# Combined Light Therapy and Group Psychotherapy as Treatment for Delayed Sleep Phase Syndrome in University Students

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**Abstract**

Delayed sleep phase syndrome is a circadian rhythm sleep disorder, prevalent amongst adolescents and young adults, the symptoms of which can cause distress and impairment in daily functioning. This study evaluated the effectiveness of a four week treatment programme integrating light therapy and group psychotherapy for university students with delayed sleep phase syndrome. A multiple-baseline across-groups design followed 18 university students (17-45 years of age) meeting ICSD-3 criteria for delayed sleep phase syndrome, over pre-treatment, intervention, and post-treatment phases, and at a 3-month follow-up. Primary outcome measures included the DASS-21, WHOQOL-BREF, Epworth Sleepiness Scale, and a measure of academic self-efficacy. Secondary outcome measures were sleep parameters recorded using a sleep diary. Results revealed improvements in some outcome variables (physical quality of life, stress), but no statistically or clinically significant change in the majority of outcome variables at post-treatment or follow-up. Suggestions to improve the effectiveness of subsequent programmes are discussed, alongside comparisons to similar programmes aimed at improving delayed sleep phase syndrome and its associated symptoms.

# Combined Light Therapy and Group Psychotherapy as Treatment for Delayed Sleep Phase Syndrome in University Students

The human sleep-wake cycle is driven by an endogenous 'body clock' located in the hypothalamus, which drives a set of physiological processes that cycle over a ~24 hour period. This cycle is known as the circadian rhythm. Although the circadian rhythm is endogenous, it is susceptible to alterations from environmental stimuli, known as Zeitgebers ('time givers'). Light is the primary Zeitgeber, which helps entrain (reset) our circadian rhythm to match the light-dark cycle of a 24 hour day. Entrainment in this capacity allows us to maintain a sleep-wake pattern that fits our environment (Moore, 1997). Circadian rhythm sleep disorders can develop when environmental or social cues (Zeitgebers) delay or advance the circadian rhythm so it no longer aligns with the typical light-dark cycle, and causes the individual distress or impairment in functioning.

Delayed Sleep Phase Syndrome (DSPS) is a circadian rhythm sleep-wake disorder characterised by an inability to fall asleep at a specified conventional clock time so that the major sleep episode is delayed until later (resulting in a shift in the timing of the sleep phase; hence the name), difficulty waking at a conventional rising time specified by clock time, and increased daytime sleepiness.

Many individuals who seek help for circadian rhythm sleep disorders report the onset of the disorder occurring in childhood or adolescence (Dagan & Eisenstein, 1999). This is particularly true for DSPS (Weitzman et al., 1981). Adolescents will typically exhibit a delay in sleep wake cycle when they enter puberty (Gradisar, Gardner, & Dohnt, 2011). Bed times and weekend wake times are often delayed, and daytime sleepiness increases as adolescents transition through puberty (Crowley, Acebo, & Carskadon, 2007).

**Prevalence of Delayed Sleep Phase Syndrome**

Estimates of prevalence rates of DSPS in the general population vary significantly, ranging from 0.17% (Schrader, Bovim, & Sand, 1993) to up to 3.1% (Wyatt, 2004). The prevalence of DSPS among adolescents is higher than the general population, with recent estimates of 7.3% (Saxvig, Pallesen, Wilhelmsen-Langeland, Molde, & Bjorvatn, 2012) and ranging from 1-16% for young adults (Gradisar & Crowley, 2013). The prevalence of DSPS in university students appears to be higher still, with studies revealing prevalence rates of 11.5% (Brown, Soper, & Buboltz Jr, 2001), 17% (Lack, 1986), and as high as 24.9% (Samaranayake, Arroll, & Fernando 3rd, 2014).

**Classification and Diagnosis**

The classification and diagnosis of DSPS is primarily based on diagnostic criteria from the International Classification of Sleep Disorders (ICSD) or the Diagnostic and Statistical Manual of Mental Disorders (DSM). The ISCD-3 (Sateia, 2014), published in 2014, is the most recent edition. Evidence of DSPS symptoms is first gathered using a clinical interview and later confirmed through monitoring of sleep wake schedules via a sleep diary, and/or actigraphy, for a period of 7-14 days (Dagan & Borodkin, 2005). More objective measures of polysomnography and dim light melatonin (a hormone that regulates the sleep/wake cycle) secretion can also be employed to confirm the diagnosis (see below for a discussion of assessment methods, including actigrapy and polysomonography). Criteria for DSPS (called 'Delayed Sleep-Wake Phase Disorder' in the ICSD third edition) include the following: (1) a sleep phase that is significantly delayed in comparison to the individual's desired sleep time; (2) symptoms present for longer than three months; (3) improved sleep quality when the individual is able to choose their own sleep

timing; (4) a significant delay in sleep timing as revealed by a sleep diary kept for 7-14 consecutive days reveals; and (5) the sleep problem cannot be otherwise explained by another sleep disorder or medical, psychological or neurological disorder (Sateia, 2014). Individuals must meet all the above criteria in order to be diagnosed with DSPS (Sateia, 2014).

**Assessment Methods for DSPS**

Clinical interviews assessing the prevalence of DSPS typically involve gathering information about the patient's current sleep complaints, sleeping habits, impairment in daily functioning, ability to adapt to a new sleep schedule, physical, family and psychiatric history, and attempts to cope with sleep difficulties (Dagan & Borodkin, 2005). The patient is then asked to keep sleep diary or log for a period of seven to fourteen days to record their sleep onset and wake times, sleep latency, and any other sleep parameters of interest (Morgenthaler et al., 2007). During this time, the patient may also be instructed to wear an actigraph (a device worn on the wrist to measure hand movement), which can provide a more objective measure of sleep and wake times (Dagan & Borodkin, 2005). In some cases, the patient may be required to undergo polysomnography (a type of sleep study whereby the patient attends a sleep clinical and is attached to a device that records brain activity, heart rate, breathing, and blood oxygen level during sleep), however this is not common practice unless to exclude another sleep disorder (Morgenthaler et al., 2007). Additionally, physiological markers of circadian phase, including core body temperature and melatonin secretion can be taken to further aid assessment. Core body temperature is measured rectally, while melatonin secretion is monitored through excretions in the saliva and urine. While useful, these measures may be time consuming and require equipment and testing facilities that may not be readily available, and there remains

insufficient evidence to recommend their routine use in the assessment of DSPS (Morgenthaler et al., 2007).

**Etiology**

The etiology of DSPS is likely due to a combination of physiological, genetic, and behavioural elements. An abnormality in circadian timing is the key physiological component contributing to DSPS. The human sleep/wake cycle is driven by an endogenous circadian rhythm, controlled by the suprachiasmatic nucleus (SCN) of the hypothalamus (Crowley et al., 2007). The SCN coordinates physiological and endocrine functions essential for circadian rhythm. Light, whether natural or artificial, assists the SCN to co-ordinate the processes involved in the human circadian rhythm. Light is classified as a Zeitgeber, which means it provides an external cue that allows our circadian rhythm to entrain and follow an approximately 24 hour cycle that follows the onset of night and day (Moore, 1997). Without cues from light, humans will exhibit a 'free-running' sleep/wake schedule in a pattern that can differ dramatically from the conventional ~24 hour sleep/wake schedule (Moore, 1997).

The SCN receives messages from photoreceptors in the retina in response to light. The SCN is particularly sensitive to short wavelength blue light, which has a more detrimental effect on melatonin suppression and sleep phase delay than light of other wavelengths(Bjorvatn & Pallesen, 2009). Information received by photorecpetors is relayed to the pineal gland (which is responsible for the secretion of the hormone melatonin), and the reticular formation regions in the hypothalamus and brainstem (which control metabolism, core body temperature, and timing of sleep-wake cycles) (Moore, 1997). Core body temperature and levels of the hormone melatonin, are two elements that oscillate with the circadian rhythm to impact on our sleep. In individuals with a typically functioning circadian phase, body temperature

reaches its lowest point (nadir), in the early hours of the morning, and peaks in the later afternoon or early evening (Bjorvatn & Pallesen, 2009). Sleep occurs when core body temperature is declining, and ends approximately two hours after the core body temperature nadir (Bjorvatn & Pallesen, 2009). A similar relationship can be observed between sleep and melatonin secretion. Melatonin secretion begins to rise with the onset of darkness, reaches peak levels in the middle of the night, and declines from there. Individuals with DSPS exhibit a delay in core body temperature nadir and melatonin secretion onset (Ozaki, Uchiyama, Shirakawa, & Okawa, 1996; Shibui, Uchiyama, & Okawa, 1999).

The timing of core body temperature nadir in individuals with DSPS has been found to be delayed by an average of 2.5 hours when compared to controls, and dim light melatonin onset time is delayed by more than two hours (Chang, Reid, Gourineni, & Zee, 2009). This follows the pattern of sleep schedules observed in individuals with DSPS compared with individuals following a conventional sleep schedule. Therefore, delays in these physiological processes are intrinsically linked to the delay in sleep phase DSPS individuals experience. Furthermore, Ozaki et al. (1996) demonstrated that individuals with DSPS experience a significantly longer interval between core body temperature nadir and sleep offset than those without the disorder. This suggests that these individuals sleep longer, on average, after they reach nadir than normal sleepers. These findings may help explain why individuals with DSPS find it difficult to advance their sleep phase (i.e. fall asleep and wake earlier than they currently are) because they continue to sleep during the key period (following nadir) when light exposure can aid this process.

Further research has demonstrated that individuals with DSPS experience a longer circadian rhythm period (tau) than normal sleepers (Micic et al., 2013). Longer

tau can have a significant effect on one's ability to advance one's sleep schedule (Saxvig et al., 2012), as it is associated with a longer sleep latency, delayed sleep timing, and less satisfaction with sleep (Lazar et al., 2013). Longer tau is, therefore, also thought to contribute to the struggle DSPS sufferers face when attempting to advance their sleep schedule.

While some progress has been made towards determining the genetic component of DSPS, we still know relatively little regarding genetic factors. Genes are known to influence an individual's chronotype, i.e., their tendency to being an "owl" (i.e., to have a preference for later sleep onset and waking) or a "lark" (i.e., early morning waking and early evening sleep onset) (Taillard, Philip, & Bioulac, 1999). Ancoli-Israel, Schnierow, Kelsoe, and Fink (2001) examined first and second degree relatives of a person presenting with DSPS and revealed 22% of the extended family showed a preference for eveningness, much higher than the percentage in the general population. Investigations into the link between the circadian clock gene *Per3* and Circadian Rhythm Sleep Disorders found that the frequency of specific variations in this gene is significantly higher in people with DSPS (Archer et al., 2003).

Behavioural factors thought to contribute to, and exacerbate DSPS in adolescence include pressure from peers to stay up later; reduced parental influence on bedtimes; access to activities promoting stimulation and arousal such as computer games, movies and television programmes; and consumption of stimulants, such as caffeine and nicotine (Dahl & Lewin, 2002; Saxvig et al., 2012). Gradisar and Crowley (2013) also propose that, given that adolescents and youth with DSPS typically have longer sleep latencies than typical sleepers (Gradisar, Dohnt, et al., 2011; Saxvig et al., 2012), they have more time available for unhelpful pre-sleep rumination or intrusive thinking. Gradisar, Dohnt, et al. (2011) found that

approximately 90 percent of their sample of adolescents with DSPS reported racing thoughts in bed while trying to initiate sleep. Hiller, Lovato, Gradisar, Oliver, and Slater (2014) found an indirect association between sleep latency and the number of catastrophizing thoughts, via anxiety experienced in their sample of adolescents with DSPS. This suggests that individuals with DSPS may experience a significant amount of racing or catastrophizing thoughts while trying to initiate sleep, which in turn may promote anxiety, extend the sleep latency period, and possibly contribute to delays in sleep phase. Given these findings, it seems important to address cognitive activity prior to sleep when considering a treatment plan for those with DSPS.

An individual's chronotype may also affect their likelihood of developing DSPS. Horne and Ostberg (1975) developed the Morningness-Eveningness Questionnaire, a self-report measure which determines an individual's preference for wake, daily activity, and sleep time, and categorises them into one of five morningness/eveningness subtypes ranging from definite evening, through to definite morning. Forty-eight subjects who completed the questionnaire were then subjected to oral temperature tests over a period of time to determine whether their body temperature fluctuations were related to their morningness/eveningness scores. Results revealed participants who scored as morning types had a significantly earlier peak in core body temperature timing than those who scored as evening types, suggesting a relationship between scores on this questionnaire and individual differences in core body temperature fluctuation, an important marker in DSPS (Horne & Ostberg, 1975).

The delay in circadian timing central to DSPS appears to be due to an interaction between these genetic, behavioural and physiological elements, which put the individual at risk for adapting and adhering to a delayed sleep schedule. This

delay becomes prevalent in adolescence, and may become further engrained in young adulthood if problems do not resolve.

**Delayed Sleep Phase Syndrome in University Students**

Students entering university, especially those of an age typical for this event, namely late adolescence, face a number of academic and social pressures as well as changes in routine and lifestyle additional to those normally experienced at that age and specific to being a participant in higher education. In order to cope with these pressures, students may exhibit sleep habits and behaviours that have consequences for their physical and mental health, and academic performance, and may also exacerbate or initiate DSPS symptoms. Recent literature suggests a significant proportion of university students appear to experience poor sleep quality, with several studies demonstrating that approximately 60% of students are classified as 'poor-quality sleepers' under the Pittsburgh Sleep Quality Index (PSQI) (Galambos, Vargas Lascano, Howard, & Maggs, 2013; Mesquita & Reimão, 2010; Suen, Ellis Hon, & Tam, 2008). Common sleep difficulties reported by university students include difficulty falling asleep, insufficient sleep, and morning tiredness (Buboltz Jr, Brown, & Soper, 2001; Lund, Reider, Whiting, & Prichard, 2010). Poor sleep quality can have a range of negative consequences for university students, including impaired cognitive functioning, heightened stress, mental and physical health problems, and increased drug and alcohol use (Lund et al., 2010; Pilcher, Ginter, & Sadowsky, 1997; Singleton & Wolfson, 2009).

Further, a significant proportion of university students may be at risk of developing a sleep disorder (Gaultney, 2010). Lack (1986) and Brown et al. (2001) investigated the occurrence of DSPS in university students. Findings from Lack's (1986) research revealed that participants with DSPS did not differ significantly in

age or sex from participants without DSPS. Individuals with DSPS more frequently reported difficulty getting to sleep and insufficient sleep on weeknights, and slept significantly longer on weekend nights, as well as obtaining lower grades than students without the disorder (Lack, 1986).

**Consequences of Delayed Sleep Phase Syndrome**

DSPS and its associated symptoms can have far reaching consequences in many aspects of one's life. While a delay in sleep timing is the most prominent symptom, insomnia, impairment in daytime functioning and inadequate sleep hygiene often co-occur (Gradisar, Dohnt, et al., 2011). Sleep hygiene is the practice of a number of techniques including maintaining a bedroom environment conducive to sleep (light and noise levels, avoiding activities other than sleep in bed), avoiding caffeine and other stimulants in the hours before sleep, that aid in promoting sleep onset. Gradisar, Dohnt, et al. (2011) found the majority of their sample of 23 adolescents with DSPS reported daytime sleepiness, fatigue, inattention, irritability, and low energy. The primary contributor to poor sleep hygiene was the use of electronic media in the bedroom. Furthermore, cognitions pertaining to sleep, including racing thoughts, worries, and anxiety about sleep were experienced by many adolescents in this sample (Gradisar, Dohnt, et al., 2011). Attempts to make up for lost sleep by taking daytime naps and consuming caffeine may exacerbate and even perpetuate the problem.

Adolescents with DSPS are likely to achieve lower average school grades than their peers, and have a higher rate of non-attendance at school or other learning institutions (Saxvig et al., 2012; Sivertsen, Harvey, Pallesen, & Hysing, 2015). They are also more likely to smoke, consume alcohol and suffer from anxiety, depression or inattention (Saxvig et al., 2012; Sivertsen et al., 2015). These problems may be

further compounded in university students. Gaultney (2010) investigated the risk of sleep disorders among American college students and their relationship with grade point average (GPA). Results revealed that students who reported having a sleep disorder, and those who identified as 'evening' people had a lower GPA than students who did not report a sleep disorder and those who identified as 'morning' people. Furthermore, students who were assessed as being at risk of developing at least one sleep disorder were more likely to be at risk of failing their courses. This finding was, however accompanied by a small effect size, so should be interpreted with caution. Gaultney (2010) proposes several mechanisms behind the relationship between sleep disorders and low GPA found in this study. They theorise that sleep disorders can lead to day time sleepiness, which in turn affects attention, memory and decision making. Additionally, they propose insufficient sleep may interrupt memory consolidation and thus affect academic performance. Alternatively, Gaultney (2010) suggests poor sleep may influence academic performance indirectly, through reduced motivation, health problems, or depressive symptoms.

Symptoms of DSPS can also have an impact on an individual's perceived quality of life. Nagtegaal et al. (2000) investigated the implications of DSPS on health-related quality of life in Dutch adults. Results revealed self-rated quality of life was significantly lower in all domains in individuals with DSPS compared to age and gender adjusted norms, however these findings were accompanied by small to medium effect sizes. Reports of poor physical health were found to interfere most with participants' typical daily physical activities, while fatigue interfered most with their daily social activities.

**Gender differences in Delayed Sleep Phase Syndrome**

Adan and Natale (2002) investigated gender differences in scores on the Morningness-Eveningness Questionnaire (Horne & Ostberg, 1975). Findings revealed that significantly more men than women scored as 'evening types'. Upon further analysis, Adan and Natale (2002) discovered that several scale items specifically differentiated males and females. Women preferred to go to bed and wake earlier than men, and felt tired earlier in the evening, whereas men preferred to sleep 1-2 hours later, woke later, and felt less alert on waking than women.

Biological factors may contribute to a gender difference in DSPS prevalence during young adulthood. On average, women's chronotypes (sleep schedules based on circadian rhythm) develop earlier than men's. Both genders develop a delay in their chronotype as they progress through adolescence and early adulthood, and will reach a peak delay in chronotype around their early 20s (Roenneberg et al., 2004). As women typically develop ahead of men, they will reach their peak delay before men, at an average age of 19.5 years, while men will reach their peak delay at an average of 20.9 years. Furthermore, the age at when chronotype is delayed the most (approximately 19.5 years in women) roughly coincides with the end of puberty. While males' delay in chronotype occurs later than females', after this point their chronotype remains later on average than females, until the age of about 50 (coinciding with the beginning of menopause for women). These findings suggest that the endocrine system may contribute to the relationship between gender/sex and sleep (Roenneberg et al., 2004).

