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Jääkallio, Pirita Katariina; Kuula, Liisa Katri Mirjami; Partonen, Timo; Pesonen, Anu-Katriina

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Continuity and Change of Delayed Sleep Phase From Adolescence to Early Adulthood

Pirita Jääkallio, MD,[1] Liisa Kuula, PhD,[1] Timo Partonen, MD,[2] Anu-Katriina Pesonen, PhD[1]

abstract

BACKGROUND AND OBJECTIVES: The developmental trajectories and stability of delayed sleep phase (DSP) are poorly known. We investigated how behavioral and psychiatric characteristics, circadian thermoregulation, and the amount and intensity of physical activity (PA) were associated with the continuity of DSP from late adolescence to early adulthood.

METHODS: We created a population-based longitudinal study in which 1374 adolescents (mean age, 16.8 years; SD, 0.6; 66% girls) self-reported their sleep and psychiatric and behavioral symptoms at timepoint 1 (T1). A DSP-weighted subsample at T2 (7 months from T1; N = 315) and at T3 (19 months from T1; N = 207) provided sleep, PA (GENEActiv actigraphy, 8 days and nights), and skin temperature data (iButton 1922L thermologgers; 3 days and nights) twice.

RESULTS: Cluster analysis indicated 2 behavioral profiles underlying DSP: 1 pointing to game addiction alone and the other to widely spread behavioral challenges. The stability of DSP was 70%, such that poor self-control, attention-deficit hyperactivity disorder symptoms, and alcohol use together accounted for 18%. Alcohol use and poor self-control accounted for 16% of the emergence of DSP over time. Circadian thermoregulation or the duration or intensity of PA did not associate with the continuity of DSP.

CONCLUSIONS: DSP is a highly stable condition from late adolescence to early adulthood. Behavioral challenges related to self-control, attention-deficit hyperactivity disorder symptoms, and alcohol use underlie the trajectories of DSP over time. The outcomes emphasize behavioral over the potential physiological components of DSP measured in this study and give clear directions to develop new interventional targets to overcome DSP.

WHAT'S KNOWN ON THIS SUBJECT: Delayed sleep phase disorder is the most common sleep disturbance in adolescence, leading to negative consequences for those suffering from it. There is a lack of understanding of how the condition develops in time and what the individual underlying factors are.

WHAT THIS STUDY ADDS: Delayed sleep phase is a highly enduring condition, and poor self-control, alcohol use, and attention-deficit hyperactivity disorder symptoms are associated with its stability. Physiological attributes related to circadian thermoregulation or physical activity did not play a role for delayed sleep phase trajectories.

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[1]*Faculty of Medicine, SleepWell Research Program, University of Helsinki, Finland; and* [2]*Finnish Institute for Health and Welfare, Helsinki, Finland*

Address correspondence to: Pirita Jääkallio, MD, SleepWell Research Program, Faculty of Medicine, University of Helsinki, Haartmaninkatu 3, 00290, Helsinki, Finland. pirita.jaakallio@helsinki.fi

Dr Jääkallio carried out the initial analyses, drafted the initial manuscript, critically reviewed and revised the manuscript, and took part in the study conceptualization. Dr Kuula critically reviewed and revised the manuscript and was involved in designing the study, in coordinating and supervising data collection and data curation, and in the study conceptualization. Dr Partonen critically reviewed and revised the manuscript and participated in the study conceptualization. Dr Pesonen conceptualized and designed the study, oversaw resources and funding acquisition, coordinated and supervised data collection, and critically reviewed and revised the manuscript and the statistical analysis strategy. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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INTRODUCTION

Adolescence is accompanied by substantial changes in sleep toward a later phase of circadian rhythms,[1] irregular sleep patterns, and insufficient sleep duration.[2] The phase delay is initiated in early adolescence,[3,4] and there is a progressive development to later sleep times over the transition period into adulthood, up to a turning point to an earlier phase of circadian rhythms at the average age of 16 to 23 years depending on the study.[5–8]

As the phase of circadian rhythms shifts later, some adolescents develop delayed sleep phase disorder (DSPD),[9] which is characterized by persistently late bedtimes (ie, at least 2 hours later than the social norm)[10] as well as problems with falling asleep and waking up at the desired time,[11] resulting in significant disruption in daytime activities.[12]

DSPD is a clinical disorder defined according to the Diagnostic and Statistical Manual of Mental Disorders (DSM)[13] or the International Classification of Sleep Disorders (ICSD)[14] criteria and has a prevalence of 3% to 4%.[11,15,16] Across the scientific studies on DSPD, there is a great variation in methodology, definitions, or diagnostic criteria used. Usually, the clinical definition is not fully met[15–20] or is not verified by clinicians. The majority of the existing literature thus refers to its subclinical form, delayed sleep phase (DSP), estimated to have a prevalence of 4.6% to 8.4%.[16,20]

The developmental process or etiology of DSP(D) is not well known. Evidence points toward the etiology as changes in endogenous biological factors, such as lower initial levels of melatonin secretion,[21] reduced homeostatic sleep pressure, lengthened circadian period, and reduced sensitivity to morning light or increased sensitivity to evening light in synchronizing the circadian rhythm.[22] In addition, there are several emotional and behavioral correlates of DSP(D). These include, for example, personality traits,[23–25] negative emotional thoughts related to sleep onset,[22] poor academic performance,[20] and affective and neurodevelopmental disorders.[11,20] Moreover, the timing and intensity of physical activity (PA) influence the phase and amplitude of circadian rhythms.[26–29] It has been demonstrated that individuals with DSP had a lower share of moderate to vigorous PA compared with those without DSP.[30]

Above all, the existing evidence is cross-sectional, and longitudinal evidence on the development and continuity of DSP is clearly lacking. Among the few, a study showed that DSP in adolescents aged 16 to 19 years was preceded by more sleep disruptions and mental health problems in adolescents aged 11 to 13 years.[31] We have previously reported that DSP in early adulthood was preceded by elevated anxiety during adolescence.[32]

The current study provides a multimodal and longitudinal analysis of DSP trajectories. Our aim is to investigate whether individuals with DSP are characterized by different psychiatric and behavioral profiles and whether these individual profiles are associated with the continuity and change of DSP from late adolescence to early adulthood. Additionally, we aim to find out whether the circadian thermoregulation,[33,34] or PA patterns, would underlie temporal changes in DSP from late adolescence to early adulthood. We hypothesize that (1) certain behavioral and psychiatric characteristics are associated with the temporal change or stability of DSP across time, and (2) physiological components (circadian temperature rhythms) underlie these changes.

