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# Sleep variability in delayed sleep-wake phase disorder and insomnia disorder in youth: A case-control study

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ABSTRACT

*Objectives:* To compare intraindividual sleep variability in youths with delayed sleep-wake phase disorder (DSWPD) and insomnia disorder (ID), and to examine the association of sleep variability with depressive symptoms and circadian measures.

*Methods:* Youths with DSWPD (n = 34, $M_{age} = 20.7 \pm 1.7$, 70.6% female), ID (n = 40, $M_{age} = 20.3 \pm 2.4$, 70.0% female) and healthy sleepers (n = 39, $M_{age} = 19.7 \pm 2.1$, 66.7% female) completed a battery of self-report questionnaires, sleep diary for eight days with actigraphy monitoring, and laboratory-based dim light melatonin onset (DLMO) assessment. Subjective and objective intraindividual variability in sleep parameters were derived from sleep diary and actigraphy data, respectively.

*Results:* Compared with youths with ID, those with DSWPD showed greater variability in diary-derived bedtime ($p = .006$), time in bed ($p = .010$), and total sleep time ($p = .013$). Relative to healthy sleepers, the DSWPD group showed greater variability in diary-derived total sleep time ($p = .017$) and actigraphy-derived wake after sleep onset ($p = .040$). Both clinical groups showed greater variability in diary-derived sleep onset latency compared with healthy sleepers (DSWPD: $p = .011$; ID: $p < .001$). Later DLMO times and higher levels of depressive symptoms were significantly associated with increased sleep variability across the full sample.

*Conclusions:* Youths with DSWPD and ID show heightened sleep variability, linked to delayed circadian rhythm and elevated depressive symptoms. Findings highlight the clinical relevance of intraindividual sleep variability. Further prospective and interventional studies are needed to delineate the mechanistic processes associated with increased sleep variability in these conditions.

## 1. Introduction

Youth is a transitional stage accompanied by changes in sleep patterns and a shift towards eveningness, which is driven by physiological and psychosocial changes associated with pubertal development [1,2]. It is also a sensitive period often linked with increased vulnerability to developing sleep and mental health problems [3]. Delayed sleep-wake phase disorder (DSWPD) is a common sleep disorder during adolescence and young adulthood, with an estimated prevalence of 3%-16% [4,5], compared to approximately 1.5% in the general adult population (aged 20-59 years) [6]. DSWPD is often considered an extreme manifestation of "eveningness" (or "night owl"), characterized by a marked and persistent delay of the internal circadian rhythm that results in insomnia or excessive sleepiness [7,8]. Individuals with DSWPD primarily have a delay in the timing of their sleep and wake times relative to their desired schedules. This misalignment between their endogenous circadian rhythms and social obligations often results in difficulty falling asleep at socially-prescribed bedtimes [9]. An intrinsic circadian delay, characterized by a later melatonin rise time, has been observed in individuals with DSWPD [10]. DSWPD has been linked to increased

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fatigue [8,11], impaired concentration [11], as well as a higher risk of developing behavioral problems and mental health issues [4,12,13].

DSWPD shares overlapping clinical features, most notably difficulty falling asleep, with another clinical sleep disorder - insomnia disorder [9]. Individuals with DSWPD often report complaints of insomnia (54.4%) [14], and more than half of adolescents with DSWPD meet the criteria for insomnia disorder [4]. Insomnia disorder is typically characterized by subjective complaints of difficulty falling asleep, waking up in the middle of the night or too early in the morning, which can negatively affect one's physical and mental wellbeing [15]. The prevalence of insomnia disorder in youths ranges from 8% to 24%, depending on the defining criteria employed [16–19]. Youth insomnia, if left untreated, may persist over time, potentially becoming a chronic sleep problem [20,21]. Previous cross-sectional and longitudinal studies have reported an association of insomnia with significant impairments in daytime functioning (e.g., deficits in memory and attention, excessive daytime sleepiness) [21–23], academic performance (especially in math) [22,24], psychological well-being (e.g., depression, anxiety and stress) [20,23] and an increased risk of suicidality [20,23].

