These psychosocial changes are coupled with rapid physiological changes in sleep (e.g., delay and/or lengthening of circadian rhythm, slower accumulation of sleep pressure), which places adolescents at risk of Delayed Sleep-Wake Phase Disorder (Gradisar & Crowley, 2013). DSWPD is defined as difficulty initiating sleep and an inability to wake for early morning commitments, as a result of delayed timing of the major sleep period (American Academy of Sleep Medicine [AASM], 2014). DSWPD is thought to affect between 1-16% of young people (AASM, 2014; Lovato, Gradisar, et al., 2013; Saxvig et al., 2012; Sivertsen et al., 2013) and can significantly impact functioning, including daytime sleepiness, fatigue, inattention, low motivation, poor academic performance and impaired mood (Giannotti et al., 2002; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lovato, Gradisar, et al., 2013; Meijer et al., 2000; Rajaratnam et al., 2015; Reid et al., 2012; Richardson, Cain, et al., Under Review; Saxvig et al., 2012; Shochat et al., 2014).

Arguably, the strongest contributing factor to DSWPD is a substantial delay of the 24-hr circadian rhythm of dim light melatonin onset, core body temperature, and relevant to the present study - sleep onset (i.e., compared to good sleepers, Micic et al., 2013; Saxvig et al., 2013). Young people with DSWPD may attempt sleep at a socially-acceptable time (e.g., 9.00pm, Czeisler et al., 1981), which is significantly earlier than their circadian timing and more aligned with their peak physiological alertness (i.e., their wake-maintenance zone; Lack & Wright, 2007). Due to the misalignment between their circadian timing and earlier desired sleep time, young people with DSWPD may spend a long time attempting to fall asleep, until

they pass their wake-maintenance zone. In turn, this delayed sleep timing may lead to an experience similar to 'sleep-onset insomnia' (Weitzman et al., 1981).

The prolonged wakefulness in bed experienced by many young people with DSWPD (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; Weitzman et al., 1981) can give rise to repetitive negative thinking (i.e., worry and rumination), which can worsen sleep-onset difficulties (Carney, Harris, Moss, & Edinger, 2010; Gross & Borkovec, 1982; Wicklow & Espie, 2000). Evidence shows a significant proportion of adolescents with DSWPD report disorder-specific worries (68%), a racing mind (89%, Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), and catastrophic thinking (Hiller et al., 2014), with such dysfunctional thought akin to insomnia and other psychological disorders (Gellatly & Beck, 2016; Harvey, 2004; Harvey & Greenall, 2003). Aside from repetitive negative thinking, there are several additional insomnia processes that have been proposed, all of which are inter-related (Espie, 2002; Harvey, 2002; Lundh & Broman, 2000). These include hyperarousal and distress, attentional bias towards sleep-related 'threats', misperception of one's sleep and functioning, and safety behaviours (Espie, 2002; Harvey, 2002; Lundh & Broman, 2000). There is emerging evidence for some of these insomnia processes occurring in those diagnosed with DSWPD (i.e., attentional bias, Espie, Broomfield, et al., 2006; MacMahon et al., 2006; Marchetti et al., 2006; misperception, Billows, 2009; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013; safety behaviours, Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lovato, Gradisar, et al., 2013; Saxvig et al., 2012; Wilhelmsen-Langeland et al., 2012). Whilst these preliminary data indicate the presence of insomnia symptoms and processes in young people diagnosed with DSWPD, these are not always directly or systematically studied. For example, there are limited data as to whether insomnia symptoms are more common in young people with DSWPD compared to good sleepers. Thus, the first

aim of the present study is to compare cognitive insomnia processes between young people diagnosed with DSWPD and good sleepers. If those with DSWPD show insomnia symptoms, one question that arises is: can best practice treatments for this circadian rhythm sleep disorder reduce insomnia symptoms?

One form of chronobiological treatment for DSWPD is bright light therapy. When bright light is administered gradually earlier in a young person's biological morning (i.e., upon natural waking), advancement of sleep timing occurs (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; Sharkey, Carskadon, Figueiro, Zhu, & Rea, 2011). Over two to three weeks, this reduces sleep latency (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014), thus reducing the opportunity for repetitive negative thinking. A reduction in repetitive negative thinking may then result in a cascade of improvements in inter-related insomnia processes. Hyperarousal and distress would likely improve alongside a reduction in repetitive negative thinking (Bonnet & Arand, 2010; Harvey, 2002; Riemann et al., 2010), as could attentional bias (Marchetti et al., 2006), safety behaviours (e.g., reduced napping and caffeine use, Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011) due to improved daytime functioning (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), and ultimately sleep misperception. The second aim of the present study is to evaluate changes in cognitive insomnia processes (i.e., repetitive negative thinking, physiological hyperarousal and distress, sleep-related attentional bias, sleep misperception) in response to the chronobiological treatment of bright light therapy (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014). If improvements in cognitive insomnia processes are found, then a third and final aim is to investigate theoretical associations

between these insomnia processes. For example, is a reduction in sleep onset latency associated with a reduction in repetitive negative thinking?

## Method

### Participants

DSWPD participants were enrolled in a randomised controlled trial at the Child & Adolescent Sleep Clinic at Flinders University, Adelaide, Australia (Richardson, Cain, et al., Under Review). Of 83 adolescents assessed, 63 met International Classification of Sleep Disorders-Third Edition (AASM, 2014) criteria for DSWPD. Pre-treatment data were collected from all 63 adolescents (mean= 15.8±2.2yrs, range 13-24, 63.5% f) and 60 were randomised to receive treatment (mean= 15.9±2.2 y, 63% f). Three participants did not undergo treatment as they no longer wanted treatment (*n*=1), declined research involvement (*n*=1) or had severe suicidal ideation (*n*=1). Adolescents were randomly allocated to receive one of four treatments; green bright light therapy (BLT) plus sedentary activity (SA) (*N*=15, mean age= 15.9±1.8yrs, 60% f), green BLT plus physical activity (PA) (*N*=15, mean= 16.2±2.8yrs, 67% f), red LT plus SA (*N*=15, mean= 15.5±1.6yrs, 67% f) or red LT plus PA (*N*=15, mean= 15.7±2.8yrs, 60% f). Physical activity was added to light therapy, as post-awakening exercise, when gradually advanced, may have the propensity to phase advance the human circadian rhythm (Miyazaki et al., 2001; Richardson, Cain, et al., Under Review; Richardson et al., 2017). However, as no significant interaction effects were found, data were collapsed for the purposes of this study (Richardson, Cain, et al., Under Review).

Good-sleeping adolescents and young adults (GS), aged 13-24yrs (mean= 15.9±2.4yrs, 75% f), were recruited via advertisements in school newsletters, community notice boards and social media. 370 adolescents completed an online screening questionnaire assessing inclusion criteria: mean sleep onset latency (SOL) ≤30 mins, wake after sleep onset

(WASO) ≤20 mins, total sleep time (TST) ≥eight hours, weekday-to-weekend wake-up time discrepancy of ≤two hours, no difficulty falling asleep, staying asleep or unrefreshing sleep and no co-morbid psychiatric, physical or sleep disorders.

The trial was registered with the Australian and New Zealand Trials Registry (ACTRN12614000308695) and the study was approved by the Southern Adelaide Clinical Human Research Ethics Committee and the Social and Behavioural Research Ethics Committee.

## Materials and Apparatus

### Clinical Sleep History Interview (CSHI)

A revised semi-structured interview (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011) was used to diagnose Delayed Sleep-Wake Phase Disorder (AASM, 2014) as per International Classification of Sleep Disorders- Third Edition (ICSD-3) criteria. The CSHI elicited information pertaining to Criterion A (chronic difficulty initiating sleep and awakening at a desired and/or required clock time); Criterion B (symptoms present for ≥three months) and Criterion E (differential diagnosis). Participants were excluded if their sleep disorder was better accounted for by another sleep problem (i.e., Chronic Insomnia, Non-24-hr Sleep-Wake Disorder). Sixty six percent of participants reported a comorbid psychological condition (i.e., anxiety: *n*=27, depression: *n*=26). However, their sleep problem was not better accounted for by the psychological condition (i.e., it did not occur exclusively within the course of anxiety or depression).

### Sleep Diary

A 7-day sleep diary was completed online and measured sleep timing (sleep onset time [SOT] and wake-up time [WUT]) and sleep quality (sleep onset latency [SOL], wake

after sleep onset [WASO] and total sleep time [TST]). Similarly to a previous study (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), adolescents completed the online sleep diary daily throughout assessment and treatment, which allowed sleep diary entries to be time and date stamped, and reduce recall bias (Buysse, Ancoli-lsrael, Edinger, Lichstein, & Morin, 2006). Sleep parameters were averaged for school nights (Sun-Thurs) and weekend nights (Fri-Sat) separately.

**Wrist Actigraphy**

Participants wore a MicroMini Motionlogger wrist actigraphy monitor (Ambulatory Monitoring Inc., Ardsley, NY) following their initial assessment and throughout treatment. The monitor collected data in one minute epochs across the 24-hr day, using zero-crossing mode. Sleep onset and offset times were scored by author CR to confirm a delay in the habitual sleep period at baseline (Criterion D), to monitor changes in sleep timing across treatment, and for the present study, to assess sleep misperception (see *Sleep Misperception* below). Actigraphy data were scored using the Sadeh algorithm (AMI, Action 4, Ambulatory Monitoring Inc, Ardsley, NY), which has been validated for adolescents (Meltzer et al., 2012; Sadeh et al., 1994). Sleep onset time (SOT) was scored as the first minute of at least three consecutive minutes of sleep occurring from 15min before sleep diary lights out time. Wake-up time (WUT) was scored as the last minute of at least five minutes of consecutive sleep, occurring from 15min before sleep diary wake-up time.

**Daytime Functioning**

Daytime sleepiness, fatigue and functional impairment are common symptoms of DSWPD (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Micic et al., 2015). The Paediatric Daytime Sleepiness Scale (PDSS) is an 8-item self-report scale of daytime sleepiness (e.g., "*How often do you fall asleep or feel drowsy in class*?"; Drake et al., 2003),

with total scores ranging from 0-32 (higher scores indicate greater sleepiness). The PDSS has good internal consistency in the current study (Cronbach α = 0.87). The Flinders Fatigue Scale (FFS) is a seven item self-report measure of global fatigue (e.g., "*Was fatigue a problem for you*?"; Gradisar et al., 2007), with total scores ranging from 0-31 (higher scores indicate greater fatigue). The FFS had good reliability in the current study (Cronbach α = 0.90). The Sheehan Disability Scale (SDS) is a 5-item measure of the disruption to one's work/school, social and family/home lives caused by symptoms of their sleeping pattern (e.g., "*To what extent has your sleep pattern disrupted your work/school work*"; Sheehan et al., 1996), with total scores range from 0-47 (higher scores indicate more lifestyle disruption and impairment). The SDS had adequate reliability in the current study (Cronbach α = 0.88). The PDSS, FFS and SDS were used to confirm daytime impairment in the DSWPD group, relative to the GS group.

**Repetitive Negative Thinking**

Repetitive negative thinking was measured using the cognitive subscale of the Pre-Sleep Arousal Scale (PSAS-C; Nicassio et al., 1985) and the Catastrophising Interview (CI; Gregory, Noone, Eley, & Harvey, 2010; Hiller et al., 2014).

The PSAS-C measures the intensity of state cognitive arousal when individuals attempt sleep and includes eight items (e.g., *"worry about falling asleep", "Can't shut off your thoughts").* Responses range from 1= *Not at all*, to 5= *Extremely*, with total scores ranging from 8-40. Higher scores indicate worse pre-sleep cognitive arousal. The PSAS-C had good internal consistency (α = 0.90), as consistent with previous reports (α = 0.88; Nicassio et al., 1985).

The Catastrophising Interview was a clinician-administered semi-structured interview (Gregory et al., 2010; Hiller et al., 2014) and began with the question "*You've said you have*

*trouble falling asleep, does it worry you?*" If adolescents indicated some level of worry, the clinician followed with the question "*What is it that worries you about nights when you have problems sleeping?"* Subsequent questioning followed the format *"What is it that worries you about <previous worry>?"* This same question was repeated until the adolescent was unable to provide a response, or until they had generated seven catastrophising responses (steps). As such, total scores ranged from 0-7.

**Physiological Arousal and Distress**

The somatic subscale of the Pre-Sleep Arousal Scale (PSAS-S) measured the intensity of state somatic arousal when individuals attempt sleep and includes eight items (e.g., *"Heart racing, pounding or beating irregularly").* Responses are measured on a 5-point Likert scale (1= *Not at all*, 5= *Extremely*) with the total subscale score ranging from 8-40. Higher scores indicate worse pre-sleep somatic arousal. The PSAS-S had good internal consistency (α = 0.88).

The Ford Insomnia Response to Stress Test (FIRST) measures trait vulnerability to hyperarousal (Drake, Richardson, Roehrs, Scofield, & Roth, 2004; e.g., "*How likely is it for your to have difficulty sleeping before an important event the next day*?"). Responses are measured on a 4-point Likert scale (1=*Not Likely*, 4= *Very Likely*), with total scores ranging from 9-36. Higher scores indicate greater susceptibility to hyperarousal and sleep disturbance. The FIRST had good internal consistency (α = 0.99).

A modified version of the Insomnia Severity Index (ISI; Morin, 1993; Short, Gradisar, Lack, & Wright, 2013) was used to detect insomnia in adolescents. The ISI was adapted to a 6-item measure, assessing the severity and impact of sleep disturbance (e.g., *"Do you have difficulty falling asleep? If yes, how severe is this difficulty?")*. Item five (e.g., "*How worried/ distressed are you about your sleep at the moment?")* was also used as a

measure of sleep-related distress. Items were measured on a 5-point Likert scale from 0 to 4, with higher scores indicating worse severity and distress. The Modified ISI had acceptable internal consistency (α=0.93).

**Sleep-Related Attention and Monitoring**

A computerized dot-probe task (Neurobehavioural Systems Presentation, v14.9) was used to objectively measure sleep-related attentional bias. In the task, a fixation point is presented in the centre of the screen (1,000ms), before being replaced with a pair of words (500ms); one sleep-related and one matched neutral word. A probe (left or right facing arrow) then appears in the previous location of one of the words. Adolescents pressed the left or right arrow key to reflect the direction of the arrow. Response times are quicker when the probe appears in the location of the previously attended word (e.g., *Naps*) and slower when the probe replaces the unattended word (e.g., *Pear*). The 20 sleep-related word pairs have been used empirically (MacMahon et al., 2006; Taylor et al., 2003; Wicklow & Espie, 2000) and the task has previously been validated for use with an Australian sample (Richardson et al., 2015). The placement of the word pairs and direction of the dot-probe were counterbalanced across all participants and trials, therefore, participants completed 80 experimental trials in total. Trials were considered valid if responses were correct (i.e. indicated correct direction of probe) and reaction time fell between 150-1500ms. Data were excluded from analyses if they had an error rate exceeding 5% (MacMahon et al., 2006). Attentional bias scores were calculated from valid trials as follows; [(Target Up, Probe Low + Target Low, Probe Up) - (Target Up, Probe Up + Target Low, Probe Low)]/2]; where Target Up/Low reflects the spatial location of the sleep-related word and Probe Up/Low reflects the location of the arrow (Harris et al., 2015; Lancee et al., 2017; MacMahon et al., 2006; Milkins, Notebaert, MacLeod, & Clarke, 2016). Positive scores indicate an attentional bias towards sleep stimuli, whereas negative scores indicate attentional bias away from sleep

stimuli (i.e., towards neutral stimuli). Adolescents completed the dot-probe task in the morning (M=11:25am±107min), on a 21.5" iMac computer in the Flinders University Sleep Laboratory.

The Sleep Associated Monitoring Index (SAMI) is a 30-item subjective assessment of sleep-related attentional bias (e.g., "*Before or as you go to bed, how often do you calculate the number of hours of sleep you are likely to get?"*; Neitzert Semler & Harvey, 2004b). Items are measured on a 5-point Likert scale (1=*Not at all*, 5= *All the time*), with total scores ranging from 30-150. Higher scores indicate greater sleep-related monitoring. There are eight subscales; i) daytime monitoring for body sensations, ii) calculation of time, iii) waking monitoring for body sensations, iv) pre-sleep monitoring for body sensations consistent with falling asleep, v) daytime monitoring of functioning, vi) pre-sleep monitoring for body sensations inconsistent with falling asleep, vii) pre-sleep monitoring of the clock and viii) pre-sleep monitoring the environment. The SAMI has previously shown to have high internal consistency ($\alpha = 0.91$), as was the case in the present study ($\alpha = 0.95$).

**Sleep Misperception**

Objective sleep data collected by wrist actigraphy were compared to subjective sleep data from the online sleep diary, to assess sleep misperception. Actigraphy was used as an objective measure of sleep as it is minimally invasive and cost effective (relative to polysomnography), allowed for week-long measurement of sleep misperception in an ecologically valid way, and has previously been used to address sleep misperception in clinical samples (Tang & Harvey, 2006). Although misperception of sleep onset latency is central to theory (Harvey, 2002) and observations of insomnia (Carskadon et al., 1976; Coates et al., 1982; Frankel, Coursey, Buchbinder, & Snyder, 1976), misperception of sleep onset time (SOT) was used in the present study. Actigraphy watches did not have the capacity to mark lights out time, and using subjective lights out time to calculate objective sleep onset

latency would have compromised the independence of the two measures. SOT can be reliably measured in adolescents following three nights of recording (Acebo et al., 1999). Therefore, sleep misperception scores were calculated when participants had corresponding sleep diary and actigraphy records for at least three school nights (Sun-Thurs) and/or at least one weekend night (Fri-Sat).

Sleep diary SOT was calculated by adding self-reported Sleep Onset Latency to Lights Out Time. Actigraphy defined SOT was subtracted from sleep diary SOT, so that positive discrepancy scores indicated overestimation of the sleep problem (misperception) and negative scores indicated underestimation.

**Safety Behaviours**

To better understand whether adolescents with DSWPD may use maladaptive ways of coping with their sleep and daytime dysfunction, relative to their good-sleeping counterparts, single-item questions were asked in the CSHI to assess alcohol use, nicotine use and caffeine use (e.g., *"How much alcohol do you have on average each day?"*), technology use (e.g., "*What sorts of things do you have in your bedroom?*") and napping (e.g., *"Do you nap during the day?", "If yes, how often, what time and what length on average?"*).

**Treatment Materials**

Adolescents used LED light devices (e.g., Re-Time Pty Ltd., Adelaide, Australia) during light therapy. These light glasses have two LED lights built into the frame for each eye. Adolescents received either green (~507nm, ~112 lux per diode at 2cm distance, commercially available) or red (~643nm, ~54 lux per diode at 2cm distance, placebo) light therapy. Red light is typically used as a placebo condition in clinical trials (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), as was the case in the present study.

During light therapy, adolescents completed either sedentary activity (i.e., sitting watching TV) or mild morning activity (i.e., standing, playing motion-sensing video games). Active video games (e.g., XBox Kinect, Nintendo Wii, Playstation Move) increase heart rate, oxygen consumption and energy expenditure, relative to sedentary video games (Graves et al., 2008; Peng et al., 2011). There is some evidence that timed morning exercise may phase advance the human circadian rhythm (Richardson et al., 2017; Youngstedt et al., 2002), especially when advanced in small daily increments (Miyazaki et al., 2001). However, as there was no meaningful difference in activity frequency between the two conditions, or any differential impact of activity or light condition (i.e., red vs green light) on treatment outcomes, data were collapsed for the present study (Richardson, Cain, et al., Under Review).

**Procedure**

Recruitment of DSWPD adolescents occurred from July 2014 - December 2016 and good sleepers were recruited from June 2015 - January 2017. The 63 DSWPD adolescents were diagnosed by a clinical, registered or provisional psychologist undergoing clinical psychology training, after completing a 7-day sleep diary, measures of insomnia (PSAS, CI, ISI, dot-probe, SAMI) and daytime functioning (PDSS, FFS, SDS) and undergoing the CSHI. DSWPD participants completed another 7-day sleep diary and wore actigraphy in the week following the initial assessment, to assist in confirming the DSWPD diagnosis (AASM, 2014) and so that pre-treatment sleep misperception data could be calculated. DSWPD diagnosis was confirmed at a consensus meeting between therapists and the clinic supervisor (MG).

The 40 good sleepers were invited to participate in the study after completing an online screening questionnaire (Qualtrics, Utah, USA). Screening questions were adapted from the CSHI (i.e., technology use, stimulant and alcohol use, napping, comorbidities,

differential diagnosis) and also included the ISI, and self-reported estimates of sleep (bed time (BT), lights out time (LOT), sleep onset latency (SOL), wake after sleep onset (WASO), wake-up time (WUT) and out of bed time (OOBT)), for both weekdays and weekends separately (Wolfwon & Carskadon, 2003). After meeting eligibility criteria, good-sleeping adolescents were invited to the Flinders University Sleep Laboratory, where they were provided with actigraphy and information necessary to access the online 7-day sleep diary. After recording their sleep for one week at home, participants returned to the Sleep Laboratory to complete questionnaires (PSAS, CI, SAMI) and the dot-probe task.

After pre-treatment completion, 60 DSWPD participants received their chronobiologic sleep treatment over 3, weekly 50min sessions. Treatment involved psychoeducation about circadian rhythms, plans to reduce evening light and administer light therapy to advance sleep timing and instructions about the type of activity they should complete during morning light therapy. Adolescents slept in on the first day of therapy and wore the portable light glasses for 30-60mins immediately after rising from bed. Out of bed time and light therapy commenced 30mins earlier each subsequent day until they reached a wake-up time of 6:00am (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Gradisar et al., 2014). Once participants achieved this target time, they ceased light therapy and maintained a wake-up time no later than 7:30am. Participants completed sleep diaries and actigraphy monitoring throughout treatment, and sleep misperception scores were averaged weekly. Cognitive insomnia processes (PSAS, CI, ISI, dot-probe) were measured pre- and post-treatment, then again at a 3-month follow-up, along with another 7-day sleep diary.

**Statistical Analyses**

Forty six adolescents met good sleeper criteria and were included in the study (mean= 16.0±2.6yrs, 76.1% f). However, six participants were excluded from data analysis as sleep measures (i.e., sleep diary, actigraphy) indicated they no longer met inclusion criteria for good sleepers (i.e., discrepant weekday to weekend sleep timing, elevated average SOL or WASO and/or reduced total sleep time). Therefore, 40 good-sleeping adolescents (mean= 15.9±2.4yrs, 75% f) were included in the final analysis. Participant characteristics for the DSWPD and GS groups are summarised in Table 6.1 (see Results).