METHODS

Participants and Procedure

Participants came from the population-based SleepHelsinki! cohort[35] described in Figure 1. Using the Finnish Population Register, we invited an urban cohort (ie, all inhabitants of the capital city of Helsinki born between 1999–2000 with Finnish registered as their native language; 7539 out of 10 4576 residents). At timepoint 1 (T1), 1374 participants (18.2% of the invited; 66.4% girls; mean age, 16.8 years; SD, 0.6) filled in a questionnaire (N = 1218 with complete data). From this cohort, we invited 552 (45.3% of T1) adolescents to the follow-up study (T2), weighted with the presence of DSP (N = 364 for DSP, N = 188 without). Of these, 353 (63.9%) agreed to participate. Of these, 315 participants provided skin surface temperature data, actigraphy data, and questionnaire data at T2 and 207 (59%) participants 12.2 months (SD, 0.8) later at T3.

The participants agreed to participate in this study and gave written informed consent for the data to be used for research purposes. All procedures were in accordance with the Declaration of Helsinki and its amendments. Ethical permission was obtained from the Hospital District of Helsinki and Uusimaa Ethics Committee for Gynecology and Obstetrics, Pediatrics and Psychiatry (Decision Number 50/13/03/03/2016). A part of the sample (N = 66) underwent an intervention that has been registered under Clinical Trials 1287174 and reported elsewhere (Kuula L, Pesonen AK., unpublished data, 2025).

Background Information

Participant age and sex were derived from the Finnish Population Register. Parental education (highest in the family) was used as a proxy for socioeconomic status (SES) as low (minimum compulsory education), middle (ie, vocational school, technical college), or upper SES (university-level degree).[35]

PA and Sleep Actigraphy

Sleep and PA were measured using wrist-worn, triaxial actigraphy GENEActiv Original devices (Activinsights; T2 mean, 8.2; T2 SD, 1.3; T3 mean, 8.0; T3 SD, 1.6 days/nights;

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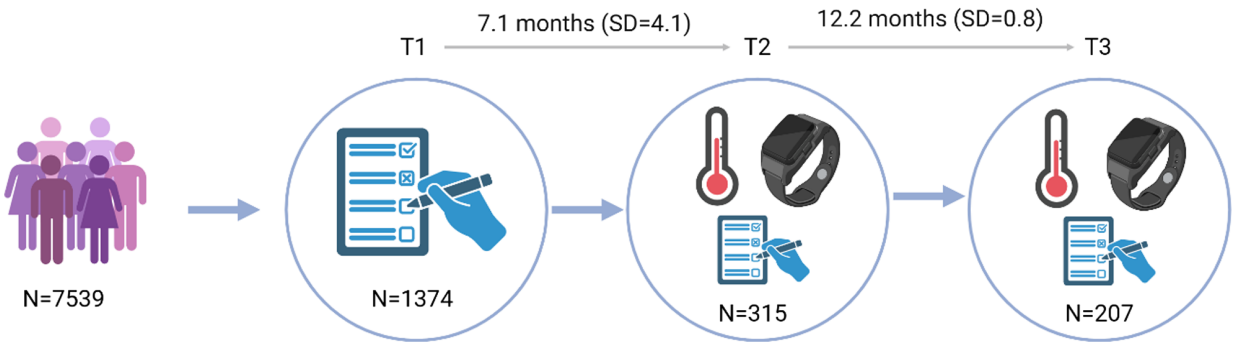


FIGURE 1.
Timeline of SleepHelsinki! study. Created with BioRender.
Abbreviations: T1 timepoint 1; T2, timepoint 2; T3, timepoint 3.

range, 3–14) along with a sleep log booklet. We converted the GENEActiv data to correspond epoch format used by a validated actigraphy algorithm both in adult[36] and adolescent populations[37] and applied this algorithm to have the following sleep parameters: Sleep duration was the assumed total sleep time. Sleep midpoint refers to the mean midpoint of the entire sleep period between sleep onset and offset. Sleep efficiency refers to the percentage of time spent asleep after sleep onset. Sleep regularity was derived from the individual SD of sleep midpoint. Nights showing marked deviations in the sleep log, or those affected by exceptional circumstances (eg, illness, travel), were excluded from the data. We defined DSP dichotomously (0 = no, 1 = yes) as having a bedtime at or after 1 AM on at least 3 nights per week, as defined previously in a similar age group[16] based on questionnaire data at T1 and actigraphy measurements at T2 and T3, reflecting the core diagnostic criterion of circadian misalignment between internal rhythm and the required sleep–wake schedule defined in both the DSM fifth edition[13] and ICSD third edition.[14]

PA was operationalized as time spent in sedentary behavior (SB), light PA (LPA) and moderate to vigorous PA (MVPA) defined from the mean metabolic equivalents from the actigraphy databased on validated cutpoints.[38]

Circadian Rhythm Temperature Measurements

As described,[39] circadian thermoregulation was measured from the skin temperature at 1-minute intervals over 3 days and nights with iButton 1922L thermologgers (DS1922 L, Maxim Integrated)[40] attached onto the inner side of the wrist near the radial artery using adhesive medical tape. Participants were instructed on how to reattach the iButton if needed and to report any removals. All records were set to start from 12 PM, and we imputed missing values using the regularized iterative principal components algorithm in the missMDA R package.[41] Temperature measures were smoothed using loess (locally estimated scatterplot smoothing) using the span of 0.3. A cosinor model[42] was fitted separately for each individual time series.

Circadian rhythm measures included circadian period length (duration of 1 cycle), amplitude (half the extent of predictable temperature variation within a cycle), and mesor (midline of temperature across the cycles). Circadian period length included values between 21 and 29 hours; outliers of other variables of more than 3 SD from the mean were filtered out.

Psychiatric and Behavioral Characteristics

Participant characteristics were derived from the self-reports at T1, all scales being converted such that higher scores indicated more challenges. Sleep hygiene was measured with the Adolescent Sleep Hygiene Scale.[43] Conduct problems were assessed with the Strengths and Difficulties Questionnaire.[44] Alcohol use was assessed with questions on the frequency and quantity of alcohol.[45] Game addiction was measured with the Game Addiction Scale.[46] Self-control was measured with the Self-Control Scale.[47] Attention-deficit hyperactivity disorder (ADHD) symptoms were measured with the ADHD Rating Scale-IV.[48] Depressive symptoms were measured with the Beck Depression Inventory-II.[49,50] Anxiety symptoms were measured with the Generalized Anxiety Disorder assessment 7.[51] Fatigue was derived from the question, "How many whole days during the past 12 months were you away from school or work due to tiredness or fatigue?" such that those reporting more than 15 days (10%) scored 1 (<15 days = 0).

