Delayed Sleep Phase Disorder

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Research question: Based on current research, would Delayed Sleep Phase Disorder be considered permanent? What is the prognosis? What are the most effective treatments and outcomes of treatment?

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1. Contents

2. Summary

Delayed Sleep Phase Disorder (DSPD) is the most common circadian rhythm sleep-wake disorder. DSPD is the inability to initiate sleep at a conventional bedtime and subsequent difficulty to wake for morning commitments. The prevalence of DSPD is unclear, however is estimated to be between 0.5-16% of the population with a higher prevalence in adolescents. True DSPD is an intrinsic sleep disorder affecting the circadian rhythm, however there are psychological, social and behavioural influences on the natural sleep-wake cycle. These factors include evening exposure to artificial light and use of electronic devices late at night. Main symptoms of DSPD include: daytime sleepiness, fatigue, low mood, irritability, poor concentration, poor memory and decreased attention.

DSPD can be episodic, persistent or recurrent. Most often it follows a **persistent** course as **relapse after treatment is common**. Left untreated, DSPD can increase the risk of poor life outcomes such as job loss, truancy, school failure, less participation in sport and social difficulties. Treatment aims to bring forward the sleep phase to enable early bedtimes and therefore wake more easily in the morning without experiencing tiredness. Treatments include **exogenous melatonin**, **light therapy** and **sleep hygiene/behavioural changes**. However, correction of the sleep-wake cycle is rarely permanent. After relapse, individuals should be encouraged to go back to their original treatment plan.

DSPD is associated with depression, anxiety, OCD, ADHD and ASD. It is important to note these **comorbidities may impact implementation and/or adherence of the treatment protocol**.

3. Delayed Sleep Phase Disorder

3.1 Background

Delayed Sleep Phase Disorder is the most common of all circadian rhythm sleep-wake disorders (Nesbitt, 2018; Prihodova et al, 2022). DSPD is defined as difficulty initiating sleep and an inability to wake for morning commitments as a result of the delayed timing of the major sleep period (Micic et al, 2016; Richardson et al, 2019). Despite the delay in the onset of sleep and wake up time, the physiological structure of sleep is preserved (Gomes et al, 2021). That is, a major sleep episode occurs every 24 hours with usual REM and non-REM sleep, but the sleep timing is inappropriate to the day-night cycle (Nesbitt, 2018).

The prevalence of DSPD is difficult to determine due to the blurred boundary between individuals with an extreme evening chronotype ('night owls') and those with DSPD (Nesbitt, 2018). Broad estimates place the prevalence between 0.5-16% of the population (Micic et al, 2016; Nesbitt, 2018) with the highest prevalence among adolescents and young adults (Nesbitt, 2018; Richardson et al, 2017).

DSPD is associated with depression, anxiety, obsessive compulsive disorder, ADHD and ASD (Nesbitt, 2018; Prihodova et al, 2022; Snitselaar et al, 2019). Research suggests 52% of patients with DSPD have a comorbid mood disorder, and 45% have a diagnosis of attention deficit hyperactivity disorder (Prihodova et al, 2022). It is suggested that these comorbid diagnoses may impact the course of DSPD as they are associated with less motivation to start and continue treatment (Danielsson et al, 2018).

3.2 Etiology

The pathophysiology of DSPD is multifactorial. True DSPD is a primary intrinsic sleep disorder where the circadian rhythm and sleep timing are delayed relative to solar cycle (Nesbitt, 2018). However, a high proportion of people diagnosed with DSPD have delayed sleep episodes with normally aligned circadian timing (Nesbitt, 2018). So while alterations in the natural circadian rhythm may play a role, there are also psychological, behavioural and social contributors to the etiology (Prihodova et al, 2022). Highlighting this point, research in a controlled laboratory environment found the majority of adolescents, all with a diagnosis of DSPD, fell asleep at a conventional time when they had no access to technology or other distractions (Prihodova et al, 2022).