Intraindividual sleep variability refers to the night-to-night fluctuations in an individual's sleep patterns [25]. For example, two individuals may have the same average sleep duration over a week but differ in their daily sleep duration. Therefore, 'sleep variability' has the potential to provide insights beyond the mean values of the measured sleep metrics [26]. Higher sleep variability has been reported in association with more severe depressive symptoms [26–30], poorer subjective well-being [31], and mood instability [29]. Importantly, independent of mean sleep parameters, intraindividual variability in sleep duration and timing has been prospectively linked to more severe depressive symptoms across diverse populations (e.g., college students [32], adolescents [33,34], physicians [29]). Although there has been some research suggesting sleep irregularity in both DSWPD and insomnia [35,36], the patterns of sleep variability have not been clearly differentiated and compared. Previous research has shown that high school students with an eveningness chronotype tended to have later bedtimes and wake times compared to those with morning- or intermediate-types [37]. Individuals with DSWPD also tend to exhibit greater sleep variability in wake time and total sleep time compared to healthy sleepers [38]. While those with insomnia show increased variability primarily in initiating or maintaining sleep (i.e., sleep latency, wake after sleep onset), compared to healthy controls [35,39]. However, to date, no studies have directly compared intraindividual sleep variability between individuals with DSWPD and those with insomnia disorder. One previous study compared sleep features between both diagnostic groups adopted 7-day actigraphy and sleep diaries but did not report variability analyses [40]. Given the overlapping symptoms of DSWPD and insomnia, it is important to further explore their potential differential sleep variability characteristics. Such an analysis could potentially enhance understanding of the clinical phenomenology and underlying pathophysiological differences between these two conditions. Moreover, delayed sleep timing may be linked to an intrinsic delayed circadian rhythm relative to social schedules, resulting in irregular sleep schedule and social jetlag [28,41,42]. Nonetheless, previous studies have been limited by a reliance on subjective circadian measures (e.g., the Morningness and Eveningness Questionnaires, the Morningness–Eveningness Composite Scale) [28,41]. As such, it remains unclear whether increased sleep variability is directly associated with underlying delayed biological circadian rhythm.

Considering these research gaps, the present study aimed to examine sleep variability in youths with DSWPD and insomnia disorder, as well as the association of sleep variability with subjective and objective circadian rhythm measures and depressive symptoms. Our primary hypotheses were: 1) youths with DSWPD would show greater variability in sleep timing and duration compared to those with insomnia disorder; 2) both youths with DSWPD and insomnia disorder would show greater variability in sleep duration, initiation, and maintenance compared to healthy sleepers. Our secondary hypotheses were that, across the full sample, 1) later DLMO would be associated with greater variability in sleep schedule, timing, and duration parameters; 2) while higher levels of depressive symptoms would be associated with greater variability in timing, schedule, initiation, and maintenance.

## 2. Methods

### 2.1. Study procedure

The present study was a case-control study which aimed to recruit participants aged 15-24 years. This age range was chosen to broadly include the adolescence period and the early phase of young adulthood, with a reference to the United Nations' definition of youth [43]. Potential participants were recruited from the local community (e.g., secondary schools, universities) via flyers and social media promotions. Interested participants completed an online pre-screening form and were subsequently invited to attend a clinical interview to ascertain their eligibility. Two semi-structured clinical interviews (the Diagnostic Interview for Sleep Patterns and Disorders (DISP) [44] and the Mini International Neuropsychological Interview (MINI) [45]) were used to ascertain the presence of any sleep disorder and psychiatric disorder. The clinical interviews were conducted by a trained doctoral research student, supervised by a clinical psychologist with training in sleep medicine. Eligible participants were instructed to complete a battery of self-reported questionnaires and an 8-day sleep diary with actigraphy monitoring in a home setting, followed by a single-night laboratory-based dim light melatonin onset (DLMO) assessment. Participants were instructed not to consume excessive caffeine, alcohol and tobacco products that deviated from their normal intake amount during the study period. Participants received an incentive of HKD500 (USD$65) after completing the whole study procedure. The study protocol was approved by the Human Research Ethics Committee of the University of Hong Kong (Ref No. EA1903024). Informed consent was obtained from all the participants, and additionally from their parent/guardian for those aged under 18 years old.