Statistical Methods

We used IBM Statistics SPSS 29 for the statistical analyses. For the cluster analyses, all variables at T1 were standardized (mean, 0; SD, 1). We used hierarchical cluster analysis, and dendrograms were visually examined to decide on the number of clusters. The final analyses were done with k-means cluster analysis. We used analysis of variance (ANOVA) to compare memberships across clusters with DSP status. Backward stepwise binary logistic regression analysis was used to define the contributions of behavioral and psychiatric characteristics to DSP continuity. We used

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logistic regression analyses with bootstrapping to analyze if physiological variables and PA were associated with DSP at T2 and T3 and ANOVA models to analyze DSP continuity. $P < .05$ was considered to be statistically significant.

RESULTS

Initial Analyses

Table 1 shows the demographic and sleep variables of those with and without DSP at T1, T2, and T3. Average time between T1 and T2 was 7.1 months (SD, 4.1; range, 1.1–14.8 months) and between T2 and T3 was 12.2 months (SD, 0.8; range, 9.7–15 months). There were significantly fewer girls with DSP compared to those without DSP at T2 (63% and 78%, respectively) and at T3 (69% and 84%, respectively). The prevalence of DSP at T1 was 35%, 30%, and 25% in the SES groups from lowest to highest, respectively ($P = .003$). Those with DSP expressed more symptoms of depression and anxiety at T1 and shorter sleep duration (T2, −44 min; T3, −24 min), later sleep start time (T2, −2 h 4 min; T3, −2 h 17 min), later sleep midpoint (T2, −1 h 40 min; T3, −2 h 2 min), lower sleep efficiency (T2, −2.2%; T3, −2.4%), and more irregular sleep (T2, 31 min; T3, 26 min) both at T2 and T3 than those without DSP.

There was a significant negative correlation between amplitude and game addiction ($r = −0.12$; $P = .049$) at T2 and a positive correlation between mesor and depressive symptoms ($r = 0.15$; $P = .025$) and poor self-control ($r = 0.14$; $P = .049$) at T3. Regarding PA, there was a significant

TABLE 1. Participant Characteristics at Each Timepoint

	T1			T2			T3		
	DSP (N = 373)	No DSP (N = 1001)	*P* Value	DSP (N = 172)	No DSP (N = 143)	*P* Value	DSP (N = 113)	No DSP (N = 94)	*P* Value
Age, mean (SD), yrs	16.9 (0.6)	16.8 (0.6)	.004	17.5 (0.7)	17.4 (0.7)	.363	18.5 (0.6)	18.4 (0.7)	.482
Sex (%), girls	63.0	67.7	.099	62.8	78.3	.003	69.0	84.0	.012
GAD-7 (%)[a]									
0–4	39.2	55.3	.006				56.6	75.5	.002
5–9	32.9	30.6					27.4	19.1	
10–21	28.0	14.1					15.9	5.3	
BDI-II (%)[b]									
0–12	53.4	66.7	.007				66.4	81.9	.007
13–18	13.7	18.4					16.8	11.7	
19–63	32.9	14.9					16.8	6.4	
Psych med, n (%)				4 (2.3)	4 (2.8)	.792			
SES (%)									
Lower	2.8	2.0	<.001						
Middle	30.4	21.8							
Upper	66.9	76.2							
Sleep, mean (SD), hh:mm									
Sleep latency				0:21 (0:25)	0:16 (0:19)	.053	0:24 (0:26)	0:17 (0:16)	.028
Sleep start				1:50 (1:02)	23:46 (0:42)	<.001	2:08 (1:14)	23:51 (0:48)	<.001
Sleep end				9:12 (1:20)	7:55 (0:43)	<.001	9:53 (1:23)	8:00 (0:58)	<.001
Get-up time				9:15 (1:20)	8:02 (0:46)	<.001	9:56 (1:23)	8:03 (0:59)	<.001
Sleep duration				7:21 (0:58)	8:05 (0:40)	<.001	7:44 (0:51)	8:08 (0:52)	.001
Sleep midpoint				5:28 (1:00)	3:48 (0:37)	<.001	5:57 (1:06)	3:55 (0:46)	<.001
Sleep efficiency (%)				83.0 (5.7)	85.2 (5.0)	.004	82.6 (6.3)	85.0 (4.4)	.002
Sleep regularity[c]				1:29 (0:33)	0:58 (0:24)	<.001	1:23 (0:33)	0:57 (0:24)	<.001

Abbreviations: BDI-II, Beck Depression Inventory-II; DSP, delayed sleep phase; GAD-7, Generalized Anxiety Disorder 7-item questionnaire; psych med, psychiatric medication; SES, socioeconomic status; T1 = timepoint 1, T2 = timepoint 2, T3 = timepoint 3.

[a] GAD-7 point results: 0–4, minimal; 5–9, mild; 10–21, moderate or severe.

[b] BDI-II point results: 0–12, normal; 13–18, mild; 19–63, moderate or severe.

[c] SD of the sleep midpoint.

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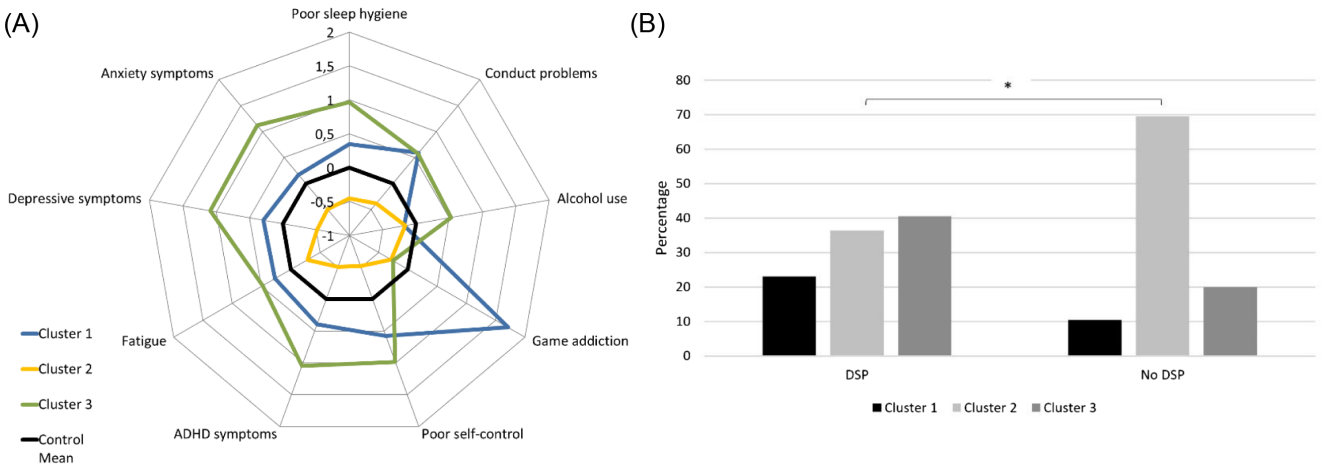