In healthy individuals, melatonin levels cycle through the day in response to light intensity – melatonin levels are negligible in the morning with bright light and rise in the early evening when light intensity reduces (Micic et al, 2016). The intensity of light is sensed by photosensitive retinal cells in the eye and conveyed to the natural timekeeper, the suprachiasmatic nucleus (Micic et al, 2016; Nesbitt, 2018). It is most likely that people with true DSPD get too much light exposure in the phase delaying portion of the response to light and too little in the phase advancing portion because they are asleep (Nesbitt, 2018). Alternatively, some people may have longer biological feedback loops within this neural circuit, meaning it takes longer for the evening sleep phase to be triggered (Micic et al, 2016; Nesbitt, 2018).

Other influences that can impact the sleep-wake cycle are:

- Onset of puberty, which is typically characterised by a biological delay in the habitual sleep pattern, tending to reversal to earlier sleep pattern in 20s, however the delay can be maintained by some individuals into adulthood (Micic et al, 2016)
- Abnormal melatonin secretion (Prihodova et al, 2022)
- Brain injury or head trauma (Snitselaar et al, 2019)

- Psychological disorders – mood disorders, specific personality traits (such as neuroticism) (Prihodova et al, 2022)
- Social and behavioural factors – natural evening chronotype, use of electronic devices, regularly attending late evening events (e.g., classes or sport scheduled in late evening) (Prihodova et al, 2022) – that increase artificial light exposure in the evening and decrease it in the morning hours (Micic et al, 2016; Nesbitt, 2018)
- Jet lag (Snitselaar et al, 2019)

3.3 Symptoms/functional impact

Individuals with DSPD demonstrate difficulties initiating sleep, so, when woken at a conventional time, experience markedly reduced daytime functioning (e.g., excessive daytime sleepiness, fatigue, low mood, irritability), impaired cognitive performance (e.g., concentration, memory and attention), and poor social functioning (Gomes et al, 2021; Micic et al, 2016; Prihodova et al, 2022; Richardson et al, 2017). These symptoms are most notable in the morning (Richardson et al, 2016), and can impact work capacity and attention due to decreased cognitive and motor skills (Gomes et al, 2021).

Over the long term, unmanaged DSPD can cause permanent health and social disruptions, impacting quality of life. Job loss, truancy, school failure, less participation in sport and social difficulties are potential risks for people with DSPD (Micic et al, 2016). Some studies suggest depression, medication use (antacids, hypnotics), tobacco, alcohol and caffeine consumption are greater for people with DSPD than controls (Micic et al, 2016).

It should be noted, when individuals diagnosed with DSPD are allowed to set their own sleep schedule – meaning they do not experience forced awakening in the morning and chronic sleep deprivation – they often experience normal sleep quality and duration for their age and experience remission of symptoms (American Psychiatric Association, 2013).

3.4 Diagnosis

DSPD is characterised by significantly later sleep onset times compared to social convention and long sleep latencies when attempting sleep at conventional bedtimes, often between 2-6 hours after the desired sleep time (American Psychiatric Association, 2013; Micic et al, 2016).

DSPD falls under the Circadian Rhythm Sleep-Wake Disorders (CRSWD) in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (American Psychiatric Association, 2013); the following outlines the criteria for CRSWD and subtype DSPD:

A. A persistent or recurrent pattern of sleep disruption that is primarily due to an alteration of the circadian system or misalignment between the endogenous

circadian rhythm and the sleep-wake schedule required by an individual's physical environment or social or professional schedule, and

B. The sleep disruption leads to excessive sleepiness, insomnia, or both, and

C. The sleep disturbance causes clinically significant distress or impairment in social, occupational, and other important areas of functioning

Specify if: Delayed sleep phase type – a pattern of delayed sleep onset and awakening times with an inability to fall asleep and awaken at a desired or conventionally acceptable earlier time.

For DSPD, specify if: **episodic** – symptoms last at least one month but less than three; **persistent** – symptoms last for 3 months or longer; **recurrent** – two or more episodes are experienced in the space of one year

The clinical diagnostic criteria for DSPD, along with sleep diaries and actigraphy (sleep monitoring devices), is typically not sufficient to distinguish between DSPD related to disruption of endogenous sleep-wake regulation and sleep-wake difficulties caused by factors such as poor sleep hygiene and behavioural delay of sleep onset (Prihodova et al, 2022). A delay in the dim light melatonin onset (DLMO) – a biological marker in saliva and plasma – can be used to identify disorders of the endogenous circadian rhythm (Snitselaar et al, 2019).