Independent-samples t-test and Chi-Square analyses were used to compare cognitive "insomnia" processes between the DSWPD and good-sleeping adolescent groups. Effect sizes were calculated to establish the magnitude of between-subjects differences. Cohen's *d* was calculated as *d*=M1-M2/(SDpooled). To minimise the potential impact of missing data (~21% from pre-treatment to 3-month follow-up), Linear Mixed Modelling (LMM) was used to analyse treatment data. LMM analyses impute missing data using expectation maximisation, based on a regression line for each individual, and is therefore preferable to other statistical techniques (e.g., ANOVA; Landau & Everitt, 2004). Cohen's *d* was also calculated to establish the magnitude of within-subjects differences. Change variables were calculated for sleep, daytime functioning and insomnia symptom variables, by subtracting post-treatment values from pre-treatment values. The relationships between the change in sleep, daytime functioning and insomnia symptoms, were investigated using Pearson product-moment correlations.

## Results

### Do adolescents with DSWPD possess insomnia symptoms?

Adolescents with DSWPD had significantly later subjective and objective sleep timing and were significantly more impaired in their daytime functioning, relative to their good-sleeping counterparts (see Table 6.1). The DSWPD group also reported significantly longer sleep onset latencies, less total sleep time and greater insomnia severity compared to the good-sleeper group. Self-reported SOL for the DSWPD group met suggested criteria for sleep-onset insomnia (i.e., ≥30mins; Buysse et al., 2006; Lichstein, Durrence, Taylor, Bush, & Riedel, 2003).

Between-group differences in insomnia processes are presented in Table 6.2. Relative to good-sleepers, adolescents with DSWPD reported significantly more repetitive negative thinking and physiological arousal and distress. Adolescents with DSWPD also displayed a trait vulnerability to hyperarousal. Although there was no significant difference in the objective measure of sleep-related attentional bias, subjective reports highlight that adolescents with DSWPD monitor for both internal and external sleep-related threats. Sleep-onset misperception was significantly more prominent within the DSWPD group, compared to good sleepers. In terms of safety behaviours, good-sleeping adolescents reported consuming significantly more caffeinated foods per day; yet this difference is unlikely to be clinically meaningful (i.e., mean intake was less than one item/day). In terms of technology located within the bedroom, good-sleeping adolescents were more likely to have a DVD player present; whereas, DSWPD adolescents were more likely to have more stimulating devices, such as an iPod, video gaming console and/or internet connection. DSWPD adolescents were also significantly more likely to report napping.

Table 6.1

*Sleep and Daytime Functioning of Good-Sleeping Adolescents and Adolescents with DSWPD.*

| | **Good Sleepers Mean±SD** | **DSWPD Mean±SD** | ***p*** | ***d*** |
|---|---|---|---|---|
| Sleep Diary | | | | |
| SOT (School Nights)* | 23:01±57 | 01:09±107 | **<0.001** | **1.50** |
| SOT (Weekend Nights)* | 23:10±54 | 01:40±110 | **<0.001** | **1.73** |
| WUT (School Days)* | 07:26±48 | 08:46±103 | **<0.001** | **1.00** |
| WUT (Weekend Days)* | 07:47±58 | 09:36±114 | **<0.001** | **1.21** |
| SOL (School Nights) (mins) | 18±12 | 76±71 | **<0.001** | **1.14** |
| SOL (Weekend Nights) | 15±12 | 66±67 | **<0.001** | **1.06** |
| WASO (School Nights) (mins) | 6±6 | 15±34 | 0.057 | 0.37 |
| WASO (Weekend Nights (mins) | 6±9 | 13±35 | 0.154 | 0.27 |
| TST (School Nights) (hrs) | 8.32±0.66 | 7.36±1.46 | **<0.001** | **0.85** |
| TST (Weekend Nights) (hrs) | 8.53±0.93 | 7.74±1.78 | **0.008** | **0.56** |
| Actigraphy | | | | |
| SOT (School Nights)* | 22:52±55 | 00:24±78 | **<0.001** | **1.18** |
| SOT (Weekend Nights)* | 22:59±52 | 01:11±108 | **<0.001** | **1.55** |
| WUT (School Days)* | 07:24±48 | 08:50±112 | **<0.001** | **0.99** |
| WUT (Weekend Days)* | 08:06±68 | 10:04±103 | **<0.001** | **1.36** |
| Insomnia Severity (ISI) | 3.00±2.35 | 16.13±3.75 | **<0.001** | **4.20** |
| Daytime Functioning | | | | |
| Daytime Sleepiness (PDSS) | 10.93±3.25 | 21.65±4.63 | **<0.001** | **2.68** |
| Fatigue (FFS) | 6.68±4.64 | 9.54±4.01 | **0.003** | **0.66** |
| Functional Impairment (SDS) | 5.00±5.97 | 23.11±9.60 | **<0.001** | **2.27** |
| Daytime Consequences of Sleep | | | | ***phi*** |
| Tired/ Fatigued | 33% | 98% | **<0.001** | **0.72** |
| Poor Attention/ Concentration/Memory | 10% | 93% | **<0.001** | **0.83** |
| Problems Socialising | 3% | 48% | **<0.001** | **0.49** |
| Poor School Grades | 10% | 65% | **<0.001** | **0.54** |
| Moody/ Irritable | 23% | 82% | **<0.001** | **0.59** |
| Daytime Sleepiness | 15% | 93% | **<0.001** | **0.79** |
| Low Energy/ Motivation | 20% | 85% | **<0.001** | **0.65** |
| Behavioural Issues (e.g., hyperactivity) | 3% | 37% | **<0.001** | **0.40** |
| Accident Prone | 5% | 40% | **<0.001** | **0.39** |
| Somatic Complains (e.g., headache, stomach problems) | 5% | 55% | **<0.001** | **0.51** |
| Worries about Sleep | 10% | 70% | **<0.001** | **0.59** |
| Needing to be Repeatedly Woken | 20% | 68% | **<0.001** | **0.56** |

Table 6.2

*Comparison of Cognitive "Insomnia" Symptoms between Good-Sleeping Adolescents and Adolescents with DSWPD.*

| | **Good Sleepers Mean±SD** | **DSWPD Mean±SD** | ***p*** | ***d*** |
|---|---|---|---|---|
| Repetitive Negative Thinking | | | | |
| Catastrophic Thinking (Steps) | 1.00±2.28 | 4.08±2.27 | **<0.001** | **1.35** |
| Cognitive Pre-Sleep Arousal (PSAS-C) | 17.30±6.60 | 26.42±6.45 | **<0.001** | **1.40** |
| Physiological Arousal and Distress | | | | |
| Somatic Pre-Sleep Arousal (PSAS-S) | 10.50±3.08 | 16.40±6.77 | **<0.001** | **1.12** |
| Vulnerability to Stress (FIRST) | 18.45±4.67 | 25.59±7.74 | **<0.001** | **1.12** |
| Distress | 0.35±0.58 | 2.68±0.95 | **<0.001** | **2.96** |
| Sleep-Related Attention and Monitoring | | | | |
| Sleep-Related Attentional Bias (Dot-Probe) | 6.45±15.05 | 0.36±23.85 | 0.241 | -0.31 |
| Sleep Associated Monitoring (SAMI) | 66.18±18.11 | 97.68±22.61 | **<0.001** | **1.54** |
| Daytime Sensations | 9.68±4.27 | 15.50±6.00 | **<0.001** | **1.12** |
| Calculation of Time | 11.28±4.10 | 12.44±4.59 | 0.197 | 0.27 |
| Waking Sensations | 9.65±3.93 | 19.69±4.37 | **<0.001** | **2.42** |
| Sensations Consistent with Sleep | 10.48±3.80 | 10.65±3.85 | 0.827 | 0.04 |
| Daytime Functioning | 8.68±3.96 | 14.52±3.90 | **<0.001** | **1.49** |
| Sensations Inconsistent with Sleep | 6.48±2.33 | 11.19±4.63 | **<0.001** | **1.29** |
| Monitoring Clock | 4.50±2.05 | 7.02±2.30 | **<0.001** | **1.16** |
| Monitoring Environment | 5.45±2.5 | 6.68±2.63 | **0.021** | **0.48** |
| Sleep Misperception (mins) | | | | |
| SOT (School Days) | 9±22 | 71±62 | **<0.001** | **1.34** |
| SOT (Weekend Days) | 10±24 | 56±60 | **<0.001** | **1.00** |
| Safety Behaviours | | | | |
| Caffeinated Beverages/ Day | 0.83±0.90 | 1.07±1.67 | 0.404 | 0.18 |
| Caffeinated Foods/day (e.g., chocolate) | 0.95±0.85 | 0.28±0.52 | **<0.001** | **-0.95** |
| Standard Alcoholic Drinks/ Weekday | 0.10±0.63 | 0.00±0.00 | 0.222 | 0.22 |
| Standard Alcoholic Drinks/ Weekend day | 0.70±2.52 | 0.23±1.32 | 0.287 | 0.23 |
| Technology in Bedroom | | | | ***phi*** |
| iPod | 35% | 63% | **0.010** | **0.28** |
| Television | 35% | 37% | 1.000 | 0.02 |
| Tablet | 15% | 23% | 0.444 | 0.10 |
| DVD Player | 63% | 27% | **0.001** | **-0.36** |
| Computer | 75% | 75% | 1.000 | 0.00 |
| Smart Phone | 90% | 85% | 0.671 | 0.07 |
| Internet Connection | 13% | 87% | **<0.001** | **0.73** |
| Video Game Console | 8% | 25% | **0.049** | **0.22** |
| Smoke Cigarettes | 3% | 5% | 0.917 | 0.06 |
| Nap | 15% | 38% | **0.022** | **0.25** |

**Does chronobiological treatment reduce insomnia?**

Figures 6.1-6.4 outline changes in objective sleep timing across treatment and subjective sleep, daytime functioning and insomnia symptoms across treatment and follow-up, respectively. Actigraphy-defined SOT, $F(3,34)= 5.20$, $p=0.005$, $d= 0.35$, and WUT, $F(3,31)= 3.92$, $p=0.017$, $d= 0.28$, on school nights became significantly earlier across treatment (see Figure 6.1).

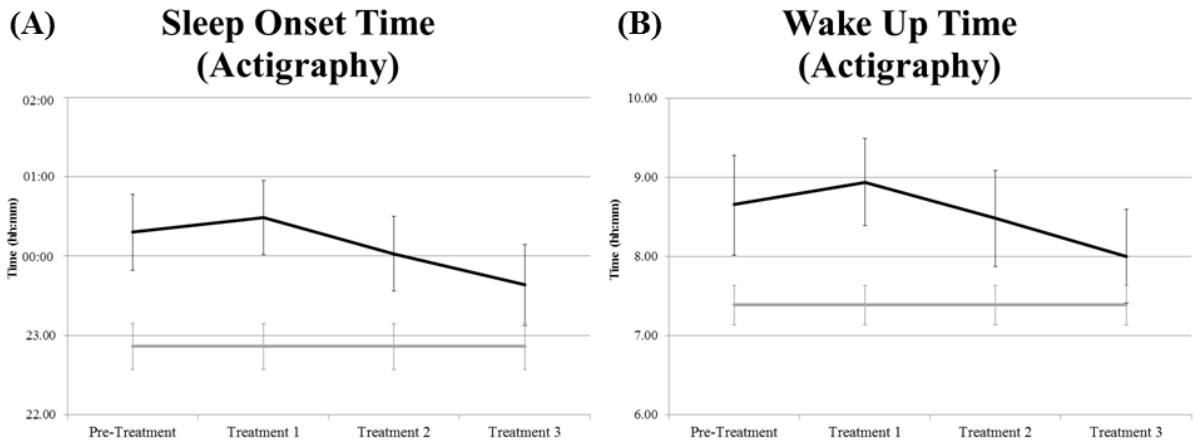


*Figure 6.1* Mean actigraphy defined school night (A) sleep onset time and (B) wake-up time for adolescents across treatment. Grey line indicates good sleeper adolescent mean at baseline. Error bars indicate 95% confidence interval.

Sleep diary estimates support these findings, with improvements in SOT, $F(2,30)=15.90$, $p<0.001$, $d=0.62$, and WUT, $F(2,30)= 10.04$, $p<0.001$, $d=0.54$, being maintained at the 3-month follow-up. Importantly, SOL, $F(2,33)= 6.32$, $p=0.005$, $d=0.53$, and TST, $F(2,28)= 4.96$, $p=0.014$, $d=0.49$ on school nights also significantly improved across treatment and follow-up (see Figure 6.2). Daytime sleepiness, $F(2,31)= 30.45$, $p<0.001$, $d=1.00$, fatigue, $F(2,35)=10.49$, $p<0.001$, $d= 0.66$, and functional impairment, $F(2,33)=9.81$, $p<0.001$, $d=0.46$, showed similar patterns of improvement across treatment and follow-up (see Figure 6.3).

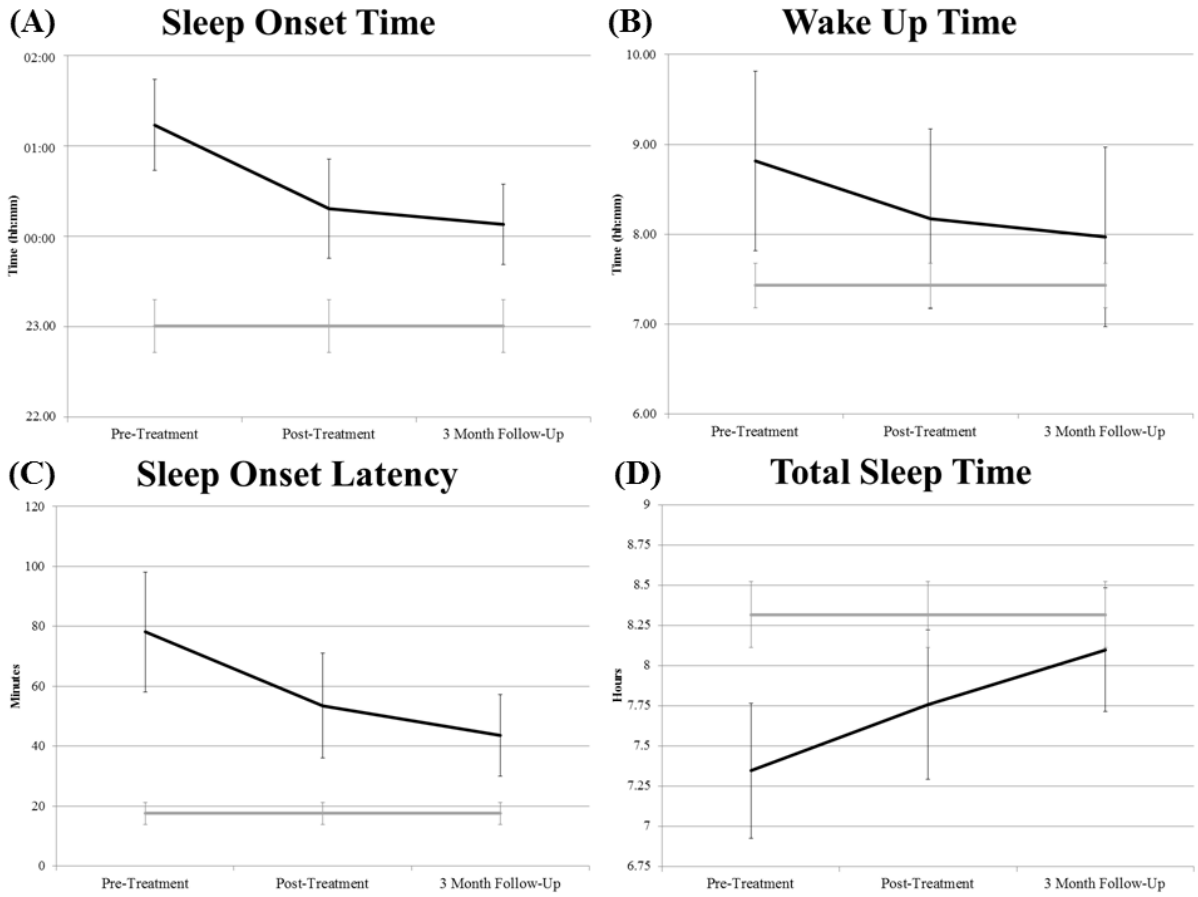


*Figure 6.2* Mean school night sleep diary (A) sleep onset time, (B) wake-up time, (C) sleep onset latency and (D) total sleep time for adolescents across treatment and follow-up. Grey line indicates good sleeper adolescent mean at baseline. Error bars indicate 95% confidence interval.

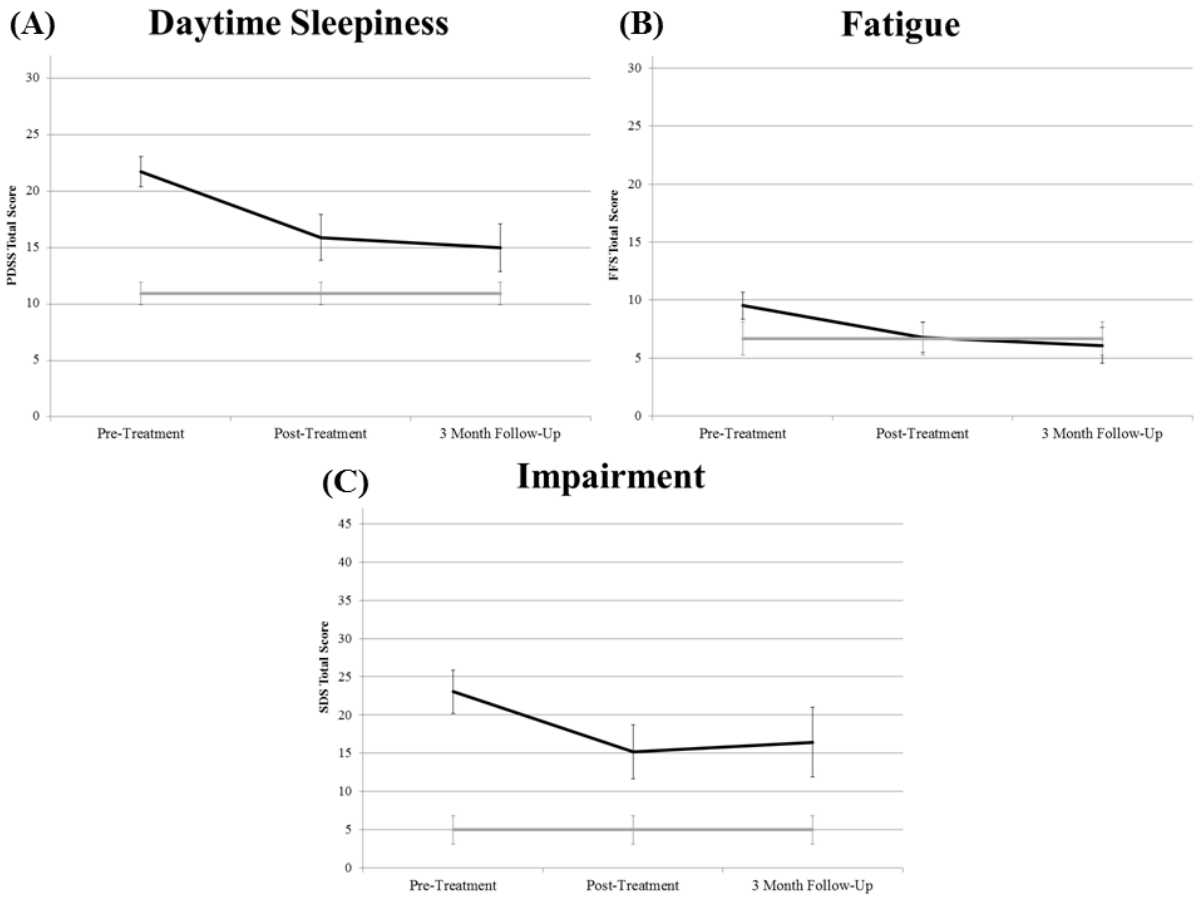


*Figure 6.3* Mean (A) daytime sleepiness, (B) fatigue and (C) functional impairment for adolescents across treatment and follow-up. Grey line indicates good sleeper adolescent mean at baseline. Error bars indicate 95% confidence interval.

Overall, insomnia severity reduced across treatment and follow-up, $F(2,35)=46.68$, $p<0.001$, $d=1.39$. Repetitive negative thinking, as measured by the catastrophising interview, $F(1,35)=12.48$, $p<0.001$, $d=0.64$, and PSAS-C, $F(2,30)=27.82$, $p<0.001$, $d=0.96$, significantly reduced across treatment and follow-up (see Figure 6.4). Physiological arousal (PSAS-S), $F(2,34)=19.01$, $p<0.001$, $d=0.69$, and distress, $F(2,36)=25.07$, $p<0.001$, $d=0.87$, also showed improvement across treatment and follow-up. However, sleep-related attentional bias (dot-probe), $F(2,21)=0.325$, $p=0.726$, $d= 0.09$, did not significantly change over time. Actigraphy was only worn across treatment, however there was some evidence of the amelioration of sleep onset misperception, $F(3,20)=2.23$, $p=0.116$, $d= 0.37$, albeit not statistically significant.