FIGURE 2.
(A) Characteristics of cluster members in clusters 1–3 in relation to each other and control mean. (B) Percentage of DSP at T1 in clusters 1–3. Differences in clusters compared to each other with regard to DSP are presented. * indicates $P < .05$.
Abbreviations: ADHD, attention-deficit hyperactivity disorder; DSP, delayed sleep phase; T1, timepoint 1.

correlation between amplitude and SB% (r = −0.12; $P = .038$), LPA% (r = 0.12; $P = .033$), and MVPA% (r = 0.27; $P < .001$) and between mesor and MVPA% (r = −0.22; $P < .001$) at T2 and between mesor and MVPA% (r = −0.20; $P = .003$) at T3. Please see the Supplementary Material for more data.

Cluster Analysis of Psychiatric and Behavioral Characteristics at T1

Hierarchical cluster analyses suggested an optimal number of 3 clusters for the psychiatric and behavioral characteristics at T1. Figure 2A shows the 3-cluster solution of the k-means cluster analysis as a radar chart (N = 1218, 66.3% females). In cluster 1 (N = 170, 35.9% females), game addiction with a z-score of 1.7 was clearly higher compared with the 2 other clusters ($P < .001$), and it was characterized by an average level of challenges in other psychiatric and behavioral scores. Cluster 2 (N = 735, 63.8% females) was characterized by a low level of challenges compared with the 2 other clusters ($P < .001$) and to the sample average. Cluster 3 (N = 313, 88.8% females) shows a significantly higher level of behavioral and psychiatric challenges compared with the 2 other clusters. See Table 2 for cluster comparisons. There was a significant difference between no DSP and DSP in cluster membership ($P = .046$) as shown in Figure 2B. The shares of clusters 1 to 3 were 23.0%, 36.4%, and 40.5%, respectively, for those with DSP and 10.5%, 69.5%, and 20.0%, respectively, for those without DSP.

Continuity of DSP Based on Cluster Membership

To find out whether the psychiatric and behavioral cluster profiles at T1 would associate with the continuity of DSP over time, we formed 4 DSP groups based on whether the participants had DSP at T2 and/or T3, in which the DSP stays group had DSP at both time points (N = 77; 39%), DSP emerges group had DSP at T3 but not T2 (N = 32; 16%), DSP dissipates group had DSP at T2 but not T3 (N = 24; 12%), and no DSP group (N = 65; 33%) had no DSP at either time point. A total of 70% of those who had DSP at T1 and 76% at T2 had it also at T3, and

TABLE 2. Comparison of the Psychiatric and Behavioral Characteristics Across Clusters 1 to 3

	Poor Sleep Hygiene	Conduct Problems	Alcohol Use	Game Addiction	Poor Self-Control	ADHD Symptoms	Fatigue	Depressive symptoms	Anxiety symptoms
Model	$F(2) = 384.05$ $P < .001$	$F(2) = 174.04$ $P < .001$	$F(2) = 59.47$ $P < .001$	$F(2) = 531.04$ $P < .001$	$F(2) = 515.64$ $P < .001$	$F(2) = 517.08$ $P < .001$	$F(2) = 82.75$ $P < .001$	$F(2) = 550.66$ $P < .001$	$F(2) = 544.66$ $P < .001$
2 vs 1 (*P*)	<.001	<.001	.832	<.001	<.001	<.001	<.001	<.001	<.001
3 vs 1 (*P*)	<.001	.840	<.001	<.001	<.001	<.001	.017	<.001	<.001
3 vs 2 (*P*)	<.001	<.001	<.001	.555	<.001	<.001	<.001	<.001	<.001

Abbreviations: ADHD, attention-deficit hyperactivity disorder; F, *F* value.
Cluster 1 N = 170. Cluster 2 N = 735. Cluster 3 N = 313.

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TABLE 3. Binary Logistic Regression Backward Stepwise Analysis for DSP Continuity

	DSP Dissipates vs DSP Stays From T1 to T3							No DSP vs DSP Emerges From T1 to T3						
	Initial Model			Final Model				Initial Model			Final Model			
	OR	95% CI	*P*	OR	95% CI	*P*	R^2	OR	95% CI	*P*	OR	95% CI	*P*	R^2
Poor sleep hygiene	1.43	0.42–4.82	.569					3.37	0.81–14.05	.095				
Depressive symptoms	0.96	0.89–1.04	.340					1.05	0.97–1.13	.255				
Conduct problems	1.20	0.84–1.72	.310					1.40	0.91–2.16	.121				
Anxiety symptoms	1.12	0.95–1.32	.166					0.96	0.81–1.13	.606				
Game addiction	1.03	1.00–1.07	.090	1.03	0.99–1.07	.100	0.175	1.02	0.98–1.06	.360				
Fatigue	2.54	0.57–11.21	.220	2.88	0.75–11.04	.123		0.08	0.01–0.81	.032				
ADHD symptoms	1.05	0.98–1.13	.174	1.08	1.02–1.15	.014		0.91	0.84–0.98	.018	0.95	0.89–1.00	.063	0.163
Poor self-control	0.96	0.92–1.00	.026	0.96	0.93–1.00	.038		1.04	0.99–1.08	.098	1.04	1.00–1.08	.035	
Alcohol use	1.86	1.03–3.35	.040	1.92	1.09–3.39	.025		1.71	0.95–3.09	.074	1.89	1.10–3.25	.021	

Abbreviations: ADHD, attention-deficit hyperactivity disorder; DSP, delayed sleep phase; OR, odds ratio; R^2, Nagelkerke R-squared; T1, timepoint 1; T3, timepoint 3.
Initial model includes all variables, and final model shows optimal model fit.