**Co-occurrence of Psychological Disorders**

Several studies have indicated the prevalence of psychological disorders is higher in those with delayed sleep phase syndrome than in the general population.

Abe et al. (2011) investigated the prevalence of depressive symptoms in a group of 90 adults diagnosed with DSPS. Sixty four percent of individuals reported moderate to severe depressive states (as measured by the Zung self-rating depression scale (SDS)). The primary depressive symptom items reported included sleep disturbance, fatigue, diurnal variation and psychomotor retardation. It should be noted, however, that while these symptoms can be characteristic of depression, they can also occur in DSPS when depression is not present. A full assessment gathering detailed information regarding both cognitive and somatic symptoms, their onset and stability may help clarify their source. Robillard et al. (2013) monitored young adults with unipolar depression or bipolar disorders, alongside a control group, using actigraphy over a 7 day period to investigate sleep-wake cycle. Sixty two percent of participants with bipolar disorders and 30% of those with depression met the criteria for DSPS, compared to 10% of the control group. Participants with bipolar disorders woke the latest, followed by those with depression (Robillard et al., 2013).

Reid et al. (2012) explored the relationship between DSM IV Axis-I disorders and DSPS and evening-type circadian preference. Results revealed more than 70% of those who met the criteria for DSPS also met the criteria for at least one Axis-I disorder. Roughly 40% of those meeting DSPS criteria and those with an evening-preference met criteria for an historic diagnosis of mood, substance use or anxiety disorders (Reid et al., 2012). Interestingly, there was no significant difference in prevalence of Axis-I disorders between those with DSPS and those with only evening-type circadian preference, suggesting that these disorders may be influenced by circadian type as opposed to the occurrence of DSPS. Research from Chelminski, Ferraro, Petros, and Plaud (1999) indicates that students who score as ‘evening types’ on the Horne-Ostberg morningness-eveningness dimension report significantly more

depressive symptoms than those who score as ‘morning types’. Additionally, Sheaves et al. (2015) discovered university students reporting higher occurrences of psychiatric symptoms, including hallucinations, paranoia, depression, anxiety and (hypo)mania, had significantly later chronotypes than those reporting lower occurrences of psychiatric symptoms.

The mechanism behind the relationship between evening-type circadian preference, DSPS, and mental illness is not fully understood. Reid et al. (2012) theorise that low levels of light exposure may contribute to the development of depression in these individuals. Individuals with evening-type circadian preference may have limited light exposure due to their sleep schedule. Low light exposure during the winter months has been linked to seasonal affective disorder, an affective disorder with symptoms similar to depression. Shirayama et al. (2003) posit that sufferers of DSPS fit a certain psychological profile. They discovered that individuals with DSPS are more likely to exhibit certain personality traits, including defensiveness, neurosis (including hypochondriasis), compulsivity, and impulsivity. Shirayama et al. (2003) suggest these traits may encourage withdrawal or removal from social situations, and in turn cause a loss of social cues that allow their circadian rhythms to synchronise to a socially accepted schedule.

**Treatment of Delayed Sleep Phase Syndrome**

Recommended treatments for delayed sleep phase syndrome include light therapy, exogenous melatonin, and chronotherapy (progressively delaying sleep phase until a desired sleep onset time is reached) (Morgenthaler et al., 2007). Light therapy is a treatment approach that involves exposing an individual to light of a certain wavelength and intensity, either using natural outdoor light or a light box for indoor use. The aim of light therapy in the treatment of DSPS is to ‘reset’ an individual’s

subjective biological clock so that their sleep phase aligns with their desired sleep time, and preferably synchronises with their social environment (Shirani & Louis, 2009). Bright (or even low) levels of light, can suppress melatonin (Knauer, 1980) thus the circadian rhythm is sensitive to light, and can be shifted with light exposure (Minors, Waterhouse, & Wirz-Justice, 1991). Light exposure can phase advance or phase delay the circadian rhythm, depending on what time of day it is administered. Light exposure in the morning (after the core body temperature nadir) can advance circadian sleep phase (Khalsa, Jewett, Cajochen, & Czeisler, 2003; Minors et al., 1991). Light exposure towards the end of the day or start of the night (before temperature nadir) delays the circadian sleep phase.

The American Academy of Sleep Medicine has published practice parameters for the clinical evaluation and treatment of circadian rhythm sleep disorders (Morgenthaler et al., 2007). They suggest that light therapy can be useful in the treatment of delayed sleep phase syndrome, and recommend broad spectrum light at an intensity of 2,000-10,000 lux, administered in the early morning. No guidelines exist to recommend the exact ideal frequency, length, or duration of light therapy. Light therapy can be self-administered by the individual in their own home via a portable plug in light box following treatment guidelines given by a professional (Bjorvatn & Pallesen, 2009). Many light boxes today have a brightness of approximately 10,000 lux when placed at an appropriate distance from the individual. The client is instructed to direct their focus to the light box, ensuring the light enters through their eyes, but not to continuously stare directly into the light. Typical exposure time is 30-45 minutes daily (Bjorvatn & Pallesen, 2009).

Several studies have assessed side effects of light therapy treatment, including eye strain, headaches, and ocular abnormalities. Gallin et al. (1995) provided

ophthalmologic examinations to patients receiving light therapy at an intensity of 10,000 lux for two to eight weeks, and a separate group of patients receiving light therapy at the same intensity for three to six years. No ocular abnormalities were observed in patients in either group. Pallesen, Nordhus, Skelton, Bjorvatn, and Skjerve (2005) found a proportion of older adults who had undergone light therapy at 10,000 lux reported eyestrain (29%) and headaches (19%). However, when compared with a control group, who were administered light therapy at 200 lux, the treatment group (10,000 lux) actually reported fewer symptoms. The symptoms that were reported were short lasting. Terman and Terman (1999) examined reported symptoms from 83 participants exposed to light therapy at 10,000 lux for 30 minutes a day over 10-14 days. Mild dizziness, headache, and nausea were reported by a small proportion of participants, however, Terman and Terman (1999) reported a substantial benefit-to-risk-ratio. These studies conclude that if patients experience side effects from light therapy, they are likely to be mild and short-lasting.

**Light Therapy and Delayed Sleep Phase Syndrome Research**

Rosenthal et al. (1990) pioneered the use of light therapy to treat DSPS. Core body temperature measures revealed that after treatment with 2,500lux for two hours in the morning, participants had a significant average phase advance of 1 hour 25 minutes. More recent research (Cole, Smith, Alcal, Elliott, & Kripke, 2002; Danielsson, Jansson-Fröjmark, Broman, & Markström, 2013; Gradisar, Dohnt, et al., 2011; Sharkey, Carskadon, Figueiro, Zhu, & Rea, 2011; Wilhelmsen-Langeland et al., 2013) has validated these results, and found light therapy can shorten sleep onset latency, encourage earlier sleep onset and wake times, and advance circadian phase in youth and adults with DSPS.

Objective measures of sleep parameters, including polysomnography and core body temperature, provide reliable records of changes in sleep following light therapy. Watanabe, Kajimura, Kato, Sekimoto, and Takahashi (1999) examined changes in their polysomnogram in six individuals with DSPS who were administered three hours of morning light therapy for five days. Sleep onset and offset times were significantly advanced following light therapy, while total sleep time and REM were decreased. However, this study required participants to spend large amounts of time in a sleep lab, which is not practical for many studies. Thus, the majority of trials using light therapy for DSPS have been conducted outside the lab.

Rosenthal et al. (1990) woke subjects at a standard early morning time (approximately 6am) for light exposure. It is also acceptable to begin by exposing an individual to light at their normal wake time and have them gradually wake earlier day by day, alongside light exposure on awakening, until they reach their desired wake time (Terman et al., 1995). This is known as timed light exposure. In fact, it may be beneficial to do so, as initially exposing individuals to light in the early morning before core body temperature nadir (~2 hours before waking naturally) may not have the desired effects of phase advance, and instead can cause phase delay (Bjorvatn & Pallesen, 2009). Additionally, light exposure that occurs close to nadir has a greater effect on phase advance or delay than exposure further away from the timing of nadir, so it follows that early morning light exposure just before nadir can have a significant effect on phase delay (Minors et al., 1991), a detrimental effect if the goal is a phase advance. Cole et al. (2002) administered timed light exposure using face masks emitting bright white light (2,700 lux), or a dim red light placebo condition, which participants wore at night. Light emitted from the masks gradually increased in brightness from 4 hours before wake time until participants woke.

Participants in the bright light condition showed a significant advance in timing of melatonin secretion, alongside an advance in sleep phase, compared to those in the dim red light placebo condition (Cole et al., 2002). This suggests timed light exposure is an effective method of administering light therapy, and demonstrates the link between melatonin secretion and sleep phase timing.

Light exposure can also be used alongside other methods to improve sleep in individuals with DSPS. Wilhelmsen-Langeland et al. (2013) demonstrated that long term (three month) melatonin supplementation and bright light treatment, alongside a gradually advanced wake time, resulted in improvements in subjective daytime sleepiness, cognitive function, and fatigue. Two published studies have used light therapy alongside cognitive behavioural therapy (CBT) to treat DSPS. Gradisar, Dohnt, et al. (2011) recruited 40 adolescents from the Child and Adolescent Sleep Clinic at Flinders University, Adelaide. Adolescents in the treatment group (N=23) were treated with light therapy at an intensity of ~1000 lux each morning for a maximum of two hours, and attended four weekly and two bi-weekly individual sessions with a psychologist, to address sleep hygiene and cognitions related to sleep. The control group (N=17) remained waitlisted throughout the study. Compared to the waitlist group, adolescents in the treatment group reported moderate to large post-treatment improvements in sleep onset latency; sleep onset and rise time; and day time sleepiness and fatigue. These improvements remained stable at a six-month follow up (Gradisar, Dohnt, et al., 2011). Danielsson, Jansson-Fröjmark, Broman and Markström (2013) presented light therapy and CBT components separately to young adults (mean age of 22 years) with DSPS to examine whether CBT would enhance or maintain the effects of light therapy. They found a non-significant trend that suggested that compared to light therapy alone, four weeks of CBT presented after

two weeks of light therapy maintained the positive effects on sleep schedule and slightly decreased symptoms associated with poor sleep.

**Present Study**

The purpose of the present study was to extend previous work (Danielsson et al., 2013; Gradisar, Dohnt, et al., 2011) by evaluating the effectiveness of timed bright light exposure (light therapy) and group cognitive behavioural therapy, on young adults (university students) with DSPS. However, while Gradisar, Dohnt, et al. (2011) and Danielsson et al. (2013) used randomised control trials (RCTs), this study employed a multiple-baseline across-groups design (Cooper, Heron & Heward, 2007). This design was chosen as the research was conducted in a health service setting and was evaluating a treatment package established in this setting. Therefore, this study was concerned with effectiveness, and not with efficacy (Gartlehner, Hansen, Nissman, Lohr & Carey, 2006). Such research is often more appropriately conducted using single-case designs, as opposed to RCTs, as they don't require as large a group of participants, focus on clinical significance and change at an individual level, and encourage a graphical presentation of data to allow viewers to apply their own judgment (Blampied, 2013). Furthermore, where previous research used one-on-one sessions to administer CBT, this study employed techniques to aid sleep in a group session format. Group therapy is a lower cost and time saving alternative to individual therapy and has been found to be as effective at improving both beliefs about sleep and sleep itself in individuals with insomnia (Bastien, Morin, Ouellet, Blais, & Bouchard, 2004). Additionally, it has been demonstrated that after four to six 50-minute group or individual sessions, individual and group therapy are equally as effective at increasing total sleep time, sleep quality and efficiency, insomnia severity,

and dysfunctional beliefs and attitudes about sleep in individuals with insomnia (Bastien et al., 2004).

Participants in this study attended in four weekly group treatment sessions, while receiving daily timed light exposure for one hour per day. Sleep and wake onset times were measured throughout the study, as well as pre- and post-treatment, using a daily sleep diary. Pre-treatment, post-treatment and three month follow up measures of quality of life, emotional state, academic performance, and daytime sleepiness were collected. It was predicted that treatment with light therapy and group treatment sessions would result in significant improvements in sleep onset and wake times, quality of life, emotional state, academic performance, and daytime sleepiness. It was further predicted that, similar to Gradisar et al.'s (2011) findings, improvements would remain at a three month follow up.

## Method

### Participants

Participants were students at the University of Canterbury (Christchurch, New Zealand; see www.canterbury.ac.nz), enrolled in a variety of Arts, Science, Law and Social Science courses. Participants were selected for the study based on their response to advertisements posted around the University (detailed below).

A total of 77 students responded to the advertisements for the programme by expressing interest in participation. Of these, 51 responded to follow up emails detailing the programme and time commitment expected. The remaining students did not respond to follow up emails so were excluded. The remaining 51 participants were given a 'pre-screening' questionnaire (detailed below), which established whether they met basic criteria for DSPS. Of the 51, 26 were further excluded at this stage. Reasons for exclusions from the programme included not meeting pre -

screening criteria for DSPS, disclosure of significant mental health problems that would likely interfere with adherence and response to the programme, symptoms of another sleep disorder, frequent night shift work, and not being able to meet the time commitments needed for the programme. The remaining 29 participants were interviewed (by master's student R.M.) in a semi-structured format (detailed below). A further 11 were excluded from the programme at this stage, for the same reasons as listed above. Figure 1 summarizes participant inclusion/exclusion, completion, and dropout over the course of the study.

The remaining 18 students accepted entry into the programme. These participants ranged in age from 17 to 45 years, with a mean age of 23.47 (SD = 7.09). Thirteen (72%) were male, five (28%) were female. Nine participants (50%) identified their ethnicity as 'NZ European', three identified as 'NZ Maori/NZ European', one identified as 'NZ Maori', and four identified their ethnicity as 'other' (Brazilian, Chinese, Sri Lankan, Indian). These 18 participants completed the programme in three groups of six. Groups received treatment at different points during the university year. Participants were allocated to groups based on when they expressed interest in the study. The first six students who contacted the researcher, attended the assessment interview and met the ICSD-3 criteria for Delayed Sleep Phase Syndrome, were given preference for the first group, beginning during the mid-semester break in the second semester of the 2015 university year (August 2015).

Another group of six subjects were recruited during the second semester and completed the programme during the end of year study break and exam period (October- November 2015). The final group of six subjects was recruited over the 2015/2016 summer school period, and completed the programme at the beginning of

the first semester of 2016 (February - March 2016). Unfortunately, there were drop-outs from each group.

One participant from group 1 dropped out, after completing one week of the programme. Two participants from group 2 dropped out. The first participant left after completing two weeks of the programme, while the second left after completing three weeks. Three participants from group 3 dropped out. The first participant left after completing one week, while the second and third left after completing three weeks of the programme. Participants cited the inability to fulfill the time commitment required from the programme and personal problems, as reasons for dropping out. These drop-outs are summarized in Figure 1.

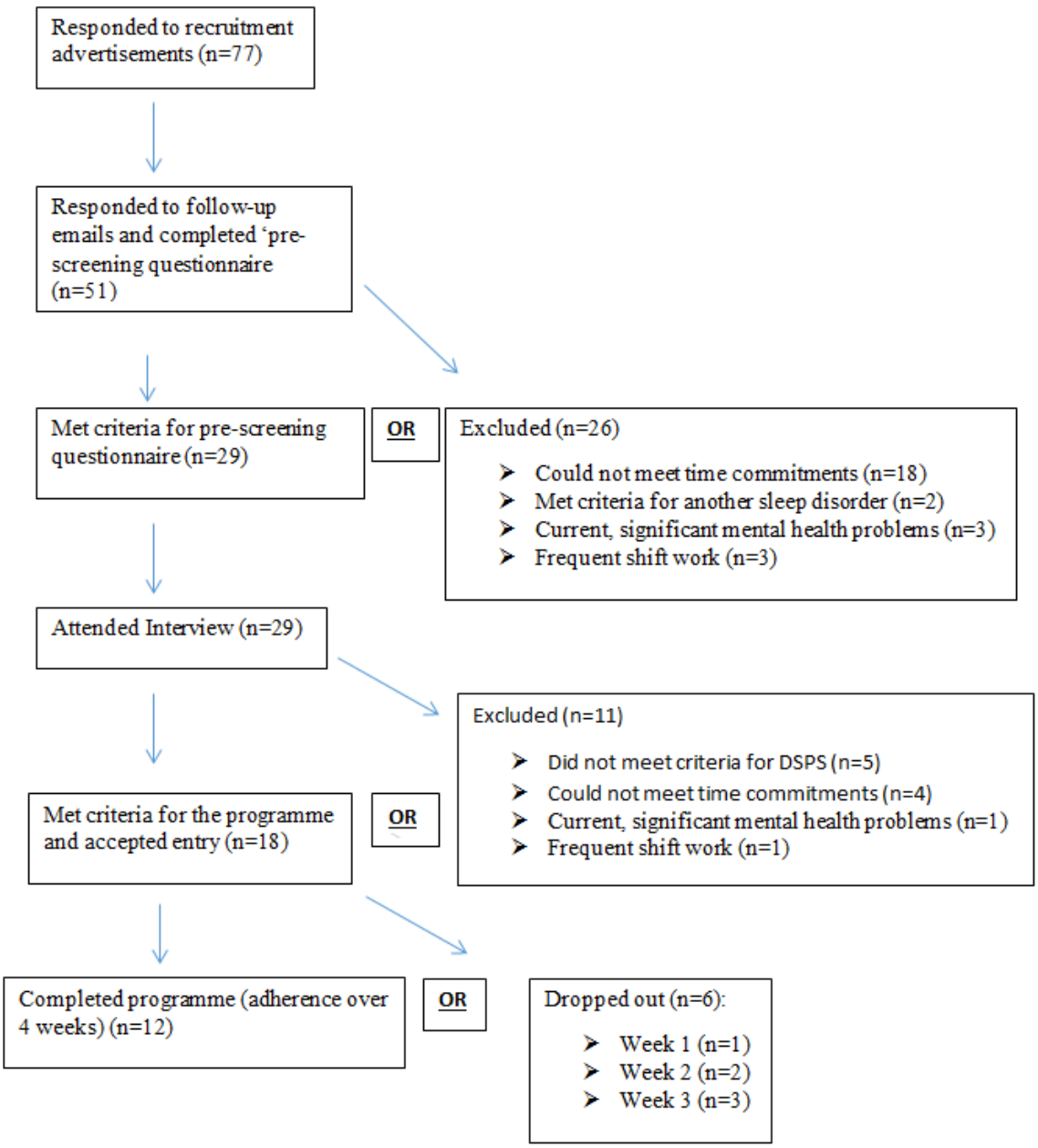


*Figure 1.* Flow diagram detailing participant inclusion/exclusion, completion, and dropout over the course of the study

**Apparatus and Materials**

Light therapy was administered using light boxes (Bright Light Daylight Simulator TL40) purchased from Beurer Products LTD. Beurer Products LTD specify that The TL40 has a standard light intensity of 10,000 lux when placed 20cm away from the face, and a Kelvin colour temperature of 6,500 (to simulate natural daylight).