### 2.2. Participants

Two case groups and one control group were included. Participants were eligible for inclusion in the insomnia disorder (ID) group if they: 1) met the DSM-5 diagnostic criteria for insomnia disorder (i.e., complaints of difficulties initiating or maintaining sleep, or early morning awakening for at least three nights a week and lasted for at least three months that caused clinically significant distress and impairments) and 2) scored $\geq$9 on the Insomnia Severity Index (ISI) [46]. Participants were eligible for inclusion in the DSWPD group if they 1) met the diagnostic criteria of DSWPD according to the DSM-5 (i.e., a persistent pattern ($>$3 months) of delayed sleep onset and offset with difficulty falling asleep or awakening at conventionally earlier times; symptoms of insomnia and/or excessive sleepiness; and clinically significant distress or impairment), and 2) had sleep onset time later than 11:15 p.m. for 12 year old, 11:30 p.m. for 13-14 year old, and 12:00 p.m. for 15-20 year old at least 3 nights per week in the past 3 months, as confirmed by the sleep diary [47]. Participants included in the control group met the research diagnostic criteria of healthy sleepers [48]: 1) absence of any sleep disturbance or daytime symptoms associated with unsatisfactory sleep; 2) free from any current sleep and psychiatric disorders; 3) scored $\leq$9 on ISI; 4) self-reported average sleep duration $\geq$7 h and $\leq$10 h.

Participants were excluded from the present study if they met any of the following criteria: 1) concurrent and regular use of medications that could affect sleep or cortisol and melatonin rhythms; 2) having medical conditions that could affect normal hormone regulation (e.g. Addison's disease, hypothyroidism, melatonin deficiency); 3) having a visual impairment; 4) working night shifts; 5) having travelled on transmeridian flights in the past month.

### 2.3. Measures

#### 2.3.1. Subjective sleep measures

Insomnia symptoms were assessed using the Insomnia Severity Index (ISI) [49]. ISI consists of 7 items, with each item rated on a 5-point Likert scale. It has been widely used in both clinical and research settings to evaluate the impact of insomnia on one's daily life. Aggregate scores ranged from 0 to 28, with higher scores indicating more severe insomnia symptoms. The Chinese version of ISI has demonstrated good psychometric properties in adolescents [46]. It showed excellent reliability in the current sample, with a Cronbach's alpha of 0.90.

Participants were instructed to complete a consensus sleep diary for eight days [50], starting from the day of the screening interview to the laboratory visit [50]. Prior work indicated that five to six nights of actigraphy measures yielded acceptable stability for intraindividual variability metrics [38,39]. We chose an eight-day window in this study because it encompassed both school/work days and free days while balancing the demand for participants and maintaining a good reliability of sleep estimates [51]. Subjective sleep parameters, including time in bed (TIB), total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), sleep efficiency (SE), sleep onset time (SON), sleep offset time (SOFF), bedtime (BT), and rise time (RT) were derived from the sleep diary.

#### 2.3.2. Objective sleep measures

Participants wore an Actiwatch Spectrum Plus (Philips Respironics, Bend, OR) on their non-dominant wrist to monitor all-day activities other than water activities (e.g., shower, swimming) for a duration of eight days. The activity levels were logged with a 60-s epoch length. The following sleep parameters were generated, including TIB, TST, SOL, WASO, SE, SON, and SOFF. Manual scoring of the start and the end of the rest intervals was conducted, following the suggestions of Patel (2015) by considering the reference indexes in the following order of importance: 1) event marker; 2) sleep diary, 3) white light intensity, and 4) activity level [52].