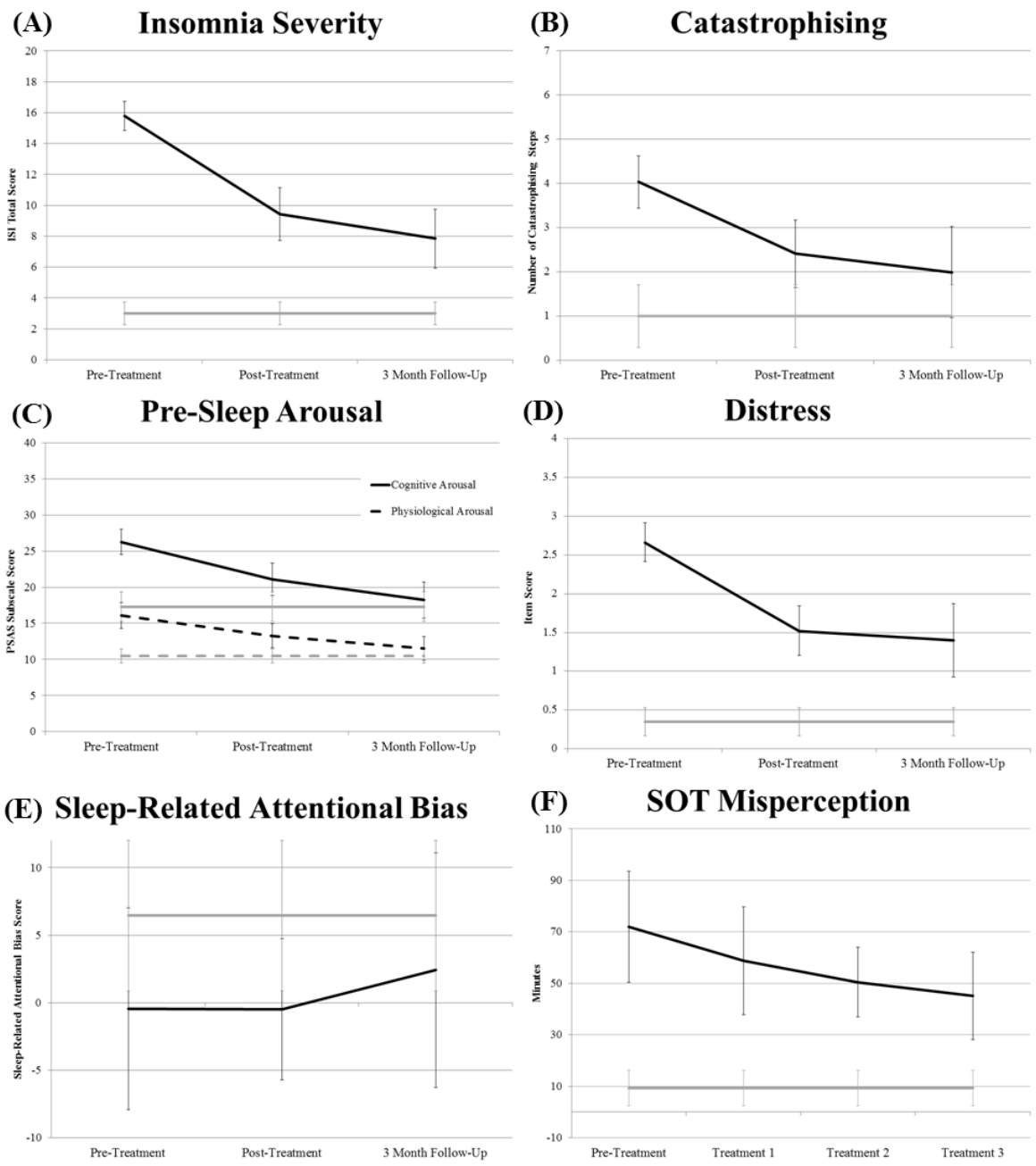


*Figure 6.4* Mean (A) insomnia severity, (B) catastrophising, (C) pre-sleep arousal, (D) distress and (E) sleep-related attentional bias score for adolescents across treatment and follow-up and (F) sleep onset misperception across treatment. Grey line indicates good sleeper adolescent mean at baseline. Error bars indicate 95% confidence interval.

**Relationship between sleep, functioning and cognitive insomnia processes**

Relationships between the change in sleep, daytime functioning and cognitive "insomnia" processes from pre- to post-treatment are highlighted in full in Appendix B. Consistent with our hypothesis, changes in subjective sleep onset latency, $r$=0.31, $p$=0.033,

objective sleep onset time, $r$=0.52, $p$=0.006, and subjective sleep onset time, $r$=0.46, $p$=0.001, were correlated with changes in cognitive pre-sleep arousal (RNT). Changes in subjective sleep onset time were also related to changes in catastrophic thinking, $r$=0.33, $p$=0.022. As per theoretical models of insomnia (Espie, 2002; Harvey, 2002) improvement in measures of repetitive negative thinking (PSAS-C) were related to improvements in pre-sleep physiological arousal (PSAS-S), $r$=0.32, $p$=0.030. Reductions in cognitive pre-sleep arousal, $r$=0.46, $p$<0.001 and catastrophising, $r$=0.37, $p$=0.011, were also related to reductions in distress.

Harvey (2002) hypothesised that distress drives changes in attentional processing in insomnia; however, change in distress, $r$=-0.24, $p$=0.25, was not significantly correlated with change in sleep-related attentional bias in the present study. Misperception of sleep is thought to be driven by repetitive negative thinking, physiological arousal, distress and sleep-related attentional bias (Harvey, 2002). Improvements in cognitive arousal were moderately related to improvements in sleep onset misperception, $r$=0.37, $p$=0.079, albeit not statistically significant. However, there was almost no relationship with other measures of repetitive negative thinking, arousal, distress or sleep-related attentional bias. Importantly, changes in subjective sleep onset latency, $r$=0.63, $p$=0.001, and sleep onset time, $r$=0.46, $p$=0.019, were strongly related to changes in sleep onset misperception.

## Discussion

The present study is the first to systematically investigate whether a diverse range of cognitive "insomnia" processes exist in adolescents diagnosed with Delayed Sleep-Wake Phase Disorder (DSWPD). Compared to good-sleeping peers, adolescents with DSWPD experienced significantly more repetitive negative thinking. This finding is consistent with previous observations of DSWPD (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al.,

2011; Hiller et al., 2014; Richardson et al., 2016; Takahashi et al., 2000). Cognitive activity was sleep-focused and unhelpful patterns of thinking (i.e., catastrophising) were also evident. These collective findings suggest that circadian rhythm sleep disorders and insomnia symptoms are not mutually exclusive.

The DSWPD group showed predisposition to sleep disturbance through increased vulnerability to stress, and reported heightened pre-sleep physiological arousal when compared to good sleepers. In a prospective study, the risk of developing insomnia was increased when both cognitive intrusion and sleep reactivity were high (Drake, Pillai, & Roth, 2014). Results from the present study suggest that the same "risk factors" may exist for the development of DSWPD. If true, repetitive negative thinking and reactivity to stress could be two important transdiagnostic processes to target in the prevention and treatment of adolescent sleep problems.

To our knowledge, the present study was the third to measure sleep-related attentional bias in DSWPD. Objectively-measured attentional bias scores (i.e., dot-probe) did not differ between the DSWPD and good sleepers. Previously, Marchetti and colleagues (2006) found significant between-group differences in sleep-related processing, using an objective inducing change blindness (image-based) task. The word-based dot-probe task used in the present study replicated the methodology of MacMahon and colleagues (2006), who found a small ($d$=0.40) difference between DSWPD individuals and good sleepers, albeit not statistically significant. The opposite effect occurred in the present study ($d$=-0.31), although again, not statistically significant. It is possible that the method of objectively measuring attentional bias has an influence on findings. Although there were no differences in objective sleep-related attentional bias, subjective reports highlighted a tendency for the DSWPD group to monitor bodily sensations upon awakening, throughout the day, and when falling asleep (for signs inconsistent with sleep). Adolescents also monitored their functioning throughout the day,

and attended to the clock and external environment during the pre-sleep period. Sleep-related monitoring increases the likelihood that sleep-threat stimuli are detected, consequently worsening repetitive negative thinking and its sequelae. Therefore, it is possible that sleep-related monitoring may play a role in maintaining DSWPD. Monitoring of sleep and daytime functioning also increases the likelihood that sleep and daytime consequences will be inaccurately perceived (Harvey, 2002). In support of this hypothesis, the DSWPD group significantly overestimated their sleep onset time, on both school days and weekend days, relative to their good-sleeping peers.

Adolescents with DSWPD may also engage in safety behaviours to aid sleep onset or overcome the daytime consequences of poor sleep (Harvey, 2002; Richardson et al., 2016). DSWPD adolescents were more likely to possess interactive forms of technology (Gradisar et al., 2013), such as an iPod, video-game console and/or internet connection, within their bedroom. However, as the timing, frequency and function of technology was not measured, it is not clear whether technology was used to aid sleep onset, as has been previously suggested (Eggermont & Van den Bulck, 2006; Exelmans & Van den Bulck, 2016). It has also been suggested that adolescents with DSWPD may engage in stimulant use to overcome daytime impairment; however, stimulant use in the present study was low (e.g., low prevalence of caffeine and tobacco use) and did not differ between the two groups, contrary to previous findings (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lovato, Gradisar, et al., 2013; Saxvig et al., 2012). However, the DSWPD group did report a higher prevalence of napping, so it is possible that adolescents use naps, rather than stimulants, to overcome negative daytime consequences.

**Are cognitive "insomnia" processes responsive to chronobiological treatment?**

Adolescents with DSWPD completed three weeks of light therapy, which resulted in significantly advanced sleep timing (i.e., SOT, WUT) and improved sleep quality (i.e., SOL, TST), across treatment and follow-up. Improvements in daytime functioning (i.e., daytime sleepiness, fatigue, functional impairment) were also evident.

In terms of cognitive "insomnia" processes, measures of repetitive negative thinking decreased over time, and scores were comparable to those of good sleepers by the 3-month follow-up. Physiological pre-sleep arousal and distress showed a similar pattern of improvement. However, there was no significant change over time for the objective measure of sleep-related attentional bias. This is not surprising, given the lack of between-group differences at baseline. There was some evidence of the amelioration of sleep-onset misperception across treatment (i.e., 27min reduction); however, relative to the good sleepers, a meaningful discrepancy was still present post-treatment (i.e., 36min).

In investigating how changes in sleep, daytime functioning and insomnia symptoms are interrelated, we provide some evidence for links between cognitive "insomnia" processes (Harvey, 2002). As sleep onset time advanced, and sleep onset latency reduced, repetitive negative thinking declined. Repetitive negative thinking and physiological arousal improved similarly (Harvey, 2002). Reductions in distress were closely aligned with reductions in repetitive negative thinking (Harvey, 2002). However changes in distress (i.e., sleep threat) were not related to changes in sleep-related attentional bias (Marchetti et al., 2006).

In terms of improvements in sleep onset misperception, there was a moderate relationship with improvement in cognitive arousal. It is possible that unhelpful styles of thinking, could be implicated in the development and maintenance of sleep misperception (Harvey, 2002). The present study therefore adds to evidence from the recent study by

Herbert and colleagues (2017), who found a relationship between pre-sleep arousal and misperception of total sleep time. Alternatively, it is possible that simply recording one's sleep over time can improve the accuracy of sleep perception (Acebo, Carskadon, Time, & Min, 1997).

**Clinical Implications**

Findings from the present study suggest that cognitive "insomnia" processes may play an important role in DSWPD (Richardson et al., 2016). As such, we suggest that these processes be assessed when DSWPD is suspected. Assessment of cognitive processes could occur through informal questioning (i.e., "*Do you experience intrusive thoughts or worry when trying to fall asleep*?", "*Is there anything that you do or think to try and improve your sleep?*"), or through standardised self-report questionnaires. Appropriate measures include those used in the present study (i.e., Insomnia Severity Index) and potentially those not used (i.e., Dysfunctional Beliefs and Attitudes about Sleep Scale, Morin, 1993). By assessing cognitive "insomnia" processes, psychoeducation at the commencement of therapy could be case-conceptualised (i.e., individually tailored to target safety behaviours, such as napping, technology and stimulant use and sleep-related monitoring, such as clock-watching; Harvey, 2006). Cognitive "insomnia" processes should also be monitored across treatment. The present study illustrates that these processes are responsive to chronobiological treatment. However, some variables remained elevated relative to good sleepers, which may increase adolescents' risk of relapse. Indeed, poor treatment outcome or relapse of DSWPD is common following chronobiological treatment (Abu-Salah & Auger, 2013; Alvarez et al., 1992; Richardson, Cain, et al., Under Review; Saxvig et al., 2014) and the failure to address cognitive sleep-disordered processes in treatment may be a contributing factor for relapse. Treatment approaches combining light therapy and cognitive behavioural therapy (CBT) have produced meaningful improvements in sleep, daytime functioning and insomnia symptoms

for adolescents and young adults with DSWPD (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Importantly, young adults receiving combined light therapy and CBT appear to have superior long-term outcomes (i.e., six months), relative to those who received light therapy alone (Danielsson et al., 2015).

If cognitive sleep-disordered processes remain elevated following chronobiological treatment, clinicians could use cognitive restructuring (Belanger, Savard, & Morin, 2006; Harvey et al., 2007), mindfulness (Gross et al., 2011; Larouche, Cote, Belisle, & Lorrain, 2014; Ong, Shapiro, & Manber, 2009; Waloszek et al., 2015), mental imagery (Harvey & Payne, 2002) and/or relaxation training to target repetitive negative thinking and physiological hyper-arousal. Psychoeducation about the role of sleep-related monitoring in perpetuating sleep disturbance may facilitate behaviour change (i.e., eliminating clock-watching) by increasing the client's awareness of unhelpful patterns of thinking and behaviour (Neitzert Semler & Harvey, 2004a). Although not yet widely available, novel treatments to target sleep-related attentional bias (i.e., computer based training) may be available to clinicians in the near future (Milkins et al., 2016). Behavioural experiments could also be used to target dysfunctional beliefs about sleep (Bennett-Levy et al., 2004) and correct sleep misperception (i.e., by providing feedback about the accuracy of self-reported sleep; Tang & Harvey, 2006). In summary, adjunct cognitive behavioural therapy strategies could be case-conceptualised to target any remaining cognitive sleep-disordered processes, to improve long-term outcomes for DSWPD adolescents.

**Limitations and Future Directions**

An extensive battery of self-report and objective measures were used to investigate cognitive "insomnia" processes in the present study. We did not want to burden participants further by comprehensively measuring sleep-related beliefs, which could be assessed in

future research. Some measures were only completed at baseline (i.e., FIRST, SAMI, safety behaviours), or from baseline until post-treatment (i.e., sleep misperception) to minimise participant burden. As adolescents with DSWPD differed from good sleepers in regard to their vulnerability to stress, sleep-associated monitoring and sleep misperception, it would be useful to know how these processes may change across the course of treatment and follow-up.

This is particularly the case for attentional bias, as the objective dot-probe task did not appear to be as sensitive as the self-reported Sleep-Associated Monitoring Index. It is possible that time of day compromised the ecological validity of this measure, as the dot-probe task was completed in the morning, rather than during the more pertinent time in the evening (Milkins et al., 2016; Richardson et al., 2015). Finally, actigraphy is the most ecologically valid and cost effective way of measuring sleep objectively. As such, actigraphy data were compared with sleep diary reports to calculate sleep misperception scores. This method has clinical utility (Tang & Harvey, 2006); however from a research perspective, it would be important to replicate our findings using more rigorous methods (i.e., home-based polysomnography).

**Conclusions**

The present study suggests that a number of cognitive "insomnia" processes exist for young people diagnosed with the circadian sleep disorder DSWPD. Many of these processes are amendable to chronobiological treatment; however residual symptoms may place adolescents at risk of poor treatment outcome or relapse. Therefore, it is important for clinicians to assess and track changes in cognitive "insomnia" processes over treatment and address these as part of case-conceptualised therapy, if indicated. Combining cognitive behavioural therapy with light therapy has resulted in more promising long-term outcomes

for adolescents and young adults with DSWPD (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). However, more clinical trials measuring cognitive "insomnia" processes are needed in order to understand whether individuals with DSWPD can benefit from adjunct cognitive behavioural therapies.

# Chapter 7

## Discussion on a Randomised Controlled Trial of Bright Light Therapy and Physical Activity for Delayed Sleep-Wake Phase Disorder in Adolescents

# Discussion on a Randomised Controlled Trial of Bright Light Therapy and Physical Activity for Delayed Sleep-Wake Phase Disorder in Adolescents

## Summary of Findings

Overall, this thesis provides further evidence for the efficacy of bright light therapy for the treatment of adolescent DSWPD. Across treatment and follow-up, adolescents had earlier sleep timing (i.e., sleep onset time, wake-up time), reduced sleep onset latency and increased total sleep time on school days, and improved daytime functioning (i.e., daytime sleepiness, fatigue, functional impairment; **Chapter 4**). Although a review of the literature provided initial evidence that appropriately timed physical activity may phase advance circadian and sleep timing (**Chapter 2**), the manipulation attempted in the randomised controlled trial did not result in an objective increase in physical activity. Consequently, it is unclear from this thesis whether physical activity can supplement bright light therapy to improve outcomes for adolescents with DSWPD.

Theoretical explanations and the empirical literature suggest that adolescents with DSWPD would have impaired cognitive performance. However, a comparison with good-sleeping adolescents highlighted a lack of statistically significant differences in working memory and processing speed (**Chapter 5**). There was some evidence for small-to-moderate performance decrements on the task which placed a higher cognitive load on adolescents (Operation Span Task), which suggests further research is needed to confirm this effect. Results from the randomised controlled trial (RCT) of light therapy and morning activity highlighted significant improvement in processing speed and some measures of working memory across treatment and follow-up. Although there was weak evidence for a relationship between changes in sleep and changes in working memory, relationships between sleep and processing speed were more consistent.

Some researchers have called for further research investigating the aetiology of DSWPD (Micic, Lovato, Gradisar, Ferguson, et al., 2016) and a review of the literature provided emerging evidence for the role that cognitive "insomnia" processes may play in the development and maintenance of DSWPD (**Chapter 3**). A comparison of good-sleeping adolescents and adolescents with DSWPD demonstrated that cognitive "insomnia" processes (i.e., repetitive negative thinking, physiological arousal, distress, selective attention and monitoring, sleep misperception, safety behaviours) exist in DSWPD (**Chapter 6**). Data collected as part of the RCT of light therapy and morning activity provided insight into whether standard chronobiological treatments reduce these cognitive "insomnia" processes. Indeed, repetitive negative thinking (i.e., catastrophising, pre-sleep cognitive arousal), physiological arousal, distress and sleep misperception showed improvement across treatment. Changes between some sleep and cognitive "insomnia" processes were inter-related, providing initial evidence that theoretical models of insomnia may also be applicable to DSWPD. Importantly, sleep onset latency, distress and sleep misperception remained elevated, relative to good-sleeping adolescents, and as such, it is possible that residual cognitive "insomnia" symptoms may place adolescents at increased risk of DSWPD relapse. Indeed, a large proportion of adolescents (38%) required treatment in addition to the three sessions of light therapy and activity. At the 3-month follow-up, 17% of adolescents met full DSWPD criteria and partial relapse of DSWPD symptomology was common (i.e., 38% of adolescents reported sleep-onset difficulties and 63% reported difficulty awakening; **Chapter 4**).

## Contribution of Thesis Findings to What We Know

### Confirmation of Past Research

Over the past decade, the field of psychology has been challenged to improve the replicability of published research findings (Ioannidis, 2005). For example, 92% of published psychological studies found confirmation of hypotheses; a proportion which is much higher than in other disciplines (e.g., space science, 70%) and unlikely to be a true reflection of the research being undertaken (Asendorpf et al., 2013). There are a number of risk factors for "false" research findings, including small sample size, small effect size, financial prejudice and having a large number of research groups within the same scientific field (Ioannidis, 2005). In order to increase confidence in research findings, and to infer causality, replication is needed (Moonesinghe, Khoury, & Janssens, 2007).

To this end, the present thesis aimed to replicate research findings relating to the efficacy of bright light therapy for adolescent DSWPD (**Chapter 4**). Importantly, the RCT reported upon in this thesis recruited the largest sample of adolescents with DSWPD to date and did not receive external funding, thus reducing bias from stakeholders with a vested interest (e.g., Re-Time Pty Ltd., who sell the Re-Timers used in this thesis). There are currently only three research groups who have conducted trials for adolescent DSWPD, with the current RCT recruiting the largest sample to date. The small number of research groups investigating treatment for adolescent DSWPD, minimises competition and subsequently, the appeal of refuting findings published in prestigious journals (Ioannidis, 2005). Importantly, the present RCT is the second to be conducted at the Child & Adolescent Sleep Clinic in Australia, and builds upon evidence from the first Australian trial (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011) and trials conducted in Norway (Saxvig et al., 2014;

Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013) and Sweden (Danielsson et al., 2015).

In relation to effect sizes, the sleep field is unique in that treatment outcomes (e.g., sleep timing and total sleep time) are often reported in time, which is an inherently meaningful unit of measurement (i.e., allows for easier comparisons). Following two weeks of treatment with bright white light therapy (i.e., active treatment), adolescents and young adults in the Norwegian study advanced their bed time by 56mins and their rise time by 2hr 57mins, however obtained 81mins less total sleep (Saxvig et al., 2014). Similarly, in the Swedish study, adolescents and young adults receiving bright white light therapy (i.e., without CBT) advanced their sleep onset time by 1hr 48mins and their wake-up time by 2hr 8mins, and maintained sleep duration (i.e., one minute less than baseline; Danielsson et al., 2015). Importantly, both of these trials averaged sleep data over the whole week (i.e., seven days), rather than focusing on impairment experienced on school nights only (i.e., Sun-Thurs), as was the case for the two Australian trials (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; **Chapter 4**). At pre-treatment, adolescents in the present trial woke early to attend school, which limited the potential for improvement (i.e., after sleeping in and gradually advancing wake-up times, adolescents returned to their usual school day wake-up time). Therefore, averaging sleep data across the school week would reduce treatment effects on wake-up time. Conversely, treatment effects for total sleep time would be inflated.

In the first Australian trial, adolescents advanced their school night sleep onset time by 38mins, their school morning rise time by 26mins and increased their school night total sleep time by 60mins, following eight weeks of bright white light therapy and CBT (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Comparable results were seen in the current RCT, which administered light therapy (green, red), without CBT, over three

consecutive weeks of treatment (**Chapter 4**). Adolescents advanced their school night sleep onset time by 55mins, their school morning wake-up time by 39mins and increased their school night total sleep time by 25mins. Importantly, treatment outcomes were maintained or improved upon (i.e., TST) from post-treatment to the 3-month follow-up.

Despite slight variations in effect sizes between studies, likely due to differences in sleep diary calculation (e.g., overall vs school night averaging) and length of therapy, these results suggest that bright light therapy can meaningfully improve sleep timing and school night sleep duration for adolescents with DSWPD. These collective results also suggest that 2-3 weeks of light therapy may be sufficient to improve the sleep of adolescents with DSWPD, which is particularly significant given participants in the current trial had experienced their sleep problem for approximately 3.5yrs. The RCT presented in this thesis plays an important role in replicating results from previously conducted trials and serves to increase our confidence in the efficacy of bright light therapy for the treatment of adolescent DSWPD.

### Extension to Knowledge

#### *Method of Bright Light Administration*

This thesis extends upon current knowledge by evaluating the utility of a newly developed portable bright light device (Re-Timer; Re-Time Pty Ltd., Adelaide, Australia). There is evidence that 1hr of short wavelength light (500nm) emitted by the Re-Timer device delays circadian timing in good sleepers, when worn on two consecutive nights (i.e., 46min delay in DLMO, Lovato & Lack, 2015). Additionally, early prototypes of the Re-Timer glasses (525nm) have shown to produce phase advances of the circadian rhythm following use for two hour on two consecutive mornings (i.e., 35-40min advance in DLMO, Wright et al., 2004). Most recently, adolescents with delayed sleep timing have benefited, in terms of

sleep onset latency and total sleep time, from using the green Re-Timer device as part of a school-based sleep education program (Bonnar et al., 2015). However, prior to this thesis, the Re-Timer device had not been evaluated in clinical samples. Therefore, this thesis extends our knowledge by being the first large clinical trial to administer bright light therapy for adolescent DSWPD via a portable light device.