Lux output from the light boxes was also measured by the researcher using a digital light meter, once the product had been received. With the light box positioned 20cm from the user, the light meter recorded ~6300 lux. Positioned 30cm away from the user, the light meter recorded ~3000 lux, and positioned 60cm away recorded ~1000 lux. To obtain a reading of 10,000 lux the light box needed to be positioned 13cm away from the user.

## Measures

**Continuous measures.** Participants were instructed to keep sleep diaries prior to, during, and after the treatment programme, recording basic sleep parameters each morning. The Consensus Sleep Diary (Carney et al., 2012) provided a template for participants to use, and also provided instructions on how to fill out their sleep diary.

***The consensus sleep diary (Carney et al., 2012).*** The Consensus Sleep Diary is a project that began with the collaboration of insomnia researchers at the 2005 Insomnia Assessment Conference in an attempt to provide a standardized assessment of insomnia and other sleep disorders (Carney et al., 2012). The core section of the sleep diary, which was used in this study, provides space for participants to record basic sleep parameters, namely (1) the time the participants went to bed; (2) they time they tried to sleep; (3) how long it took them to get to sleep; (4) how many times they woke during the night, (5) how long these awakenings lasted; (6) the final awakening time; (7) the time out of bed; and (8) the perceived quality of sleep. There was room for one week's worth of recorded parameters on each sleep diary, which fit on an A4 sheet of paper.

Attached to the Consensus Sleep Diary were instructions on how to fill it out, including clarification on how to answer each item, how often to fill out the dairy, and what to do if participants missed a day. Participants filled out the sleep dairy for two

consecutive weeks before attending the interview. This provided a baseline measurement of their sleep, and also helped determine whether they met DSPS criteria. Participants also completed the diary for four consecutive weeks during the treatment programme, and two consecutive weeks post-treatment.

No information is currently available regarding the validity and reliability of the Consensus Sleep Diary.

**Pre- and Post-treatment Measures.** At the beginning and the end of the four week treatment period, participants were instructed to complete the WHOQOL-BREF; Epworth Sleepiness Scale; DASS-21; and The Perceived Self-Efficacy subscale from the Student Approaches to Learning, and Self-Efficacy for learning and performance subscale from the Motivated Strategies for Learning Questionnaire (MSQL) for College students. These measures were used to determine post-treatment quality of life, day time sleepiness, and emotional state and academic performance, respectively. Three months after finishing the treatment, participants were requested to complete the same questionnaires, in an online format, in order to obtain follow up data.

***The WHOQOL-BREF (Harper, 1998).*** The WHOQOL-BREF (Harper, 1998) is a short version of the WHOQOL-100, a quality of life assessment created by the WHOQOL group at the World Health Organisation. The WHOQOL-BREF contains 26 questions, including 24 questions from the 24 facets of quality of life (as determined in the WHOQOL-100) grouped into the categories ‘Physical Health’, ‘Psychological’, ‘Social Relationships’, and ‘Environment’ (Harper, 1998).

Participants were instructed to rate between 5 and 1 (5 being ‘not at all’ and 1 being ‘an extreme amount’, ‘extremely’, or ‘completely’) the extent to which they were affected various health issues, and their ability in various aspects of their life.

They were also instructed to rate between 1 and 5 (1 being 'very dissatisfied' and 5 being 'very satisfied') their satisfaction with various elements of their lives. Participants were instructed to answer all questions based on how they had felt over the past four weeks.

Domain Scores were created for questions pertaining to the domains 'Physical Health', 'Psychological', Social Relationships', and 'Environment'. The scores of the questions within each domain were summed to create a total raw score for each domain. Transformed scores between 0-100 were calculated according to WHOQOL (1996) guidelines (Harper, 1998). Internal consistency within each domain (other than for Social) has been demonstrated to be adequate across populations in a number of countries (Physical $\alpha$= 0.82, Psychological $\alpha$= 0.81, Social $\alpha$= 0.68, Environment $\alpha$= 0.80) (Skevington, Lotfy, & O'Connell, 2004).

The WHOQOL-BREF has been shown to discriminate significantly between 'well' and 'sick' individuals, across all domains (Physical, Psychological, Social, and Environmental) (Skevington et al., 2004).

***The Epworth Sleepiness Scale (Johns, 1991).*** The Epworth Sleepiness Scale (ESS) is a questionnaire to assess an individual's likelihood of dozing in various everyday situations. Participants were instructed to rate between 0 and 3 (0 being 'would never doze' and 3 being 'high chance of dozing') how likely they believed they were to doze in situations including 'sitting and reading', 'watching television', and 'sitting inactive in a public place'. The scores from each item were summed to give a total score between 0 and 24. Participants completed the questionnaire before and after the treatment programme. The Epworth Sleepiness Scale has been shown to have good test-retest reliability (r=0.82), and a high level of internal consistency ($\alpha$=0.88) in a student sample (Johns, 1992).

***Depression, Anxiety and Stress Scale (DASS) -21 (Lovibond & Lovibond, 1995).*** The DASS-21 is a short form version of the 42 item Depression, Anxiety and Stress Scale, a measure of negative emotional states developed by Lovibond and Lovibond (1995). The DASS-21 has been demonstrated to have high reliability and moderate convergent and discriminate validity (Imam, 2007). Participants were instructed to read each of the 21 statements in the questionnaire and circle a number from 0 (to indicate 'Never') to 3 (to indicate 'Almost Always') that corresponded with how much the statement had applied to them over the past week. The scores for the items in each of the three subscales (Depression, Anxiety and Stress) were totaled to give a score for each domain. Participants' subscale scores were rated as 'Normal', 'Mild', 'Moderate', 'Severe', or 'Extremely Severe'. It is also permissible to use the sum of the DASS21 scores as an overall index of distress. For comparability with DASSS-42 scores it is conventional to double DASS-21 scores (Lovibond & Lovibond, 1995).

***The Self-Efficacy for Learning and Performance subscale from the Motivated Strategies for Learning Questionnaire (MSQL) for College Students (Pintrich, Smith, Garcia and McKeachie, 1991).*** The Self Efficacy for Learning Questionnaire is a subscale of the Motivated Strategies for Learning Questionnaire for College Students, a questionnaire designed to assess university students' motivation to learn and ability to use effective learning strategies in their course of study (Pintrich, Smith, García, & McKeachie, 1993). The Self Efficacy for Learning and Performance subscale assess the students' expectancy of success in their course of study, and their academic self-efficacy (belief in one's ability to master a task) (Pintrich et al., 1993). Participants were asked to rate between 1 ('Not at all true of me') and 7 ('Very True of me'), how true to them 8 statements about academic

performance were. Scores were summed to give students a total score. The Self Efficacy for Learning and Performance Subscale has been demonstrated to have a high level of internal consistency ($\alpha$=0.93) and correlate moderately (r= 0.41) with final university course grade, in a student sample (Pintrich et al., 1993).

**Procedure**

This study was approved by the University of Canterbury's Human Ethics Committee (see Appendix for a copy of this approval letter). Participants were selected for the study based on their response to flyers, a post on the University of Canterbury Student's Association Facebook page, an entry on a University of Canterbury Communications blog, an interview on the student radio station (RDU), and emails sent to department administrators and lecturers, all requesting participants for the study. Flyers advertising the study were posted on noticeboards around campus. Flyers and other recruitment methods requested that interested students contact the researcher by email if they had trouble 'going to sleep and waking up at a normal time, and had been unable to fix this', and if they also wished to participate in a free four-week treatment programme that may help them to get to sleep earlier and wake up more easily.

When individuals first contacted the researcher by email, they were sent a reply answering any questions they had asked about the study. Participants were also given a copy of an information sheet, a consent form, and a short pre-screening questionnaire containing five yes/no questions pertaining to whether the participant believed he/she was committed and motivated enough to change their sleep habits using our programme, and whether the participant had trouble both waking up and going to sleep at a conventional time. If the participant had answered 'yes' to all questions, they were sent an email inviting them to attend a semi-structured

assessment interview with the researcher (R.M, a postgraduate student and author of this Master's thesis) to determine whether they met ICSD-3 criteria for DSPS. Participants were given a link to follow in order to book an appointment time via an online appointment scheduling app. In this same email, participants were also given two one-page sleep diary templates (Consensus Sleep Diary, Carney et al., 2012), which required daily entries over two consecutive weeks, alongside instructions on how to fill it in. Participants were asked to fill in this sleep diary every morning for two weeks, to determine their baseline sleep habits, and bring the completed diary to the Assessment Interview.

The Assessment Interview was adapted from Gradisar et al.'s (2011) Clinical Sleep History interview (see Appendix for revised version), developed in 2011 at Flinders University in Adelaide, for adolescents with suspected sleep phase delay. This interview assessed sleep habits, sleep history, daytime functioning, worries surrounding sleep, bedtime routine, sleep hygiene, and screened for differential diagnoses. Participants were informed that any information collected by the researcher would remain confidential and anonymous. The interviews were conducted by the author (under the supervision of Alex Mortlock (registered clinical psychologist). Participants were asked to set aside one hour for this interview. The interviews took place in a private office space in the Department of Psychology, and when this was unavailable, a small, private discussion room in the University Central Library.

Following the interview, participants were asked to complete several questionnaires: (1) the WHOQOL-BREF to measure quality of life; (2) the Epworth Sleepiness Scale (Johns, 1991) to assess daytime sleepiness; (3) The Self-Efficacy for Learning and Performance subscale from the MSQL for College Students (Pintrich et

al., 1991) to assess self-efficacy in the university courses the participants were currently taking; and (4) the DASS-21 , to asses emotional state. After each interview the researcher and clinical supervisor met to discuss the participant's suitability for the programme. Participants who met ISCD-3 criteria for DSPS, and did not have a differential diagnosis, medical problem, or any other confounding problems, were accepted into the study, providing the researcher and supervisor agreed on the participant's suitability.

Participants were notified by email whether they had been accepted into the study. Those who had not been accepted were advised of the reason the researcher and supervisor believed they should not participate. These participants were also advised that they could make an appointment with Alex Mortlock (-a clinical psychologist) at the Student Health Centre or a General Practitioner for no charge, to discuss an individualised treatment plan that could address their specific needs.

Group sessions were scheduled based on the participants' and facilitators' availability. These were conducted by Alex Mortlock , assisted by the researcher, and took place in a private meeting room in the Health Centre at The University of Canterbury. Four weekly group treatment sessions took place over the four weeks of the treatment programme, each with a different theme. Prior to each session, participants were given a handout, which included a summary of the information covered during the session.

Participants were instructed to self-administer one hour of timed light exposure via their choice of a light box or natural outdoor light, each morning during the four week treatment programme. On the first day of use, participants were instructed to wake up naturally (i.e. without the use of an alarm or wakeup call), and sit with the light box in their field of vision at a distance of 20cm, or as close as

comfortable, for 1 hour, immediately after they wake. Thereafter, every second day, participants were instructed to wake with an alarm set 20 minutes earlier than their last wake up time two days before. Participants were instructed to wake up 20 minutes earlier every second day until they reached their desired wake time (which was determined by the participant, under the guidance of the researcher, in the first group session). Participants were given a chart to help them plan and remember light exposure times. Over the four week period, participants had the opportunity to wake approximately 1 hour earlier each week. When participants reached their desired wake time they were instructed to cease using the light box, and attempt to continue to wake at their desired wake time each morning. If participants experienced any difficulties with timed light exposure, such as missing a day, not being able to commit to the full hour of light exposure, problems with the light box (amongst other things) they were encouraged to contact the researcher by email. Each weekly group session also included a check in on how participants were finding the timed light exposure and any problems that arose were discussed and a solution was offered.

Alongside bright light exposure on waking, participants were instructed to reduce exposure to artificial lighting in the two hours before bed. This included dimming the lights in the room, and limiting computer, laptop, tablet, phone and other screen use. If participants could not avoid using screens in this time frame, they were advised to download and use the application ‘f.lux’, which changes the colour of the screen display to reduce blue light output. During the first group treatment session, participants were given hard copies of sleep diary templates to last for the four weeks of the programme. Participants were instructed to use the Consensus Sleep Diary to record basic sleep parameters, corresponding to the previous night’s sleep, each morning when they woke up. Participants were encouraged to contact the researcher

by email as soon as possible if they had any problems with the sleep diary, or entering basic sleep parameters.

In the weeks following the fourth group session, participants made an appointment to see the researcher individually. Participants returned their final week of sleep diary entries and their light box. They were given an evaluation form and asked to evaluate the treatment programme, rating various aspects of the group treatment sessions on a scale ranging from 1 (strongly agree) to 5 (strongly disagree). They were also asked to provide any relevant feedback regarding any changes they felt could be made to improve the programme.

Participants were contacted by email approximately three months after the final group session took place. Online versions of each of the four questionnaires provided pre- and post-treatment were created using the software Survey Monkey (https://www.surveymonkey.com/). Participants were sent hyperlinks to access these surveys and were asked to complete them at their earliest possible convenience. Once the surveys were completed and returned, participants were thanked for their time and effort.

**Programme Outline**

The programme integrated a number of insomnia treatment components to address physiogical, behavioural, and emotion-regulation aspects of delayed sleep phase syndrome. It was hypothesised that including treatment strategies for insomnia would enhance treatment aimed at circadian entrainment.

**Stimulus Control**. Stimulus control therapy is a well-established behavioural treatment for insomnia. The intervention was originally developed by Bootzin (1972) and has a good evidence base for use both as a stand-alone treatment and as a

component of a more comprehensive cognitive-behavioural therapy package (Morgenthaler, et al., 2006).

The rationale for stimulus control is based on the idea that *falling asleep* is a behaviour influenced by antecedent stimuli that are amenable to modification. The intervention aims to enhance stimuli promoting sleep and reduce stimuli associated with wakefulness. This should lead to sleep coming under 'stimulus control' as is hypothesised to be typical of good sleepers.

**Mindfulness**. Mindfulness-based interventions for sleep problems are a relatively recent development. Training sleep-disordered patients in mindfulness skills is hypothesised to be particularly useful for reducing the distress and emotional reactivity that contribute to insomnia (Ong & Manber, 2011). Mindfulness interventions have shown promise both as a stand-alone treatment (e.g., Carlson & Garland, 2005) and as one component of a cognitive-behavioural therapy package for chronic insomnia patients (e.g., Ong, Shapiro, & Manber, 2008).

**Relapse Prevention**. Relapse prevention is a cognitive-behavioural therapy component included at the end of treatment (Beck, 2011). The aim is to teach the client(s) to maintain their therapeutic gains independently. A common approach is to help clients identify high risk situations for possible relapse (e.g., changes in study schedule or late-night socialising), identify early warning signs of relapse (e.g., a desire to sleep later or mild early insomnia), identify positive coping strategies (e.g., adhering more consistently to treatment strategies), and to avoid unhelpful strategies (e.g. drinking more coffee, trying to 'catch up' on missed sleep by sleeping in).

The first group session began with an expression of appreciation for participants' attendance and participation. Participants were encouraged to introduce themselves and share what they hoped to gain from the treatment programme. Privacy

and confidentiality for participants and researchers was discussed. Participants signed a confidentiality agreement stating they would refrain from discussing other group members or any personal information disclosed, outside of group sessions. Furthermore, expectations of group members' attendance at all sessions and responsibility to do their best to adhere to the intervention were discussed. Participants were given the opportunity to ask questions at any time. Table 1 outlines the content covered in each group session.

Table 1. The content of each of the four group treatment sessions

| Session | Content |
|---|---|
| 1 | *Education on circadian physiology, DSPS, and the rationale for the treatment approach.*<br>• Basic information about DSPS (prevalence, age of onset, and symptoms)<br>• Introduction of information about circadian rhythms, their capability to delay, the underlying physiology, and the effect of light in delaying and advancing the body clock.<br>• Participants assisted to develop individual light therapy plans for each participant to implement over the 4-week period of treatment. |
| 2 | *Stimulus Control*<br>• Introduction to behavioural stimulus control sleep management protocol.<br>• Focus on the rationale for using stimulus control to help with sleep, highlighting internal and external factors that may disrupt sleep.<br>• Participants given stimulus control instructions to apply to encourage them to associate their bed with sleeping (going to sleep only when sleepy; avoiding using the bed for any activity other than sleep; getting up for 20 minutes if not able to fall asleep quickly; and setting an alarm for the same time every morning) |
| 3 | *Mindfulness to promote sleep*<br>• Introduction to the concept of mindfulness and how it can be applied to sleep, in order to obtain a better quality sleep.<br>• The concepts of non-striving, letting go, non-judging, acceptance, beginner's mind, trust, and patience, and how these can be applied to sleep explained and discussed.<br>• Participants taken through a ten minute mindfulness exercise led by the researcher. (Participants were later given an audio recording of |

| | |
|---|---|
| | this exercise to use at home). |
| 4 | *Long Term Management plan/Relapse Prevention*<br>• Participants introduced to strategies to improve sleep hygiene (maintaining a bedroom environment conducive to sleep, best practice for food, drink and exercise prior to sleep).<br>Participants asked to identify and share high risk situations that may cause them to relapse, early warning signs, and good and bad responses to this. |

**Design and Data Analysis**

This study employed a multiple-baseline across-groups research design Cooper et al., 2007), a modification of the standard multiple-baseline across individuals design where groups replace individuals; unfortunately for operational reasons the different groups had the same baseline durations (but there was replication across three groups). Baseline measures of continuous variables (sleep diary data) were taken over a period of 14 consecutive days, prior to the beginning of the programme. Baseline measures of quality of life, day time sleepiness, emotional state and academic self-efficacy were recorded once prior to the beginning of the programme. Sleep diary variables were recorded throughout the duration of the four week programme, and for 14 consecutive days immediately following the programme. Questionnaire data was recorded once in the weeks following the programme, and again three months after the end of the programme.