#### 2.3.3. Subjective circadian measures

Circadian preferences were assessed using the Morningness-Eveningness Questionnaire (MEQ) [53], which consists of 19 self-assessment items. This questionnaire assesses individuals' sleep-wake habits, peak alertness times, and preferred activity levels throughout the day. Aggregate scores ranged from 16 to 86, with lower scores indicating eveningness preference and higher scores indicating morningness preference. The current sample demonstrated good reliability, with a Cronbach's alpha of 0.82.

Midpoint of sleep on free days corrected for sleep debt on workdays (MSFsc) was measured by sleep diary and served as a marker for subjective circadian preferences. This measure provides greater accuracy than the typical midpoint of sleep (MSF) by adjusting for the compensatory sleep during free days. MSFsc was calculated by the following formula adopted in previous research [54], where $SD_f$ refers to the sleep duration during free days, and $SD_{w.avg}$ refers to the weighted average sleep duration.

$$MSFsc = MSF - \left(SD_f - SD_{w.avg}\right) / 2$$

#### 2.3.4. Objective circadian measures

MSFsc measured by actigraphy was used to assess the objective circadian phase. It was calculated using the same formula as that derived from the sleep diary.

Endogenous circadian phase was estimated by DLMO timing. DLMO assessment was conducted in the Sleep Laboratory at the University of Hong Kong. Prior to the lab-based DLMO assessment, participants were asked to continue their habitual rest-wake schedule and complete the sleep diary prospectively for eight days whilst wearing actigraphy. On the day of the DLMO assessment, participants were instructed to arrive at the laboratory 7.5 h before their habitual bedtime. They stayed in a room with an ambient light level below 10 lux as confirmed by a digital light meter throughout the assessment period. Saliva samples were collected 6 h before and 2 h after their habitual bedtime with a 30-min interval sampling rate under dim light circumstances [55]. A total of 17 samples were collected for each participant. An ultra-performance liquid chromatography-tandem mass spectrometry (UPLC-MS/MS) with positive electrospray ionization (Waters Acquity Xevo TQ-XS system, Waters Corporation, Milford, MA, USA) was used to assay the melatonin level of the collected salivary samples. The assay procedure was conducted at the Biomedical Mass Spectrometry Unit of the Department of Chemical Pathology, Prince of Wales Hospital. Salivary samples were diluted with an ascorbic acid solution that contained a deuterium-labeled melatonin internal standard, then centrifuged and injected into the UPLC-MS/MS for chromatographic separation from matrix interference and tandem mass spectrometric quantification. This method achieved a lower limit of quantification of 4.65 pmol/L and had a linear analytical range extending up to 2000 pmol/L. The DLMO clock time was estimated using linear interpolation with a threshold of 12.9 pmol/L (3.5 pg/mL) for at least two additional samples [56].

#### 2.3.5. Mood-related measures

Depressive symptoms were assessed using the Beck Depression Inventory – Short Form (BDI-SF) [57], a 13-item self-report measure with each item rated on a 4-point Likert scale. It is widely used in both clinical practice and research to evaluate the severity of depression. Aggregate scores range from 0 to 16, with higher scores indicating more severe depressive symptoms. In the current sample, BDI-SF demonstrated good internal consistency (Cronbach's $\alpha = .84$).

Suicidality was assessed during the clinical interview using the Suicidality Module of the MINI [45]. This module assesses suicidal thoughts and behaviors over the past month. Based on the participants' responses, their suicidality level was rated as: none, low, moderate, and high.