3.5 Treatment

Current treatments for DSPD aim to advance the natural circadian phase and bring sleep timing earlier to increase sleep duration and reduce daytime impairments (Gomes et al, 2021; Micic et al, 2016; Nesbitt, 2018). However as large scale randomised controlled studies in patient population are lacking there is little supporting clinical evidence how to best manage the disorder (Nesbitt, 2018). Main treatments to regulate the circadian rhythm and induce earlier sleep phases are chronotherapy or pharmacotherapy, light therapy and behavioural lifestyle changes (Lu et al, 2022).

Exogenous melatonin supplementation has been a traditional therapy for DSPD, which provides a sleep time-cue for the body (Nesbitt, 2018). A strategically timed 0.5mg daily dose taken consistently 10-12 hours prior to the mean mid-point of desired sleep time or 6-8 hours prior to the desired sleep onset time can be effective in changing sleep phases (Nesbitt, 2018). Using melatonin in chronotherapy should be closely monitored by a sleep specialist to avoid desynchronisation to a non-24hr sleep-wake disorder (Nesbitt, 2018).

Light therapy is another treatment that has been used to alter sleep timing to a more desired schedule as light is the predominant timekeeper of the circadian cycle (Richardson et al, 2017). Morning light is most helpful in phase-advancing, so it should be delivered immediately on waking. This can be natural light, by going out for a walk or sitting near a window, bright artificial light, dim light or blue light therapy (Nesbitt, 2018). While there is some mixed data

regarding the use of artificial light therapy, many studies do show advancement of sleep onset and sleep offset when artificial light therapy is used consistently in the morning (Danielsson et al, 2018). Research by Danielsson et al (2018) into the use of artificial light therapy for DSPS found daily light therapy more predictive of better outcomes than light intensity exposure or length of time of the exposure. However, a systematic review suggested that while the sleep-wake cycle may be altered, there was often no corresponding biological change in the circadian phase as measured by the biomarker DLMO (Gomes et al, 2021).

For optimal effect of morning light therapy, it is suggested it should be combined with minimising exposure to artificial light from dusk onwards in the evening (Danielsson et al, 2018; Richardson et al, 2017). Sources of artificial light that should be avoided include light from television screens and handheld devices (Cardinali et al, 2021). Use of blue light filter goggles or glasses with amber lenses may effectively reduce the stimulation from artificial light in the evenings (Nesbitt, 2018). It is suggested that light therapy, including natural, artificial bright, dim and blue light, can result in improvements in sleep quality and daytime functioning (i.e., reductions in daytime sleepiness, fatigue and functional impairment) (Lu et al, 2022; Richardson et al, 2019), however these improvements are often not sustained long-term (Gomes et al, 2021).

Another novel technique described in the literature that may manipulate sleep-onset timing is the use of **exercise** to advance the circadian timing. For individuals with DSPD, this may be undertaking 1 hour of moderate exercise in the subjective “morning” (i.e., soon after waking) and advancing wake up time and exercise timing by 20-30 minutes earlier each day until desired sleep timing is achieved (Richardson et al, 2017). This also suggests that people with DSPD should try to avoid intense exercise late in the day in order to reduce dysregulation of the circadian rhythm.

Behaviours that promote **good sleep hygiene** have also been found to support other DSPD treatment protocols. Some behaviours include (Mennella & Shiebel, 2018):

- Avoid alcohol, tobacco, and other stimulant prior to bedtime
- Avoid behaviours that require a high level of concentration immediately before bed, including exciting or emotionally disturbing activities
- Maintain a low-light environment in the evening and avoid using the computer or watching television
- Invest in good quality bedding and keep the room dark, quiet and cool
- Avoid staying in bed longer than 7.5 hours, use an alarm clock to wake
- Avoid a difference of 2 hours in wake time on weekends

For any DSPD treatment protocol, it is important to consider coexisting conditions that may either exacerbate DSPD symptoms or make difficult to follow the protocol (Nesbitt, 2018). For example, patients with comorbid ADHD often show reduced compliance with treatment

potentially because of the difficulties these patients have with planning and organisation in their daily life, which can lead to irregular intake of medication, such as melatonin, and difficulties following lifestyle guidance such as regular bedtimes and minimising artificial light at night (Snitselaar et al, 2019).