Data collected from participants' sleep diaries was plotted as individual time series. Self-reported number of awakenings, total length of awakenings, sleep onset latency, and sleep quality were analysed for change over time. Single case effect sizes for these sleep diary variables were obtained using Percentage Exceeding the Median (PEM) ((Ma, 2006). Medians were calculated for each participant's baseline phase. The percentage of values in each participant's intervention phase (during the

programme), and post treatment phase (calculated separately) exceeding the median value in their baseline phase, was used to determine PEM and establish an effect size. The percentage of values falling above or below the median line was calculated, depending on the direction of therapeutic change for each variable (i.e. whether increase or decrease signalled improvement) (Ma, 2006). In this instance, it was calculated as percent below the median for variables where the clinical direction was reduction. Effect size was calculated following the convention that PEM between 70% and 90% suggests a moderate effect size, while PEM exceeding 90% suggests a large effect size (Ma, 2009). The mean and standard deviation for each participant's baseline phase was calculated as a measure of baseline variance.

Estimated sleep onset and wake times, obtained from participants' sleep diaries, were entered into an excel spreadsheet as 24 hour clock times. These times were then converted to the equivalent number of minutes passed since midnight the previous night using the formula =IF(HOUR( insert 24 hr time value' )>12,HOUR(")*60+MINUTE("),(HOUR(" )+24)*60+MINUTE( ")), where each 24 hour time value is inserted within all of the brackets provided by the formula. The number of minutes past previous midnight (MPPM) was averaged over baseline, treatment, and post treatment phases for each participant, and the group. Standard deviations were calculated alongside mean values. Mean MPPM were then presented as 24 hour clock times.

**Modified Brinley Plots.** Modified Brinley Plots (Blampied, 2016) were used to analyse individual changes over time from (a) pre-treatment to post treatment, (b) pre-treatment to follow-up, and (c) post-treatment to follow up, on all questionnaire variables (quality of life (WHOQOL-BREF), day time sleepiness (Epworth Sleepiness Scale), emotional state (DASS-21) and academic self-efficacy (SELPs). Modified Brinley plots are a variation of Brinley plots (Brinley, 1965), which are scatter plots designed to display data examining the average cognitive performance of different groups (i.e. young and old participants) in certain conditions.

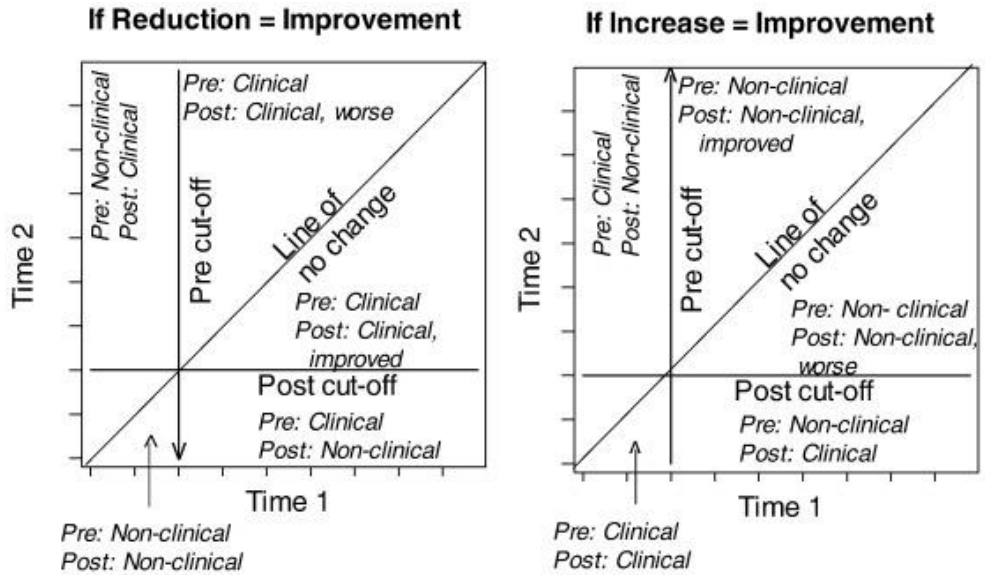


*Figure 2.* A guideline for interpreting modified Brinley plots, including the use of clinical cut offs to determine therapeutic effect (from Blampied, 2016).

Modified Brinley plots are scatter plots suited to single-case research. These plots are useful in single-case research as they are able to detect systematic effects of an intervention on a group as a whole, and also provide a visual display of each individual participant's data. Each individual participant's score from one point in time (i.e. baseline, post-treatment, or follow-up) is plotted against scores from another

point in time, and displayed as one data point. Data points for each participant are plotted on the same graph. A diagonal line through the centre of the graph represents a line of no change, so if an individual's baseline score and intervention score are the same, their data point would fall on this line. Data points that deviate from the line of no change line represent systematic effects of an intervention. Points falling above the line represent an improvement in score over time, while points falling below the line represent a worsening in score. Lines can also be placed on the graph to indicate clinical cut-off scores, providing a visual display of the clinical significance of outcomes (Blampied, 2016). Cohen's d effect sizes (Cohen, 1988) were calculated for pooled group data for each variable using software provided by Cumming (2013) and Lakens (2013).

## Results

Throughout the course of the programme, 6 participants dropped out at various points, resulting in incomplete data sets (see flow diagram in Figure 1). As these participants ceased attendance at group sessions and adherence to the daily light exposure regime, they were deemed to not have complied fully with the intervention. Therefore, data collected from their pre-treatment questionnaire variables and sleep diaries was not included in data analysis. Data sets from the 12 participants who completed the programme were analysed. All 12 participants provided baseline and post-treatment questionnaire data – i.e., these participants provided sleep diaries for 2 weeks of baseline, and 4 weeks of intervention, however, only 10 of the 12 participants provided 2 weeks of post-treatment sleep diaries and only 9 provided 3-month follow up questionnaire data.

## Questionnaire Data

**Daytime Sleepiness (Epworth Sleepiness Scale).** Scores of 10 and above on the Epworth Sleepiness Scale are considered problematic (Kendzerska, Smith, Brignardello-Petersen, Leung, & Tomlinson, 2014),therefore, a score of 10 was used as the clinical cut-off to separate 'normal' levels of daytime sleepiness from problematic levels. This is threshold is shown as a horizontal line in Figure 3.

At baseline, three participants scored above the clinical cut-off (participants 12, 44 and 74). A small reduction in daytime sleepiness was observed from pre-treatment to follow up (Cohen's d = 0.21). One participant remained in the clinical range following intervention, but reported reduced sleepiness, levels, while two participants no longer remained above the threshold, signifying reduced problematic sleepiness following intervention. No significant change in daytime sleepiness was observed between pre-treatment and post-treatment, or post-treatment and follow-up. Eight participants (67%) scored within the non-clinical range (signifying no significant problems with daytime sleepiness) at baseline, and remained in the non-clinical range following intervention.

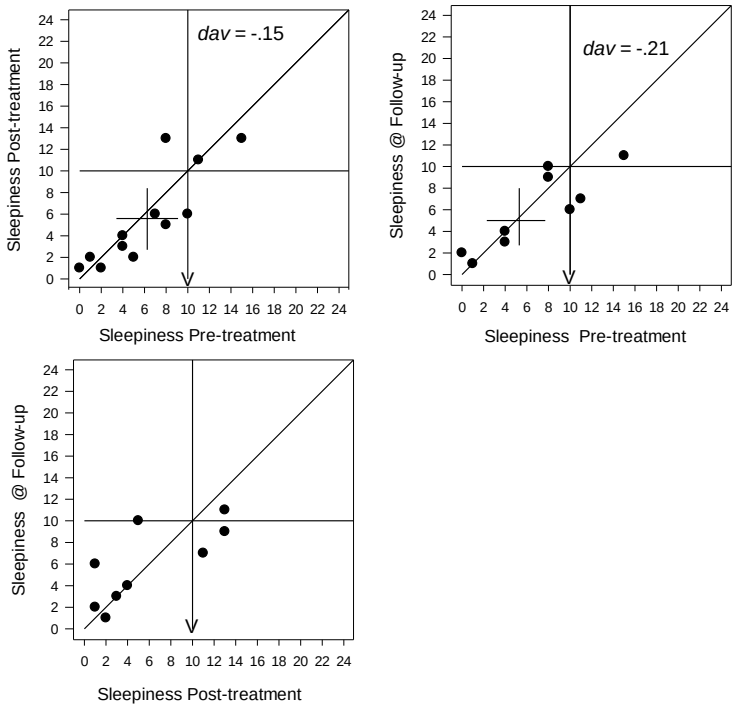


*Figure 3.* Data from the Epworth Sleepiness Scale as modified Brinley plots, completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up.

**Academic Self-efficacy (Self-Efficacy for Learning and Performance subscale from the MSLQ)**. A small reduction in academic self-efficacy was observed from pre-treatment to post- treatment (Cohen's d = 0.22). This is reflected in figure 4, which shows that six participants (50%) reported a reduction in academic self-efficacy (data points to the left of the diagonal line), five reported an increase (data points to the right of the diagonal line), and one participant reported no change. A small increase in academic self-efficacy was reported from pre-treatment to follow-up (Cohen's d = 0.2).

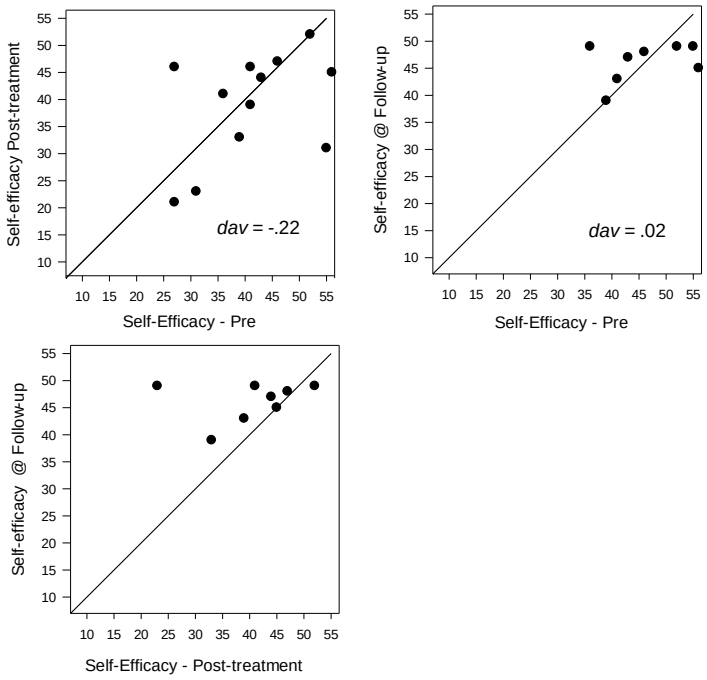


Figure 4. Data from the Self-Efficacy for Learning and Performance subscale from the MSLQ, as modified Brinley plots. The questionnaire was completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up.

**Quality of Life (WHOQOL-BREF).** Changes in self-rated physical, psychological, social and environmental quality of life are shown in Figures 5-8. A large increase in physical quality of life was observed between pre-treatment and post-treatment (Cohen's *d* = 0.92), with seven of the ten participants who completed both measures reporting an improvement. A moderate increase was observed between pre-treatment and follow-up (Cohen's *d* = 0.39), with five participants reporting improvements in physical quality of life, three reporting no change, and one reporting a reduction.

A moderate reduction in psychological quality of life was observed between pre-treatment and follow-up (Cohen's *d* = -0.58), with four of the eight participants who completed the measure at both time points reporting a reduction over time, two reporting no change, and one reporting an improvement. No significant change in

psychological quality of life was reported between pre-treatment and post-treatment or between post-treatment and follow-up.

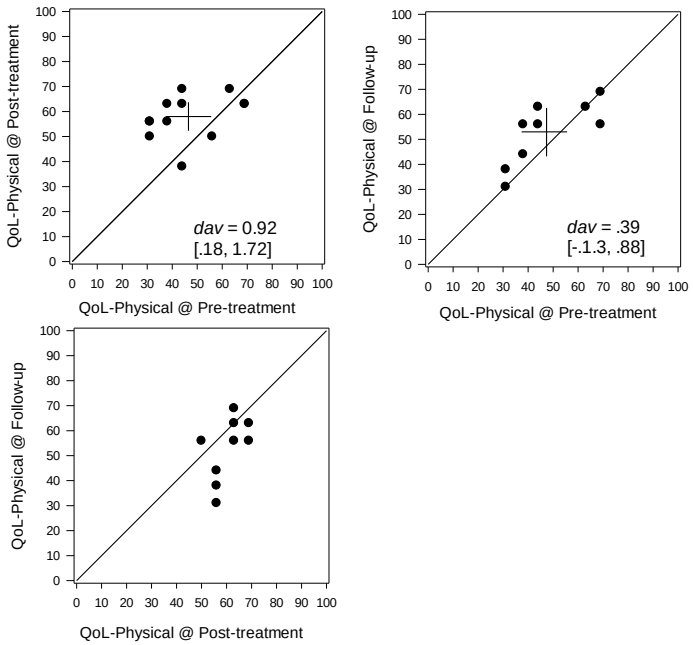


*Figure 5.* Data from the WHOQOL-BREF Physical QOL subscale, presented as modified Brinley plots, completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up.

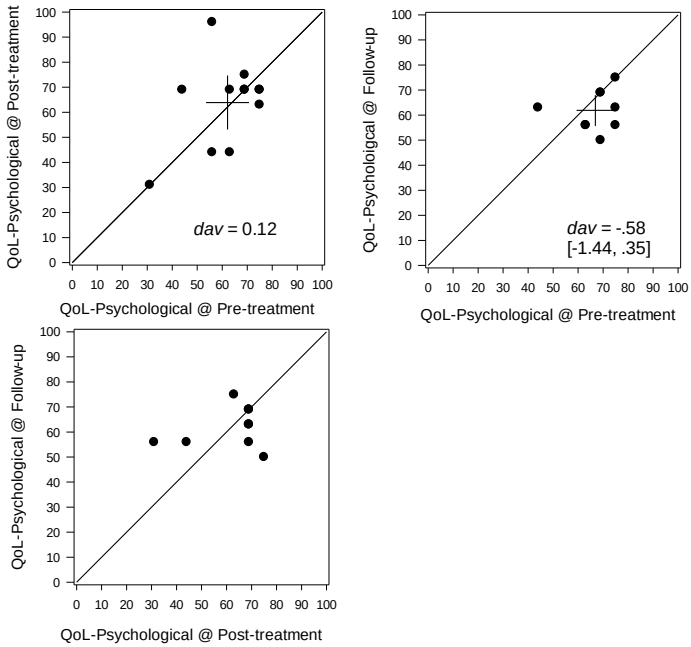


*Figure 6.* Data from the WHOQOL-BREF Psychological QOL subscale, presented as modified Brinley plots, completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up.

A moderate increase in environmental quality of life was observed between pre-treatment and follow-up (Cohen's d = 0.67), with five participants reporting an increase over time. No significant change in psychological quality of life was reported between pre-treatment and post-treatment or between post-treatment and follow-up.

A small reduction in social quality of life was observed between pre-treatment and follow-up (Cohen's *d* = -0.44), with five of the eight participants who completed the measure at both time points reporting a reduction over time, one reporting no change, and three reporting an improvement in social QOL. Similar to psychological and environmental quality of life, no significant change in social quality of life was reported between pre-treatment and post-treatment or between post-treatment and follow-up.

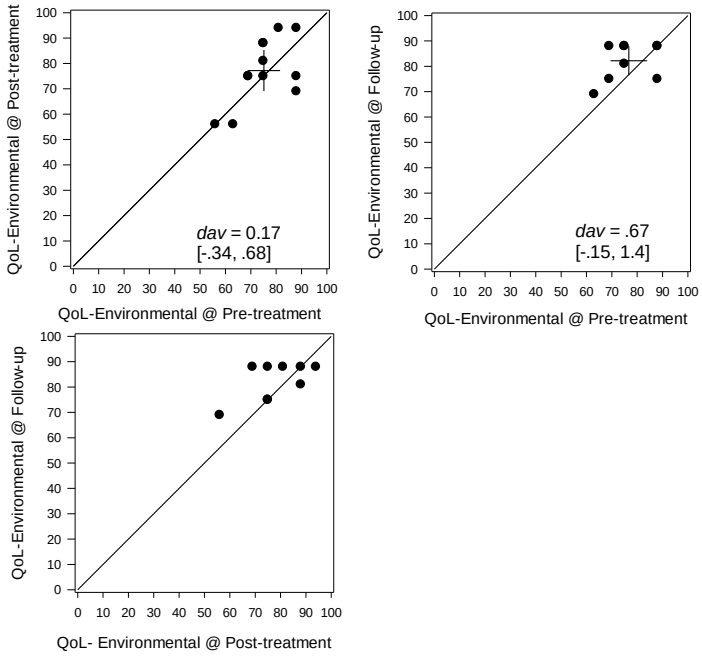


*Figure 7.* Data from the WHOQOL-BREF Environmental QOL subscale, presented as modified Brinley plots, completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up.