As previously mentioned, it is difficult to directly compare treatment outcomes between the four RCTs for adolescent DSWPD; however, effect sizes from the current thesis are comparable to previous findings. Although beyond the scope of the present thesis, preliminary data suggests that portable bright light emitting devices may be as effective as clinically-endorsed methods of artificial light administration (i.e., light box/ lamp) in the treatment of adolescent DSWPD (**Chapter 4**; Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014). Light boxes and lamps are currently the only light emitting devices that are clinically recommended for the treatment of DSWPD, and practice guidelines are unlikely to be changed unless a direct comparison of devices (i.e., portable devices vs light box/lamp) is undertaken (Auger et al., 2015). Therefore, a comparison of light emitting devices may be a future priority. As it has been hypothesised that portable light devices may promote treatment compliance (Burgess & Emens, 2016), this avenue of research may also provide further insight into whether treatment compliance influences treatment outcome (Danielsson, Jansson-Fröjmark, Broman, & Markström, 2016).

### *Wavelength of Bright Light Administration*

Another novel aspect of this thesis is the comparison of the efficacy of short wavelength (green) light therapy vs long wavelength (red) light therapy. This comparison was particularly important given the call to replicate experimental (i.e., lab-based) research

findings within less tightly controlled settings (i.e., with clinical samples at home; Auger et al., 2015). There is a robust experimental evidence base for the human circadian rhythm's optimal sensitivity to short wavelength light, with larger phase advances occurring following exposure to short (i.e., blue-green) versus long wavelength (i.e., red) or broad spectrum (i.e., white) light (Warman et al., 2003; Wright et al., 2004). This is thought to occur, due to greater melatonin suppression from shorter, rather than longer wavelength or broad spectrum light (Cajochen et al., 2005; Gabel et al., 2017). Importantly, the human phase response curve to low intensity short wavelength light (480nm, 11.2 lux) overlaps with the phase response curve to high intensity white light (10,000lux; Rüger et al., 2013). This may have benefits for chronobiological treatment, in that lower, more tolerable, intensities of short wavelength light may exert the same influence as higher intensity white light (Rüger et al., 2013). Consequently, treatment compliance and outcomes may be better for lower intensity short wavelength light therapy, relative to high intensity bright broad spectrum light therapy.

Importantly for adolescent DSWPD, experimental research has also shown that short wavelength light (470nm) is more effective at increasing daytime alertness and performance than long wavelength light (600nm), even when administered at lower intensities (Cajochen et al., 2005; Lockley et al., 2006; Revell, Arendt, et al., 2006). These experimental findings suggest that adolescents with DSWPD may benefit more, in terms of their sleep and daytime functioning, from short wavelength bright light therapy, relative to long wavelength light therapy.

The Norwegian research group were the first to compare different "wavelengths" of light in their randomised controlled trial of bright light therapy for adolescent DSWPD, by comparing bright white light (10,000lux) with dim red light (400lux; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). However, contrary to their hypotheses, both light conditions advanced sleep and circadian timing and improved

daytime functioning (i.e., daytime sleepiness, fatigue, cognitive function), with no between-group differences (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). These results reinforced the importance of translating experimental research findings into less tightly controlled clinical settings. However, as a singular finding, replication was needed. Additionally, participants in the Norwegian trial were "re-randomised" following the initial two weeks of treatment, to either i) receive bright white light therapy and melatonin for three months or to ii) cease treatment altogether. As such, it is unclear whether the wavelength of light had differential effects on sleep, circadian timing and daytime functioning in the longer term.

The RCT reported in this thesis aimed to replicate research findings from the Norwegian group, whilst also *extending* upon knowledge by comparing short wavelength light (~500nm, ~112lux per diode) with long wavelength light (~643nm, ~54 lux per diode). Results from the trial highlighted no difference between the light groups in outcomes relating to subjective and objective sleep, or daytime functioning (i.e., morning alertness, daytime sleepiness, fatigue, functional impairment). Participants tended to maintain or extend upon improvements (i.e., TST) across the follow-up period, however, the lack of between-group differences remained consistent. As a limitation of the current study, measures of circadian timing (e.g., DLMO, core body temperature) were not taken, and thus it is unclear whether the wavelength of light had a differential effect on circadian timing (i.e., phase shifts).

Although there is a robust evidence base for heightened sensitivity of the human circadian rhythm to short wavelength light, there is some suggestion that red light may also phase delay (Mien et al., 2014) and phase advance (Zeitzer, Kronauer, & Czeisler, 1997) the human melatonin rhythm. However, the intensity of light is likely to greatly influence this relationship (Figueiro, Bierman, Plitnick, & Rea, 2009). Additionally, there is some evidence that moderate intensity red light (i.e., 213lux) can induce physiological alertness (i.e.

increases heart rate) and improved performance (Figueiro et al., 2009; Sahin, Wood, Plitnick, & Figueiro, 2014). Mien and colleagues (2014) have hypothesised that red light may influence sleep and alertness via cone photoreceptors in the eye, rather than via melanopsin[10], with multiple photoreception pathways exerting control over the circadian system. Consequently, it is possible that the red light administered in the Norwegian and Australian trials was of a sufficient intensity to exert influence over adolescents' sleep timing and daytime functioning (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013).

Alternatively, it is possible than changes in behaviour (i.e., having a fixed earlier sleep schedule) contributed to improvements in sleep and daytime functioning. For example, the small pilot trial (*N*=10) investigating the portable light device Luminette® did not appear to gradually advance adolescents' sleep timing alongside bright light administration (Langevin et al., 2014). Instead adolescents were instructed to minimise light exposure within their bedrooms after 9pm and wear the Luminette® light device (emitting either blue enriched white light, or orange light) for 45mins promptly after awakening in the morning. With such a small sample size (*N*=10) the results of this study need to be interpreted with caution. Their results suggested that short wavelength enriched white light (2,000lux) was more effective at advancing sleep onset time, increasing total sleep time, and reducing daytime sleepiness, relative to long wavelength light (2,000lux, Langevin et al., 2014). The primary difference between Langevin and colleagues' (2014) protocol, and the protocol used in previous RCTs is the inclusion of sleep scheduling. In the Australian trials, adolescents were instructed to sleep in on the first day of therapy and wake up 30min earlier each subsequent day (**Chapter 4**; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Similarly, the Norwegian group instructed adolescents to sleep in on the first day of treatment

[10] Melanopsin is a photopigment in retinal ganglion cells within the eye, and is most sensitive to short wavelengths of light (Morin & Allen, 2006).

and wake up 60mins earlier each subsequent morning (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Danielsson and colleagues (2015) scheduled adolescents' wake-up time as 8.5hrs after their dim light melatonin onset time. Contrastingly, Langevin and colleagues (2014) did not appear to provide adolescents with any instructions about when they should wake. Therefore, it is possible that the behavioural components of bright light therapy (i.e., fixed advanced sleep times) reduce the differential effect of the wavelength of light administered. Given the limited data to support this hypothesis, further research is needed.

Two independent clinical trials have now shown that the wavelength of light administered in bright light therapy did not significantly alter treatment outcomes (**Chapter 4**; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). These results suggest the need to refine our understanding of how and why bright light therapy is *clinically* effective, particularly given that much of our current understanding is informed by tightly controlled lab studies (Auger et al., 2015). Consequently, results from the present thesis may indicate a need to return to a broader research question, "*What components of bright light therapy drive improvements in sleep and daytime functioning for adolescents with DSWPD*". To answer this question, it may be useful to dismantle the components of bright light therapy. For example, future research could compare the efficacy of i) sleep scheduling alone, ii) sleep scheduling with evening light restriction, and iii) sleep scheduling with evening light restriction and post awakening (short vs long wavelength) light. It has been over 35 years since DSWPD was first described within the literature (Weitzman et al., 1981) and it has long been known that adolescents' have particular vulnerability to DSWPD (Carskadon et al., 1993). Thus, it is surprising there are so few randomised controlled trials which have evaluated treatment for adolescent DSWPD.

Consequently, it should be a priority of future research to gather more evidence about the efficacy of bright light therapy.

### *Physical Activity as an Adjunct to Bright Light Therapy*

In addition to the novelty of evaluating alternate methods of bright light administration, this thesis includes the first clinical trial to evaluate the utility of physical activity as a chronobiological treatment for adolescent DSWPD. Previous to this trial, physical activity interventions have been implemented with non-clinical samples of adolescents (e.g., Kalak et al., 2012). Due to a lack of clinical trials, there is no clinical recommendation regarding the use of exercise as a treatment component (Auger et al., 2015). However, a literature review outlined in **Chapter 2**, highlighted that nocturnal physical activity can phase delay the circadian rhythm (Baehr et al., 2003; Baehr et al., 1999; Barger et al., 2004; Buxton et al., 1997; Eastman et al., 1995; Van Reeth et al., 1994; Youngstedt et al., 2002). In contrast, exercise at other times of the day may result in phase advances (Buxton et al., 2003; Van Reeth et al., 1994), particularly if advanced in gradual daily increments (e.g., 20min/day, Miyazaki et al., 2001). There is a lack of consensus about the time of day exercise needs to be completed to yield a phase advance of circadian timing, as a phase response curve to exercise had not been comprehensively mapped and initial hypotheses were extrapolated from studies investigating nocturnal exercise (Buxton et al., 2003).

Since the publication of **Chapter 2** in 2017, unpublished data have become available which support the timing of physical activity administered in the RCT of bright light and morning activity in the present thesis (**Chapter 4**). In mapping a phase response curve to exercise, Youngstedt and colleagues (2017) found two phase advance regions; 6-9am and 1-5pm. The first phase advance portion would occur proximal to habitual wake time for normal sleepers (i.e., who slept 11pm-7am). Consequently, it is likely that adolescents with DSWPD

would need to complete exercise later, closer to their natural wake-up time. These data, along with the results from Miyazaki and colleagues (2001), provide evidence that physical activity completed after natural awakening, then gradually advanced by small daily increments (i.e., 30min/day), may facilitate circadian phase advances for adolescents with DSWPD. This protocol overlaps with bright light therapy (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Gradisar et al., 2014) and consequently, reinforces why the two chronobiological approaches were combined in this thesis.

Results from the RCT in this thesis (**Chapter 4**), found no evidence that physical activity provided additional benefits for adolescents with DSWPD, relative to bright light therapy alone. However, posteriori analyses showed no objective increases in physical activity in the one hour after waking for adolescents completing morning exercise, relative to adolescents within the sedentary conditions. As a limitation, physical activity was scored using movement data collected from actigraphy. Although this method of measurement is more ecologically valid, relative to empirically used measures (e.g., heart rate, maximal oxygen consumption), it is likely not as sensitive a measure of exercise intensity (Lang, Kalak, et al., 2016). Regardless, it is premature to draw conclusions about the potential utility of morning physical activity from these initial data. Instead, this thesis highlights a range of new research questions.

It is likely that physical activity was timed appropriately to induce phase advances in circadian timing (Youngstedt et al., 2017), however, the physical activity manipulation did not result in objective increases in movement. Therefore, future research may aim to manipulate the duration, intensity and modality of the exercise stimulus, to align more closely with empirical research; for example, 45-60min moderate intensity exercise, on a treadmill (Barger et al., 2004; Buxton et al., 1997; Eastman et al., 1995; Youngstedt et al., 2017). A well-controlled lab based study currently underway is administering exercise of this duration,

intensity and modality, with a sub-clinical sample of adolescents with extreme evening chronotype (Lang, Richardson, & Gradisar, 2016). Results from this study will shed further light on whether adolescents with DSWPD may benefit from scheduled physical activity in the morning, of a longer duration and higher intensity.

### *Cognitive Performance in Adolescents with DSWPD*

Prior to this thesis, only one trial had measured the effect of treatment for DSWPD on cognitive performance (Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). This is an important area of enquiry, as adolescents with DSWPD are at increased risk of school lateness, absenteeism, dropout and poor academic performance (Dewald et al., 2010; Giannotti et al., 2002; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Lack, 1986; Meijer et al., 2000; Saxvig et al., 2012), and cognitive performance is hypothesised to link adolescent sleep with these adverse academic outcomes. However, a comparison of cognitive performance between adolescents diagnosed with DSWPD and good-sleeping adolescents had never been undertaken, thus highlighting another novel contribution from this thesis.

Results from the between-group comparison (**Chapter 5**) demonstrated the importance of conducting research in clinical samples, rather than inferring knowledge from non-clinical, cross-sectional, and experimental research. Namely, this study revealed a lack of between-group differences in cognitive performance, on measures assessing working memory and processing speed. However, a small-to-moderate difference in performance on the Operation Span Task (i.e., a dual-task, with high cognitive load), suggests that further research is needed to elucidate whether differences in cognitive functioning exist. For example, future research may wish to use more complex and standardised school-like tasks to assess performance (e.g., Wechsler Individual Achievement Test, Wechsler, 2009), at the

beginning of the school day (e.g., 8.30am, Short, Gradisar, Lack, Wright, Dewald, et al., 2013), to increase the ecological validity of research findings (Curcio, Ferrara, & De Gennaro, 2006).

Adolescent's performance on tasks assessing working memory (i.e., Operation Span, Letter Number Sequencing) and processing speed (i.e., Digit-Symbol Substitution) improved across treatment and follow-up, with small-to-moderate effects ($d$= 0.45-0.57). The inclusion of a control group (i.e., red light therapy, sedentary activity) was designed to help elucidate practice effects from treatment effects. However, as there were no between-group differences in treatment outcomes, other forms of statistical analysis were employed. Correlations between changes in sleep and cognitive performance highlighted evidence for a relationship between wake-up time and working memory (i.e., Op Span, Digit Span Backwards). However, relationships between changes in sleep (i.e., sleep onset time, total sleep time), daytime functioning (i.e., sleepiness, fatigue, impairment) and processing speed (i.e., Digit-Symbol Substitution) were more consistent. These results support empirical literature, which suggests that sleep interventions may improve adolescents' processing speed (Dewald-Kaufmann et al., 2013); thus, highlighting a potential point of intervention for educational psychologists. Higher order cognitive abilities rely upon processing speed, therefore, processing speed is thought to influence academic achievement through its effect on intelligence (Rindermann & Neubauer, 2004). Consequently, sleep interventions may have positive flow on effects for adolescents' school performance, through improved processing speed (Dewald-Kaufmann et al., 2013; Dewald et al., 2010; Rindermann & Neubauer, 2004).

### *Cognitive Insomnia Symptoms in DSWPD*

Two recent reviews, focusing on the aetiology of DSWPD (Gradisar & Crowley, 2013; Micic, Lovato, Gradisar, Ferguson, et al., 2016) highlighted the role that psychological

factors may play in the development and maintenance of DSWPD. For example, sleep-onset difficulties, physiological hyperarousal and avoidant morning behaviours may contribute to the disorder, and relevant to adolescents, repetitive negative thinking may perpetuate sleep-onset difficulties (Gradisar & Crowley, 2013; Micic, Lovato, Gradisar, Ferguson, et al., 2016). Many of these factors are common to conceptualisations of insomnia (Espie, 2002; Espie, Broomfield, et al., 2006; Harvey, 2002; Lundh & Broman, 2000). However, this thesis is the first to propose the role of cognitive "insomnia" processes in DSWPD and to provide systematic evidence (i.e., comparing groups across time points and using standardised measures) for this hypothesis.

More specifically, a review of the literature (**Chapter 3**) provided initial evidence of cognitive and physiological pre-sleep arousal (e.g., Barbero, 2013, unpublished thesis; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Hiller et al., 2014), sleep-related attentional bias (MacMahon et al., 2006; Marchetti et al., 2006), misperception of sleep and daytime functioning (Billows, 2009, unpublished thesis; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), dysfunctional beliefs (Wilhelmsen-Langeland et al., 2012) and safety behaviours (Jansson-Fröjmark, Danielsson, Broman, & Markström, 2016; e.g., Lovato, Gradisar, et al., 2013; Pasch et al., 2010; Saxvig et al., 2012) in DSWPD. Importantly, many of these studies were conducted with adolescent samples. However, this thesis included the first systematic comparison of cognitive "insomnia" processes between adolescents with DSWPD and good sleepers (**Chapter 6**). Results showed that adolescents with DSWPD experienced elevated repetitive negative thinking, physiological arousal and distress, sleep-related monitoring and sleep-onset misperception, relative to good-sleeping adolescents. Additionally, there was some evidence of safety behaviours, such as napping and technology use in those with DSWPD (i.e., internet and iPod use).

Following treatment, adolescents with DSWPD reported reduced repetitive negative thinking and physiological arousal and distress. Sleep-onset misperception also showed signs of amelioration across three weeks of treatment. As per theoretical models of insomnia (Espie, 2002; Harvey, 2002), there was some evidence that repetitive negative thinking (i.e., catastrophising, cognitive arousal) was related to sleep-onset difficulties. Physiological arousal and distress improved alongside repetitive negative thinking, and repetitive negative thinking was associated with sleep-onset misperception. These novel research findings suggest that cognitive "insomnia" processes are inter-related and are implicated in DSWPD. Consequently, these findings may have implications for the theoretical conceptualisation of DSWPD.

## Theoretical Implications

### Aetiology of DSWPD

Many theories pertaining to the aetiology of DSWPD have limited empirical support. For example, there is conflicting evidence regarding the role of abnormal phase angle of entrainment[11] (e.g., Watanabe et al., 2003; Wilhelmsen-Langeland et al., 2012). Only one study has suggested sleep pressure may accumulate more slowly (Uchiyama et al., 1999). Finally, there are currently no studies evaluating sensitivity to morning light in DSWPD.

Results from this thesis (**Chapter 4**) support the theory that circadian misalignment is a primary contributing factor for DSWPD (Micic, Lovato, Gradisar, Ferguson, et al., 2016). Although circadian timing was not measured, sleep timing can be viewed as a proxy (Ancoli-Israel et al., 2003; Youngstedt, Kripke, Elliott, & Klauber, 2001). Three weeks of chronobiological treatment was sufficient to advance sleep timing, increase total sleep time and improve daytime functioning, which suggests circadian timing phase advanced. There is

[11] Phase Angle of Entrainment refers to the relationship between sleep timing (e.g., sleep onset and offset time) and circadian timing (e.g., core body temperature minimum).

some evidence that DSWPD may develop in late childhood (i.e., from 10yrs of age; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), and a majority of adolescents with DSWPD cite gradual onset of their sleep problem (**Chapter 4**). Sleep timing (and presumably circadian timing) in the general population continues to become progressively later until the age of approximately 20 years (Roenneberg et al., 2004). Consequently, circadian rhythm delay is likely a contributing factor to adolescent DSWPD.

Cognitive "insomnia" symptoms reduced alongside improvements in sleep timing. Despite cognitive "insomnia" symptoms improving, some still remained elevated following chronobiological treatment (e.g., sleep onset latency >30 min, distress, sleep misperception). Nevertheless, the present thesis has provided evidence that cognitive "insomnia" symptoms exist in those diagnosed with DSWPD, and thus has the potential to add to current theoretical explanations of DSWPD. However, there still remains a gap in current knowledge regarding how this might be the case. For example, these data suggest that cognitive processes in DSWPD are inter-related with, but perhaps not exclusive to the physiological mechanisms underlying delayed sleep timing (i.e., circadian delay).

Current theoretical models suggest that physiological changes underlie later bedtimes and wake times in adolescence. However, given the need to attend school, adolescents with DSWPD may attempt sleep at an earlier, more socially-desirable bedtime (i.e., during the wake maintenance zone; Micic, Lovato, Gradisar, Ferguson, et al., 2016). Consequently, earlier bedtimes may contribute to sleep-onset difficulties. With repeated "exposure" to being alert when attempting sleep, sleep-onset difficulties may become conditioned, providing multiple (and in many cases, consecutive) opportunities for cognitive "insomnia" processes to develop. Within the insomnia literature, cognitive processes are thought to contribute to the maintenance of disturbed sleep (Harvey, 2002). For example, repetitive negative thinking prevents the cognitive and physiological de-arousal necessary for sleep onset to occur, thus

extending latency to sleep (Espie, 2002; Harvey, 2002). As sleep disturbance continues, individuals are likely to become increasingly distressed, which inadvertently reinforces sleep-related attentional bias. Attentional biases increase the likelihood that threat stimuli are detected, and thus, may serve to worsen repetitive negative thinking and sleep-onset difficulties. For example, individuals may preferentially attend to the eye region of their face upon awakening (Akram, Ellis, Myachykov, & Barclay, 2017) and deduce "*I look tired, I didn't sleep well and am unlikely to perform well today*". Maladaptive beliefs about sleep (i.e., "*In order to get more sleep, I need to spend more time in bed*", "*I need caffeine to help me function*"), may lead to behaviour change (i.e., safety behaviours); such as, going to bed earlier, or consuming high levels of caffeine throughout the day (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Safety behaviours can maintain sleep disturbance by increasing the probability that the feared outcome will occur (i.e., increased sleep effort extends sleep latency and caffeine use further prevents pre-sleep de-arousal, thus, extending sleep latency; Ree & Harvey, 2004). Cognitive "insomnia" processes are thought to culminate in the misperception of the sleep deficit (i.e., sleep-onset difficulties) and daytime impairment (i.e., daytime sleepiness, school performance; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), which can worsen repetitive negative thinking and its' sequelae (Harvey, 2002). In this regard, the vicious cycle of cognitive "insomnia" processes may maintain or worsen sleep disturbance in adolescents. More broadly, the existence of cognitive "insomnia" processes may also account for why DSWPD often persists into adulthood despite the amelioration of physiological contributing factors (i.e., delayed circadian timing in adolescence; Paine, Fink, Gander, & Warman, 2014; see Appendix C[12]).

[12] Although not included within the body of this thesis, a qualitative study was conducted during this thesis by the candidate that provides evidence of insomnia symptoms for adults diagnosed with a circadian rhythm disorder.

In summary, whilst it is probable that physiological factors (i.e., circadian timing, sleep homeostasis) precipitate delayed sleep timing, sleep-onset difficulties and the development of sleep-disordered cognitive processes in adolescents, it is likely that both cognitive and physiological factors interact in the perpetuation of DSWPD. To this end, a model for explaining contributing factors to adolescent DSWPD is proposed here (see Figure 7.1).

*Figure 7.1* A conceptualisation of how cognitive "insomnia" processes may be implicated in DSWPD. Green arrows indicate the temporal sequence implicated in the development of DSWPD, red arrows represent how DSWPD is maintained. Note: # = This aetiological theory currently has limited empirical support (Uchiyama et al., 1999).