In general, basic treatment of DSPD involves good sleep hygiene, consistent regular bedtime with habitual lights off, appropriate light exposure and exogenous melatonin taken at the right time (Prihodova et al, 2022; Snetselaar et al, 2019). However, implementation of treatment can be difficult due to lack of patient motivation and compliance (Prihodova et al, 2022), which may be secondary to the fatigue and daytime sleepiness experienced by patients with DSPD. It has been found that psychoeducation can increase adherence to treatment and increase the chance of successful results (Danielsson et al, 2018). However, with any treatment for DSPD relapse is common as correction of the circadian delay is rarely permanent (Nesbitt, 2018).

3.6 Prognosis

DSPD can be episodic, persistent or recurrent (see section 3.4 Diagnosis) (American Psychiatric Association, 2013). While it is more common in adolescents and young adults, it can continue into adulthood if not managed effectively (Richardson et al, 2017).

DSPD often follows a **persistent** course and can be refractory to therapeutic interventions (Prihodova et al 2022). A **relapse of symptoms is common**, and patients should be prepared for this eventuality and encouraged to go back to their original treatment plan (American Psychiatric Association, 2013; Nesbitt, 2018).

Exacerbations or relapses are often triggered by a change in schedule that requires an early awakening time, such as a change in work or school hours (American Psychiatric Association, 2013). Individuals who can manage their schedule to accommodate the delayed sleep phase timing demonstrate greater resilience to experience remission of symptoms (American Psychiatric Association, 2013).

4. References

American Psychiatric Association. (2013). Attention Deficit Hyperactivity Disorder. In *Diagnostic and statistical manual of mental disorders* (5th ed.).

Danielsson, K., Jansson-Frojmark, m., Broman, J.-E., & Markstrom, A. (2018). Light therapy with scheduled rise times in young adults with delayed sleep phase disorder: therapeutic outcomes and possible predictors. *Behavioural Sleep Medicine, 16*(4), 325-336. https://doi.org/10.1080/15402002.2016.1210150

Gomes, J. N., Dias, C., Brito, R. S., Lopes, J. R., Oliveira, I. A., Silva, A. N., & Salles, C. (2021). Light therapy for the treatment of delayed sleep-wake phase disorder in adults: a systemic review. *Sleep Science, 14*(2), 155-163. DOI: 10.5935/1984-0063.20200074

Li, D., Fag, P., Liu, H., Chen, L., Fu, Y., Liu, J., Xie, B., Liu, Y., Ye, H., & Gu, P. (2022). The clinical effect of blue light therapy on patients with delayed sleep-wake phase disorder. *Nature and Science of Sleep, 14,* 75-82. https://doi.org/10.2147/NSS.S344616

Mennella, H., & Schiebel, D. A. (2018). Delayed sleep phase syndrome. *CINAHL.*

Micic, G., Lovato, N., Gradisar, M., Ferguson, S. A., Burgess, H. J., & Lack, L. C. (2016). The etiology of delayed sleep phase disorder. *Sleep Medicine Reviews, 27*, 29-38. http://dx.doi.org/10.1016/j.smrv.2015.06.004

Nesbitt, A. D. (2018). Delayed sleep-wake phase disorder. *Journal of Thoracic Disease, 10*(Suppl 1), S103-S111. http://dx.doi.org/10.21037/jtd.2018.01.11

Prihodova, I., Dostalova, S., Bielicki, P., Skibova, J., Nevsimalova, S., & Sonka, K. (2022). Delayed sleep-wake phase disorder: can polysomnography by useful? *Pediatric neurology, 127*, 28-31. https://doi.org/10.1016/j.pediatrneurol.2021.11.010

Richardson, C. E., Gradisar, M., Short, M. A., & Lang, C. (2017). Can exercise regulate the circadian system of adolescents? Novel implications for the treatment of delayed sleep-wake phase disorder. *Sleep Medicine Reviews, 34*, 122-129. http://dx.doi.org/10.1016/j.smrv.2016.06.010

Snitselaar, M. A., Smits, M. G., & Spijker, J. (2019). ADHD symptoms in adults with delayed sleep phase disorder. *Sleep & Biological Rhythms, 17*(1), 11-18. http://dx.doi.org/10.1007/s41105-018-0188-0

5. Version control

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