### 2.4. Statistical analysis

Intraindividual sleep variability was derived from both sleep diary (subjective) and actigraphy (objective) data. We calculated intraindividual sleep variability for the following parameters: TIB, TST, SOL, WASO, SON, SOFF, BT, RT. The root mean squared successive differences (RMSSD) was employed to quantify the variability for each parameter, as indicated in the formula below, where $n$ represents the number of days [38,39,58]. It can detect the changes from one night ($i$) to the next ($i+1$) and be less sensitive to systematic changes (e.g., a gradually delayed bedtime over time), when compared to using the deviations from individuals' mean [59].

$$RMSSD = \sqrt{\frac{\sum_{i=1}^{n-1} (x_{i+1} - x_i)^2}{n-1}}$$

Descriptive statistics were computed for all variables. One-way ANOVA or Chi-square test was used for between-group comparison. For the primary hypotheses, ANCOVA was used to compare sleep variability across the three groups while controlling for mean values of corresponding sleep parameters, as suggested by previous studies [26,30]. Post-hoc tests were conducted with Bonferroni correction to consider the overall rate of false positives of multiple comparisons. For the secondary hypotheses, a series of multiple regression models were used to examine the associations of intrinsic circadian phase and depressive symptoms with sleep variability using the data from the overall sample. The dependent variables included intraindividual variability of each sleep parameter (TIB, TST, SOL, WASO, SON, SOFF, BT, RT), while the independent variables comprised DLMO clock times, BDI-SF scores, and the diagnostic group (DSWPD, ID, Control). Age, sex, ISI scores, and mean values of corresponding sleep parameters were adjusted for in the regression models. All analyses were conducted using

R (version 4.4.3). A p-value <0.05 was considered the threshold for statistical significance.

## 3. Results

### 3.1. Sample characteristics

A total of 113 youths were recruited (DSWPD: n = 34, $M_{age} = 20.8 \pm 1.7$, 70.6% female; ID: n = 40, $M_{age} = 20.3 \pm 2.4$, 70.0% female; Control: n = 39, $M_{age} = 19.7 \pm 2.1$, 66.7% female). Sample characteristics are presented in Table 1. No significant differences in age, sex, and occupational status were found across the groups. The DSWPD group exhibited both a subjectively delayed circadian preference and an objectively delayed biological clock. They had significantly lower MEQ scores, later MSFsc, and later DLMO clock times than the ID group (MEQ: $M_{diff} = 7.96$, $p < .001$; sMSFsc: $M_{diff} = -1.08$, $p < .001$; oMSFsc: $M_{diff} = -1.20$, $p = .002$; DLMO: $M_{diff} = -1.55$, $p < .001$) and the control group (MEQ: $M_{diff} = 10.51$, $p < .001$; sMSFsc: $M_{diff} = -1.29$, $p < .001$; oMSFsc: $M_{diff} = -1.39$, $p < .001$; DLMO: $M_{diff} = -2.10$, $p < .001$). The DSWPD group also had the highest BDI-SF scores (compared to the ID group: $M_{diff} = -3.56$, $p = .017$; compared to the controls: $M_{diff} = -6.45$, $p < .001$), followed by the ID group (compared to the controls: $M_{diff} = -2.88$, $p = .010$) and the control group. Both case groups had higher ISI scores compared to the control group (DSWPD: $M_{diff} = -10.84$, $p < .001$; ID: $M_{diff} = -10.83$, $p < .001$), but there was no significant difference in ISI scores between the DSWPD group and ID group ($M_{diff} = -0.01$, $p > 0.05$).