Importantly, an Australian Research Council funded study (Project ID: DP150100215) currently being undertaken at Flinders University, aims to elucidate when physiological changes in sleep homeostasis and the circadian rhythm occur, in relation to the development of sleep problems during early adolescence (i.e., before or after onset of a sleep problem). Depending upon the outcome of this research, the theorised temporal sequence of events leading to the development and maintenance of DSWPD (Figure 7.1) may need to be revised. In the RCT, 28% of families reported that adolescents had previously experienced a sleep problem (e.g., Chronic Insomnia Disorder) during childhood (**Chapter 4**). This preliminary evidence suggests it is possible that the development of cognitive “insomnia” processes preceded the onset of DSWPD for some adolescents. However, with a lack of prospective data tracking sleep across childhood and adolescence, it is premature to speculate

further. Consequently, the field of paediatric sleep research eagerly awaits results from longitudinal studies.

### Approaches to the Treatment of DSWPD

To date, researchers and clinicians (including this thesis) have taken a diagnosis-specific approach to the treatment of adolescent DSWPD. Regarding the aetiology of DSWPD, the "circadian misalignment" hypothesis has the strongest evidence base to date (Gradisar & Crowley, 2013; Micic, Lovato, Gradisar, Ferguson, et al., 2016). Consequently, each of the four RCTs for adolescent DSWPD have administered one or more chronobiological agents, in order to advance circadian timing and ameliorate misalignment between the circadian rhythm and the 24-hr social world (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; **Chapter 4**). However, the accumulating evidence suggests that more than one aetiological factor may contribute to the development and maintenance of DSWPD (i.e., physiological and psychological). Therefore, it is likely that treatment addressing just one underlying cause (i.e., circadian misalignment) will not ameliorate the disorder. Indeed, there is some evidence to suggest that treatments aiming to advance circadian timing fail to produce robust improvements, particularly in the long-term (Abu-Salah & Auger, 2013; Alvarez et al., 1992; Saxvig et al., 2014). Results from the present RCT (**Chapter 4**) support these empirical findings, in that partial or full relapse of DSWPD symptomology was common and 38% of adolescents required additional treatment by the 3-month follow-up.

To date, the primary strategy to improve the overall efficacy of treatment for adolescent DSWPD has been to combine treatment components. For example, trials have compared the relative and combined efficacy of the chronobiological treatments of bright light therapy (BLT) and melatonin (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig,

Pallesen, Nordhus, Vedaa, et al., 2013), or chonobiological and psychological treatments of BLT and CBT (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011) and in this thesis, BLT and physical activity (**Chapter 4**). Results from these trials suggest that, overall, treatment is effective. However, the addition of extra treatment components has not significantly improved treatment outcomes related to sleep. For example, over two weeks of treatment, there was no difference in the efficacy of BLT, exogenous melatonin administration, or an approach combining the two (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Bright light therapy combined with cognitive behavioural therapy (CBT) has shown to be an effective treatment for adolescent DSWPD (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). However, in attempting to disentangle the effects of BLT and cognitive behavioural therapy, Danielsson and colleagues (2015) found that the addition of CBT did not improve outcomes related to sleep. Worth noting though is that the addition of CBT did result in greater improvements in symptoms of depression and anxiety (Danielsson et al., 2015). This finding is clinically meaningful given the high rate of psychiatric comorbidity in DSWPD (Reid et al., 2012; Sivertsen et al., 2015), and that psychiatric comorbidity could lead to DSWPD relapse (e.g., social withdrawal associated with depression could lead to sleeping in and circadian delay). It is possible that CBT supplements BLT because the two treatment components are targeting different aetiological factors (i.e., psychological and circadian, respectively), unlike other treatment approaches (i.e., BLT and melatonin, BLT and physical activity). This initial finding suggests that supplementing chronobiological treatment with psychological treatments (e.g., CBT) may lead to better outcomes. However, many more studies are needed in this area to confirm the hypothesis of using a psychobiological approach to the treatment for adolescent DSWPD.

Importantly, Danielsson and colleagues (2015) suggested that not all adolescents with DSWPD may have benefited from the inclusion of CBT. Therefore, future research could aim to identify which adolescents benefit from additional treatment (e.g., adolescents with a comorbid psychological condition). Additionally, as CBT includes a great number of strategies (i.e., psychoeducation, sleep regularisation, stimulus control, progressive muscle relaxation, cognitive restructuring; Blake, Sheeber, Youssef, Raniti, & Allen, 2017), it is unclear which are the active components of treatment. Future research could aim to tease apart treatment components via dismantling research designs.

One major flaw of disorder-specific treatment is that individuals commonly experience more than one disorder (e.g., DSWPD, depression, chronic fatigue) at the same time (**Chapter 4**; Dudley et al., 2011; Sivertsen et al., 2015) and current theories pertaining to DSWPD do not explain how other conditions (i.e., depression, anxiety) may contribute to the development and maintenance of the disorder. In this regard, a transdiagnostic approach to treatment could be preferable to a disorder-specific model (Dudley et al., 2011). Given the bidirectional relationship between emotion regulation difficulties and sleep (e.g., Alvaro, Roberts, & Harris, 2013; American Psychiatric Association, 2013; Lovato & Gradisar, 2014), and the neurobiological correlates (i.e., genes, neurotransmitters) between sleep disturbance and other psychological disorders (Lamont, Legault-Coutu, Cermakian, & Boivin, 2007; Mansour et al., 2006; Serretti et al., 2003), Harvey (2011) has proposed that sleep is a transdiagnostic process globally related to psychopathology. This theoretical reframing has led to the development of a 4-10 session transdiagnostic intervention for adolescent sleep (TranS-C Youth; Harvey, 2016). This intervention includes functional analysis, psychoeducation, motivational interviewing and goal setting across treatment, to target irregular sleep timing, pre-sleep hyperarousal, morning resistance, daytime impairment, dysfunctional beliefs and relapse (Harvey, 2016). Additional modules targeting reduced sleep

quality, delayed sleep phase and sleep-related worry can be added if clinically indicated (i.e., these are optional modules). In this regard, transdiagnostic interventions for adolescent sleep inherently encourage case conceptualisation, as opposed to manualised disorder-specific approaches. A transdiagnostic approach allows for the treatment of comorbid sleep problems (i.e., DSWPD with comorbid sleep-onset insomnia), and targets processes common to many other physical or psychological conditions. Additionally, transdiagnostic treatments may reduce the burden on clinicians, in that they would learn to administer one treatment, rather than multiple protocols (Harvey, 2016). However, further research is needed to evaluate whether transdiagnostic treatment for adolescent sleep is efficacious, and ultimately cost effective, particularly in relation to traditional disorder-specific treatment (Dudley et al., 2011). Consequently, the sleep health field eagerly awaits results from clinical trials evaluating transdiagnostic treatments for sleep (e.g., Harvey et al., 2016). In the future, cost benefit analysis of each treatment approach may need to be undertaken, with particular focus placed on balancing long-term treatment outcomes with the time investment and financial cost associated with each treatment.

**Clinical Implications**

Findings from this thesis may have implications for the way in which treatment for adolescent DSWPD is administered. Results from the RCT (**Chapter 4**) provided the first evidence regarding the efficacy of portable light emitting glasses for the treatment of adolescent DSWPD. Although portable devices have not been directly compared with traditional methods of bright light administration (i.e., light boxes and lamps), these initial data may help to increase clinician's confidence in their use. Additionally, results from this thesis could help to inform future clinical practice guidelines (Auger et al., 2015).

In addition, data from this thesis may help to inform clinicians' judgements regarding the type of light administered in bright light therapy. In particular, growing evidence suggests the wavelength of light is not the most important component of bright light therapy for adolescent DSWPD (**Chapter 4**; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). These findings may influence whether clinicians recommend the purchase of commercially available short-wavelength light emitting devices (i.e., Re-Timer, Luminette®), as opposed to traditional methods of bright light delivery (e.g., natural ambient light, white light boxes/lamps).

This thesis also provides insight into whether physical activity could be used to supplement traditional chronobiological agents (i.e., bright light therapy, exogenous melatonin) in the treatment of adolescent DSWPD. Despite reports of good compliance in the RCT (**Chapter 4**), the physical activity manipulation was not of sufficient strength to induce objective increases in adolescents' movement. The addition of physical activity, therefore, did not improve treatment outcomes. In terms of application to clinical practice, it is unlikely that ~30min of mild post-awakening physical activity has a significant impact upon the sleep or daytime functioning of adolescents with DSWPD. It is possible that exercise of a longer duration, or higher intensity, can exert influence over the adolescent circadian system (**Chapter 2**). However, more clinical trials are needed before clinical recommendations can be made (Auger et al., 2015). Importantly, given evidence that nocturnal activity can phase delay the human circadian rhythm, clinicians may need to assess adolescents' pre-sleep routine and advise against physical activity close to bedtime.

This thesis also presents the first systematic evidence that cognitive "insomnia" processes may be implicated in the development and maintenance of adolescent DSWPD (**Chapter 6**). Consequently, it is important for clinicians to assess cognitive insomnia symptoms when DSWPD is suspected. Standardised measures administered in this thesis

(e.g., PSAS, catastrophising interview, ISI, actigraphy for sleep misperception) and some that were beyond the scope of this thesis (e.g., Dysfunctional Beliefs and Attitudes about Sleep Scale, Sleep-Related Behaviours Questionnaire) could be used to assist in the assessment of cognitive "insomnia" symptoms (Hiller et al., 2014; Morin, 1993; Nicassio et al., 1985; Ree & Harvey, 2004). Informal questioning could also be used; for example, "*Do you experience intrusive thoughts or worry when trying to fall asleep?*"[13] Standardised measures have inherent benefits in that they can be used to reliably track clients' progress across treatment. Suboptimal improvement on these measures may "flag" to the clinician that additional treatment may be indicated. If left untreated, it is possible that residual symptoms of insomnia may increase the risk of DSWPD relapse.

A number of additional treatment components could be used to supplement traditional chronobiological treatments for adolescent DSWPD. As per clinical guidelines for insomnia, treatment components should be informed by the individual's symptom pattern (Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008). Repetitive negative thinking could be targeted in treatment via cognitive restructuring (i.e., identifying and challenging negative automatic thoughts, generating alternate thoughts, etc.; Belanger et al., 2006), mental imagery and/or mindfulness, as cognitive therapies and mindfulness-based cognitive therapies have shown to improve adult insomnia, by reducing repetitive negative thinking (Harvey et al., 2007; Yook et al., 2008). Treating cognitive hyperarousal should inadvertently target physiological hyperarousal, since the relationship between these processes is theorised to be causally bidirectional (Espie, 2002). However, heightened pre-sleep physiological arousal can be directly targeted in treatment through relaxation training (e.g., progressive muscle relaxation; Nicassio, Boylan, & McCabe, 1982). Relaxation training is a recommended treatment for insomnia (Morgenthaler et al., 2006), and has good empirical support

[13] Further lines of questioning can be found in supplementary materials in Appendix C.

(Lichstein, Riedel, Wilson, Lester, & Aguillard, 2001; Means, Lichstein, Epperson, & Johnson, 2000).

Psychoeducation and behavioural experiments can be used to target sleep-related attentional bias and monitoring. For example, clock watching could be targeted by reviewing and collecting evidence for and against the function of this strategy (e.g., Tang, Schmidt, & Harvey, 2007). Behavioural experiments could be used to better understand the consequences of threat-related monitoring. For example, testing how monitoring for internal sensations (e.g., energy/ fatigue) vs neutral external stimuli (e.g., flowers, people) can influence perceptions of daytime functioning (Harvey et al., 2007). Once further clinical testing has been conducted, novel methods of altering attentional biases (i.e., computerised attention-bias modification training) may also become more readily available to clinicians (Milkins et al., 2016).

Similar to sleep-related attentional bias, behavioural experiments can be used to target maladaptive sleep-related beliefs and sleep misperception. For example, clinicians could directly test their clients' beliefs, such as, "*In order to gain more sleep, I need to go to bed earlier*". Alternatively, socratic questioning could be used to challenge beliefs (e.g., "*What do you think the impact of seeing yourself as having a sleep problem is*?"; Lichstein, 2017). In relation to sleep misperception, the therapist and client can collaboratively compare subjective (i.e., sleep diary) and objective (i.e., actigraphy) measures of sleep, until they become more closely aligned. By providing feedback regularly throughout treatment, it may be possible to improve sleep misperception more quickly, compared to when a chronobiological treatment is provided alone (i.e., greater slope of change; **Chapter 6**). The current thesis also provides evidence that the cognitive performance of adolescents with DSWPD is not markedly different from their good-sleeping peers, despite the perception of impairment (**Chapter 5**). Consequently, these results in themselves can be used by clinicians

to challenge common beliefs and misperceptions regarding daytime functioning (e.g., that sleep-onset difficulties are going to influence school performance; Hiller et al., 2014). Within the insomnia field, it has been hypothesised that sleep *perceptions* (i.e., rather than actual deficit) may impair treatment response and increase the likelihood of relapse (Lichstein, 2017). Additionally, change in sleep-disordered cognitions is related to better long term maintenance of improvements in insomnia (Morin et al., 2002). Therefore, these findings highlight how important the inclusion of treatment strategies targeting cognitive "insomnia" processes may be for adolescents with DSWPD.

**Limitations of Research**

The interpretations that can be drawn from this thesis were somewhat limited by the exclusion of additional measures. For example, it is unclear whether underlying circadian timing phase advanced following treatment, as objective measures of circadian timing (i.e., 24-hr core body temperature, dim light melatonin onset; Bjorvatn & Pallesen, 2009) were not taken. Additionally, wrist actigraphy was used to measure exercise intensity, rather than more robust measures, such as heart rate, or maximal oxygen consumption. Wrist actigraphy was likely a less sensitive measure of exercise intensity and thus, may not have been able to detect small differences in physical activity between the treatment groups. Adolescents were asked to report daily activity duration; however, this measure may have been susceptible to socially desirable reporting. It is also a limitation that ambient light within participants' homes was not measured. Participants were instructed to limit ambient light during light therapy (i.e., by closing blinds); however it cannot be ruled out that changes in sleep and daytime functioning were driven by exposure to natural light, rather than light emitted by the Re-Timer device. Although this additional data could have provided further insight into why there were no between-group differences in treatment outcomes, the clinical utility of such information is questionable, given that ambient light is almost always going to be present in adolescents'

homes. In summary, the interpretation of results from this thesis may have been limited by the exclusion of additional measures. However, these measures were not included as i) we did not have access to such materials (i.e., actigraphy watches that measure light), ii) inclusion would have been financially expensive (i.e., DLMO sampling and assaying), iii) these measures would have decreased the ecological validity of the study and most importantly, iv) inclusion would have led to undue participant burden.

Another limitation of the research design is the failure to include an inactive waitlist control. The sedentary red light therapy group was designed as an active control group. However, as all treatment groups improved similarly, this limited the conclusions that could be drawn, particularly in relation to cognitive performance (i.e., controlling for practice effects). On the other hand, there are pitfalls of an inactive waitlist condition, including the inability to account for non-specific intervention effects (e.g., therapeutic alliance, belief in treatment; Cuijpers et al., 2012). Additionally, the inclusion of another treatment group would have further reduced statistical power. Regardless, these methodological issues need to be taken into consideration when planning clinical trials for adolescent DSWPD in the future.

**Future Directions for Research**

In addition to the potential avenues for future research that have been outlined earlier in the Discussion, there are a number of other areas that warrant further consideration. Namely, as a result of this thesis, the field may like to further investigate i) methods for disseminating bright light therapy, ii) ways to prevent long-term relapse of DSWPD, iii) how cognitive "insomnia" processes could be implicated in the development and maintenance of DSWPD and iv) the relationship between sleep and emotional wellbeing in adolescence.

### Bright Light Therapy

Results from this thesis add to empirical evidence supporting the use of bright light therapy for adolescent DSWPD. Once an efficacious treatment has been established, emphasis may be placed on dissemination. From a global perspective, bright light therapy is being successfully implemented in a number of sleep clinics around the world, with clinical trials being conducted in Australia, Norway and Sweden (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Asian adolescents have later bedtimes and greater school day sleep restriction than their counterparts in North America and Europe (Gradisar, Gardner, et al., 2011). However, controlled clinical trials for adolescent DSWPD have not been conducted in Asian countries to date and it is unclear to what extent these adolescents may benefit from bright light therapy as a single treatment component (Okawa, Uchiyama, Ozaki, Shibui, & Ichikawa, 1998). In the present trial, adolescents commenced bright light therapy during the school term. However, there may be cultural barriers to applying findings from this thesis to other countries (e.g., due to early school start times in the USA (e.g., 7.45am, Short, Gradisar, Lack, Wright, Dewald, et al., 2013) and academic pressures in Asian countries, meaning adolescents cannot miss school during treatment (Liu, Liu, Owens, & Kaplan, 2005; Liu, Uchiyama, Okawa, & Kurita, 2000)). Therefore, future clinical trials for adolescent DSWPD could be rolled out in other countries (i.e., Asia, USA).

More generally, given the large number of adolescents who could be experiencing DSWPD worldwide (e.g., ~6.5 million adolescents), it should be a priority of future research to investigate ways to widely disseminate treatment. Importantly, doing so may also reduce the time taken for adolescents to receive treatment (i.e., ~3.5 years in this thesis; **Chapter 4**),

which may help to reduce the burden of disordered sleep on adolescents in the wider community.

As adolescents may initially report symptoms of sleep disturbance to their medical practitioner, it may be possible to implement bright light therapy within this clinical setting. More specifically, medical staff (i.e., nurses) could be trained to assess DSWPD and administer manualised bright light therapy, as per current clinical practice guidelines (e.g., Gradisar et al., 2014). Empirically, training has increased the recognition and diagnosis of sleep problems within a general medical setting (Zozula, Rosen, & Jahn, 2005). Additionally, the dissemination of treatment in medical settings has shown to be clinically effective for adult insomnia (Espie, Inglis, Tessier, & Harvey, 2001). Alternatively, given that a majority of adolescents attend school, it may be possible to train teachers or school counsellors to assess for and treat adolescent DSWPD. Although not directly targeting adolescents with DSWPD, school based interventions encouraging morning bright light exposure have shown promise (Bonnar et al., 2015; Cain, Gradisar, & Moseley, 2011; Moseley & Gradisar, 2009), thus highlighting another area for future enquiry.

Adolescents' have an affinity for technology, thus it may be more viable to disseminate bright light therapy via an internet-based intervention. Generally, internet-based psychological interventions have shown to produce similar effect sizes to traditional face-to-face therapy (Barak, Hen, Boniel-Nissim, & Shapira, 2008). More specifically, internet-based interventions are effective for the treatment of infant and adult sleep problems (e.g., Mindell et al., 2011; Ritterband et al., 2009), and more recently, insomnia in adolescents (de Bruin, Bögels, Oort, & Meijer, 2015). Internet-based interventions are relatively inexpensive (i.e., do not require a sleep specialist to administer treatment) and can be provided to adolescents in rural or remote areas, who otherwise would go without treatment. Thus, the potential of internet-delivered bright light therapy warrants investigation. In summary, bright light

therapy is an effective treatment for adolescent DSWPD and has the potential to be disseminated widely. Consequently, it should be a priority of future research to investigate ways to increase adolescents' access to treatment for DSWPD.

### Relapse Prevention

Although bright light therapy for adolescent DSWPD has the most empirical support to date (i.e., relative to other treatment approaches, such as exogenous melatonin and chronotherapy), adolescents remain susceptible to poor treatment outcome and relapse (**Chapter 4**; Saxvig et al., 2014). The long-term efficacy of transdiagnostic intervention for adolescent sleep is not yet known. Therefore, emphasis should be placed upon further refining disorder-specific treatment for adolescent DSWPD. In this respect, one aspect of treatment that could be improved upon is relapse prevention. Although 2-3 weeks of chronobiological treatment is likely to improve the sleep and daytime functioning of adolescents with DSWPD, findings from Saxvig and colleagues (2014) suggest that the sleep problem is likely to return without the inclusion of a relapse prevention module of treatment. This study also demonstrated that long-term melatonin and bright light administration facilitates the maintenance of treatment gains (Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). However, adolescents are unlikely to be motivated to implement treatment indefinitely and in any case, the long-term impacts of exogenous melatonin use on young people (i.e., <18yrs) are unknown (Kennaway, 2015). Consequently, alternate methods of relapse prevention may need to be considered.

In their trial, Danielsson and colleagues (2015) formulated an individualised coping program and included content on how to deal with relapses. At the 6-month follow-up, approximately 65% of adolescents who received light therapy (without CBT) were falling asleep after 1.00am and approximately 60% were waking up after 9.00am (Danielsson et al.,

2015). These cut-off scores were designed to provide insight into symptom remission. Consequently, a majority of adolescents in this sample were experiencing DSWPD symptomology at 6-months post-treatment. In the current randomised controlled trial, relapse prevention instructions were more prescriptive (i.e., not tailored to the individual), with all adolescents being instructed to minimise evening light, maximise morning light and limit sleep-ins to no more than two hours after weekday wake-up time. Of the participants who completed the 3-month follow-up, 38% of adolescents reported sleep-onset difficulties and 63% reported difficulty awakening. Perhaps more importantly, 38% of the entire sample had returned for additional treatment by the 3-month follow-up. These data suggest that more effective relapse prevention could be provided, to improve the long-term efficacy of bright light therapy.

In order to improve the relapse prevention module of DSWPD treatment, research could be conducted to identify "risk factors" for relapse. Given the current lack of relapse prevention research within the field of adolescent DSWPD, future research may need to be informed by other fields. For example, early models of relapse prevention for addiction identified a number of "risk factors", including negative emotional states, inadequate motivation, poor initial response to treatment, lack of coping skills, and inadequate social support (Marlatt & George, 1984). Once risk factors for DSWPD relapse are identified, relapse prevention content can be tailored accordingly. For example, if low motivation is identified as a risk factor for relapse, then motivational interviewing could be integrated into relapse prevention.

Another approach, which has been adopted empirically, includes the addition of "booster" sessions to the therapy protocol (Marlatt & George, 1984). Booster sessions may be useful in that DSWPD symptomology could be detected and addressed earlier, to prevent full DSWPD relapse. Many participants who returned for additional chronobiological treatment

in the current RCT expressed a lack of confidence in being able to re-implement treatment without the support of a therapist (Micic, Richardson, & Gradisar, 2016). Therefore, therapist-led booster sessions (i.e., monthly) may help adolescents to maintain treatment gains. However, therapist-led sessions are unlikely to increase adolescents' confidence in being able to manage their own sleep problem in the long term; which is an important skill to develop given the probability of relapse. Therefore, adolescents could be encouraged to review their own progress in weekly home therapy sessions. To facilitate home therapy sessions, adolescents could collaboratively generate a therapy blueprint, with their sleep therapist, prior to completing treatment. The therapy blueprint would assist adolescents in identifying symptoms of a setback (i.e., relapse) and could outline a plan to overcome setbacks.