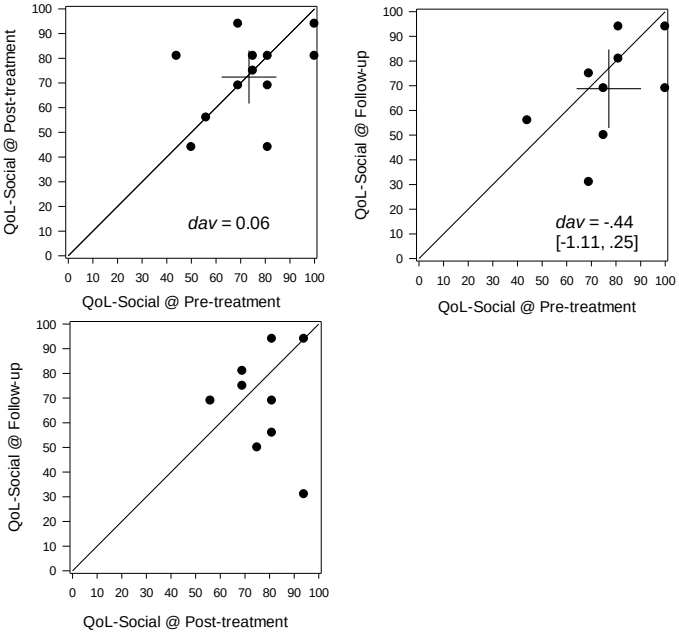


*Figure 8.* Data from the WHOQOL-BREF Social QOL subscale, presented as modified Brinley plots, completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up**.**

**Depression, Anxiety and Stress (DASS-21).** Changes in self-reported Depression, Anxiety and Stress symptoms across baseline, intervention and post-treatment phases are show in Figures 9-11.

Following the doubling of the DASS-21 scores conventional DASS clinical cut-off scores (Crawford & Henry, 2003; Lovibond & Lovibond, 1995) were used to separate those who reported symptoms within the normal range, from those who reported mild, moderate or severe depression, anxiety and stress symptoms. DASS-21 scores were doubled (as recommended by Lovibond & Lovibond (1995) to make use of DASS-42 cut-off scores already available.

***Depression.*** A cut-off score of 13 was used to separate normal and mild scores from moderate and severe depression scores. At baseline, eight participants (67%) scored below the clinical cut-off, while the remaining five scored above the cut-off. Following intervention, six participants remained below the clinical cut-off, while one participant moved from the clinical to non-clinical range, and another moved from the non-clinical to clinical range (see Figure 9). Two participants who scored in the clinical range at baseline reposted a reduction in depression symptoms following intervention, despite remaining in the clinical range. No substantive changes in depressive symptoms were observed between pre-treatment, post-treatment and follow-up.

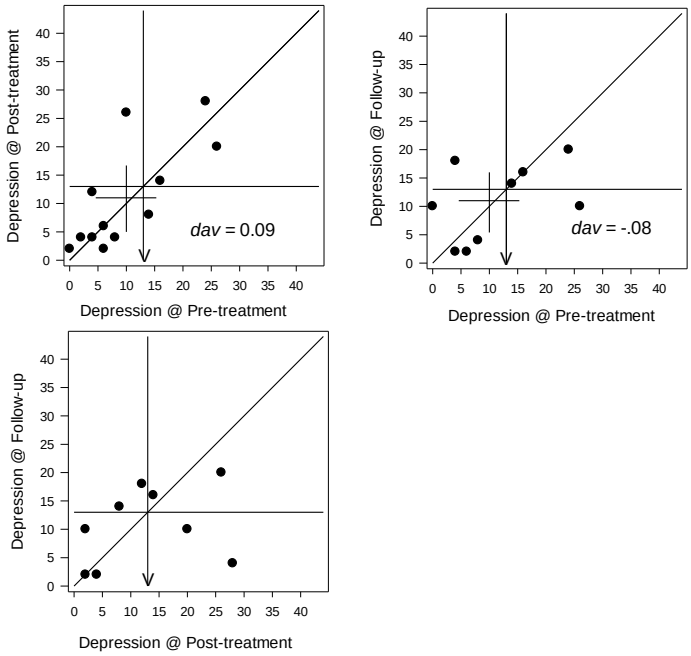


*Figure 9.* Data from the DASS-21 Depression subscale (scores doubled to accommodate DASS-42 cut-off scores) as modified Brinley plots, completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up.

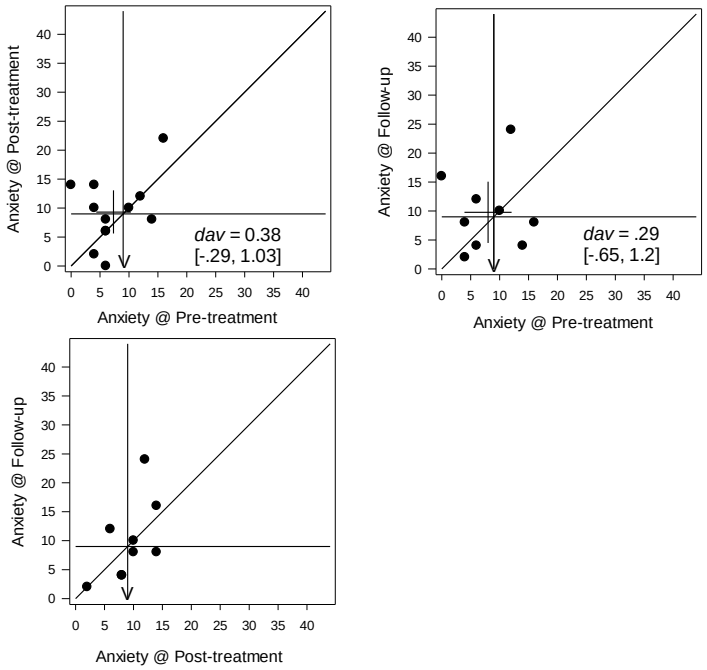


*Figure 10.* Data from the DASS-21 Anxiety subscale (scores doubled to accommodate DASS-42 cut-off scores) as modified Brinley plots, completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up.

***Anxiety.*** A clinical cut-off score of 10 was used to separate normal and mild scores from moderate and severe anxiety scores. At baseline, seven participants (64%) scored below the clinical cut-off, while the remaining four scored above the cut-off. A moderate increase in anxiety symptoms was observed between pre-treatment and post-treatment (Cohen's d = 0.38). Three participants moved from the non-clinical to the clinical range, indicating a worsening of anxiety symptoms, while two participants remained in the clinical range and reported an increase in anxiety symptoms (Figure 10). A moderate increase in anxiety symptoms was also observed between pre-treatment and follow-up (Cohen's *d* = 0.29).

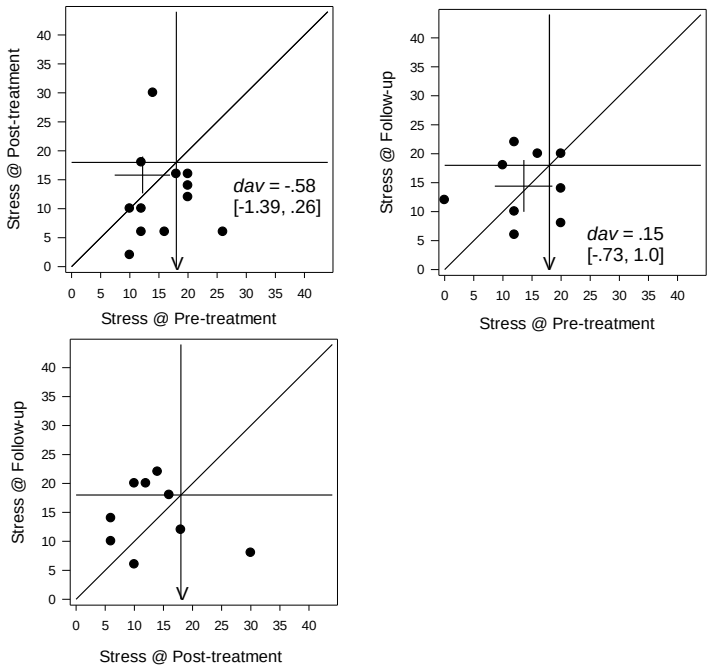


*Figure 11.* Data from the DASS-21 Stress subscale (scores doubled to accommodate DASS-42 cut-off scores) as modified Brinley plots, completed by participants at pre-treatment (baseline), post-treatment, and at a 3-month follow-up.

***Stress.*** A cut-off score of 18 was used to separate normal and mild scores from moderate and severe stress scores. At baseline, four participants (33%) reported symptoms within the clinical range. A moderate reduction in self-reported stress was

observed between pre-treatment and post-treatment (Cohen's d = -0.57). At post-treatment, all four participants in the clinical range at baseline moved to the non-clinical range, indicating an improvement in symptoms, while one participant moved from the non-clinical to the clinical range (see Figure 11). A moderate reduction in stress was also observed between pre-treatment and follow-up (Cohen's d = -0.37).

**Sleep Diary Data**

Sleep diary data was collected from participants at baseline, during the intervention, and post-treatment. The sleep dairy variables analysed included number of night time awakenings, total length of night time awakenings, sleep onset latency (time to fall asleep), and perceived quality of sleep. Baseline and Intervention sleep diary entries were completed by 12 participants and post-treatment sleep diaries by 10 participants. Data for each night of diary entry, and each variable, was plotted for each participant as separate time series. PEM was calculated to assess the outcome of the intervention, at both the intervention and post-treatment phases.

**Number and Total Length of Awakenings.** Few participants reported frequently experiencing more than 5 awakenings per night during the baseline, intervention or post treatment phase. Most participants reported the total length of awakenings as lasting less than 1 hour, aside from the occasional problematic night. However, two participants reported frequent long awakenings, namely participant 74 at baseline, and participant 44 during the intervention phase.

Two participants (96 and 60) reported few, if any, night time awakenings, and therefore minimal total length of awakenings. Therefore, the intervention had no effect on these variables as they were not problematic for these participants. One participant (12) reported a minimal number of night time awakenings at baseline, occurring for very short durations. PEMs for this participant indicate that intervention

may have had a moderate effect on the number of awakenings in the early stages, but did not continue (PEM = 62.5%), and did not have a substantive effect on the length of awakenings (PEM = 45.8%).

Three participants (31, 70, 74) reported a treatment effect on the number and length of awakenings at both the intervention and post-treatment phases. However, for participants 70 and 74 baseline number and length of awakenings are relatively variable, therefore inference of change in intervention and post-treatment phases is more tentative. Similarly, participant 58 reported a significant reduction in total length of awakenings at intervention (PEM = 85.7%) and post-treatment (PEM = 85.7%), however baseline data was variable. Participant 14's baseline number and length of awakenings are comparatively more stable, suggesting a significant decrease in these variables at both intervention (PEM = 100% (number), 85.7% (length)) and post-treatment (PEM = 92.8% (number), 100% (length)). Two participants (44, 73) reported no substantive treatment effect on number or length of awakenings. Participant 44 reported no substantial treatment effects on either variable at intervention and post-treatment phases, while participant 73 reported no effect at intervention, but did not supply post-treatment data.

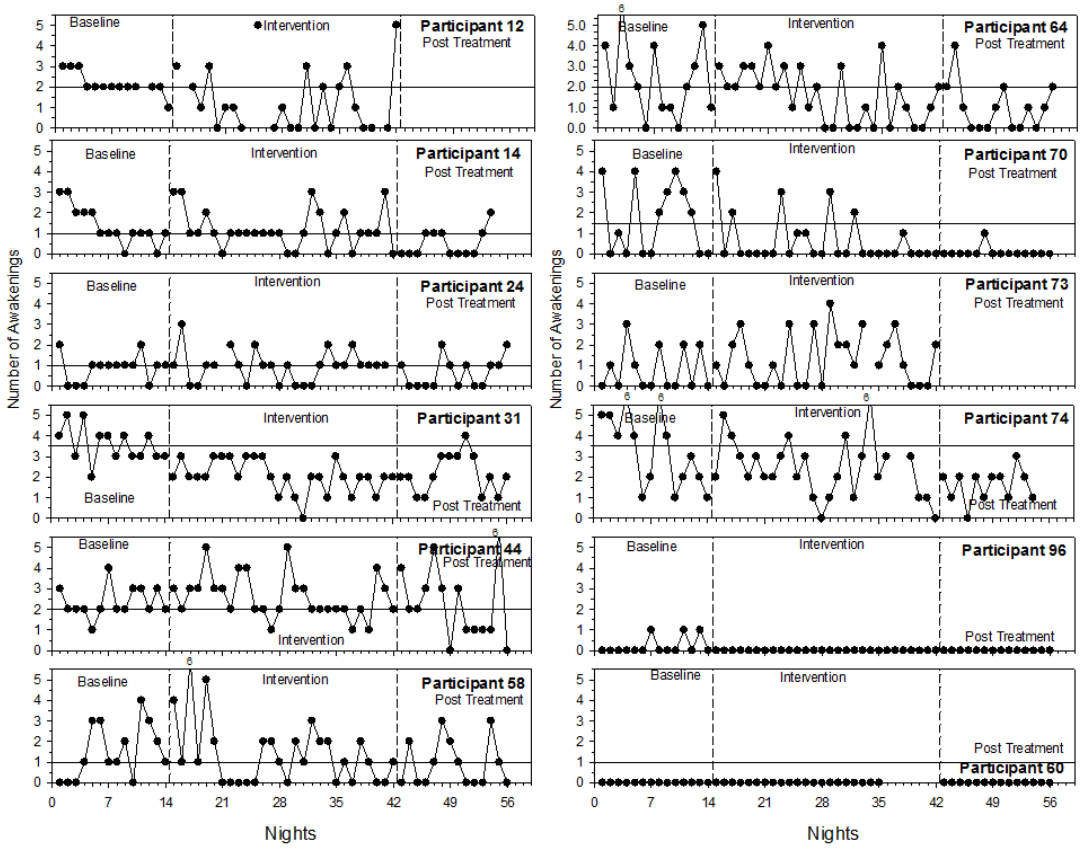


*Figure 12.* Time series graphs of self-reported number of awakenings, recorded for 2 weeks baseline, 4 weeks intervention, 2 weeks post-treatment using the Consensus Sleep Diary.

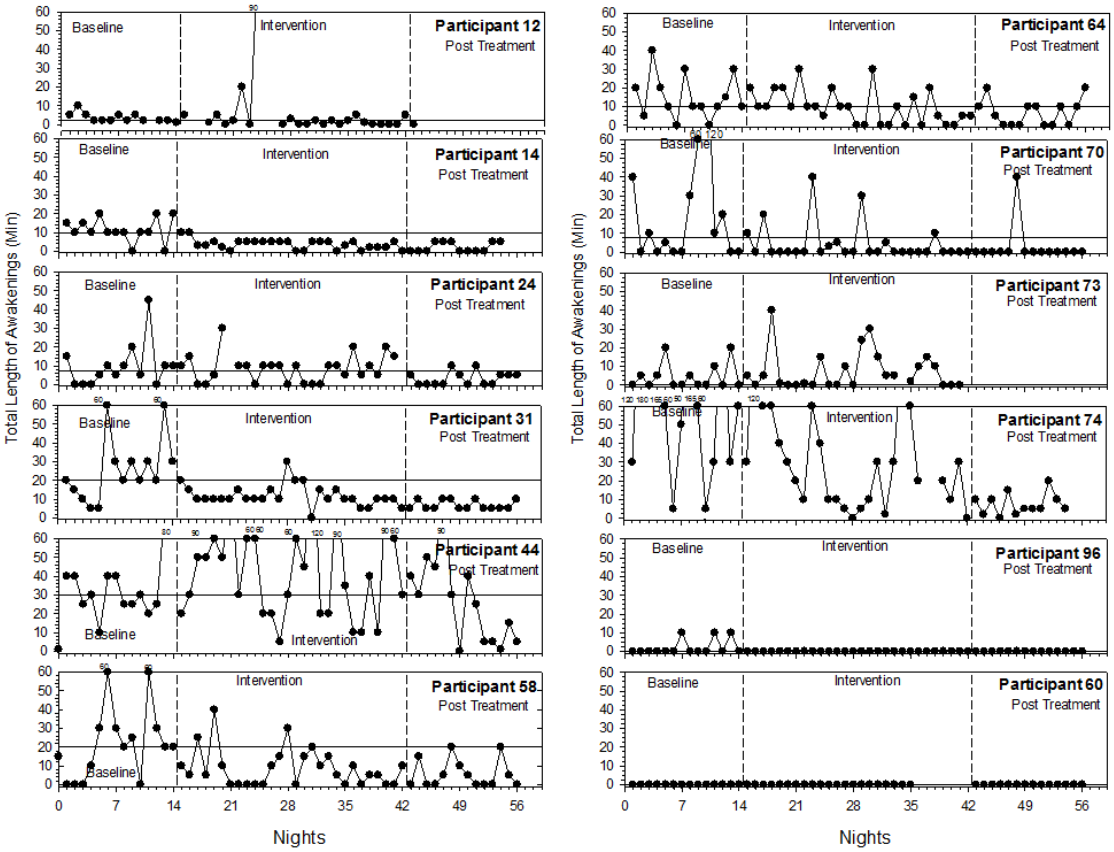


*Figure 13*. Time series graphs of self-reported total length of awakenings, recorded for 2 weeks baseline, 4 weeks intervention, 2 weeks post-treatment using the Consensus Sleep Diary.