26% of those without DSP at T1 and 33% at T2 fulfilled the criteria at T3.

Continuity of DSP Based on Psychiatric and Behavioral Characteristics

We performed binary logistic regression analyses using a backward stepwise method to analyze the specific components underlying continuity of DSP from T1 to T3. Table 3 shows the odds ratios (ORs) of DSP dissipates vs DSP stays for the initial (model 1) and final model (model 5) after exclusion of the nonsignificant stepwise components. The final model accounted for 17.5% of the continuity (Nagelkerke R-squared) and included variables alcohol use, poor self-control, and ADHD symptoms as significant components ($P < .04$), whereas game addiction and fatigue remained nonsignificant ($.05 < P < .13$). Table 3 also shows the ORs comparing no DSP vs DSP emerges. The binary logistic regression with backward stepwise method resulted in 7 steps from the initial to the final model. The final model 7 accounted for 16.3% of the continuity (Nagelkerke R-squared) and included alcohol use and poor self-control as significant components ($P < .04$) and ADHD symptoms as a nonsignificant component ($P = .063$). The Hosmer–Lemeshow test of goodness of fit shows a good fit for the models overall.

Continuity of DSP Based on Circadian Rhythm Indicators and PA

None of the circadian rhythm indicators from the skin temperature were significantly associated with DSP cross-sectionally at T2 or T3 or associated with the temporal continuity of DSP (Table 4). Regarding PA at T2, both LPA% (N = 314; OR, 0.894; 95% CI, −0.226 to −0.013; $P = .031$) and MVPA% (N = 307; OR, 0.934; 95% CI, −0.114 to −0.023; $P = .002$) were significantly lower and *SB%* significantly higher (N = 307; OR, 1.053; 95% CI, 0.028–0.078; $P < .001$) for those with DSP. The differences did not persist at T3, and the relative percentual changes from T2 to T3 were not significant between participants without DSP vs DSP emerges nor DSP stays vs DSP dissipates ($P > .1$).

DISCUSSION

We investigated the profiles of adolescents with DSP and the continuity and change of DSP across time. At the cross-sectional level, we showed that the psychiatric and behavioral profiles of adolescents with DSP have significant variation. Two groups with an increased risk of DSP were found: one characterized by a distinct game addiction and the other by a wide range of behavioral and psychiatric challenges. These characteristics were also significantly associated with the continuity of DSP to early adulthood. In our cohort, 70% of DSP cases showed continuity in delayed sleep over the transition into adulthood. Our results indicate that the persistence of DSP over time is strongly associated with regulation of behavior. This study thus complements previous studies by showing how DSP(D) and its persistency are associated with different behavioral characteristics, not only with distinct disorders such as ADHD but also with specific behaviors as related to sleep hygiene or game addiction.[52–55]

Our cohort focused on the transition period from late adolescence to early adulthood, and we did not track the timing of the initial sleep rhythm transition that probably took place earlier during the pubertal development.[56] Our study window thus coincided with a period of biological maturation processes, with a continuous normative change toward later sleep rhythms such that individuals both with and without DSP undergo similar biological changes. Yet, developing a markedly delayed rhythm may take place at any age, as 26% of participants classified as no DSP at baseline developed DSP during follow-up.

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TABLE 4. Circadian Temperature Measures in Participants With no DSP vs DSP at T2 and T3 and ANOVA Analyses for the Relative Percentual Change From T2 to T3

				T3			((T3 − T2)/T2) × 100				
	No DSP vs DSP OR (N = 249)	CI[a]	*P*[a]	No DSP vs DSP OR (N = 171)	CI[a]	*P*[a]	DSP Stays (N = 72) Mean (SD)	DSP Emerges (N = 28) Mean (SD)	DSP Dissipates (N = 20) Mean (SD)	No DSP (N = 60) Mean (SD)	*P* (No DSP vs DSP Emerges/ DSP Stays vs DSP Dissipates)
Mesor	0.71	−0.97 to 0.20	.201	1.13	−0.53 to 0.81	.702	0.64 (2.15)	0.51 (2.21)	0.15 (1.38)	0.33 (2.14)	.698/.355
Amplitude	0.66	−0.99 to 0.11	.122	1.11	−0.55 to 0.81	.742	10.61 (62.72)	9.80 (53.16)	9.45 (34.52)	5.53 (40.86)	.721/.930
Circadian period	1.01	−0.21 to 0.21	.911	1.02	−0.23 to 0.26	.894	1.22 (7.76)	−0.39 (7.27)	−0.08 (5.86)	0.81 (6.02)	.522/.499

Abbreviations: ANOVA, analysis of variance; DSP, delayed sleep phase; OR, odds ratio; T2, timepoint 2; T3, timepoint 3.
((T3 − T2)/T2) × 100 is the relative percentual change between timepoints 2 and 3.
[a] Bootstrap results based on 1000 bootstrap samples.

The temporal change in the presence of DSP, either being stable, dissipating, or emerging across the follow-up period, was associated with psychiatric and behavioral characteristics. Poor self-control, ADHD symptoms, and alcohol use accounted for up to 18% of stability vs dissipation of DSP over time. For the emergence of DSP during the follow-up period, again alcohol use and poor self-control accounted for 16% of the outcome. Interestingly, mood and anxiety symptoms did not play a significant role in the continuity of DSP in this study for which a wide range of challenges was considered, although when studied in isolation previously, they also played a role.[32] Game addiction, previously noted as a risk factor for generic sleep problems,[57] appeared a strong component for some adolescents with DSP, but it was not significantly associated with continuity of DSP in time.

Previous studies show inconclusive results in the association between delayed circadian rhythm and circadian temperature markers. We and others have shown that delayed circadian rhythm is associated with changes in endogenous circadian markers. For example, associations with amplitude and mesor of body temperature were found in cross-section,[39] and the endogenous circadian temperature period length (tau) was 25 minutes longer in participants with DSPD compared with that of good sleepers.[33] On the other hand, the study of Micic et al[58] reported subtypes of delayed sleep and found a longer tau only in a subgroup of diagnosed cases, the other group having a normative tau. This may indicate 2 sources for the condition, one being more biologically based, the other emphasizing sleep behavior. Our current sample may echo the behavioral component of DSP, as we did not find any significant temperature-related differences between DSP and no DSP groups nor changes across time. Along with a normal maturation process, more autonomy is often given to adolescents to regulate their sleep times; albeit, there is some evidence that parental control, for example, of screentime, may result in better sleep outcomes for adolescents.[59] The current findings indirectly suggest supporting adolescents in their process of building sleep autonomy and developing skills to recognize somatic cues of tiredness as a strategy to prevent DSP,[35] as the lack of self-regulation was clearly the strongest component associated with the continuity of DSP over time.