As previously mentioned, adolescents may be inclined to engage with internet-based treatment (de Bruin et al., 2015), therefore the therapy blueprint could be digitised. One study investigating young people's attitudes towards smartphone applications to promote behaviour change, showed that participants valued the ability to record and track behaviour (Dennison, Morrison, Conway, & Yardley, 2013). Therefore, a relapse prevention smartphone application could be developed. In particular, the smartphone application could prompt adolescents to track DSWPD symptomology (e.g., through weekly check-ins) and encourage adolescents to address symptoms of a setback as soon as they arise (i.e., strategies could be recommended depending upon the symptomology reported). In this regard, the smartphone application could make the therapy blueprint and weekly home therapy sessions more engaging, accessible and interactive. Consequently, adolescents may feel more supported to re-instigate treatment on their own, without the need to return for therapist-lead treatment. Future research could investigate whether a relapse prevention smartphone application could improve the long-term efficacy of DSWPD treatment.

### Cognitive "Insomnia" Processes

As bright light therapy does not directly target cognitive "insomnia" processes, it is possible that residual insomnia symptoms limit treatment success, and also increase the risk of DSWPD relapse. However, as this thesis is the first to systematically investigate cognitive "insomnia" processes in DSWPD, further research is needed. Initially, future research could aim to replicate findings from this thesis, by measuring cognitive "insomnia" symptoms in independent samples of adolescents and adults with DSWPD. Measures used in the current thesis to assess repetitive negative thinking, physiological arousal and distress would likely be appropriate for use in future research. However, measurement of sleep-related attentional bias and sleep misperception may need to be further refined. In particular, given the discrepancy in findings between subjective (i.e., Sleep Associated Monitoring Index) and objective measures of attentional bias (i.e., dot-probe), objective measures could instead be measured in the evening, when sleep-threat is most salient (Milkins et al., 2016; Richardson et al., 2015). Additionally, the words used in dot-probe were based on adults' pre-sleep cognitions (Wicklow & Espie, 2000) and thus, stimuli may need to be adapted for an adolescent sample. One way to overcome this limitation is to present pictorial stimuli, which may be less susceptible to age differences (Jansson-Fröjmark, Bermås, & Kjellén, 2013). Indeed, the insomnia field has moved towards measuring attentional bias to visual sleep-related cues, such as the eye-region of the face (Akram et al., 2017), and the clock (Woods et al., 2009). Importantly, adolescents with DSWPD reported selectively monitoring the clock and time (**Chapter 6**), which suggests such stimuli may be appropriate. In regard to sleep misperception, future research could aim to replicate and extend upon findings from this thesis by more accurately measuring sleep with polysomnography (i.e., rather than with actigraphy). Doing so will help to elucidate whether individuals with DSWPD misperceive their sleep and also, whether treatments for DSWPD correct this misperception.

Future research could also measure sleep-related beliefs and safety behaviours in adolescents with DSWPD. Sleep-related beliefs were not systematically assessed in this thesis and although common safety behaviours were measured pre-treatment (e.g., napping, substance use), it is unclear what function these behaviours served. Therefore, future research could also aim to link cognitions (e.g., "*My sleep affects how I function*") with behaviours (e.g., napping, caffeine use), and measure these processes across treatment. Replication will help to increase our confidence that cognitive "insomnia" processes are implicated in DSWPD. If future research confirms these initial findings, research effort could be funnelled into investigating whether adding treatment components to target cognitive "insomnia" processes (i.e., those outlined in *Clinical Implications*) improves the long-term efficacy of DSWPD treatment.

**Sleep and Emotion Wellbeing**

As highlighted in the **Introduction**, sleep loss can affect adolescents' ability to regulate their emotions (Brand, Kirov, et al., 2016; Dahl, 1999). Most relevant to DSWPD, later bedtimes and reduced sleep duration emerge as risk factors for emotional problems, such as anxiety, depression, bipolar disorder, internalising problems and suicidality (Asarnow et al., 2014; Chelminski, Ferraro, Petros, & Plaud, 1999; Gangwisch et al., 2010; Gaspar-Barba et al., 2009; Gau et al., 2007; Kitamura et al., 2010; Staton, 2008). Similar to past research, adolescents with DSWPD in the current study commonly reported co-morbid psychological problems, such as anxiety and depression (Danielsson, Markström, et al., 2016; Kripke et al., 2008; Reid et al., 2012; Saxvig et al., 2012; Sivertsen, Harvey, Pallesen, & Hysing, 2015; Thorpy, Korman, Spielman, & Glovinsky, 1988). A meta-analysis investigating the bidirectional relationship between sleep and depression found consistent evidence that sleep disturbance is a precursor for the development of depression (Lovato & Gradisar, 2014). However, there was limited evidence for the inverse relationship. To explain this relationship,

Lovato and Gradisar (2014) theorised that wakefulness in bed (e.g., increased sleep onset latency due to circadian delay), gives rise to repetitive negative thinking (i.e., rumination), which over time can worsen sleep disturbance, and lead to common symptoms of depression, such as irritable mood, decreased motivation, fatigue and poor concentration.

Further studies are needed to elucidate the bidirectional relationship between sleep and emotional wellbeing in adolescence. Although psychological comorbidity was measured pre-treatment, it is a limitation of the current research that emotional wellbeing (e.g., depression, anxiety) was not assessed following treatment, and at the 1- and 3-month follow-ups. Therefore, it is unclear whether adolescents' emotional wellbeing improved alongside improvements in sleep and daytime functioning, as empirical literature would suggest. Future clinical trials could include follow-up measures of emotional outcomes, such as depression and anxiety, with particular importance placed on understanding *how* sleep and mood are interrelated. To this end, future research could formally test Lovato and Gradisar's (2014) theoretical model. That is, evaluating whether repetitive negative thinking mediates the relationship between sleep disturbance (i.e., wakefulness in bed) and depression in this at risk population.

**Conclusion**

A significant number of adolescents experience Delayed Sleep-Wake Phase Disorder (DSWPD) worldwide. DSWPD is chronic and unlikely to spontaneously remit during adolescence. Therefore it is important to ensure that efficacious treatments are available. After bright light therapy, adolescents had advanced sleep timing, reduced sleep-onset difficulty (i.e., SOL) and increased total sleep time on school nights. Daytime functioning (i.e., morning alertness, daytime sleepiness, fatigue, functional impairment) and some measures of cognitive performance also improved across treatment and follow-up. However,

as there were no statistically significant differences between the cognitive performance of adolescents with DSWPD and good sleepers, it is unclear how meaningful the improvements in working memory and processing speed were, and thus further research is needed.

More broadly, results from this thesis suggest the need to better understand why bright light therapy works (i.e., disentangling the relative contribution of sleep scheduling, evening light restriction, post-awakening light administration). Further investigation of the optimal wavelength and method of bright light administration is also warranted.

This thesis investigated whether physical activity could supplement bright light therapy. Similarly to previous studies (i.e., those adding exogenous melatonin, cognitive behavioural therapy), this approach did not significantly alter treatment outcomes (Danielsson et al., 2015; Saxvig et al., 2014; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). However, due to methodological limitations, further investigation of physical activity is warranted.

Of note, this thesis provides evidence that adolescents with DSWPD experience cognitive "insomnia" symptoms. Although these processes are amenable to bright light therapy, residual symptoms of insomnia may place adolescents at risk of DSWPD relapse. Consequently, future research could also investigate whether long-term treatment outcomes are improved through the addition of targeted "cognitive" treatment components.

Findings from this thesis highlight the need to question common theoretical assumptions about adolescent DSWPD. For example, that DSWPD has no cognitive component; that cognitive performance is impaired in adolescents with DSWPD and that short wavelength light is superior in bright light therapy. In this regard, this thesis raises more research questions than it can answer and highlights a number of avenues for future research - but these are questions for another time (and possible another PhD candidate).

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# Appendices

# Appendix A

Supplementary Tables for Chapter 4.

A Randomised Controlled Trial of Bright Light Therapy and Morning Activity for Adolescents with Delayed Sleep-Wake Phase Disorder.

Table S4.1

*Means and Standard Errors of Sleep and Daytime Functioning Outcomes from Pre-treatment to 3-month Follow-up, by Treatment Group.*

| | **Green BLT + Sedentary Activity** | **Green BLT + Morning Activity** | **Red LT + Sedentary Activity** | **Red LT + Morning Activity** | *p* Values | |
|---|---|---|---|---|---|---|
| | Mean±SE | Mean±SE | Mean±SE | Mean±SE | Time | Group x Time |
| ***Sleep Diary (School Nights)*** | | | | | | |
| **Bed Time (hh:mm±mm)** | | | | | | |
| Pre-Treatment | 22:39±26 | 23:39±28 | 23:17±27 | 22:37±27 | | |
| Post-Treatment | 22:31±26 | 23:10±28 | 22:47±26 | 22:23±26 | | |
| 1-Month Follow-Up | 22:42±27 | 23:35±29 | 22:54±27 | 22:23±24 | | |
| 3-Month Follow-Up | 22:28±27 | 23:50±29 | 23:03±28 | 22:36±29 | 0.123 | 0.607 |
| **Lights Off Time (hh:mm±mm)** | | | | | | |
| Pre-Treatment | 00:04±27 | 00:02±29 | 00:01±28 | 23:40±28 | | |
| Post-Treatment | 23:36±27 | 23:32±29 | 23:21±27 | 23:13±27 | | |
| 1-Month Follow-Up | 23:43±26 | 23:44±28 | 23:22±25 | 22:56±27 | | |
| 3-Month Follow-Up | 23:35±27 | 00:07±29 | 23:26±28 | 23:05±29 | **0.049** | 0.676 |
| **Sleep Onset Time (hh:mm±mm)** | | | | | | |
| Pre-Treatment | 01:09±29 | 01:20±31 | 01:48±30 | 00:44±30 | | |
| Post-Treatment | 00:09±33 | 00:19±36 | 00:20±34 | 00:20±33 | | |
| 1-Month Follow-Up | 23:58±24 | 00:04±26 | 23:50±23 | 23:40±27 | | |
| 3 -Month Follow-Up | 00:11±25 | 00:36±27 | 00:09±25 | 23:35±27 | **<0.001** | 0.481 |
| **Wake-Up Time (hh:mm±mm)** | | | | | | |
| Pre-Treatment | 8:39±28 | 9:28±30 | 8:42±29 | 8:35±29 | | |
| Post-Treatment | 7:50±35 | 8:54±38 | 7:56±35 | 8:13±35 | | |
| 1-Month Follow-Up | 7:52±27 | 8:33±29 | 8:06±26 | 7:57±57 | | |
| 3-Month Follow-Up | 7:58±18 | 8:49±20 | 7:38±19 | 7:38±38 | **0.001** | 0.961 |
| **Out of Bed Time (hh:mm±mm)** | | | | | | |
| Pre-Treatment | 9:09±27 | 10:05±29 | 9:04±28 | 8:52±28 | | |
| Post-Treatment | 8:09±35 | 9:15±39 | 8:10±36 | 8:21±35 | | |
| 1-Month Follow-Up | 8:28±28 | 8:53±30 | 8:24±27 | 8:10±30 | | |
| 3-Month Follow-Up | 8:23±19 | 8:56±40 | 7:43±20 | 7:49±23 | **<0.001** | 0.900 |
| **Sleep Onset Latency (mins)** | | | | | | |
| Pre-Treatment | 63±19 | 78±20 | 108±20 | 65±19 | | |
| Post-Treatment | 31±18 | 64±19 | 58±18 | 67±18 | | |
| 1-Month Follow-Up | 30±14 | 51±15 | 40±13 | 61±16 | | |
| 3-Month Follow-Up | 43±13 | 48±14 | 34±14 | 38±16 | **0.008** | 0.398 |
| **Wake After Sleep Onset (mins)** | | | | | | |
| Pre-Treatment | 12±9 | 26±9 | 5±9 | 14±9 | | |
| Post-Treatment | 3±6 | 16±7 | 2±6 | 6±6 | | |
| 1-Month Follow-Up | 5±3 | 7±3 | 3±3 | 5±3 | | |
| 3-Month Follow-Up | 3±4 | 13±5 | 2±4 | 4±4 | 0.074 | 0.642 |
| **Total Sleep Time (hours)** | | | | | | |
| Pre-Treatment | 7.3±0.4 | 7.7±0.4 | 6.7±0.4 | 7.6±0.4 | | |
| Post-Treatment | 7.7±0.5 | 8.15±0.5 | 7.6±0.5 | 7.8±0.5 | | |
| 1-Month Follow-Up | 7.7±0.4 | 8.15±0.5 | 8.2±0.4 | 7.8±0.5 | | |
| 3-Month Follow-Up | 7.1±0.4 | 8.5±0.4 | 7.7±0.4 | 8.25±0.4 | **0.021** | 0.714 |
| **Sleep Efficiency (%)** | | | | | | |
| Pre-Treatment | 81±4 | 76±4 | 75±4 | 80±4 | | |
| Post-Treatment | 89±3 | 85±4 | 85±3 | 85±3 | | |
| 1-Month Follow-Up | 87±3 | 87±3 | 89±3 | 85±3 | | |
| 3-Month Follow-Up | 88±2 | 87±2 | 88±3 | 90±3 | **<0.001** | 0.666 |

| | **Green BLT + Sedentary Activity** | **Green BLT + Morning Activity** | **Red BLT + Sedentary Activity** | **Red BLT + Morning Activity** | *p* Values | |
|---|---|---|---|---|---|---|
| | Mean±SE | Mean±SE | Mean±SE | Mean±SE | Time | Group x Time |
| ***Actigraphy (School Nights)*** | | | | | | |
| **Sleep Onset Time (hh:mm±mm)** | | | | | | |
| Pre-Treatment | 00:24±28 | 00:36±32 | 23:57±30 | 00:14±29 | | |
| Treatment Week 1 | 00:29±27 | 01:07±30 | 00:12±28 | 00:11±28 | | |
| Treatment Week 2 | 23:49±28 | 00:34±29 | 23:38±30 | 00:04±27 | | |
| Treatment Week 3 | 23:34±31 | 00:25±31 | 23:15±34 | 23:21±29 | **0.007** | 0.905 |
| **Wake-Up Time (hh:mm±mm)** | | | | | | |
| Pre-Treatment | 8:51±34 | 9:40±39 | 8:05±37 | 8:06±35 | | |
| Treatment Week 1 | 9:11±30 | 9:59±34 | 8:46±32 | 7:58±31 | | |
| Treatment Week 2 | 7:48±34 | 9:16±35 | 7:36±38 | 9:02±32 | | |
| Treatment Week 3 | 7:38±36 | 8:46±35 | 7:18±40 | 8:05±32 | **0.020** | 0.244 |
| ***Daytime Functioning*** | | | | | | |
| **Morning Alertness** | | | | | | |
| Pre-Treatment | 2.78±0.27 | 3.13±0.31 | 2.88±0.29 | 3.06±0.32 | | |
| Treatment Week 1 | 3.40±0.23 | 3.27±0.24 | 3.70±0.23 | 3.33±0.24 | | |
| Treatment Week 2 | 3.37±0.29 | 3.62±0.31 | 3.20±0.28 | 3.81±0.33 | | |
| Treatment Week 3 | 3.40±0.35 | 3.68±0.35 | 3.30±0.35 | 3.78±0.40 | **0.010** | 0.215 |
| **Daytime Sleepiness (PDSS)** | | | | | | |
| Pre-Treatment | 22.17±1.28 | 21.02±1.38 | 21.72±1.38 | 21.59±1.39 | | |
| Post-Treatment | 15.58±2.04 | 15.67±2.10 | 15.19±2.00 | 16.63±2.02 | | |
| 1-Month Follow-Up | 17.29±2.32 | 12.86±2.54 | 14.30±2.12 | 15.60±2.34 | | |
| 3-Month Follow-Up | 17.26±1.94 | 14.90±1.97 | 13.42±2.11 | 12.73±2.46 | **<0.001** | 0.517 |
| **Fatigue (FFS)** | | | | | | |
| Pre-Treatment | 9.98±1.10 | 8.58±1.17 | 9.43±1.20 | 9.93±1.21 | | |
| Post-Treatment | 6.75±1.30 | 7.06±1.34 | 5.69±1.26 | 7.39±1.28 | | |
| 1-Month Follow-Up | 5.94±1.46 | 5.27±1.63 | 6.71±1.24 | 5.94±1.47 | | |
| 3-Month Follow-Up | 7.96±1.39 | 1.03±1.41 | 6.45±1.47 | 4.61±1.68 | **<0.001** | 0.248 |
| **Functional Impairment (SDS)** | | | | | | |
| Pre-Treatment | 26.48±2.54 | 19.55±2.83 | 22.25±2.94 | 22.83±2.93 | | |
| Post-Treatment | 14.68±3.56 | 15.09±3.56 | 11.91±3.51 | 18.03±3.42 | | |
| 1-Month Follow-Up | 14.08±3.54 | 9.47±3.89 | 12.15±2.91 | 16.39±3.65 | | |
| 3-Month Follow-Up | 18.40±4.13 | 14.06±4.26 | 13.82±4.34 | 14.92±4.73 | **<0.001** | 0.592 |

## Appendix B

Supplementary Tables for Chapter 6.

Cognitive "Insomnia" Processes in Delayed Sleep-Wake Phase Disorder: Do they exist and are they Responsive to Chronobiological Treatment?

Table S6.1

*Correlations between Changes in Sleep and Daytime Functioning and Cognitive "Insomnia" Processes from Pre-treatment to Post-treatment.*

| | Repetitive Negative Thinking | | Arousal and Distress | | Sleep-Related Attention and Monitoring | Sleep Misperception |
|---|---|---|---|---|---|---|
| | Catastrophic Thinking | Cognitive Pre-Sleep Arousal | Physiological Pre-sleep Arousal | Distress | Sleep-Related Attentional Bias Score | Sleep Onset Misperception |
| | *r* | *r* | *r* | *r* | *r* | *r* |
| SOL (SD) | 0.16 | **0.31*** | 0.11 | 0.12 | 0.04 | **0.63*** |
| SOT (SD) | **0.33*** | **0.46*** | 0.23 | 0.17 | -0.15 | **0.46*** |
| SOT (Act) | 0.37 | **0.52*** | 0.34 | 0.26 | -0.32 | -0.09 |
| WUT (SD) | 0.15 | 0.18 | **0.30*** | 0.16 | -0.19 | 0.20 |
| WUT (Act) | 0.29 | 0.19 | 0.22 | 0.24 | -0.44 | 0.10 |
| TST (SD) | -0.21 | **-0.35*** | -0.05 | -0.07 | -0.02 | **-0.43*** |
| Sleepiness | 0.15 | **0.74**** | **0.30*** | **0.66**** | -0.03 | -0.30 |
| Fatigue | 0.19 | **0.62**** | 0.13 | **0.59**** | -0.04 | -0.14 |
| Impairment | 0.18 | **0.60**** | **0.31*** | **0.67**** | -0.32 | 0.211 |

*$p<0.05$ **$p<0.001$

Table S6.2

*Correlations between Changes in Cognitive "Insomnia" Processes from Pre-treatment to Post-treatment.*

| | Repetitive Negative Thinking | | Arousal and Distress | | Sleep-Related Attention and Monitoring | Sleep Misperception |
|---|---|---|---|---|---|---|
| | Catastrophic Thinking | Cognitive Pre-Sleep Arousal | Physiological Pre-sleep Arousal | Distress | Sleep-Related Attentional Bias Score (Dot-Probe) | Sleep Onset Misperception |
| | *r* | *r* | *r* | *r* | *r* | *r* |
| Catastrophic Thinking | 1 | 0.13 | 0.15 | **0.37*** | 0.21 | 0.13 |
| Cognitive Arousal | | 1 | **0.32*** | **0.46*** | 0.12 | 0.37 |
| Physiological Arousal | | | 1 | 0.27 | -0.16 | -0.06 |
| Distress | | | | 1 | -0.24 | 0.14 |
| Sleep-Related AB | | | | | 1 | 0.22 |
| Sleep Onset Misperception | | | | | | 1 |

*$p<0.05$ **$p<0.001$

## Appendix C

Additional study conducted by the candidate during this thesis, resulting in preparation of a manuscript titled:

Insomnia and the Psychosocial Challenges of Delayed Sleep-Wake Phase Disorder in Middle Adulthood: A Mixed-Methods Approach.

Cele Richardson, Gorica Micic, Michael Gradisar.

Flinders University, College of Education, Psychology and Social Work, Adelaide, Australia

### Author Contributions

CR led study conceptualisation, design, recruitment, data collection, data analysis, results interpretation and manuscript preparation. GM assisted with data analysis, results interpretation and manuscript preparation. MG contributed to study conceptualisation, design, results interpretation and manuscript preparation.

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Richardson, C., Micic, G., & Gradisar, M. Insomnia and the Psychosocial Challenges of Delayed Sleep-Wake Phase Disorder in Middle Adulthood: A Mixed-Methods Approach.

## Abstract

**Study Objectives:** Delayed Sleep-Wake Phase Disorder (DSWPD) is thought to be caused by a delay in the circadian rhythms promoting sleep and alertness. Although DSWPD typically develops in adolescence, it can persist into adulthood despite the amelioration of age-related physiological contributing factors. Recent research has highlighted the role that cognitive "insomnia" processes (e.g., cognitive activity, physiological arousal, distress, selective attention, safety behaviours and erroneous beliefs) and psychosocial factors may play in the development and maintenance of DSWPD in adolescents and young adults; however further research is needed to understand whether these processes may contribute to the maintenance of DSWPD into middle adulthood. This preliminary study aimed to shed light on i) the percentage of adults experiencing cognitive "insomnia" processes, ii) adult's qualitative experience of "insomnia" and iii) the broader psychosocial context of sleep disturbance in DSWPD.

**Methods:** Thirty four middle-aged adults with chronic Delayed Sleep-Wake Phase Disorder (*M*=47.3yrs, *SD*= 14.1, 91% female) completed an online questionnaire comprised of multiple choice and short answer questions.

**Results:** Cognitive "insomnia" processes were common, with qualitative reports highlighting a similarity in phenomenology with insomnia. Behavioural treatments had a low level of acceptability in this sample. A number of psychosocial challenges (e.g., employment, social isolation, lack of social understanding) were identified. Participants desired more awareness, advocacy and support surrounding their sleep disorder.