Table 2. Percentage Exceeding the Median (PEM) for each participant and each dependent variable from the Consensus Sleep Diary, at Intervention (Int.) and Post-treatment (Post.) phases

| | Number of Awakenings | | Length of Awakenings | | Sleep Onset Latency | | Quality of Sleep | | Mean |
|---|---|---|---|---|---|---|---|---|---|
| Participant | Int. | Post. | Int. | Post. | Int. | Post. | Int. | Post. | |
| 12 | 62.50 | | 45.8 | | 40.00 | | 12.00 | | 40.07 |
| 14 | 21.43 | 58.33 | **92.86** | **100** | **85.71** | **75.00** | 40.74 | 25.00 | 62.38 |
| 24 | 26.92 | 50.00 | 42.31 | **78.57** | 60.00 | **78.57** | 13.00 | 0.00 | 43.67 |
| 31 | **100** | **92.86** | **85.71** | **100** | **89.29** | **78.57** | **100** | **84.62** | 91.38 |
| 44 | 10.71 | 42.86 | 32.14 | 50.00 | **85.71** | **78.57** | 37.04 | 64.3 | 50.17 |
| 58 | 35.71 | 50.00 | **85.71** | **85.71** | 64.29 | 42.86 | 41.67 | 42.86 | 56.10 |
| 64 | 46.42 | 71.43 | 42.86 | 50.00 | 67.9 | 50.00 | 25.00 | | 50.52 |
| 70 | **82.14** | **92.86** | **82.14** | **92.86** | **78.57** | 28.57 | **75.00** | 57.14 | 73.66 |
| 73 | 0.0 | | 0.0 | | 0.0 | | 14.81 | | 3.70 |
| 74 | **80.77** | **100** | **76.92** | **100** | **80.77** | **100** | 19.23 | 50.00 | 75.96 |
| 96 | 0.0 | 0.0 | 0.0 | 0.0 | **85.71** | **85.71** | 44.44 | 53.85 | 33.71 |
| 60 | 0.0 | 0.0 | 0.0 | 0.0 | 42.86 | 35.71 | 33.33 | 57.14 | 21.13 |
| Mean | 38.88 | 55.83 | 48.87 | **65.71** | **65.07** | **65.36** | 38.01 | 48.32 | |

Note: Bold numbers indicate a moderate or large effect size (PEM > 70%)

Table 3. Means and Standard Deviations of baseline data for each participant and each dependent variable derived from the Consensus Sleep Diary

| | Number of Awakenings | | Length of Awakenings | | Sleep Onset Latency | | Quality of Sleep | |
|---|---|---|---|---|---|---|---|---|
| Participant | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| 12 | 2.03 | 0.51 | 3.33 | 2.53 | 5.85 | 3.31 | 3.55 | 0.77 |
| 14 | 1.36 | 0.93 | 11.42 | 6.33 | 20.00 | 5.55 | 3.43 | 0.65 |
| 24 | 0.86 | 0.66 | 9.64 | 11.84 | 36.01 | 38.39 | 3.83 | 1.19 |
| 31 | 3.57 | 0.85 | 25.36 | 17.04 | 28.04 | 12.49 | 2.38 | 0.65 |
| 44 | 2.36 | 0.74 | 35.71 | 18.90 | 14.64 | 9.09 | 2.07 | 0.62 |
| 58 | 1.5 | 1.34 | 21.79 | 19.96 | 13.21 | 8.23 | 2.93 | 0.83 |
| 64 | 2.36 | 1.86 | 15.0 | 11.77 | 29.64 | 25.68 | 2.86 | 1.17 |
| 70 | 1.64 | 1.69 | 21.07 | 33.87 | 77.14 | 20.91 | 1.79 | 0.58 |
| 73 | 0.79 | 1.05 | 4.64 | 7.20 | 12.43 | 9.79 | 2.64 | 0.63 |
| 74 | 3.29 | 1.82 | 77.14 | 61.01 | 69.64 | 46.18 | 2.93 | 0.83 |
| 96 | 0.21 | 0.43 | 2.14 | 4.26 | 63.21 | 53.01 | 2.71 | 0.91 |
| 60 | 0.00 | 0.00 | 0.00 | 0.00 | 14.32 | 10.67 | 2.86 | 0.77 |

**Sleep Onset Latency.** Each participant's baseline level and stability of sleep onset latency were analysed. Baseline mean and standard deviations are presented in table 3. Five participants (12, 14, 31, 58, 73) appeared to have relatively stable baseline levels of sleep onset latency. The remaining eight participants appeared to have relatively unstable baselines, with three (24, 60, 64) showing a trend towards increasing baseline levels of sleep onset latency, and two participants (44, 70) showing a trend towards reduction in baseline levels.

Six participants (96, 74, 24, 44, 14, 31) reported substantive treatment effects for sleep onset latency at both intervention and post treatment phases. Of these participants, two (14, 31) demonstrated baseline stability, while the others showed relatively unstable baselines. The majority of PEMs obtained were moderate (between 70% and 90%), with the exception of participant 24's intervention phase PEM of 60%.

Three participants (12, 73, 60) reported no substantive treatment effects for sleep onset latency at either intervention or treatment phase. Participants 12 and 73 did not supply data for the post-treatment phase, and reported no substantive treatment effect for the intervention phase. Both participants appeared to have relatively stable baseline levels. Participant 60 reported no substantive treatment effect at the intervention or post-treatment phase. This participant demonstrated a somewhat unstable baseline.

Participants 64 and 70 reported mixed findings at intervention and post-treatment phases. Participant 60 reported a small treatment effect (PEM = 67.9%), mainly evident towards the beginning of the intervention phase, while participant 70 reported a moderate treatment effect (PEM= 78.6%), mainly evident towards the end of the intervention phase (as determined by visual analysis of time series plots – see

figure 7). The treatment effect was not sustained into the post-treatment phase for either participant

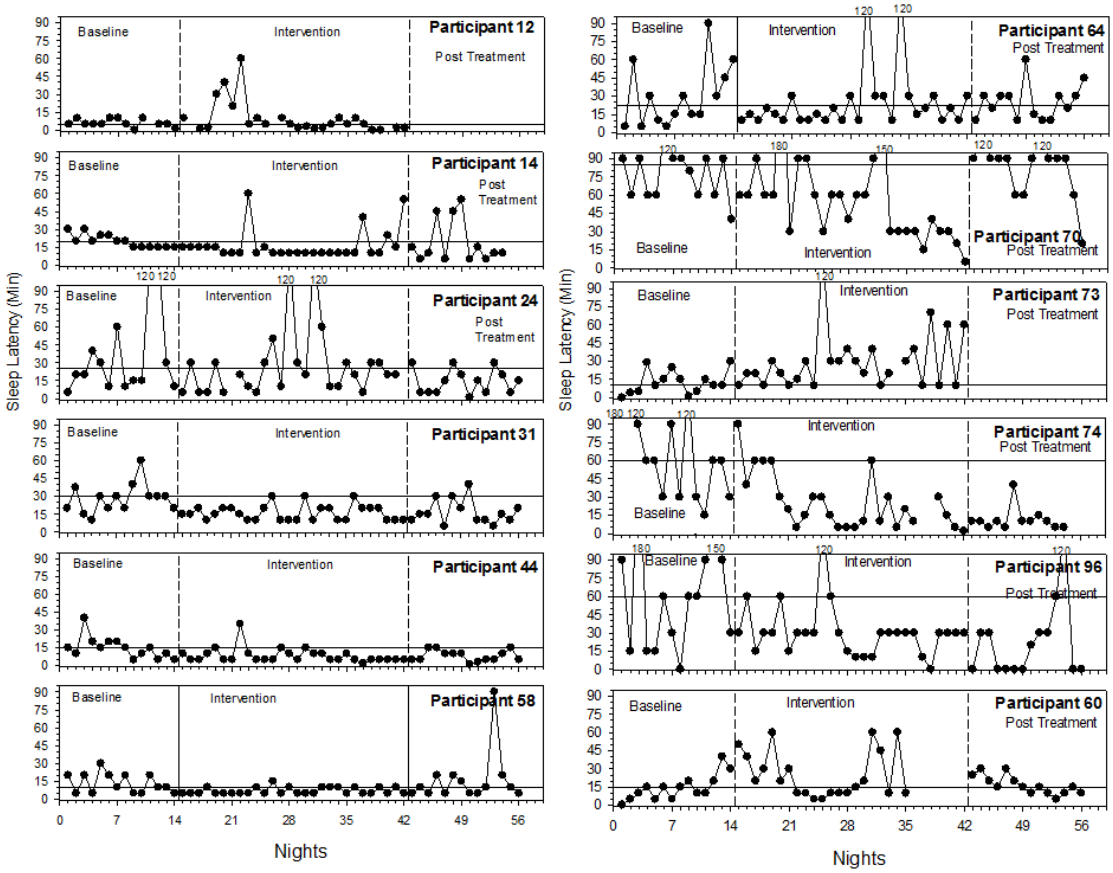


*Figure 14.* Time series graphs of self-reported sleep onset latency, recorded for 2 weeks baseline, 4 weeks intervention, 2 weeks post-treatment using the Consensus Sleep Diary.

**Quality of Sleep.** PEM results revealed that eight participants (12, 73, 14, 24, 74, 96, 58, 64) reported no substantive treatment effect on quality of sleep, at both intervention and post-treatment phases. Participants 12 and 73 did not supply post-treatment data, but reported no substantial treatment effect at the intervention phase. While the PEM from participant 58 revealed no substantial change following intervention, visual analysis of time series graphs (figure 8) suggested that towards

the end of the intervention phase some an increase in sleep quality may have occurred, which was not reflected in the overall PEM score.

Only one participant (31) reported moderate to large treatment effects at both intervention (PEM = 100%) and baseline (PEM = 84.62%) phases.

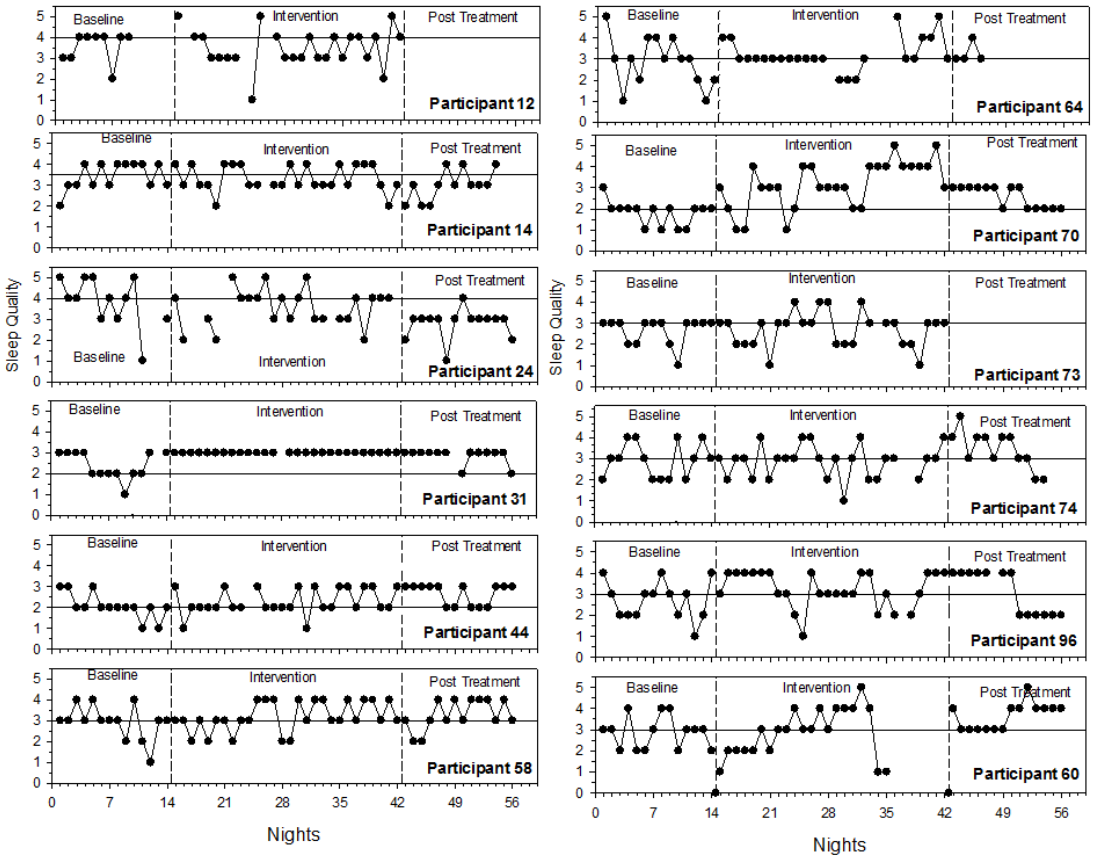


*Figure 15*. Time series graphs of self-reported sleep quality, recorded for 2 weeks baseline, 4 weeks intervention, 2 weeks post-treatment using the Consensus Sleep Diary.

Three participants (44, 60, 70) reported mixed findings of treatment effect at intervention and post-treatment. PEM scores and visual analysis of plots from participants 44 and 60 suggest that while there was no substantive treatment effect during the intervention phase, a small effect was detected at post-treatment.

Conversely, participant 70 reported a moderate treatment effect (PEM = 75.0%), but this was not sustained into post treatment.

**Patterns of change across individual data sets.** Three participants reported a consistent reduction in symptoms across sleep diary variables. Participant 31 reported a reduction in symptoms across all sleep diary variables at both intervention and post-treatment.Participant 74 reported a reduction in symptoms across all sleep diary variables except sleep quality, at both the intervention and post-treatment phases. Participant 70 reported a reduction in the number and length of awakenings across intervention and post-treatment phases, and an improvement in sleep quality during the intervention phase, which was not sustained during the post-treatment phase. Participants 12 and 73 supplied data for the intervention phase, but not the post-treatment phase. Both participants reported no substantive reduction in symptoms in all sleep variables across intervention.

Participants 60 and 96 reported a median of 0 instances of awakenings at baseline, intervention and post-treatment phases, therefore a reduction in number and length of awakenings was not able to be calculated. Both participants reported no substantive improvement in quality of sleep at intervention and post-treatment, and participant 60 further reported no substantive improvement in sleep onset latency.

Participant 58 reported no significant reduction in number of awakenings and sleep onset latency, and no improvement in sleep quality at intervention and post-treatment, however a significant reduction in length of awakenings was observed during both phases. Participants 14, 24 and 44 reported a more varied treatment effect, and did not demonstrate a clear pattern of a reduction in symptoms or lack thereof across variables. These participants did, however, report that any significant

improvements in variables seen during the intervention phase were sustained into the post-treatment phase.

**Sleep Onset and Wake Times.** Table 4 presents the baseline, treatment and post-treatment mean sleep onset and wake times for each participant and the group mean. These times are recorded in 24 hour clock time, which was calculated from the raw scores (number of minutes lapsed following midnight the previous night, corresponding to each clock time).

Table 4. Means (in 24-hour clock time) and Standard Deviations (in minutes) of individual and group baseline, treatment and post-treatment sleep onset and wake times (from the Consensus Sleep Diary).

| Participant | Baseline sleep | Baseline wake | Treatment sleep | Treatment wake | Post sleep | Post wake |
|---|---|---|---|---|---|---|
| 12 | 1:24 (73.54min) | 9:44 (75.15min) | 0:48 (67.78min) | 8:48 (166.41min) | | |
| 14 | 1:20 (57.57min) | 8:43 (82.62min) | 1:42 (61.94min) | 8:34 (83.90min) | 3:51 (229.26min) | 9:22 (89.41min) |
| 24 | 1:45 (64.85min) | 9:07 (80.34min) | 1:17 (76.83min) | 9:44 (59.03min) | 1:12 (72.14min) | 8:46 (41.35min) |
| 31 | 1:32 (41.91min) | 10:25 (83.40min) | 1:11 (35.23min) | 8:54 (43.66min) | 1:33 (36.35min) | 9:20 (64.11min) |
| 44 | 2:17 (132.62 min) | 9:05 (84.86min) | 3:04 (178.12min) | 9:29 (131.01min) | 3:01 (132.45min) | 10:06 (86.34min) |
| 58 | 6:13 (51.47min) | 0:49 (94.11min) | 5:15 (49.65min) | 11:45 (122.72min) | 5:04 (51.34min) | 13:00 (41.51min) |
| 64 | 3:57 (209.08min) | 11:08 (118.51min) | 3:41 (70.15min) | 11:07 (95.64min) | 1:40 (135.52min) | 10:25 (134.89min) |
| 70 | 4:44 (59.11min) | 12:44 (96.85min) | 1:52 (108.42min) | 10:11 (132.75min) | 2:06 (86.98min) | 9:23 (113.37min) |
| 73 | 3:02 (44.49 min) | 9:08 (182.87min) | 2:31 (55.01min) | 9:19 (124.54min) | 2:02 (40.24min) | 9:48 (183.08min) |
| 74 | 1:11 (191.32min) | 7:59 (136.24min) | 23:53 (71.33min) | 8:15 (86.28min) | 23:05 (37.97min) | 6:34 (83.83min) |
| 96 | 2:16 (91.93min) | 8:51 (84.48min) | 0:58 (88.46min) | 8:45 (51.15min) | | |
| 60 | 1:32 (65.07min) | 10:11 (67.13min) | 1:48 (79.01min) | 10:43 (93.70min) | 1:25 (17.65min) | 9:22 (18.58min) |
| **Mean** | **2:37 (95.55min)** | **10:00 (92.98min)** | **1:41 (61.79min)** | **9:38 (78.34min)** | **2:00 (85.22min)** | **9:37 (95.21min)** |

These raw scores were used to perform paired t-tests and calculate effect sizes (Cohen's dav) (Lakens, 2013) comparing participants' baseline, treatment and post-treatment mean sleep onset times, and baseline, treatment and post-treatment mean wake times. Comparing mean baseline and treatment sleep onset time, a moderate

treatment effect, d = -.68 (95% CI [-1.2, -0.128], t(11) = -2.88 , p< 0.05), was recorded. No significant treatment effects were observed between treatment and post-treatment sleep onset times, or between the baseline and post-treatment sleep onset times. Comparing mean baseline, treatment, and post-treatment wake times, no significant treatment effects were observed.

## Discussion

This study investigated an intervention for university students with delayed sleep phase syndrome (DSPS), involving a novel combination of light therapy and group psychotherapy sessions. This intervention was associated with improvements in some outcome variables, but no statistically or clinically significant change in the majority of outcome variables. Daytime sleepiness, academic self-efficacy, and symptoms of depression, appeared largely unaffected by the intervention, both at completion and at 3-month follow up. Variables that appear to have improved include physical quality of life, which showed a large increase from baseline to post-treatment (Cohen's *d* = 0.92), and perceived stress, which showed a moderate reduction from baseline to post-treatment (Cohen's *d* = 0.57). Interestingly, participants reported a moderate increase in anxiety symptoms from baseline to post-treatment and follow-up, and a moderate reduction in psychological quality of life from baseline to follow up**.**

Some participants reported improvements in variables related to sleep, as recorded in daily sleep diary entries. Sleep onset latency appears to be the variable most affected by the intervention. Six participants (50%) reported a reduction in sleep onset latency over both intervention and post-treatment phases, while three participants reported no substantive treatment effect over either phase. The effect of the intervention on the number and total length of night time awakenings was

variable. Three participants reported a moderate to large treatment effect on both the number and length of awakenings over intervention and post-treatment phases, while two participants reported no substantive effect at either phase. Quality of sleep appears to be the variable least affected by the intervention, with eight participants (67%) reporting no substantive treatment effect over both the intervention and post-treatment phases, and only one participant reporting a moderate to large effect over both phases.

