

Delayed Sleep-Phase Disorder

Leon C. Lack, PhD^{a,*}, Helen R. Wright, PhD^a,
Richard R. Bootzin, PhD^b

KEYWORDS

- Circadian rhythms • Bright light • Melatonin
- Delayed sleep phase • Period length • Insomnia

Weitzman and colleagues^{1,2} first defined delayed sleep-phase syndrome as a syndrome characterized by a delay of the usual sleep period by as much as 2 to 6 hours. According to the most recent *International Classification of Sleep Disorders (ICSD-2)*,³ the terminology has been altered slightly to delayed sleep-phase disorder (DSPD). Individuals with DSPD have difficulty falling asleep at their desired bedtime and an inability to wake spontaneously at the desired time in the morning. For example, individuals with DSPD may wish to sleep between midnight and 8 AM to meet social or employment obligations. However, if they do go to bed at midnight, they experience sleep-onset insomnia and, if they must awaken at 8 AM, their total sleep time is curtailed. Repeated reduction of total sleep time results in an accumulated sleep debt and daytime sleepiness (especially in the mornings), irritability, and a lack of concentration, all of which can subsequently impair safety, performance at work, and family life.

PREVALENCE AND ETIOLOGY

Estimated prevalence rates for DSPD have ranged from 0.2% to 10% of the population.^{1,2,4-6} The apparently large range of these prevalence estimates may be due to variation of the severity criteria for the delayed sleep period. Less severe cases of DSPD (eg, 2 AM to 10 AM sleep period) are likely to be more prevalent than the more extreme delayed cases (eg, 5 AM to 2 PM sleep period). A recent study found the habitual sleep-onset times of patients diagnosed with DSPD

varied between about 11:30 PM and 5:15 AM with a majority before 2 AM.⁷

DSPD is associated with delayed endogenous circadian rhythms.⁸⁻¹⁴ This includes delayed sleep-timing parameters, melatonin circadian rhythms, and core body temperature rhythms.^{11,13,15-17} When core body temperature rhythms and melatonin circadian rhythms are delayed, then the “wake maintenance zone” (WMZ), which occurs about 6 PM and 9 PM in a normally entrained person, is also delayed.¹⁸ For example, if the temperature minimum (CTmin) is delayed until 8 AM to 9 AM, the WMZ will occur from 10 PM to 2 AM, resulting in significant difficulty falling asleep until after 2 AM.¹⁹ In such cases, a person attempting to awaken early for work or other obligations, at 8 AM for example, would be trying to arise around CTmin, the most sleepy circadian time. For this person, waking up would be especially difficult.

Researchers have speculated at the cause of this circadian rhythm phase delay. Putative circadian mechanisms include (1) a diminished response of phase advance to morning light stimulation, (2) enhanced phase-delay response to evening light stimulation, and (3) a longer than normal circadian period length (the time to complete one circadian cycle).²⁰ Just recently, Campbell and Murphy²¹ explored the third possibility in a time-free isolation experiment with one DSPD participant in comparison with three normal control healthy sleepers. The DSPD person showed a consistent circadian period length of about 25.4 hours, 1 hour longer than the average for the three controls. This suggests that DSPD may arise from an inherently longer circadian

^a School of Psychology, Flinders University, GPO Box 2100, Adelaide, SA 5001 Australia

^b Department of Psychology, University of Arizona, Tucson, AZ 85721-0068, USA

* Corresponding author.

E-mail address: leon.lack@flinders.edu.au (L.C. Lack).

period length, resulting in an unrelenting tendency to phase delay in our 24-hour world. This would imply that the DSPD patient is required to phase advance by 1 to 1.5 hours every day to remain synchronized with the 24-hour world. The fact that this seems to be possible most of the time for DSPD patients is somewhat surprising. It perhaps can be explained by the likelihood that their awakenings are usually later in the morning in the presence of generally stronger phase-advancing ambient light and the likelihood that the timing of this exposure to light is when it is most effective for phase-advancing their rhythms (eg, very soon after their temperature minimums). The possible genetic basis for this inherent tendency is being investigated.²²