**Conclusions:** These results highlight the importance of assessing the psychosocial context of DSWPD in adult clients. In addressing these factors as part of a case-conceptualised treatment, the acceptability, uptake and outcome of treatment might be improved.

## Introduction

Delayed Sleep-Wake Phase Disorder (DSWPD) is a circadian rhythm sleep disorder, whereby biological rhythms determining sleep and alertness are timed significantly later than the 24-hr social world (American Psychiatric Association, 2013). Individuals with DSWPD typically report sleep-onset difficulties and a diminished ability to awaken for morning commitments. Forced early awakening decreases total sleep time and results in significant daytime impairments (American Psychiatric Association, 2013). Onset of the disorder typically occurs in adolescence (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), as physiological changes place teens at risk of delayed bedtimes (e.g., delay in circadian timing, reduced homeostatic sleep pressure; Gradisar & Crowley, 2013). However, DSWPD can persist into adulthood, with a recent study estimating the prevalence of DSWPD in adulthood to be as high as 8.9% (Paine et al., 2014). As such, it is important to investigate what processes maintain DSWPD, given that age-related physiological contributing factors ameliorate in early adulthood (Roenneberg et al., 2004).

Historically, DSWPD has been conceptualised as a physiologically-driven sleep disorder, with relatively little input from psychological factors. However, potential cognitive and psychosocial factors which may contribute to the development and maintenance of DSWPD have been acknowledged more recently (Danielsson et al., 2015; Micic, Lovato, Gradisar, & Lack, 2016; Richardson et al., 2016; Wilhelmsen-Langeland et al., 2012; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Specifically, difficulty initiating sleep may give rise to negatively-toned cognitive activity, such as rumination and worry (Espie, 2007; Harvey, 2002; Lundh & Broman, 2000). Unsurprisingly, 89% of adolescents with DSWPD have reported experiencing a racing mind in bed (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), with a similar number reporting catastrophic pre-sleep thinking (Hiller et al., 2014). Many of the catastrophic thoughts

pertained to sleep-related effects on daytime and interpersonal functioning (Hiller et al., 2014), mirroring themes of pre-sleep cognitive activity in individuals with insomnia (Wicklow & Espie, 2000). Models of insomnia propose a bidirectional and causal relationship between cognitive and physiological pre-sleep arousal (e.g., heart rate, core body temperature, metabolic rate), in that cognitive arousal prevents physiological de-arousal and thus, sleep onset, and vice versa (Espie, 2002; Lundh & Broman, 2000). However, these relationships have received very little attention in the DSWPD field to date, and little is known about how these processes may be implicated in the maintenance of disordered sleep into adulthood.

Pre-sleep cognitive and physiological hyperarousal, when paired with distress, can lead to the development of an attentional bias towards sleep-threat stimuli (Espie, Broomfield, et al., 2006; Harvey, 2002). Importantly, selectively attending to internal (e.g., physical sensations in body) and external (e.g., the clock) sleep-related stimuli during the day and at night, can serve to worsen or maintain disturbed sleep, by: i) worsening negatively toned cognitive activity and ii) contributing to overestimation of sleep problems and associated daytime impairments (Espie, 2002; Harvey, 2002; Harvey & Tang, 2012). To date, two studies have shown that individuals with DSWPD display attentional bias to sleep-related stimuli, relative to good sleepers (MacMahon et al., 2006; Marchetti et al., 2006). Furthermore, there is some evidence that individuals with DSWPD misperceive their sleep (Billows & Gradisar, 2009, unpublished thesis, Saxvig et al., 2014) and daytime functioning (e.g., sleepiness; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), which can serve to perpetuate disturbed sleep.

Finally, models of insomnia suggest that an individual's erroneous beliefs about sleep and related safety behaviours (e.g., increased effort to sleep), can maintain disturbed sleep, by contributing to negatively toned cognitive activity and its sequelae (Espie, 2002; Harvey,

2002). One qualitative study has shed some light on beliefs held by adolescents and young adults with DSWPD (e.g., loss of control over sleep, the impact that sleep has on their mood and psychological health; Wilhelmsen-Langeland et al., 2012). However, further research is needed to gain a broader understanding of the types of sleep-related beliefs that adults with DSWPD hold. Similarly, there is some indirect evidence that adolescents with DSWPD may use maladaptive ways of coping with their sleep and daytime impairment (e.g., alcohol, Lovato, Gradisar, et al., 2013; Saxvig et al., 2012, caffeine, Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Saxvig et al., 2012, technology use, Tavernier & Willoughby, 2014 and napping , Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011), which may inadvertently be maintaining disordered sleep. However, erroneous beliefs have not yet been linked with safety behaviours, or measured in adults, within the DSWPD literature.

As highlighted by Wilhelmsen-Langeland and colleagues (2012), DSWPD is also associated with a plethora of psychosocial challenges, such as interpersonal conflict, challenges relating to self-image (e.g., *"I do look at myself as a little, a little lazy",* pp. 55*)* and an inability to attend and perform well at school or work, to name a few. However, this study was conducted with a small number of "well-functioning" adolescents with DSWPD (Wilhelmsen-Langeland et al., 2012) and as such, the present study aimed to understand the broader psychosocial issues that adults with DSWPD face (i.e., adults with more severe and/or chronic DSWPD).

Given current conceptualisations of DSWPD focus on the physiological underpinnings of the disorder, it is unsurprising that primary treatment approaches are behaviourally-based methods to manipulate underlying physiology (e.g., bright light therapy, exogenous melatonin; Saxvig et al., 2014; Sharkey et al., 2011; van Maanen et al., 2013). However, treatment compliance, drop-out and relapse are significant issues facing the field (Alvarez et al., 1992; Sack, Auckley, Carskadon, et al., 2007) and may contribute to the

persistence of DSWPD into adulthood. Poor treatment outcomes may partly be attributable to clinicians' failure to recognise and address cognitive "insomnia" processes and other psychosocial issues associated with DSWPD. More recently, two randomised controlled trials evaluated a combined cognitive and behavioural approach to the treatment of DSWPD, with promising results (Danielsson et al., 2015; Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). However, further research into the phenomenology of DSWPD is needed to refine assessment and treatment techniques to improve treatment outcomes. Consequently, this preliminary study aimed to shed light on i) the percentage of individuals experiencing cognitive "insomnia" processes, ii) individual's qualitative experience of "insomnia" and iii) the broader psychosocial challenges associated with sleep disturbance in adults with chronic DSWPD, using a sample of convenience. The study employs a mixed-methods approach, using both quantitative measures of sleep, insomnia and daytime functioning, as well as rich qualitative data from participant responses to open-ended questions. Qualitative data allows for a more comprehensive understanding of complex processes which are poorly understood; as is the case for cognitive "insomnia" symptoms in DSWPD (Curry, Nembhard, & Bradley, 2009).

## Methods

### Participants

As a preliminary study, a sample of convenience was recruited. Participants were part of an American-based international online support group for individuals with Delayed Sleep-Wake Phase Disorder and Non-24-hr Sleep-Wake Rhythm Disorder. The online community is likely to have ~1,000 members; however, a significant proportion are no longer active members. A subsample of the online community ($N$=48) expressed interest to the authors, to conduct research investigating the experience of individuals with DSWPD. Although the

subsample represent a small proportion of the total number of members of the online support group (~5%), the sample was deemed appropriate for a preliminary study examining the existence of insomnia symptoms in DSWPD in middle adulthood. Additionally, as respondents were not biased by knowing the study was investigating insomnia symptoms in DSWPD, the results are likely to be representative of other adults with DSWPD. This study was exempt from institutional review board approval as the research conducted for the online community (a not-for-profit organisation) involved subjective reports of public behaviour where human subject data were de-identified (NHMRC, ARC, & AVVC, 2007).

Of the 48 online community members who expressed interest in participating in the study, 44 participants commenced the online survey. Nine participants had incomplete responses, to the extent that prevented inclusion in data analysis. One participant was excluded as she did not currently have a sleep problem. Three participants also indicated they did not believe they currently had a sleep problem. However, the authors (CR, MG) included these participants in analyses as survey responses (e.g., sleep patterns, daytime functioning, insomnia) were consistent with a current diagnosis of DSWPD. As such, data from 34 participants were included in final analyses. Participant demographics are outlined in Table 1 in Results.

As per ICSD-3 criteria for DSWPD, participants reported moderate sleep-onset difficulties (*M*=2.15, *SD*=1.40)[14], which occurred approximately 3.5 days per week (M=3.68 days, *SD*=2.95), and difficulty waking up (*M*=3.95, *SD*=1.30)[15] (Criterion A; see Table 2). Furthermore, in line with DSM-5 diagnostic criteria for DSWPD (American Psychiatric Association, 2013), all participants reported daytime consequences that occurred at least three times per week (see Table 3). All participants had experienced these symptoms for at least

[14]Item scores ranged from 0-4, with higher scores indicating worse severity and greater impact of sleep disturbance.
[15] Item scores ranged from 0-4, with 0 indicating "Not at all difficult" and 4 indicating "Very difficult".

three months (Criterion B). Participants maintained a delayed sleep-wake pattern when on an *ad libitum* schedule (i.e., weekends), however had less difficulty awakening and improved morning alertness of weekend mornings compared to weekday mornings (see Table 2; Criterion C). Comorbid conditions were assessed and did not better explain the sleep disturbance (Criterion E, see Table 3).

## Procedure

A mixed-methods (quantitative and qualitative) online survey, including both multiple choice (50%) and free text responses (50%), was developed and distributed using the survey website Qualtrics (see supplementary materials for full survey). Data collection occurred from July to August 2016. Prior to completing the survey, participants were fully informed of the purpose of the study (i.e., to better understand the experience of DSWPD). They volunteered to participate, could choose not to answer questions at any time and were not offered reimbursement for their involvement.

## Materials

**Demographics.** Participants reported their age, gender, country of residence, employment status, working hours and reasons for their work schedule.

**Delayed circadian timing.** To assess sleep timing, participants were asked to report their typical bedtime, sleep onset time, wake-up time and out of bed time. Sleep timing and sleep onset latency were assessed for both weekdays and weekends separately.

Participants were asked whether they woke naturally, then rated their difficulty awakening and alertness in the morning, for both weekend and weekday mornings. Participants were asked whether they nap, frequency of nap, the most common time and length of nap.

**Primary sleep diagnosis.** Participants were asked whether they thought they currently had a sleep problem and if so, questions were asked to assess the onset, chronicity and type of the sleep problem, and whether (and by whom) the sleep problem had been diagnosed.

**Comorbidities.** Participants identified whether they had sleep, physical or psychological comorbid conditions. If the participant self-reported comorbidity, they were asked to list their comorbid condition/s.

**Daytime Consequences.** Participants were asked to indicate whether they currently experienced common daytime consequences of their sleep, from a list provided (e.g., tiredness/fatigue, problems socialising, poor mood/ irritability, lack of energy/ motivation, worries about sleep, etc.). If participants identified one or more daytime consequence, they rated whether these symptoms were present at least three days per week, in line with diagnostic criteria for Insomnia Disorder (American Psychiatric Association, 2013).

**Treatment.** Participants indicated whether they had received treatment for their sleep problem. Those who *had not* received treatment, were asked to list reasons why, using free response text. Participants who *had* received treatment, listed what treatments they had tried, and which ones they found the most useful.

**Insomnia.** A modified version of the Insomnia Severity Index (ISI; Morin, 1993) was used to screen for "insomnia" in DSWPD. The ISI was adapted to a six item measure, such that it did not bias respondents to assume they had insomnia. The adapted ISI measured the severity and impact of sleep disturbance, on a 5-point likert scale from 0 to 4, with higher scores indicating higher insomnia severity. The Modified ISI had acceptable internal consistency ($\alpha$=0.66).

As a clinical indicator of "insomnia", participants were asked to report their average sleep onset latency and amount of wake after sleep-onset. The frequency of sleep-onset difficulties was also assessed by asking participants how many nights per week they have trouble getting to sleep.

Authors (CR & MG) also developed a series of questions to assess for cognitive "insomnia" symptoms, by addressing each component of Harvey's (2002) model of insomnia. In particular, participants were asked if they experience excessive negatively-toned cognitive activity, physiological arousal, distress, selective attention towards and monitoring of sleep stimuli and safety behaviours. If participants endorsed a cognitive "insomnia" symptom, they were then asked to provide examples in free response text, before rating the frequency of each cognitive "insomnia" symptom. The presence and examples of each symptom were assessed during the daytime and night. Participants were also asked to share beliefs they hold about sleep.

To supplement the assessment of safety behaviours, participants were also explicitly asked to rate their average daily intake of caffeinated drinks, caffeinated foods, alcohol and cigarettes. If participants indicated a value >0, they were asked to list reasons why they use each substance. Similarly, if participants napped, they listed reasons for napping.

**Psychosocial Challenges.** To gain insight into the experience of DSWPD, participants used free text to comment on challenges they face, living with a circadian rhythm sleep disorder and finally, whether there was anything they wanted sleep researchers and clinicians to know.

### Statistical Analysis

Descriptive statistics, *t*-test and chi-square analyses were used to assess participant characteristics and the quantitative insomnia data, using IBM SPSS Statistics 22 (IBM Corporation, United States of America). Percentages presented throughout are expressed as the proportion of the *valid* sample (e.g., percentage of those who endorsed each symptom). However, estimates of comorbidity (i.e., sleep, physiological and psychological) are expressed as a percentage of the total sample (*N*=34). Qualitative data were independently categorised by two authors (CR, GM) according to emerging themes (Thomas, 2006). The raters had between 3-6 years of clinical experience in assessing and treating DSWPD and insomnia and used the cognitive model of insomnia as a theoretical guide when classifying responses (Harvey, 2002). Any discrepancy in independently identified themes was discussed in a meeting with the senior sleep psychologist (MG) and the two raters, until a consensus was reached. Qualitative data (i.e., quotes) are used to support or represent key findings.

## Results

### Participant Characteristics

Participant characteristics including demographics, employment and features of the disorder are presented in Table 1, and Table 2 defines subjectively reported sleep parameters. Average sleep data suggest that the sample were typical of those experiencing DSWPD.

Table 1.

*Participant Demographics and Disorder-related Characteristics.*

| | | | | | |
|---|---|---|---|---|---|
| **Age** | 47.3 ± 14.1 years | | | | |
| **Gender** | Male | | Female | | |
| | 9% | | 91% | | |
| **Country of Residence** | USA | UK | Canada | Brazil | Norway |
| | 85% | 6% | 3% | 3% | 3% |
| **Employment Status** | Full time | | Part time | | Retired |
| | 29.4 % | | 20.6 % | | 17.6 % |
| **Work Times** | Conventional | | Flexible | | |
| | 17.4% | | 88.9% ^ | | |
| **Diagnosis** | DSWPD | | N24SWD * | | OSA ~ |
| | 91.7% | | 4.2% | | 4.2% |
| **Diagnosis Approach** | Professional | | Informal | | |
| | 71% | | 29% | | |
| **Diagnostician** | Sleep Physician | | Psychiatrist | Psychologist | GP |
| | 50% | | 23% | 18% | 9% |
| **Course of Development** | Gradual | | Sudden onset | | |
| | 87% | | 13% | | |
| **Chronicity** | 3-12 years | | ≥ 12 years | | |
| | 19.4% | | 80.6% | | |

~ DSWPD was secondary to Obstructive Sleep Apnoea (OSA) diagnosis

^ 89% cited sleep pattern as the reason for flexible hours

* The individual with Non-24 hour Sleep-Wake Disorder was included due to similarities in phenomenology between the two disorders (Micic, Lovato, Gradisar, Burgess, et al., 2016; Micic, Lovato, Gradisar, & Lack, 2016).

Table 2.

*Subjectively Reported Sleep Parameters.*

| | Weekday | | Weekend | | | | |
|---|---|---|---|---|---|---|---|
| | **Mean** | **SD** | **Mean** | **SD** | ***t*** | ***df*** | ***p*** |
| **Bedtime** | 02:40 | 2.48hrs | 02:55 | 2.26hrs | | n.s. | |
| **Sleep Onset Time** | 03:37 | 2.93hrs | 04:11 | 1.75hrs | | n.s. | |
| **Wake-Up Time** | 11:16 | 2.49hrs | 12:23 | 2.10hrs | -4.11 | 31 | < .001 |
| **Out of Bed Time** | 11:44 | 2.45hrs | 12:56 | 2.09hrs | -3.91 | 31 | < .001 |
| **Difficulty Waking Up*** | 3.95 | 1.30 | 3.15 | 1.26 | 4.03 | 33 | < .001 |
| **Morning Alertness^** | 1.35 | 0.99 | 2.12 | 1.04 | 3.71 | 33 | .001 |
| **Spontaneous Wake Up** | 44% | | 76.5% | | | | |
| | **Portion of sample** | | **Length** | | **Time** | | |
| **Naps** | 32.4% | | 92.7 ± 48.8min | | 16:33 ± 2.5hrs | | |

* 0: Not at all difficult; 4: Very difficult

^ 0: Not at all alert; 4: Very alert

**Comorbidity and Treatment**

As per Table 3, there was a high level of comorbidity within the sample; including secondary sleep, physical and psychological conditions. A majority of the sample (70.6%) received treatment for their sleep problem. Table 3 suggests that although recommended behavioural treatment strategies (i.e., morning light therapy, evening exogenous melatonin, evening dim light) were the most frequently used, participants found medication to be the most useful treatment approach (i.e., hypnotics, tetracyclic antidepressants, benzodiazepines).

Importantly, a large proportion of the sample (30.4%) reported that none of the treatment strategies they have tried have been useful.

*"I have received very little ongoing monitoring of how these treatments should be applied, very little trouble shooting on what other conditions may be contributing and how to address them, and very little intervention for the psychological and emotional components of my sleep disorder, which I consider to be a lynch pin of my problem."*

Interestingly, the most common reasons for not seeking treatment were a lack of faith in health professionals and treatments available (60%), the expense of treatments (30%) and preferring to alter lifestyle, rather than advance sleep timing (30%).

Table 3.

*Daytime Consequences, Comorbidities and Treatment Strategies Associated with DSPWD According to Self-report Responses.*

| | | | | | |
|---|---|---|---|---|---|
| **Daytime Consequences** | Tiredness / fatigue | Problems socialising | Daytime Sleepiness | Lack of energy / motivation | Worries about sleep |
| | 79.4% | 76.5% | 73.5% | 70.6% | 70.6% |
| **Comorbidities** | Sleep Disorder | Physical Condition | Psychological Disorder | | |
| | 58.8% | 67.6% | 67.6% | | |
| **Comorbid Sleep Disorders** | CID | OSA | RLS | | |
| | 23.5% | 20.5% | 11.8% | | |
| **Comorbid Physical Conditions** | Fibromyalgia | Neurological conditions | Thyroid conditions | Digestive conditions | |
| | 26.5% | 11.8% | 11.8% | 11.8% | |
| **Comorbid Psychological Disorders** | Depression | Anxiety | PTSD | ADHD | BD |
| | 41.1% | 35.3% | 14.7% | 14.7% | 11.8% |
| **Attempted treatments** | Morning BLT | Melatonin | Dim Light | Medications | |
| | 56.5% | 56.5% | 47.8% | 47.8% | |
| **Other treatment strategies** | Sleep Hygiene | Sleep Scheduling | Relaxation | Dietary Supplements | Chronotherapy | CBT |
| | 26.1% | 21.7% | 17.4% | 13% | 13% | 13% |
| **Most effective treatments** | Medication | Morning BLT | Evening Melatonin | Evening Dim Light | |
| | 43.5% | 26.1% | 21.7% | 21.7% | |

**CID**: Chronic Insomnia Disorder; **OSA**: Obstructive Sleep Apnoea; **RLS**: Restless Legs Syndrome; **BD**; Bipolar Disorder; **PTSD**: Post-Traumatic Stress Disorder; **ADHD**: Attention-Deficit Hyperactivity Disorder; **BLT**: Bright Light Therapy; **CBT**: Cognitive Behavioural Therapy

**Aim 1: Frequency of Insomnia Symptoms in DSWPD**

Participants reported several symptoms consistent with insomnia. Participants were unhappy with their sleep pattern (*M*=3.15, *SD*=0.96) and reported moderate sleep-onset difficulties (*M*=2.15, *SD*=1.40)[16], which occurred approximately 3.5 days per week (M=3.68 days, *SD*=2.95). Average self-reported sleep onset latency was above the clinical cut off for "sleep-onset insomnia" (i.e., ≥31 mins) on both weekday (*M*=60.7mins, *SD*=84.8) and weekends nights (*M*= 55.0mins, *SD*=81.5; Lichstein et al., 2003). Participants found their sleep to be moderately unrefreshing (*M*=2.29, *SD*=1.32) and sleep maintenance difficulties were rated as mild (*M*=1.12, *SD*=1.27). Self-reported amount of wake after sleep onset approached the clinical cut off (i.e., ≥31 mins; *M*= 28.8mins, *SD*= 54.6; Lichstein et al., 2003). In terms of the broader impact of their sleep disturbance, participants reported that the impact on their quality of life was noticeable to others (*M*=3.18, *SD*= 1.06) and participants were somewhat worried and/or distressed by their sleep (*M*=2.56, *SD*=1.08).

Cognitive "insomnia" processes were commonly reported and frequently experienced (i.e. on average, 72% of individuals experienced these symptoms three or more days per week; see Table 4). Selective attention and monitoring of sleep-related stimuli, sleep-related behaviours and cognitive activity were the most commonly endorsed cognitive "insomnia" symptoms overall.

In terms of the timing of these processes, an exact McNemar's Test determined physiological arousal (*p*=.001) and sleep-related behaviours (*p*=.016) were more prevalent at night. Conversely, participants showed a trend to be more distressed by their sleep during the day (*p*=.065).

[16]Item scores ranged from 0-4, with higher scores indicating worse severity and greater impact of sleep disturbance.

Table 4.