These results are not entirely consistent with findings from previous research combining light therapy and psychological therapy as a treatment approach for DSPS. Gradisar et al. (2011), found moderate to large improvements in various sleep parameters (sleep onset latency; sleep onset and rise time; day time sleepiness) in the combination therapy group, compared to a light therapy only group. Danielsson et al. (2013) found only a non-significant trend that suggested that compared to light therapy alone, group CBT sessions, presented after 2 weeks of light therapy, maintained the positive effects on sleep schedule and slightly decreased symptoms associated with poor sleep. Several potential limitations, outlined below, speculate possible reasons for this discrepancy in findings.

The majority of subjects (72%) participating in the intervention were male. This ratio of males to females may indicate that males in this university population are more likely to have difficulties with delayed sleep phase. Alternately, it may suggest that more males than females are willing to seek help for DSPS, or perhaps have a more severe form of DSPS that requires professional help. These findings fit with findings from Adan and Natale (2002), which indicate that significantly more males than females score as ‘evening’ types on the Morningness-Eveningness Questionnaire (Horne & Ostberg, 1975) (an indicator of chronotype). As mentioned

above, women reach a peak in chronotype delay before men, at an average age of 19.5 years, while men will reach their peak delay at an average of 20.9 years (Roenneberg et al., 2004). The average age of participants in this study was 23.47 years (SD = 7.09). Therefore, it is possible that females this age with problems with sleep phase delay had already reached their peak delay and had started to advance their sleep schedule towards more conventional timing, whereas males of a comparable age may still be at, or close to, their peak delay in sleep schedule. Environmental and genetic factors can also influence chronotype, so while developmental stage may play a role in the relationship between sleep problems and gender, other factors may also interact or contribute indirectly to this relationship. Given this gender ratio, the findings from this study may not be generalizable to female university students, although previous studies administering similar treatment to adolescents and young adults (Cole et al., 2002; Danielsson et al., 2013; Gradisar et al., 2011) have not reported a discrepancy in the ratio of male to female participants, nor have they reported a differing treatment effect between males and females. Nonetheless, results obtained in this study may not generalise to females with the same pattern of sleep difficulties, and females may respond differently to treatment than males. Future trials, therefore, should seek sufficient participants of each sex so as to permit separate analyses for males and females.

This study employed a single-case research design (SCRD), unlike previous studies assessing the effectiveness of similar treatment programmes for DSPS (Gradisar et al., 2011; Danielsson et al., 2013), which utilised randomised control trials (RCTs). SCRDs can provide a unique perspective not available to RCTs that can complement and add to this field of research. SCRDs place an emphasis on the participants as individuals, allowing the researcher to understand phenomena and

change at an individual level. While in RCTs an emphasis is placed on statistical significance to indicate a successful intervention, SCRDs allow for an exploration of clinical significance (smaller changes detected at an individual level). Given the emphasis on the individual, SCRDs do not require as many participants and RCTs, thereby decreasing the cost of research and the time and resources needed to conduct it (Blampied, 2013). Furthermore, while RCTs require control groups (whether by waitlist or another form of treatment) SCRDs allow individual's baseline data to be used as a comparison to their treatment and post treatment data, providing a more ethically sound alternative. SCRDs can also increase generality, as they focus on effectiveness of treatment in atypical settings, as opposed to a focus on efficacy in a clinic or lab environment. Through systematic replication with varied cases, settings, or treatment components SCRDs encourage the exploration of these dimensions, and the treatment outcome in different environments can be assessed (Blampied, 2013). Given the valuable information SCRDs can provide, they may prove to be a useful adjunct to RCTs in this field of research.

**Limitations and Future Directions**

Participants in this study may not have been indicative of the entire DSPS population (other than university students) due to a restricted range in the severity of their condition. As a group, the mean pre-treatment sleep onset time was 2:37am (±95.55), and the mean pre-treatment wake time was 10:00am (±92.98 mins). While these sleep onset and wake times are consistent with the diagnosis and indicate the level of severity of DSPS, they may not represent the more severe cases of DSPS, as the literature suggests some individuals report later sleep onset times, ranging up to 6:00am (Bjorvatn & Pallesen, 2009; Garcia, Rosen, & Mahowald, 2001). More, or less, severe cases of DSPS may respond differently to the treatment programme

introduced in this study, therefore it may be beneficial to include participants with a wider range of sleep onset and wake times, or compare groups of different severity.

This study may also have been limited by the nature of, and adherence to, the light therapy component of the programme. Firstly, although the manufacturers of the light boxes used in this study maintained that they emitted a light intensity of 10,000 lux when placed 20cm away from the face, light intensity recorded by the researcher using digital light meter, resulted in a different lux reading. With the light box positioned 20cm from the user, the light meter recorded ~6300 lux. Positioned 30cm away from the user, the light meter recorded ~3000 lux, and positioned 60cm away recorded ~1000 lux. To obtain a reading of 10,000 lux the light box had to be positioned 13cm away from the user. The American Academy of Sleep Medicine's practice parameters for the clinical evaluation and treatment of circadian rhythm sleep disorders (Morgenthaler et al., 2007) recommend light at an intensity of 2,000-10,000 lux, administered in the early morning to treat such disorders. For practical reasons, it would have been difficult for participants in this study to position themselves 13cm from the light box, for a recommended hour at a time, in order to gain optimal light exposure. It is likely that participants positioned themselves further away, limiting the light exposure received and making it difficult to ascertain whether all participants received light exposure at a sufficient intensity for circadian entrainment, and at intensity similar to other participants.

Secondly, although participants were given a personalised timetable outlining when light exposure should occur, the timing, length and consistency of the light exposure was not closely monitored. Participants were encouraged to contact the researcher if they encountered any difficulties following their light exposure regime, and they were also encouraged to share and discuss these problems at the group

sessions, however, the programme relied on participants being honest about their adherence to light exposure, and did not include an objective measurement of light exposure. Therefore, there was no way to know whether participants were correctly and consistently following the light exposure regime. If participants deviated from the planned light exposure timing, it is likely to have reduced the effectiveness of this part of the intervention. Participants were encouraged to use the light box, natural outdoor light, or both, for light exposure. Many participants, however, reported using only the light box and foregoing outdoor light exposure as this was more convenient. Had participants used outdoor light exposure they are likely to have received light exposure at a higher intensity (lux) than from the light box, especially in groups who participated in the programme over the summer.

Adherence to planned sleep schedules, and recording sleep parameters in sleep diaries accurately, also relied on participants' honesty. Although participants were required to complete the sleep diaries each day upon waking, this was not monitored on a daily basis. If participants forgot to record their sleep parameters on waking, they may have completed sleep diaries retrospectively, reducing the accuracy of their recall. As the researcher required a physical copy of each week's entry, participants may also have felt pressure to complete sleep diaries, even if they were not able to or forgot to. This may have resulted in falsified or inaccurate entries completed in order to avoid disappointing the researcher or group, and the perceived consequences following this. The use of smart phone apps, or devices such as the fit bit, that track movement during periods of sleep and wakefulness (similar to an actigraph) to estimate sleep cycles may prove to be a useful, and cost effective addition to this area of research, however the body of research assessing the effectiveness of such devices is not particularly substantive.

Further research could benefit by addressing motivational and compliance issues with participants as they complete the programme. While this study addressed concerns at each group session, and encouraged participants to contact the researcher after hours with their concerns, participants may benefit from more frequent, individual check-ins, to address problems when they first occur. This could include daily reminders by email or text message sent automatically to participants; having 'drop in' office hours where participants could meet with the researcher and discuss difficulties; observation sessions to ensure participants were correctly following the programme; or daily messages sent by participants outlining their sleep parameters and the time and length of light therapy each morning. Participants might also benefit from a Motivational Enhancement component to therapy to target ambivalence around engaging in the programme.

The study may also have benefitted from objective measures of participant's circadian timing, including measures of dim light melatonin onset, core body temperature, or actigraphy. This study was limited in this regard due to the budget and resources available, however, inclusion of physiological measures may have helped validate sleep diary data and, if this information was collected at regular intervals, may have provided another means to monitor and encourage participants more frequently.

The current study could be further improved by adhering to a traditional multiple-baseline across-groups design, in order to control for the effects of extraneous variables and demonstrate whether changes observed an effect of the intervention. Due to recruitment difficulties and time constraints, the three groups received treatment at times spaced well apart in the university year, and each recorded baseline measures for the same duration, so the intended design was not possible.

With better planning of recruitment and facilitation of group sessions, a multiple-baseline across groups design could be employed with greater ease.

This study involved light therapy administered alongside psychoeducation and psychotherapy, in a group format. As light therapy was administered concurrently alongside group treatment sessions, it is difficult to disentangle the effects of these two treatment components. While light therapy has been demonstrated to advance sleep phase, few studies have paired it with other forms of therapy and assessed the efficacy of this treatment combination. Both Gradisar et al. (2011) and Danielsson et al. (2013) presented randomised control trials to assess the efficacy of light therapy and cognitive behavioural therapy compared to light therapy alone. Two factors distinguish the research of Gradisar et al. (2011) from that of Danielsson et al. (2013) and the current study; namely, the age of the participants and the presentation of psychological therapy (individual vs. group). It is possible that findings from the current study were not consistent with results from Gradisar et al. (2011) due to the age or life stage of participants, the use of group sessions instead of one-on-one sessions, or both these factors.

Firstly, the age and life stage of university students, compared to high school students, may make them a different population. Notably, the structure of a university student's day is likely to be different from a high school student's day. Typically, high school students' timetables and learning environments are relatively structured; they are required to attend every class and get up at a similar time each weekday morning. University students may have a different class schedule each day and lecture attendance is often not mandatory (although advised). They may experience a greater sense of freedom to socialise, engage in part time work, and participate in recreational activities—on their own terms and at their preferred time. This variation in daily

schedule and/or lack of routine may have made it particularly difficult for the university students in the current study to adhere to the structured treatment programme.

The content or method of presentation of the psychological therapy component of the treatment programme may have had an impact on its effectiveness. Topics introduced in the current study's treatment sessions included education about DSPS, sleep hygiene, stimulus control, mindfulness techniques, and relapse prevention. These topics are similar to those introduced by Danielsson et al. (2013), however, Gradisar et al. (2011) focused more on cognitive techniques, including identifying and challenging automatic thoughts, and generating alternative thoughts. Perhaps, given the relatively high prevalence of depression and anxiety among those with DSPS (Abe et al., 2011; Reid et al., 2012; Robillard et al., 2013; Sheaves et al., 2015), alongside reports of racing thoughts, worries, and anxiety related to sleep from adolescents with DSPS (Gradisar et al., 2011), cognitive techniques may prove to be a necessary component in psychological therapy to target DSPS, that should be considered for future studies. Therapist fidelity (the adherence to treatment protocol by the therapist) may also have affected treatment outcome. Therapist fidelity was not measured in this study, therefore the extent to which the content was presented similarly across sessions and groups could not be determined. The addition of a rating scale assessing therapist fidelity to the therapeutic goals and approach in future trials may help determine the extent to which these are consistently adhered to. The amount, length, frequency, and format of therapy sessions may also be important. In this study, these factors were considered and decided upon to take into account the participants' and therapist's available time and the cost of running the sessions. Further research may be beneficial to determine whether more frequent sessions,

different content, or presentation (i.e. one-on-one vs group presentation) could improve participants' outcome.

In the future, researchers and practitioners would benefit from independent replication to establish the reliability and generality of treatment effects of combined light therapy and psychotherapy for DSPS in a university student population. The current study has, however, provided insight into the plausibility of conducting such a treatment programme for university students displaying DSPS symptoms, and the challenges faced in doing so. While the programme may benefit from minor adjustments to improve motivation and adherence to the treatment components, a brighter light box, as well as content, presentation and structure, this research provides a starting point from which to base further studies to investigate the effectiveness of combined light therapy and psychotherapy in the treatment of DSPS in this population.

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# Appendix A

## Consent Form for Participants

Department of Psychology

Email: rebecca.manning@pg.canterbury.ac.nz

**Timed light exposure and group treatment sessions as treatment for delayed sleep phase syndrome in university students.**

**Consent Form for Participants**

I have been given a full explanation of this project and have had the opportunity to ask questions. I understand what is required of me if I agree to take part in the research.

I understand that participation is voluntary and I may withdraw at any time without penalty. Withdrawal of participation will also include the withdrawal of any information I have provided should this remain practically achievable.

I understand that any information or opinions I provide will be kept confidential to the researcher and that any published or reported results will not identify the participants. I understand that names and personal information shared and discussed by group members during group treatment sessions will remain confidential between participants and the researcher and clinician facilitating the sessions. I understand that a thesis is a public document and will be available through the UC Library.

I understand that all data collected for the study will be kept in locked and secure facilities and/or in password protected electronic form and will be destroyed after five years.

I understand the risks associated with taking part and how they will be managed.

I understand that I am able to receive a summary of the results of the study by contacting the researcher at the conclusion of the project.

I understand that I can contact the researcher (Rebecca Manning: rebecca.manning@pg.canterbury.ac.nz) or supervisors (Neville Blampied: neville.blampied@canterbury.ac.nz, Alex Mortlock: alex.mortlock@canterbury.ac.nz) for further information. If I have any complaints, I can contact the Chair of the University of Canterbury Human Ethics Committee, Private Bag 4800, Christchurch (human-ethics@canterbury.ac.nz)

By signing below, I agree to participate in this research project.

Name: _____ Signature: _____

Date: _____

## Appendix B

## Information Sheet for Participants

Department of Psychology

Email: rebecca.manning@pg.canterbury.ac.nz

**Timed light exposure and group treatment sessions as treatment for delayed sleep phase syndrome in university students.**

**Information Sheet for Participants**

*I am studying towards a Master of Arts in Psychology. The purpose of my research is to determine whether timed bright light exposure combined with group therapy is an effective treatment for Delayed Sleep Phase Syndrome. Delayed Sleep Phase is a circadian rhythm sleep disorder characterised by an inability to fall asleep at a desired time, resulting in a phase delay of one's major sleep episode, difficulty waking at a conventional time, and increased daytime sleepiness.*

Your involvement in this project will involve several elements. This includes 1 hour of light exposure on waking each morning and weekly 1 hour group treatment sessions, totalling approximately 32 hours of time over a four week period. Prior to and during the treatment programme you will be asked to fill in a sleep diary each morning. This will provide us with information about your sleep patterns. Before the treatment programme begins, and after it ends you will also be asked to fill out several questionnaires assessing your daytime sleepiness, perceived quality of life, emotional state, and your confidence about coping with academic work.

During the treatment phase you will be asked to use a light box or natural daylight (outdoors) in order to receive bright light exposure. You will be asked to do this for one hour, in the morning when you first wake up. Also, we will ask you to wake 20 minutes earlier every second day, with the use of an alarm, in order to gradually bring your wake up time, and your bright light exposure, earlier in the morining. You will be asked to continue to wake 20 minutes earlier every second day until you reach your desired morning wake time. Each morning when you wake you will also be asked to fill out a sleep diary, which will take no longer than 5-10 minutes.

The second part of the four week treatment programme will be weekly hour long group sessions. You will be asked to attend all four group sessions. In these group sessions you will meet with a clinical psychologist and five other participants. Topics discussed in the group sessions will include: delayed sleep phase syndrome and treatment; sleep management; mindfullness techniques; and long term management of sleep problems. Personal information shared and discussed in group sessions will remain confidential between participants and the researcher and clinician facilitating the sessions. You will be asked to sign a confidentiality agreement at the first group session to ensure this.

As a follow-up to this investigation, three months after the treatment programme ends you will be contacted and asked to complete the sleep diary and questionnaires you completed prior to treatment.

In the performance of the tasks and application of the procedures there are risks of side effects. Possible side effects from the use of light boxes include headaches, eye strain, mild dizziness and nausea. However, several studies have assessed the safety of light box use and concluded that there is a substantial benefit-to-risk ratio, and that if patients do experience side effects from light therapy, they are likely to be mild and short-lasting. I have included a list of references below, if you wish to research this further.

You may receive a copy of the project results by contacting the researcher at the conclusion of the project.

Participation is voluntary and you have the right to withdraw at any stage without penalty. If you withdraw from the study before the results are analysed, I will remove information relating to you from the study database.

The results of the project may be published in scientific and professional journals and may be presented at conferences and to social and news media, but you may be assured of complete anonymity regarding your participation. None of the data gathered will be able to be personally identified and your identity will not be disclosed without your prior consent. To ensure confidentiality, data will be stored in a locked filing cabinet and on a password protected computer, within a locked room. My supervisors and I will be the only people with access to the data. Please bear in mind that the thesis reporting the study is a public document and will be available through the UC Library.

The project is being carried out as a requirement of a Master of Arts by Rebecca Manning under the supervision of Neville Blampied, Alex Mortlock, and Jacki Henderson. They will be pleased to discuss any concerns you may have about participation in the project.

This project has been reviewed and approved by the University of Canterbury Human Ethics Committee, and participants should address any complaints to The Chair, Human Ethics Committee, University of Canterbury, Private Bag 4800, Christchurch (human-ethics@canterbury.ac.nz).

If you agree to participate in the study, please refer to the email I have sent you. Once you have read this form and completed and returned our pre-screening questionnaire and consent form (attached in this email), I will be in touch with you. Based on your answers to our pre-screening questionnaire, you may be asked to attend an assessment interview, where we will determine if you meet the diagnostic criteria for Delayed Sleep Phase Syndrome, and for our research.

Kind Regards,

Rebecca Manning.

## Appendix C

## Pre-Screening Questionnaire

Pre-Screening Questionnaire

Please place and 'X' in the yes or no box to indicate your answer. When you have filled this out, please send your completed form back to me as an email attachment.

1. Are you willing to commit 10 hours of your time per week, over a 4 week period, to this study?

YES ☐ NO ☐

2. Are you motivated to change enough to commit to this study?

YES ☐ NO ☐

3. Do you **usually** fall asleep at a time that is later than what you consider to be 'normal'?

YES ☐ NO ☐

4. Do you **usually** wake up at a time that is later than what you consider to be 'normal' (when waking without the use of an alarm)?