We have previously shown that PA is associated with endogenous circadian rhythm indicators, such that higher levels of PA are linked to stronger circadian variation of skin temperature (amplitude) and lower circadian mesor.[30] Additionally, those with DSP showed less moderate to vigorous PA and more SB.[30] In the current study, the level of PA was not associated with the temporal continuity of DSP.

Strengths and Limitations

The strengths of this study comprise a population-based and longitudinal study cohort along with the use of multimodal measurement methods, including actigraphy. As limitations, sleep phase delaying covers a longer timespan than followed up in the current study. Also, the menstrual cycle, which potentially affects body temperature,[60] was not reported. Distal skin temperature measurements with iButtons can be prone to sources of error from the environment. Parental education is a narrow definition of the family SES. In addition, the generalizability of the findings is limited, as the sample represents predominantly urban upper and middle class, and as expected in this age group, the dropout rate was large and might have influenced the results. Alcohol use was self-reported with a risk for bias and should thus be interpreted with caution. Finally, it is not possible in the scope of this study to determine how sleep behavior affects circadian temperature rhythm, whether the direction is opposite or even reciprocal.

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CONCLUSIONS

Although the shift toward later bedtimes in adolescence has a biological basis, the trajectories of DSP are more associated with behavioral components, especially related to behavioral regulation. Even 100 years ago, there were concerns expressed over the strains of modern times conveyed as "hurry" and "rush" having a negative effect on adolescent sleep.[61–63] The challenge remains, and even more self-regulation is needed to cope in contemporary times. From the clinical perspective, it is essential to recognize adolescents with DSP(D), as they often experience negative consequences in various areas of life because of their delayed circadian rhythm. Our results highlight the importance of providing behavioral support—for example, cognitive behavioral therapy–based tools.[19,64,65] At the same time, our findings underscore the variability of the condition, suggesting that tailoring therapeutic support to individual needs may be warranted.

ABBREVIATIONS

ADHD: attention-deficit hyperactivity disorder
ANOVA: analysis of variance
DSM: the Diagnostic and Statistical Manual of Mental Disorders
DSP: delayed sleep phase
DSPD: delayed sleep phase disorder
ICSD: International Classification of Sleep Disorders
LPA: light physical activity
MVPA: moderate to vigorous physical activity
PA: physical activity
SB: sedentary behavior
SES: socioeconomic status
T1: timepoint 1

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REFERENCES

1. Crowley SJ, Wolfson AR, Tarokh L, Carskadon MA. An update on adolescent sleep: new evidence informing the perfect storm model. *J Adolesc.* 2018;67(1):55–65. PubMed doi: 10.1016/j.adolescence.2018.06.001
2. Lund HG, Reider BD, Whiting AB, Prichard JR. Sleep patterns and predictors of disturbed sleep in a large population of college students. *J Adolesc Health.* 2010;46(2):124–132. PubMed doi: 10.1016/j.jadohealth.2009.06.016
3. Kuula L, Pesonen A-K, Merikanto I, et al. Development of late circadian preference: sleep timing from childhood to late adolescence. *J Pediatr.* 2018;194:182–189.e1. PubMed doi: 10.1016/j.jpeds.2017.10.068
4. Saarenpää-Heikkilä OA, Rintahaka PJ, Laippala PJ, Koivikko MJ. Sleep habits and disorders in Finnish schoolchildren. *J Sleep Res.* 1995;4(3):173–182. PubMed doi: 10.1111/j.1365-2869.1995.tb00166.x
5. Randler C, Vollmer C, Kalb N, Itzek-Greulich H. Breakpoints of time in bed, midpoint of sleep, and social jetlag from infancy to early adulthood. *Sleep Med.* 2019;57:80–86. PubMed doi: 10.1016/j.sleep.2019.01.023
6. Randler C, Faßl C, Kalb N. From lark to owl: developmental changes in morningness-eveningness from new-borns to early adulthood. *Sci Rep.* 2017;7(1):45874. PubMed doi: 10.1038/srep45874
7. Roenneberg T, Kuehnle T, Pramstaller PP, et al. A marker for the end of adolescence. *Curr Biol.* 2004;14(24):R1038–R1039. PubMed doi: 10.1016/j.cub.2004.11.039
8. Kuula L, Gradisar M, Martinmäki K, et al. Using big data to explore worldwide trends in objective sleep in the transition to adulthood. *Sleep Med.* 2019;62:69–76. PubMed doi: 10.1016/j.sleep.2019.07.024
9. Bruce ES, Lunt L, McDonagh JE. Sleep in adolescents and young adults. *Clin Med (Lond).* 2017;17(5):424–428. PubMed doi: 10.7861/clinmedicine.17-5-424
10. Feder MA, Baroni A. Just let me sleep in: identifying and treating delayed sleep phase disorder in adolescents. *Psychiatr Clin North Am.* 2024;47(1):163–178. PubMed doi: 10.1016/j.psc.2023.06.011
11. Futenma K, Takaesu Y, Komada Y, et al. Delayed sleep-wake phase disorder and its related sleep behaviors in the young generation. *Front Psychiatry.* 2023;14:1174719. PubMed doi: 10.3389/fpsyt.2023.1174719

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12. Gradisar M, Gardner G, Dohnt H. Recent worldwide sleep patterns and problems during adolescence: a review and meta-analysis of age, region, and sleep. *Sleep Med.* 2011;12(2):110–118. PubMed doi: 10.1016/j.sleep.2010.11.008

13. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR)*. American Psychiatric Association; 2013.

14. The International Classification of Sleep Disorders. *Diagnostic and Coding Manual*. 3rd ed. American Academy of Sleep Medicine Westchester; 2014.