Although circadian rhythm phase delay is seen as the major contributor to DSPD, some behavioral and psychological factors are also likely to be important contributors, which should also be addressed to improve treatment effectiveness. The circadian phase delayed individual is likely to feel better in the evenings than in the mornings as indicated by their preference for doing things in the evening according to their responses in the Morning/Evening questionnaire.²³ They may want to prolong this period by staying up later. In addition, they are likely to engage in activities that interfere with sleep onset and sleep quality, including late-night socializing, computer use, and mobile phone use.^{24–28} However, later bedtimes also tend to delay morning awakenings. “Sleeping in” leads to further delays of the circadian rhythm, which thus exacerbate the DSPD.^{29–31} Because of this vicious cycle of circadian rhythm phase delay leading to behavior that further magnifies the circadian rhythm phase delay, therapies must address both behavior and circadian rhythm phase delay to be most effective.

When anticipating a necessary early awakening (eg, 8 AM), the DSPD sufferer is likely to attempt an earlier bedtime (eg, midnight would be “early” for them) in the hopes of obtaining sufficient sleep. However, because this bedtime is still within the circadian wake-maintenance or alert zone, DSPD sufferer will take a long time to fall asleep and the experience is likely to be frustrating or worrying. This frustration or worry may activate the “fight or flight” response and heightened arousal, which further inhibits sleep onset. Repetition of this experience over many nights can lead to more persistent conditioned or psychophysiological insomnia. Even when DSPD sufferers sleep at their most preferred sleep time, they show evidence of having some sleep-onset insomnia. At these preferred later bedtimes, they had sleep-onset latencies that were still significantly

and clinically lengthened to 32 minutes versus 10 minutes for controls in one study¹⁴ and to 38 minutes versus 17 minutes for controls in another study.²¹ Therefore, in addition to a delayed circadian rhythm, DSPD sufferers are likely to have persistent sleep-onset insomnia.

In DSPD, behavioral or psychological factors, as well as the delayed circadian rhythm, may contribute to difficulty awakening in the morning. On those occasions when the DSPD sufferer is forced to awaken close to the circadian nadir (which is probably quite common), they experience lethargy, reduced motivation, irritability, or even mild depression. Falling back to sleep avoids these aversive experiences. Thus, the resumption of sleep rewards the sufferer by providing a way to avoid the morning period. Seeking this reward, the DSPD person could fall into a pattern of persistent conditioned sleep resumption. This possible conditioned sleep resumption also needs to be addressed in therapy. Furthermore, individuals with DSPD who must awaken for school or work may be more likely to use stimulants (eg, caffeine, nicotine) to stay awake. This may lead to a vicious cycle in which the use of stimulants delays sleep onset and produces further phase delays.³² Therefore, the clinician should be alert to the possibility of multiple factors playing a role in DSPD and be willing to use therapies addressing those factors in addition to therapies dealing with circadian factors.³³

CLINICAL DIAGNOSIS AND ASSESSMENT

It is suggested that virtually all DSPD patients score as extreme evening types although not all evening types report the distress required for a diagnosis of DSPD. Perhaps many of these non-distressed extreme evening types have adapted their lifestyles to accommodate delayed circadian rhythms.³⁴

Diagnosis of a delayed sleep-wake schedule is made from a 1- to 2-week sleep-wake diary and actigraphy if available.³⁵ Sleep parameters documented on the diaries include bedtime and lights-out time, sleep-onset latency (SOL), time spent awake during the night after sleep onset (wake after sleep onset [WASO]), final wake-up time (including whether or not an alarm (AI) was needed), and estimated total sleep time (TST). Time of food, caffeine, and alcohol intake during the day is also entered on the diary each evening. Any change of sleep pattern between weekdays and weekends should be noted. **Fig. 1** is the 1-week sleep-wake diary of a 19-year-old university student with DSPD. The diary illustrates a delayed sleep pattern and sleep-onset difficulty. Despite bedtimes around midnight, his average

Download English Version:

<https://daneshyari.com/en/article/3837646>

Download Persian Version:

<https://daneshyari.com/article/3837646>

[Daneshyari.com](https://daneshyari.com)