YES ☐ NO ☐

5. Have you had experienced these problems for 3 months or longer?

YES ☐ NO ☐

## Appendix D

## University of Canterbury Human Ethics Committee approval letter

UNIVERSITY OF CANTERBURY
Te Whare Wānanga o Waitaha
CHRISTCHURCH NEW ZEALAND

HUMAN ETHICS COMMITTEE

Secretary, Lynda Griffioen
Email: human-ethics@canterbury.ac.nz

Ref: HEC 2015/55

2 July 2015

Rebecca Manning
Department of Psychology
UNIVERSITY OF CANTERBURY

Dear Rebecca

The Human Ethics Committee advises that your research proposal "Timed light exposure and group cognitive behavioural therapy sessions as treatment for delayed sleep phase syndrome in university students" has been considered and approved.

Please note that this approval is subject to the incorporation of the amendments you have provided in your email of 23 June 2015.

Best wishes for your project.

Yours sincerely

Lindsey MacDonald
***Chair***
***University of Canterbury Human Ethics Committee***

## Appendix E

## Revised Clinical Sleep History Interview (adapted from Gradisar et al. (2011))

# *Assessment Interview*

**In attendance: Interviewer** **Participant Code:**

**Consent form signed and collected** ☐

**Introduction**
Explain to participant:

-What the interview involves (subjects covered)
-Personal nature of questions (can choose not to answer)
-Confidentiality and anonymity (participant code, locked filing cabinet, discussed with supervisors only)
-Free to ask questions or for clarification if required
-Can take a break at any point

***"Does the Sleep Diary show a typical week?"*** YES ☐ NO ☐

***"What is your main Sleep Problem?"***

_____

_____

_____

_____

**DAYTIME FUNCTIONING**

**1. Do any of the following occur for you due to the poor sleep?** *(tick if YES)*

Tiredness / fatigue ..... ☐
Attention, concentration, or memory problems ..... ☐
Problems socialising ..... ☐
Poor academic performance ..... ☐
Moodiness or irritability ..... ☐
Sleepy during the day ..... ☐
Lack of energy / motivation ..... ☐
Tension, headaches, or stomach problems ..... ☐
Worries about sleep ..... ☐
Other health problems ..... ☐

**2. Are these difficulties present at least 3 days per week?**

YES ☐ NO ☐

*(Alert participant to change of topic)

**SLEEP HISTORY**

**3. How long has the current sleep problem been an issue?**
Years_____or Months_____or Weeks _____

**4. Do you remember anything that may have triggered this problem?**
-If yes, clarify trigger event
-If no, "Has the sleep problem come about gradually?"

_____
_____

**5. Has the sleep problem changed/worsened since you first noticed it?**

_____
_____
_____

**6. Have there been other sleep issues in the past? (include info about type of symptoms, age of onset, duration, treatment,etc.)**

_____
_____
_____
_____
_____

**7. What was your sleep like in high school?**

_____
_____

**8. Has anyone in your family experienced sleep problems? (i.e. mum, dad, siblings)**
_____

Please elaborate:

_____
_____

**9. So you've said you have trouble falling/staying asleep. Does this worry you?**
YES ☐ NO ☐

If yes, what is it that worries you about the problems youhave sleeping?

*(Alert participant to change of topic)

**BEDTIME ROUTINE**

**10. What do you usually do in the last hour before going to bed?**

_____

_____

_____

**11. Do you put off going to bed at night?**

YES ☐ NO ☐

**Reasons given for putting off going to bed** (*eg, not feeling tired or sleepy*)

_____

_____

**12. Do you:**

| | No | Yes |
|---|---|---|
| Read in bed…… | ☐ | ☐ |
| Watch TV in bed…… | ☐ | ☐ |
| Study in bed…… | ☐ | ☐ |
| Use your cell phone in bed…. | ☐ | ☐ |
| Use your computer/laptop or play video games in bed…… | ☐ | ☐ |
| **Is your:** | | |
| Bed comfortable…… | ☐ | ☐ |
| Bedroom dark at night…… | ☐ | ☐ |
| Bedroom quiet at night …… | ☐ | ☐ |

**13. How much of the following do you have (on average) each day?**

Fizzy Drink glasses per day Last drink a.m. p.m.
Coffee/Tea cups per day Last drink a.m. p.m.
Chocolate pieces per day Last piece a.m. p.m.
Energy drinks cans per day Last drink a.m. p.m.

*Alcohol standard per day on **weekdays/workdays**
Last drink a.m. p.m.
*Alcohol standard per day on **weekends/days off**
Last drink a.m. p.m.

*Nicotine cigarettes per day Last one a.m. p.m.

**14. Do you look at a clock/phone to check the time when you're in bed?**

YES ☐ NO ☐

**When? (can tick more than one)**

trying to fall asleep at the start of the night ☐
when waking up during the night ☐
when waking up in the morning (ie, before getting out of bed) ☐

**DELAYED CIRCADIAN PHASE**

**15. Do you nap during the day?**
YES ☐ NO ☐

If YES, how often?: ☐ 1 time per week
2-3 times per week ☐
4-5 times per week ☐
6-7 times per week ☐
If YES, i) time of nap_____
ii) length of nap _____ minutes / hours

**16. Do you lack energy or motivation towards the end of the day?**
YES ☐ NO ☐

**17. Do you become more alert after dinner?**
YES ☐ NO ☐

**18. Do you have difficulty getting out of bed for a morning commitment (e.g. lecture or work)?**
YES ☐ NO ☐

**19. How many days per week do you wake up at a normal time, without the use of an alarm?**
☐ 1 time per week
☐ 2-3 times per week
☐ 4-5 times per week
☐ 6-7 times per week

**20. Do you wake up without the use of an alarm on…**
**Weekends** YES ☐ NO ☐
*describe:*_____
_____

**21. Think about the last time you were able to choose your own sleep timing (e.g., weekends or holidays, without any morning commitments)…**

- What time did you naturally wake-up? _____
- What time did you go to sleep? _____
- Do you think you slept better than normal? ☐ YES NO ☐
- Do you think you slept longer than normal? ☐ YES NO ☐

**22. Ideally, what time would you like to be waking up naturally in order to meet morning commitments (e.g. lectures, work)?** _____

**DIFFERENTIAL DIAGNOSIS**

* "Now I'm going to ask about different aspects of your life".

**23. Do you frequently travel interstate or overseas?**

YES ☐ NO ☐
**If YES, describe below:**
Destination: _____ No. of time zones travelled: _____
How recent? _____ months _____ weeks _____ days

Do you still have this sleep problem when not travelling?
YES ☐ NO ☐

**24. Do you work evening or night shifts?**
YES ☐ NO ☐
**If YES, ask:**
-What time does your shift begin and end? _____
-How often do you work this shift? _____
-Do you still have this sleep problem on your days off or on holiday?
YES ☐ NO ☐

**25. Have you been diagnosed with a MEDICAL condition?**
YES ☐ NO ☐
If YES, list:
_____ diagnosed by _____
_____ diagnosed by _____
_____ diagnosed by _____

**26. Did the sleep problem occur at the same time as** <the medical disorder>**?**
NO ☐ YES ☐
_____

**Which would you consider worse, the sleep problem or** <the medical disorder>**?**
Sleep Problem ☐ Medical Disorder ☐

**27. Have you been diagnosed with a mental health condition? (eg, depression, anxiety, etc.)?** * Remind participant about confidentiality
YES ☐ NO ☐
If YES, list:
_____ diagnosed by? _____
_____ diagnosed by? _____
_____ diagnosed by? _____

**28. Did the sleep problem occur around the same time as** <the mental health condition>**?**
☐ YES ☐ NO *further detail* _____

**29. Does the sleep problem 'come and go' with changes in the severity of the** <mental health condition>**?**
☐ YES ☐ NO

**30. Are you on any MEDICATION (please list)?**
**_____ dose_____ time taken_____**
**_____ dose_____ time taken_____**
**_____ dose_____ time taken_____**

**31. Do you experience restless legs (i.e., feel a need to urge to move your legs or experience uncomfortable sensations in your legs?)**
YES ☐ NO ☐

**If YES, how often?:**
1 night per week ☐
2-3 nights per week ☐
4-5 nights per week ☐
6-7 nights per week ☐

**32. Do you experience regular twitching legs (or arms) during sleep (e.g. is your bed in a mess in morning?)**
YES ☐ NO ☐

If YES, how often?:
1 night per week ☐
2-3 nights per week ☐
4-5 nights per week ☐
6-7 nights per week ☐

**33. Do you snore in your sleep?**
☐YES ☐ DK ☐NO

If YES, how often?:
1 night per week ☐
2-3 nights per week ☐
4-5 nights per week ☐
6-7 nights per week ☐

Generally, how loud is the snoring?
light ☐
moderately loud ☐
very loud ☐

**34. Has anyone ever told you that you stop breathing for short periods (eg, 10-20 secs) during your sleep?**
☐YES ☐ DK ☐NO

If YES, how often?: ☐

1 night per week
2-3 nights per week ☐
4-5 nights per week ☐
6-7 nights per week ☐

**35. Do you ever wake up gasping, choking, or short of breath?**
YES ☐ NO ☐

**36. Do you experience any of the following:**
☐ Headache when you wake in the morning
☐ Dry mouth when you wake in the morning
☐ Frequent trips to the bathroom during the night

**37. Have you noticed any other unusual experiences around your sleep (eg, sleep walking, sleep talking, nightmares)?**
☐YES ☐NO *describe:*_____
_____
_____

**38. Is there anything of moral or cultural importance to you that you would like us to consider, should you participate in this study? How can we best accommodate your needs?**
_____
_____
_____
_____
_____
_____

**39. Are there any other important issues we haven't covered that you would like to discuss?**
_____
_____
_____
_____
_____

**PARTICIPANT'S GOALS**

1.

2.

3.

# Appendix F

## Participant hand outs for Group Sessions 1-4

### Session 1

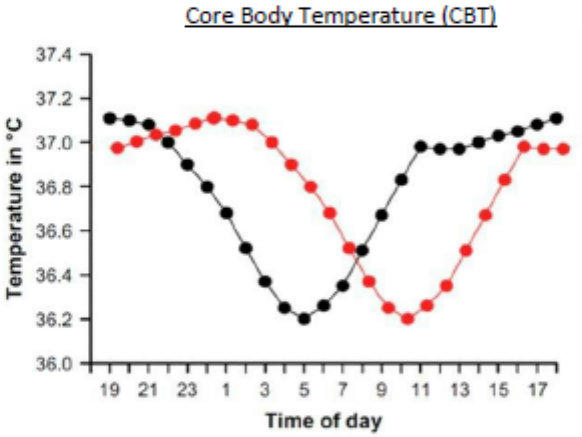
## Delayed Sleep Phase Syndrome

*Delayed Sleep Phase Syndrome*

- Delay in sleep timing
  - Difficulty *getting to sleep* at desired time
  - Difficulty *waking* at desired time
- Reasonably consistent time of falling sleep
- Easier to get up when after sleeping in
- Broken sleep is not the main problem
- Going to bed earlier and forcing self to get up early doesn't fix the problem

*Circadian Rhythms*

- Body processes that vary over 24-hour cycle
- Sleep, energy levels, appetite, hormone level, etc
- Potential for delay - 24.5 hour cycle following insufficient light exposure




*Circadian Physiology*

- Eyes - Special receptors detect blue light and report its intensity to deep brain structures
- Brain – The suprachiasmatic nucleus controls how much melatonin is produced (high levels during night; low levels in the day).
- Melatonin – Hormone that induces sleepiness and cools body temperature.
- Light - Bright light turns off melatonin production and resets the body clock each morning.

*Light therapy*

- Carefully timed light exposure can delay or advance the body clock
- The direction of change depends upon the time of exposure
  - Delay – *before* CBT minimum
  - Advance – *after* CBT minimum
- For DSPS, light exposure timed to occur *after* the CBT minimum.
- Light exposure time is gradually shifted earlier every couple of days.
- The person with DSPS starts with their *natural* sleep schedule and gradually advances this until

their sleep fits their *desired* schedule
- 60 min of bright light exposure is required.
- Sufficient light intensity requires going outside or use of provided light box
- Dim light before bed also helps
    - Low room lighting
    - Reduce computer/cellphone use
    - Turn down screen brightness
    - Install blue-light reducing software ('f.lux' on computer and 'Twilight' on smartphone)
    - Consider sunglasses or cap indoors if you are bold enough!

**Session 1: Treatment Plan**

Day 1

Evening
- Dim light in late evening (2 hrs before estimated sleep time)
- Ensure bedroom is dark (wear sleep mask if light will enter room in the morning)
- Stay up late and only go to bed when *sleepy* (often in a.m. hours)

Day 2

Morning
- Get up when you wake naturally (often late morning, perhaps early afternoon)
- Within an hour of waking, commence light therapy: 60 mins exposure (either from going outside or from sitting close to light box

Evening
- Set tomorrow morning's alarm for 20 mins earlier than expected natural wake time.
- Dim light before bed (2 hrs)
- Dark sleep environment
- Go to bed when sleepy (approx 8 hrs before morning alarm)

Day 3

Morning
- Get up when alarm goes off (even if it requires some effort)
- Within an hour of waking, commence light therapy: 60 mins exposure

Evening
- Set tomorrow morning's alarm to wake at the same time as this morning.
- Dim light before bed
- Dark sleep environment
- Go to bed when sleepy

Day 4

Morning
- Get up when alarm goes off
- Within an hour of waking, commence light therapy

Evening
- Set morning alarm for 20 mins earlier than this morning's alarm.
- Dim light before bed
- Dark sleep environment
- Go to bed when sleepy

**Session 2**

# Stimulus Control

*Rationale*

- Falling asleep is a behaviour, which is reinforced by the experience of sleep
- It is most likely to occur in the right environment (internal & external)
- Goal of Stimulus Control is to fall asleep quickly and return to sleep quickly if awoken at night

*External factors*

- An optimal environment will have clear *sleep-specific* cues, and few *activity-related* cues
- Distractions in the environment may trigger non-sleep behaviour
- Insufficient sleep-specific cues may fail to trigger falling asleep

*Internal factors*

- A 'wind down' routine can establish sleep-compatible internal states - calmness, relaxation, awareness of feeling sleepy, etc
- An internal state of arousal (anxiety, busy mind, anticipation of insomnia, etc), in contrast, will reduce likelihood of sleep.

*Stimulus Control Instructions*

1) Lie down and go to sleep only when you are sleepy.

2) Do not use your bed for anything except sleep; that is, do not read, watch television, eat, or worry in bed. Sexual activity is the only exception to this rule. On such occasions, the instructions are to be followed afterward when you intend to go to sleep.

3) If you find yourself unable to fall asleep, get up and go into another room. Stay up *for about 20 minutes* and then return to the bedroom to sleep. Although we do not want you to watch the clock, we want you to get out of bed if you do not fall asleep *quickly*.

Remember the goal is to associate your bed with falling asleep *quickly*. If you are in bed more than *about 20 minutes* without falling asleep and have not gotten up, you are not following this instruction.

4) If you still cannot fall asleep, repeat step (3). Do this as often as in necessary throughout the night.

5) Set your alarm and get up at the same time every morning irrespective of how much sleep you got during the night. This will help your body acquire a consistent sleep rhythm.

6) Do not nap during the day.

**Session 3**

# Mindfulness for Sleep

*Applying Mindfulness Principles to Sleep*

Mindfulness can help you learn the optimal state of mind for initiation of sleep (at the beginning or middle of the night). In doing so, consider changing your *relationship* to sleep rather than the amount of sleep you get each night. This may help you might attain an improvement in the quality of your seep. Later, you may see an increase in the amount of sleep you get. This approach requires discipline and consistency, but follows the principles of mindfulness.

**Non-striving:** Sleep is a process that cannot be forced, but instead should be allowed to unfold naturally. Putting *too much effort* into sleeping longer or better is counterproductive.

**Letting go:** Attachment to sleep or your ideal sleep needs usually leads to worry about the consequences of sleeplessness. That is counterproductive and inconsistent with the natural process of letting go of the day to allow sleep to come.

**Non-judging:** It is easy to automatically judge the state of being awake as negative and aversive, especially if you do not sleep well enough for several nights. However, this negative energy can interfere with the process of sleep. One's relationship to sleep can be a fruitful subject of meditation.

**Acceptance:** Recognising and accepting your current state is an important first step in choosing how to respond. If you can accept that you are not in a state of sleepiness and sleep is not likely to come soon, why not get out of bed? Many people who have trouble sleeping avoid getting out of bed. Unfortunately, spending long periods of time awake in bed might condition you to being awake in bed.

**Beginners Mind:** Remember that each night is a new night. Be open and try something different! What you have been doing to this point is probably not working well.

**Trust:** Trust your sleep system and let it work for you! Trust that your mind and body can self-regulate and self-correct for sleep loss. Knowing that short consolidated sleep often feels more satisfying than longer fragmented sleep can help you develop trust in your sleep system. Also, sleep debt can promote good sleep as long as it is not associated with increased effort to sleep.

**Patience:** Be patient! It is unlikely that both the quality and quantity of your sleep will be optimal right away.

**Session 4**

## Sleep Hygiene & Relapse Prevention

*Sleep Hygiene* - habits and practices that are conducive to sleeping well on a regular basis.

- **Bedroom environment** - cool (16-18°c), dark (or red light), quiet (or ambient sound)
- **Stimulants** - caffeine, nicotine, alcohol, illicit drugs
- **Exercise** - best in morning or afternoon
- **Food & Drink** - Light snack before bed okay. Avoid too much food late or going to bed hungry
- **Comfort** - bedding, pillow(s), etc
- **Clock watching** - best to avoid

*Relapse Prevention*

| |
|---|
| **High Risk Situations** |
| **Early Warning Signs** |
| **Good Responses** |
| **Bad Responses** |

## Appendix G

## Participant Confidentiality Statement, completed prior to group treatment

Department of Psychology

Email: rebecca.manning@pg.canterbury.ac.nz

**Timed light exposure and group cognitive behavioural therapy sessions as treatment for delayed sleep phase syndrome in university students.**

**Confidentiality Statement for Participants**

I, _____ understand that group member's names and comments, disclosure, or discussion that occurs within the group are confidential. By signing this agreement form, I agree to maintain the confidentiality of all members of the group.

I have read the points stated above, have discussed them when I was not clear about them, and have had my questions answered fully. I understand and agree to them, as shown by my signature below.

Signature_____ Date_____

**sessions**