15. Sivertsen B, Pallesen S, Stormark KM, Bøe T, Lundervold AJ, Hysing M. Delayed sleep phase syndrome in adolescents: prevalence and correlates in a large population based study. *BMC Public Health.* 2013;13(1):1163. PubMed doi: 10.1186/1471-2458-13-1163

16. Danielsson K, Markström A, Broman J-E, von Knorring L, Jansson-Fröjmark M. Delayed sleep phase disorder in a Swedish cohort of adolescents and young adults: prevalence and associated factors. *Chronobiol Int.* 2016;33(10):1331–1339. PubMed doi: 10.1080/07420528.2016.1217002

17. Tomishima S, Komada Y, Tanioka K, Okajima I, Inoue Y. Prevalence and factors associated with the risk of delayed sleep-wake phase disorder in Japanese youth. *Front Psychiatry.* 2022;13:878042. PubMed doi: 10.3389/fpsyt.2022.878042

18. Hazama GI, Inoue Y, Kojima K, Ueta T, Nakagome K. The prevalence of probable delayed-sleep-phase syndrome in students from junior high school to university in Tottori, Japan. *Tohoku J Exp Med.* 2008;216(1):95–98. PubMed doi: 10.1620/tjem.216.95

19. Sivertsen B, Harvey AG, Gradisar M, Pallesen S, Hysing M. Delayed sleep-wake phase disorder in young adults: prevalence and correlates from a national survey of Norwegian university students. *Sleep Med.* 2021;77:184–191. PubMed doi: 10.1016/j.sleep.2020.09.028

20. Saxvig IW, Pallesen S, Wilhelmsen-Langeland A, Molde H, Bjorvatn B. Prevalence and correlates of delayed sleep phase in high school students. *Sleep Med.* 2012;13(2):193–199. PubMed doi: 10.1016/j.sleep.2011.10.024

21. Micic G, Lovato N, Gradisar M, et al. Nocturnal melatonin profiles in patients with delayed sleep-wake phase disorder and control sleepers. *J Biol Rhythms.* 2015;30(5):437–448. PubMed doi: 10.1177/0748730415591753

22. Gradisar M, Crowley SJ. Delayed sleep phase disorder in youth. *Curr Opin Psychiatry.* 2013;26(6):580–585. PubMed doi: 10.1097/YCO.0b013e328365a1d4

23. Wilhelmsen-Langeland A, Saxvig IW, Pallesen S, et al. The personality profile of young adults with delayed sleep phase disorder. *Behav Sleep Med.* 2014;12(6):481–492. PubMed doi: 10.1080/15402002.2013.829063

24. Micic G, Lovato N, Gradisar M, Lack LC. Personality differences in patients with delayed sleep-wake phase disorder and non-24-h sleep-wake rhythm disorder relative to healthy sleepers. *Sleep Med.* 2017;30:128–135. PubMed doi: 10.1016/j.sleep.2016.04.002

25. Lunn J, Wilcockson T, Donovan T, Dondelinger F, Perez Algorta G, Monaghan P. The role of chronotype and reward processing in understanding social hierarchies in adolescence. *Brain Behav.* 2021;11(5):e02090. PubMed doi: 10.1002/brb3.2090

26. Youngstedt SD, Elliott JA, Kripke DF. Human circadian phase-response curves for exercise. *J Physiol.* 2019;597(8):2253–2268. PubMed doi: 10.1113/JP276943

27. Barger LK, Wright KP Jr, Hughes RJ, Czeisler CA. Daily exercise facilitates phase delays of circadian melatonin rhythm in very dim light. *Am J Physiol Regul Integr Comp Physiol.* 2004;286(6):R1077–R1084. PubMed doi: 10.1152/ajpregu.00397.2003

28. Lang C, Richardson C, Short MA, Gradisar M. Low-intensity scheduled morning exercise for adolescents with a late chronotype: a novel treatment to advance circadian phase? *Sleep Adv.* 2022;3(1):zpac021. PubMed doi: 10.1093/sleepadvances/zpac021

29. Merikanto I, Kuula L, Lahti J, Räikkönen K, Pesonen A-K. Eveningness associates with lower physical activity from pre- to late adolescence. *Sleep Med.* 2020;74:189–198. PubMed doi: 10.1016/j.sleep.2020.07.021

30. Kuula L, Lipsanen J, Partonen T, Kauramäki J, Halonen R, Pesonen AK. Endogenous circadian temperature rhythms relate to adolescents' daytime physical activity. *Front Physiol.* 2022;13:947184. PubMed doi: 10.3389/fphys.2022.947184

31. Hysing M, Harvey AG, Stormark KM, Pallesen S, Sivertsen B. Precursors of delayed sleep phase in adolescence: a population-based longitudinal study. *Sleep.* 2018;41(11):zsy163. PubMed doi: 10.1093/sleep/zsy163

32. Jääkallio P, Kuula L, Pesonen AK. Temporal pathways between circadian rhythm, depression and anxiety in the transition from adolescence to early adulthood. *J Affect Disord.* 2024;350:656–664. PubMed doi: 10.1016/j.jad.2024.01.141

33. Micic G, de Bruyn A, Lovato N, et al. The endogenous circadian temperature period length (tau) in delayed sleep phase disorder compared to good sleepers. *J Sleep Res.* 2013;22(6):617–624. PubMed doi: 10.1111/jsr.12072

34. Carpenter JS, Robillard R, Hermens DF, et al. Sleep-wake profiles and circadian rhythms of core temperature and melatonin in young people with affective disorders. *J Psychiatr Res.* 2017;94:131–138. PubMed doi: 10.1016/j.jpsychires.2017.07.007

35. Short MA, Kuula L, Gradisar M, Pesonen A-K. How internal and external cues for bedtime affect sleep and adaptive functioning in adolescents. *Sleep Med.* 2019;59:1–6. PubMed doi: 10.1016/j.sleep.2018.11.018

36. Pigeon WR, Taylor M, Bui A, Oleynk C, Walsh P, Bishop TM. Validation of the sleep-wake scoring of a new wrist-worn sleep monitoring device. *J Clin Sleep Med.* 2018;14(6):1057–1062. PubMed doi: 10.5664/jcsm.7180

37. Pesonen AK, Kuula L. The validity of a new consumer-targeted wrist device in sleep measurement: an overnight comparison against polysomnography in children and adolescents. *J Clin Sleep Med.* 2018;14(4):585–591. PubMed doi: 10.5664/jcsm.7050

38. Eslinger D, Rowlands AV, Hurst TL, Catt M, Murray P, Eston RG. Validation of the GENEA accelerometer. *Med Sci Sports Exerc.* 2011;43(6):1085–1093. PubMed doi: 10.1249/MSS.0b013e318220513be

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39. Lipsanen J, Kuula L, Elovainio M, Partonen T, Pesonen AK. Data-driven modelling approach to circadian temperature rhythm profiles in free-living conditions. *Sci Rep.* 2021;11(1):15029. PubMed doi: 10.1038/s41598-021-94522-9