*The Percentage and Frequency of Cognitive "Insomnia" Symptoms*

| **Insomnia Symptom** | **% of Sample Endorsed** | **Frequency of Symptoms (%)** | | | |
|---|---|---|---|---|---|
| | | *Less than once a week* | *1-2 days per week* | *3-5 days per week* | *6-7 days per week* |
| Night Time Cognitive Activity | 61.8% | 14.3% | 28.6% | 23.8% | 33.3% |
| Daytime Cognitive Activity | 61.8% | 4.8% | 19.0% | 38.1% | 38.1% |
| Night Time Physiological Arousal | 67.6% | 17.4% | 13.0% | 30.4% | 39.1% |
| Daytime Physiological Arousal | 35.3% | 0.0% | 0.0% | 36.4% | 63.6% |
| Night Time Distress | 48.5% | 0.0% | 20.0% | 26.7% | 53.3% |
| Daytime Distress | 69.7% | 0.0% | 30.4% | 26.1% | 43.5% |
| Night Time Attention and Monitoring | 85.3% | 20.7% | 20.7% | 37.9% | 20.7% |
| Daytime Attention and Monitoring | 94.1% | 16.7% | 26.7% | 33.3% | 23.3% |
| Night Time Sleep Related Behaviours | 87.9% | 3.4% | 17.2% | 27.6% | 51.7% |
| Daytime Sleep Related Behaviours | 66.7% | 19.2% | 7.7% | 34.6% | 38.5% |
| | **Average** | **9.7%** | **18.3%** | **31.5%** | **40.5%** |

**Aim 2: Themes of Cognitive "Insomnia" in DSWPD**

Emerging themes for each cognitive "insomnia" process are listed below. Themes were ubiquitous across the day and night, unless otherwise specified.

**Cognitive activity.**

1) ***Sleep and its consequences;*** e.g., "*You think about sleep every five minutes. Imagine you had type 1 diabetes, and how often you'd think about food*".

2) ***Rehearsal, planning and problem solving;*** e.g., "*My mind endlessly goes over the day and things I could have done differently*". Unsurprisingly, thoughts related to planning were more prevalent during the daytime e.g., "*I spend a lot of time planning out how I will cope with the sleep deprivation and how I will maximize the amount of sleep I am able to get*".

**Physiological arousal.**

1) ***Restlessness;*** e.g., tingling, urge to move, anxiety, muscular tension, numbness, hypnic jerks.
2) ***Pain;*** e.g., "*general aches and pains", "muscle pain*". Pain was the most commonly reported theme during the daytime.

**Distress.**

1) ***Sleep and its consequences;*** e.g., "*It's kind of an ongoing sense of misery, when I wake up at a time most people are starting to wind up their afternoon, when I'm sleepy throughout the first part of my day, when I commit to myself that I'll go to bed earlier that day and then don't, when I realize that there's one more event I can't make because of my schedule*".

**Selective attention and monitoring.**

The primary themes of night time selective attention and monitoring were:

1) ***Cognitive and physiological arousal;*** e.g., "*being extremely focused and alert an hour or more prior to anticipated bedtime*".
2) ***Time;*** e.g. *"when I have to get up at normal people time for an appointment or training and yet, even though I swore I would try to go to bed earlier, I don't.....then I watch the clock and freak out*".

The primary themes of day time selective attention and monitoring were:

3) ***Daytime consequences of sleep;*** e.g., '*Poor memory, forgetfulness, disorganization, scattered thinking, dozing off during the day, asking people to repeat things over and over, almost missing deadlines".*
4) ***Mood;*** e.g., *"Anything before noon feels like a blur. It's a feeling of unreality. I am teary, impulsive, and inauthentic. It feels like I am intruding in another world. I know this because I am confident when I rise later. I am 'myself' and really love life*".

**Sleep-related beliefs.**

1) ***Sleep is important;*** e.g., *"I have learned that sleep has to come first or I won't be able to function at all*".
2) ***Sleep is hard to control;*** e.g., *"It's completely out of my control. I just have to accept my different time and deal with it*".
3) ***It is better to sleep in line with one's natural circadian rhythm;*** e.g., *"It is better to just go with the natural flow of how your body works in regards to circadian rhythm. For me that means sleeping when others are awake*".
4) ***Having a delayed sleep pattern should be accepted within society;*** e.g., "*I believe some of our natural body clocks simply run later than other people's. It is only a "disorder" because society deems it to be, which is unfair".*
5) ***Circadian rhythm sleep disorders have particular origins;*** e.g., "*I honestly believe I was born with some sort of genetic mutation where my body clocks run later than a "normal" person's*".

**Sleep-related behaviours.** Participants reported engaging in a large range of behaviours to promote sleep and daytime functioning. Most commonly, participants reported:

1) ***Treatment strategies;*** e.g., "*Melatonin, blue light-blocking glasses, light box, sunlight in the day, exercise, nothing stimulating at night*".
2) ***Facilitating delayed sleep-wake pattern;*** e.g., *"I cancel many appointments and tasks from my schedule just to sleep more".*
3) ***Stimulant use;*** e.g., "*I drink caffeinated beverages and take Adderall[17]".*
4) ***Napping;*** Participants who napped, most commonly reported they did so to reduce sleepiness and improve daytime performance and productivity.

**Aim 3: Psycho-social Challenges**

In terms of the wider psychosocial context of individual's sleep disturbance, participants reported the following challenges as a result of their sleep:

1) **Employment;** e.g., *"This severely limits my employment opportunities and since I have a strong career goal in mind, the possibility of not achieving my potential has been really difficult to grapple with".*
2) **Social isolation;** e.g., "*This condition is lonely. I want to meet friends or join outings but I don't make it. I have missed friends' weddings and even stood up friends at restaurants for lunch. Heck, I was late for my own wedding!".*

[17] Adderall is a stimulant drug used to treat Narcolepsy and Attention Deficit Hyperactivity Disorder.

3) **Lack of understanding;** e.g., "*People do not understand. It is not a choice. I can't just force it to change".*
4) **Living out of sync with society;** e.g., *"making appointments, attending events or classes, sleep disturbance in the daytime, hotel check-out times".*

In line with the psychosocial challenges identified, participants expressed a desire for:

1) **Awareness;** e.g., within the general public (e.g., *"Public education on circadian rhythm disorders would be wonderful! Help to bring understanding")* and health professionals (e.g., "*it would be nice if specialists in sleep disorders could educate or inform their non-specialist colleagues in fields where it's significant*").
2) **Advocacy and support;** e.g., "*I would love an effective treatment or cure for DSPD, but barring that, I wish clinicians and researchers would focus sometimes on what happens when treatment fails (which it seems to more than not) and how to live a productive and full life, even at odd hours*".
3) **Further research;** e.g., *"It's discouraging to find so little research underway and so few treatment options… greater understanding of the mechanisms behind it are desperately needed. Solutions are desperately needed, treatment, coping, work/life accommodations, strategies, etc."*

## Discussion

Results from the current study suggest that cognitive "insomnia" processes (e.g., cognitive activity, physiological arousal, distress, selective attention and monitoring, safety behaviours and erroneous beliefs) are common in adults with chronic DSWPD, supporting previous research conducted with adolescents and young adults (Danielsson et al., 2015; Micic, Lovato, Gradisar, & Lack, 2016; Richardson et al., 2016; Wilhelmsen-Langeland et al., 2012; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013). Cognitive 'insomnia" processes may be implicated in the persistence of DSWPD into adulthood and, if left untreated, may contribute to poor long-term outcomes.

Despite being evidence-based, chronobiologic treatment strategies had a low level of acceptability in the current sample of adults with chronic and severe DSWPD. Participants reported that treatments failed to address the psychological, emotional and social components of their sleep disturbance, which is particularly important given the high prevalence of

psychological comorbidity in individuals with DSWPD (Reid et al., 2012). Although the current sample is not representative of the typical DSWPD population (i.e., participants were predominately female) and may be skewed (i.e., does not include individuals who have been successfully treated for DSWPD, females may be more likely to self-report worry and other cognitive processes; Robichaud, Dugas, & Conway, 2003), it does afford the opportunity to better understand one reason why DSWPD may persist into adulthood and how the provision of treatment to adults could be improved.

Emerging themes within qualitative responses support previous research (Hiller et al., 2014; Wilhelmsen-Langeland et al., 2012) and highlight a similarity in phenomenology with insomnia (Neitzert Semler & Harvey, 2004b; Richardson et al., 2016; Wicklow & Espie, 2000). Additionally, there was some evidence of an inter-relationship between these processes (e.g., thematic similarity between cognitive arousal and distress, i.e., sleep and its consequences; selective attention towards cognitive and physiological arousal) consistent with the prevailing models of insomnia (Espie, 2002; Espie, Broomfield, et al., 2006; Harvey, 2002; Lundh & Broman, 2000).

In comparison to previous research, the current sample provides insight into the features of DSWPD that are unique to adults with chronic and/or severe sleep disturbance. Relative to adolescents, employment and daily living tasks (e.g., parenting, attending appointments) were identified as areas of difficulty for adults with DSWPD. Additionally, problems socialising emerged as an important symptom *and* challenge for adults with DSWPD; whereas this concern was less prevalent for adolescents (Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011). Importantly, there were some key similarities identified; for example, adolescents with DSWPD have also i) demonstrated unhelpful thinking about the impact of their sleep on daytime functioning, ii) expressed similar beliefs about the cause of their sleep problem (e.g., underlying biology or society) and iii) expressed concern that

others do not understand what it is like to have DSWPD (Hiller et al., 2014; Wilhelmsen-Langeland et al., 2012).

Although these results support a growing body of evidence (e.g., Barbero & Gradisar, 2013, unpublished thesis, Billows & Gradisar, 2009, unpublished thesis, Gradisar, Dohnt, Gardner, Paine, Starkey, Menne, et al., 2011; Hiller et al., 2014; Lovato, Gradisar, et al., 2013; MacMahon et al., 2006; Marchetti et al., 2006; Saxvig et al., 2012; Wilhelmsen-Langeland, Saxvig, Pallesen, Nordhus, Vedaa, et al., 2013), further research is warranted. In particular, to extend upon these preliminary findings (i.e., proof of concept), the study could be replicated in larger and/or more representative sample (i.e., balanced gender split, including adolescents and young adults etc.). Additionally, the current study relied on participants' ability to accurately introspect on their experience of cognitive "insomnia" processes and some of these processes often occur without conscious awareness (e.g., selective attention). Therefore, the presence of cognitive "insomnia" processes should also be investigated using validated subjective (e.g., Pre-Sleep Arousal Scale, Sleep Anticipatory Anxiety Scale, Insomnia Severity Index, Sleep Associated Monitoring Index, Dysfunctional Beliefs and Attitudes about Sleep, Sleep-Related Behaviours Questionnaire) and objective measures (e.g., dot-probe task for attentional bias), where possible (Bootzin et al., 1994; MacMahon et al., 2006; Marchetti et al., 2006; Morin, 1993; Neitzert Semler & Harvey, 2004b; Nicassio et al., 1985; Ree & Harvey, 2004). Finally, as an emerging area, further research is required to better understand the potentially complex relationship between these cognitive "insomnia" processes in DSWPD. If future studies further confirm that "insomnia" processes exist in DSWPD, clinicians could target these processes in assessment and treatment.

Clinicians could use the aforementioned measures, or more informal questioning (e.g., the current study's questionnaire) to assess for cognitive "insomnia" processes. Information

gathered through thorough assessment can then inform case-conceptualised treatment (i.e., treatment that applies psychological theory with a specific focus on the unique experience of the individual; Dudley et al., 2011). Case-conceptualised treatment may involve a combination of both behavioural (e.g., morning light therapy, evening exogenous melatonin, evening dim light) and cognitive therapies. In particular, cognitive restructuring, mindfulness, mental imagery and relaxation training could be used to reduce negative cognitive activity and physiological arousal (Harvey & Payne, 2002). Psychoeducation and behavioural experiments can be used to challenge maladaptive beliefs about sleep and ameliorate sleep misperception (Tang & Harvey, 2006), whilst attentional bias modification tasks could be used to train an individual's attention away from sleep-related stimuli (Milkins et al., 2016).

Importantly, participants in the current study called for greater awareness, advocacy and support. Therefore, sleep specialists could also strive to increase awareness of circadian rhythm sleep disorders within related professions (e.g., general practitioners, psychologists, psychiatrists). Doing so may increase detection of DSWPD, which could facilitate early intervention and a multimodal treatment approach (i.e., addressing circadian delay, secondary sleep disorders and comorbid physical and psychological conditions). Additionally, clinicians may need to diversify their role, by also providing advocacy and support to those wanting to make lifestyle adjustments to accommodate their delayed sleep pattern. For example, if evidence-based treatments fail to produce acceptable improvements in sleep timing and daytime functioning, clinicians could support individuals to overcome the psychosocial barriers to their endogenous sleep-wake timing. In particular, clinicians could advocate for altered employment hours for their clients (e.g., citing the Americans with Disabilities Act or similar legislation), and provide support to overcome the inherent difficulties of sleeping out of synchrony with the 24-hr world (e.g., social isolation and ability to access services).

Linking clients with other advocacy services (e.g., Circadian Sleep Disorders Network) and online support groups could also prove beneficial.

*Conclusions*

In sum, results from the present study highlight the importance of assessing the wider psychosocial context of sleep disturbance in adults with DSWPD. In doing so, clinicians may be able to identify unique risk factors for the maintenance (or relapse) of individual's sleep disturbance. By addressing these "risk" factors as part of a case-conceptualised treatment, the acceptability, uptake and outcome of treatments might be improved. However, if evidence based treatments are not successful, clinicians and researchers need to consider how to best support those living with Delayed Sleep-Wake Phase Disorder.

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## Supplementary Materials for Appendix C: Study Questions

| Survey Question | Response Type |
|---|---|
| What is your gender | Male/ Female |
| How old are you in years? | Free text |
| What country do you live in? | Free text |
| What is your current employment status? | Employed full time/employed part time, studying full time/ studying part time, retired, unemployed, other (free text) |
| How would you describe the hours of your primary occupation (work or study)? | Regular daytime hours (e.g., 9am-5pm)/ shift work/ flexible hours, other (free text) |
| What are your reasons for undertaking shift work OR flexible hours? | Free text |
| What time do you usually get into bed at night on weekdays? | HH:MM AM/PM |
| What time do you usually fall asleep at night on weekdays? | HH:MM AM/PM |
| After turning out the lights out at night to go to sleep on weekdays, how long does it usually take you to fall asleep? | Minutes |
| What time do you usually get into bed at night on weekends? | HH:MM AM/PM |
| What time do you usually fall asleep at night on weekends? | HH:MM AM/PM |
| After turning out the lights out at night to go to sleep on weekends, how long does it usually take you to fall asleep? | Minutes |
| On average, how many minutes do you spend awake in bed after initially falling asleep at night (e.g., during night time awakenings)? | Minutes |
| How many nights per week do you have trouble getting to sleep? | I don't experience this problem/ 1 night/ 2 nights/ 3 nights/ 4 nights/ 5 nights/ 6 nights/ 7 nights |
| Do you normally wake up by yourself (e.g., without an alarm) on weekdays (Monday to Friday mornings)? | Yes/ No |
| On weekdays (Monday to Friday mornings), what time do you usually wake up finally? | HH:MM AM/PM |
| On weekdays (Monday to Friday mornings), what time do you usually get out of bed? | HH:MM AM/PM |
| How difficult is it for you to wake up and get out of bed on weekdays? | 0 (not at all difficult) to 4 (very difficult) |
| Do you normally wake up by yourself (e.g., without an alarm) on the weekend (Saturday and Sunday) mornings? | Yes/No |
| On weekend (Saturday and Sunday) mornings, what time do you usually wake up finally? | HH:MM AM/PM |
| On weekend (Saturday and Sunday mornings), what time do you usually get out of bed? | HH:MM AM/PM |
| How difficult is it for you to wake up and get out of bed on weekends? | 0 (not at all difficult) to 4 (very difficult) |
| Given the opportunity (e.g., weekends, or holidays, without morning commitments) what time would you naturally wake? | HH:MM AM/PM |
| Do you nap? | Yes/ No |
| How many times per week do you nap? | 1 time per week/ 2-3 times per week, 4-5 times per week/ 6-7 times per week |
| What time do you most commonly nap? | HH:MM AM/PM |
| What are your reasons for napping? | Free Text |
| Do you consider yourself to have a sleep problem currently? | Yes/No |

| | |
|---|---|
| How long have you had your current sleep problem? | 0-11 years, 0-11 months |
| Has your current sleep problem been diagnosed by a health professional? | Yes/ No |
| What type of health professional diagnosed your current sleep problem? | General Practitioner/ Specialist Sleep Physician/ Psychiatrist/ Psychologist/ Other (free text) |
| What is your current primary sleep disorder diagnosis? | Delayed Sleep Phase Disorder/ Advanced Sleep Phase Disorder/ Non-24 Hour Sleep Disorder/ Irregular Sleep Wake Disorder/ Primary Insomnia/ Other (free text) |
| How did your sleep problem happen? | Suddenly (means you remember exactly when it started)/ Gradually (means you are not sure when it started) |
| Have you been diagnosed with any other sleep disorder/s (e.g., sleep apnoea, restless leg syndrome, primary insomnia)? | Yes/ No |
| What other sleep disorder/s have you been diagnosed with? | Free Text |
| Have you been diagnosed with any medical (physical) condition/s (e.g., chronic fatigue)? | Yes/ No |
| What medical (physical) condition/s have you been diagnosed with? | Free Text |
| Have you been diagnosed with a psychiatric condition/s (e.g., depression, anxiety etc.)? | Yes/ No |
| What psychiatric condition/s have you been diagnosed with? | Free Text |
| Do you currently experience any of these symptoms as a result of your primary sleep problem? ( Please check all that apply ) | Tiredness/ fatigue/ Problems socializing/ Moody or irritable/ Lack of energy/ motivation/ Accident prone/ Worries about sleep/ Attention, concentration or memory problems/ Poor school/ university/ work performance/ Sleepy during the day/ Behavioural issues (hyperactivity/ aggression)/ Tension, headaches or stomach problems/ Other health problems |
| Are these difficulties present at least 3 days per week? | Yes/ No |
| How happy/ unhappy are you with your current sleep pattern? | 0 (Very happy)/ 1 (Happy)/ 2 (Okay)/ 3 (Unhappy)/ 4 (Very unhappy) |
| Do you have difficulty falling asleep and if YES, how severe is this difficulty? | 0 (No problem)/ 1 (Mild)/ 2 (Moderate)/ 3 (Severe)/ 4 (Very severe) |
| Do you have difficulty staying asleep and if YES, how severe is this difficulty? | 0 (No problem)/ 1 (Mild)/ 2 (Moderate)/ 3 (Severe)/ 4 (Very severe) |
| Do you wake up feeling that your sleep has NOT been refreshing, and if YES, how severe is this difficulty? | 0 (No problem)/ 1 (Mild)/ 2 (Moderate)/ 3 (Severe)/ 4 (Very severe) |
| How worried/ distressed are you about your sleep at the moment? | 0 (Not at all)/ 1 (A little)/ 2 (Some)/ 3 (Much)/ 4 (Very much) |

| | |
|---|---|
| Have you received treatment/s for your current sleep problem? | Yes/ No |
| Please list the treatment/s that you have received for your current sleep problem. | Free Text |
| Which treatment/s have been the most useful for your current sleep problem? | Free Text |
| Why have you not received treatment for your current sleep problem? | Free Text |
| On average, how many caffeinated beverages (e.g., tea, coffee, coke, energy drinks) do you drink per day? | Number |
| On average, how many food products containing caffeine (e.g., chocolate bars) do you eat per day? | Number |
| What are your reasons for having caffeinated food and/or beverages? | Free Text |
| On average, how many standard alcoholic drinks do you consume per day during the week? | Number |
| On average, how many standard alcoholic drinks do you consume per day over the weekend? | Number |
| What are your reasons for having alcoholic drinks? | Free Text |
| Do you smoke cigarettes? | Yes/ No |
| How many cigarettes do you smoke per day? | Number |
| What are your reasons for smoking cigarettes? | Free Text |
| Do you take any substances to help you fall sleep at night (e.g., sleeping pills, herbal supplements, hot chocolate)? | Yes/ No |
| How many days per week do you take a substance to help you fall sleep? | 0-7 nights |
| What substance/s do you take to help you fall asleep? | Free Text |
| What amount of the substance/s do you take? | Free Text |
| Are you taking any medication which may affect your sleep? | Yes/ No |
| What medications are you taking that may affect your sleep? | Free Text |
| Do you ever experience intrusive thoughts or worry when trying to fall asleep? | Yes/ No |
| Please provide examples of the types of intrusive thoughts you have and/or the content of worry when trying to fall asleep. | Free Text |
| How often do you experience intrusive thoughts or worry when trying to fall asleep? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| Do you ever experience intrusive sleep-related thoughts or sleep-related worry during the day? | Yes/ No |
| Please provide examples of the types of intrusive sleep-related thoughts you have and/or the content of sleep-related worry you have during the day. | Free Text |
| How often do you experience intrusive sleep-related thoughts or sleep-related worry during the day? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| Are you aware of physical sensations in your body when you try to fall asleep at night? | Yes/ No |
| Please provide examples of the types of physical sensations you experience in your body when trying to fall asleep at night. | Free Text |
| How often do you experience these physical sensations in your body when trying to fall sleep? | Less than once a week/ 1-2 nights a week/ 3-5 nights a |

| | |
|---|---|
| | week/ 6-7 nights a week |
| Are you aware of these physical sensations in your body during the day? | Yes/ No |
| Please provide examples of the physical sensations you also experience in your body during the day. | Free Text |
| How often do you experience these physical sensations in your body during the day? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| Are you distressed by your sleep at night time? | Yes/ No |
| What causes you distress about your sleep at night time? | Free Text |
| How often are you distressed by your sleep at night time? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| Are you distressed by your sleep during the day? | Yes/ No |
| What causes you distress about your sleep during the day? | Free Text |
| How often are you distressed by your sleep during the day? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| Do you ever notice signs that indicate that you are not going to get enough sleep? | Yes/ No |
| Please provide examples of signs you notice that indicate that you are not going to get enough sleep. | Free Text |
| How often do you notice these signs that indicate that you are not going to get enough sleep? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| Do you ever notice signs that indicate that you are not coping well or functioning well during the day? | Yes/ No |
| Please provide examples of signs you notice that indicate that you are not coping well or functioning well during the day. | Free Text |
| How often do you notice these signs that indicate that you are not coping well or functioning well during the day? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| Given that you have experienced a sleep problem for some time, what beliefs have you developed about sleep? | Free Text |
| Is there anything that you do or think to try and ensure that you will get enough sleep? | Yes/ No |
| Please provide examples of the types of things you do or think to try and ensure that you will get enough sleep. | Free Text |
| How often do use these strategies to try and ensure that you will get enough sleep? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| Is there anything that you do or think to try and ensure that you will cope well or function well during the day? | Yes/ No |
| Please provide examples of the types of things you do or think to try and ensure that you will cope well or function well during the day. | Free Text |
| How often do use these strategies to try and ensure that you will cope well or function well during the day? | Less than once a week/ 1-2 nights a week/ 3-5 nights a week/ 6-7 nights a week |
| What are some of the biggest challenges you face living with a circadian rhythm sleep disorder? | Free Text |
| Is there anything that hasn't been covered so far, that you think is important for sleep researchers and clinicians to know? | Free Text |