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Cognitive performance in adolescents with Delayed Sleep-Wake Phase Disorder: Treatment effects and a comparison with good sleepers

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ABSTRACT

The present study aimed to investigate whether Australian adolescents with Delayed Sleep-Wake Phase Disorder have impaired cognitive performance and whether chronobiological treatment for Delayed Sleep-Wake Phase Disorder improves adolescents' sleep, daytime functioning and cognitive performance. Adolescents with Delayed Sleep-Wake Phase Disorder (mean = 15.68 ± 2.1 y, 62% f) reported significantly later sleep timing (d = 1.03–1.45), less total sleep time (d = 0.82) and greater daytime sleepiness (d = 2.66), fatigue (d = 0.63) and impairment (d = 2.41), compared to good sleeping adolescents (mean = 15.9 ± 2.4 y, 75% f). However, there were no significant between-group differences (all $p > 0.05$) in performance on the Operation Span ($\eta p^2 = 0.043$), Digit Span (forwards: $\eta p^2 = 0.002$, backwards: $\eta p^2 = 0.003$), Letter Number Sequencing ($\eta p^2 < 0.001$) (working memory) and Digit-Symbol Substitution Tasks ($\eta p^2 = 0.010$) (processing speed). Adolescents with Delayed Sleep-Wake Phase Disorder went on to receive 3 weeks of light therapy. At 3 months post-treatment, adolescents with Delayed Sleep-Wake Phase Disorder reported significantly advanced sleep timing (d = 0.56–0.65), greater total sleep time (d = 0.52) and improved daytime sleepiness (d = 1.33), fatigue (d = 0.84) and impairment (d = 0.78). Performance on the Operation Span (d = 0.46), Letter Number Sequencing (d = 0.45) and Digit-Symbol Substitution tasks (d = 0.57) also significantly improved.

1. Introduction

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

It is unsurprising then, that some teenagers (~1–16%) develop a more severe form of this sleep problem, Delayed Sleep-Wake Phase Disorder (American Academy of Sleep Medicine, 2014; Lovato, Gradisar, Short, Dohnt, & Micic, 2013; Saxvig, Pallesen, Wilhelmsen-Langeland, Molde, & Bjorvatn, 2012). Delayed Sleep-Wake Phase Disorder is a circadian rhythm disorder, whereby the

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body clock of an individual (i.e., melatonin and core body temperature rhythms) is timed significantly later than the 24-hr social world (Micic et al., 2016). Due to the severity of delayed circadian timing, Delayed Sleep-Wake Phase Disorder typically presents as difficulty initiating sleep and an inability to wake early in the morning, to fulfil daytime commitments (American Academy of Sleep Medicine, 2014). Many adolescents with Delayed Sleep-Wake Phase Disorder attempt to wake at a socially conventional time to attend school, which is during their subjective night (i.e., point of maximal sleepiness) (Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998), which is associated with reduced alertness, slower reaction time, poorer working memory, and impaired grip strength (Johnson et al., 1992; K. Wright, Hull, & Czeisler, 2002). Subsequently, Delayed Sleep-Wake Phase Disorder has been associated with a plethora of negative daytime consequences (i.e., daytime sleepiness, fatigue; Gradisar, Dohnt, et al., 2011; Lovato, Gradisar, et al., 2013; Micic et al., 2015), and ultimately, school lateness, absenteeism, dropout, and poor academic performance (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010; Giannotti, Cortesi, Sebastiani, & Ottaviano, 2002; Gradisar, Dohnt, et al., 2011; Lack, 1986; Meijer, Habekothé, & Van Den, 2000; Saxvig et al., 2012). Therefore, sleep disturbance is likely a risk factor for poor school performance in adolescence (Beebe, 2011).

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

Another way in which clinically delayed sleep timing might impact upon adolescent's cognition is through reduced total sleep time. Adolescents with Delayed Sleep-Wake Phase Disorder often experience restricted sleep due to a very late time of falling asleep and the need to rise early for school (Saxvig et al., 2012). Reduced sleep duration in adolescents (e.g., total sleep time < 8hr) has been associated with impaired working memory performance (Gradisar, Terrill, Johnston, & Douglas, 2008). More extreme experimental sleep restriction (time in bed = 5hr) results in significant impairments in sustained attention, working memory, and executive function (Lo, Ong, Leong, Gooley, & Chee, 2016), particularly in the morning (Agostini, Carskadon, Dorrian, Coussens, & Short, 2017). Pertinent to adolescents with Delayed Sleep-Wake Phase Disorder, recovery sleep was not sufficient to rectify these impairments (Agostini et al., 2017; Lo et al., 2016). On the other hand, small advances in sleep timing and total sleep time, over two weeks, resulted in improvements in some aspects of adolescent cognitive performance (i.e., visuospatial processing) (Dewald-Kaufmann, Oort, & Meijer, 2013).

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

Due to the significant impact that impaired cognition may have on adolescents with Delayed Sleep-Wake Phase Disorder (i.e., poor grades and attendance, school dropout), and that sleep has been identified as a modifiable risk factor for many conditions (e.g., depression) (Clarke & Harvey, 2012), it is also important to know whether interventions for sleep improve cognitive performance. Randomised controlled trials evaluating light therapy + cognitive behaviour therapy, or light therapy + exogenous melatonin have shown clinically meaningful improvements in the sleep and daytime functioning of adolescents with Delayed Sleep-Wake Phase Disorder (Danielsson, Jansson-Fröjmark, Broman, & Markström, 2016; Gradisar, Dohnt, et al., 2011; Saxvig et al., 2014). However, only one trial to date has measured changes in cognitive performance as a result of treatment (Wilhelmsen-Langeland et al., 2013). Following 2 weeks of treatment, adolescents improved their performance on measures of working memory, processing speed, and executive functioning, maintaining sleep and daytime benefits at the 3-month follow-up (i.e., working memory, processing speed, sustained attention, verbal fluency).

Importantly, with only one study investigating whether cognitive performance improves alongside chronobiological treatment for Delayed Sleep-Wake Phase Disorder in adolescents, more trials are needed to confirm this single finding (Wilhelmsen-Langeland et al., 2013). Therefore, the second aim of the present study was to measure changes in adolescents' cognitive performance across treatment for Delayed Sleep-Wake Phase Disorder. Evidence for the efficacy of light therapy is building (Danielsson et al., 2016; Gradisar, Dohnt, et al., 2011) and as advancements in the delivery of light therapy occur (i.e., development of portable light glasses), this opens the door for novel treatments. For example, timed physical activity may be used as an adjunct to light therapy, to assist in phase advancing the human circadian rhythm (Richardson, Gradisar, Short, & Lang, 2017). We hypothesised that light therapy and timed morning activity will advance sleep timing and increase total sleep time, and consequently, improve adolescents' daytime functioning and cognitive performance across treatment and follow-up. If improvements in cognition occurred, we also aimed to shed light on whether advancement of sleep timing, extension of total sleep time, or improvements in daytime functioning (i.e., sleepiness) contributed to these improvements. Processing speed and working memory have been linked with academic attainment (Alloway & Alloway, 2010; Fry & Hale, 1996; Rindermann & Neubauer, 2004) and were previously found to be sensitive to improvements in sleep (Wilhelmsen-Langeland et al., 2013). As such, processing speed and working memory were chosen as measures of cognitive performance in the present study.