40. Hasselberg MJ, McMahon J, Parker K. The validity, reliability, and utility of the iButton® for measurement of body temperature circadian rhythms in sleep/wake research. *Sleep Med.* 2013;14 (1):5–11. PubMed doi: 10.1016/j.sleep.2010.12.011

41. Josse J, Husson F. missMDA: a package for handling missing values in multivariate data analysis. *J Stat Softw.* 2016;70(1):1–31. doi: 10.18637/jss.v070.i01

42. Tong YL. Parameter estimation in studying circadian rhythms. *Biometrics.* 1976;32(1):85–94. PubMed doi: 10.2307/2529340

43. LeBourgeois MK, Giannotti F, Cortesi F, Wolfson AR, Harsh J. The relationship between reported sleep quality and sleep hygiene in Italian and American adolescents. *Pediatrics.* 2005;115 (suppl_1):257–265. PubMed doi: 10.1542/peds.2004-0815H

44. Goodman R. Psychometric properties of the strengths and difficulties questionnaire. *J Am Acad Child Adolesc Psychiatry.* 2001;40(11):1337–1345. PubMed doi: 10.1097/00004583-200111000-00015

45. Virtanen S, Kaprio J, Viken R, Rose RJ, Latvala A. Birth cohort effects on the quantity and heritability of alcohol consumption in adulthood: a Finnish longitudinal twin study. *Addiction.* 2019; 114(5):836–846. PubMed doi: 10.1111/add.14533

46. Lemmens JS, Valkenburg PM, Peter J. Development and validation of a game addiction scale for adolescents. *Media Psychol.* 2009;12 (1):77–95. doi: 10.1080/15213260802669458

47. Tangney JP, Baumeister RF, Boone AL. High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *J Pers.* 2004;72(2):271–324. PubMed doi: 10.1111/j.0022-3506.2004.00263.x

48. DuPaul G. *ADHD Rating Scale-IV: Checklists, Norms, and Clinical Interpretation.* Guilford Press; 1998.

49. Beck AT, Steer RA, Brown GK. *Manual for the Beck Depression Inventory.* 2nd ed. Psychological Corporation; 1996.

50. Besier T, Goldbeck L, Keller F. Psychometric properties of the Beck depression inventory-II (BDI-II) among adolescent psychiatric patients. *Psychother Psychosom Med Psychol.* 2008;58(2):63–68. PubMed doi: 10.1055/s-2007-986195

51. Mossman SA, Luft MJ, Schroeder HK, et al. The Generalized Anxiety Disorder 7-item scale in adolescents with generalized anxiety disorder: Signal detection and validation. *Ann Clin Psychiatry.* 2017; 29(4):227–234. PubMed doi: 10.1177/104012371702900404

52. Wilhelmsen-Langeland A, Saxvig IW, Johnsen EH, et al. Patients with delayed sleep-wake phase disorder show poorer executive functions compared to good sleepers. *Sleep Med.* 2019;54:244–249. PubMed doi: 10.1016/j.sleep.2018.10.035

53. Kuula L, Pesonen A-K, Heinonen K, et al. Naturally occurring circadian rhythm and sleep duration are related to executive functions in early adulthood. *J Sleep Res.* 2018;27(1):113–119. PubMed doi: 10.1111/jsr.12581

54. Hysing M, Lundervold AJ, Posserud M-B, Sivertsen B. Association between sleep problems and symptoms of attention deficit hyperactivity disorder in adolescence: results from a large population-based study. *Behav Sleep Med.* 2016;14(5):550–564. PubMed doi: 10.1080/15402002.2015.1048448

55. Quaranta G, Barbuti M, Pallucchini A, et al. relationships among delayed sleep phase disorder, emotional dysregulation, and affective temperaments in adults with attention deficit hyperactivity disorder and cyclothymia. *J Nerv Ment Dis.* 2020;208(11):857–862. PubMed doi: 10.1097/NMD.0000000000001209

56. Carskadon MA, Tarokh L. Developmental changes in circadian timing and sleep: adolescence and emerging adulthood. In: Wolfson A, Montgomery-Downs H, eds. *The Oxford Handbook of Infant, Child, and Adolescent Sleep and Behavior.* Oxford University Press; 2013: 70–78.

57. Hawi NS, Samaha M, Griffiths MD. Internet gaming disorder in Lebanon: relationships with age, sleep habits, and academic achievement. *J Behav Addict.* 2018;7(1):70–78. PubMed doi: 10.1556/2006.7.2018.16

58. Micic G, Lovato N, Ferguson SA, Burgess HJ, Lack L. Circadian tau differences and rhythm associations in delayed sleep-wake phase disorder and sighted non-24-hour sleep-wake rhythm disorder. *Sleep.* 2021;44(1):zsaa132. PubMed doi: 10.1093/sleep/zsaa132

59. Hale L, Kirschen GW, LeBourgeois MK, et al. Youth screen media habits and sleep: sleep-friendly screen behavior recommendations for clinicians, educators, and parents. *Child Adolesc Psychiatr Clin N Am.* 2018;27(2):229–245. PubMed doi: 10.1016/j.chc.2017.11.014

60. Baker FC, Driver HS. Circadian rhythms, sleep, and the menstrual cycle. *Sleep Med.* 2007;8(6):613–622. PubMed doi: 10.1016/j.sleep.2006.09.011

61. Sleeplessness. *BMJ.* 1894;2(1761):719.

62. Acland T. On the hours of sleep at public schools: based on an inquiry into the arrangements existing on forty of the great public schools in England and others in the United States of America. *Lancet.* 1905;166(4272):136–142. doi: 10.1016/S0140-6736(01)13857-2

63. Lucas WP. *The Health of the Runabout Child: The Journey from His Mother's Lap to the School Gate.* Macmillan; 1923.

64. Gradisar M, Dohnt H, Gardner G, et al. A randomized controlled trial of cognitive-behavior therapy plus bright light therapy for adolescent delayed sleep phase disorder. *Sleep.* 2011;34(12): 1671–1680. PubMed doi: 10.5665/sleep.1432

65. Danielsson K, Jansson-Fröjmark M, Broman J-E, Markström A. Cognitive behavioral therapy as an adjunct treatment to light therapy for delayed sleep phase disorder in young adults: a randomized controlled feasibility study. *Behav Sleep Med.* 2016;14(2): 212–232. PubMed doi: 10.1080/15402002.2014.981817

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