Subjective and objective sleep parameters were derived from the sleep diary and actigraphy data, respectively. Only valid sleep data recorded for five or more days based on the sleep diary and actigraphy were included in the analysis (sleep diary: n = 113/113, 100.0%, actigraphy: 109/113, 96.5%) [51,60]. Significant between-group differences were found in subjective SOL, WASO, SE, SON, SOFF, BT, RT as measured by sleep diary, and objective SOL, SON, SOFF as measured by actigraphy. Post-hoc tests revealed that DSWPD group showed significant later SOFF and RT compared to ID group (subjective SOFF: $M_{diff} = -1.18$, $p = .003$; objective SOFF: $M_{diff} = -1.41$, $p < .001$; RT: $M_{diff} = -1.35$, $p < .001$) and the control group (subjective SOFF: $M_{diff} = -1.02$, $p = .016$; objective SOFF: $M_{diff} = -1.32$, $p = .002$; RT: $M_{diff} = -1.27$, $p = .003$), and later BT compared to ID group ($M_{diff} = -0.94$, $p = .013$). In terms of insomnia features, both case groups reported significantly longer subjective SOL (DSWPD: $M_{diff} = -26.44$, $p < .001$; ID: $M_{diff} = -29.40$, $p < .001$), longer objective SOL (DSWPD: $M_{diff} = -12.45$, $p = .010$; ID: $M_{diff} = -13.97$, $p = .005$) and lower subjective SE (DSWPD: $M_{diff} = 0.09$, $p < .001$; ID: $M_{diff} = 0.08$, $p < .001$) compared to the control group. DSWPD and ID groups showed comparable subjective and objective SOL, WASO, and SE (*ps* > 0.05). No significant group differences were found in subjective and objective TIB and TST.

### 3.2. Comparison of sleep variability across the groups

Table 2 shows subjectively and objectively measured intraindividual sleep variabilities among groups. Significant differences were observed in subjective variability in TIB, TST, SOL, and BT as measured by sleep diary, objective WASO as measured by actigraphy. DSWPD group showed greater subjective vTIB ($p = .010$), vTST ($p = .013$), and vBT ($p = .006$) compared to ID group, as well as greater subjective vTST ($p = .017$) and objective vWASO ($p = .040$) compared to the control group. Both DSWPD group ($p = .011$) and ID group ($p < .001$) showed greater subjective vSOL compared to the control group.

As most participants in the DSWPD group were university students, we conducted sensitivity analyses by re-running the models excluding secondary students, and the key results remained comparable (Table S1, Supplementary).

**Table 1**
Sample characteristics.

| | *DSWPD (a) n = 34* | *ID (b) n = 40* | *Control (c) n = 39* | *F test/$\chi^2$* | *Post hoc* |
|---|---|---|---|---|---|
| *Demographics* | | | | | |
| Age | 20.76 (1.74) | 20.33 (2.40) | 19.72 (2.08) | 2.72 | – |
| Female, n (%) | 24 (70.6) | 28 (70.0) | 26 (66.7%) | 0.16 | – |
| Occupation, n (%) | | | | 12.48 | – |
| Employed | 1 (2.9) | 7 (17.5) | 3 (7.7) | | |
| Students (postgraduate) | 5 (14.7) | 5 (12.5) | 3 (7.7) | | |
| Students (undergraduate) | 28 (82.4) | 24 (60.0) | 26 (66.7) | | |
| Students (secondary) | 0 (0.0) | 4 (10.0) | 7 (17.9) | | |
| *Psychiatric history,*[a] n (%) | | | | | |
| Depressive disorders | 14 (41.1) | 3 (7.5) | 0 (0.0) | 26.85*** | |
| Anxiety disorders[b]/OCD | 8 (2.4) | 6 (15.0) | 0 (0.0) | 9.56** | |
| *Suicidality* | | | | 18.90** | |
| None | 19 (55.9) | 34 (85.0) | 37 (94.9) | | |
| Low | 11 (32.4) | 4 (10.0) | 2 (5.1) | | |
| Moderate/High | 4 (11.8) | 2 (5.0) | 0 (0.0) | | |
| *Insomnia symptoms*[c] | | | | | |
| DIS | 32 (94.1) | 38 (95.0) | 0 (0.0) | 97.0*** | |
| DMS | 12 (35.3) | 16 (40.0) | 0 (0.0) | 19.8*** | |
| EMA | 8 (23.5) | 12 (30.0) | 0 (0.0) | 20.8*** | |
| *Insomnia severity* | | | | | |
| ISI score | 14.74 (4.43) | 14.73 (3.97) | 3.90 (3.08) | 121.83*** | a>c, b > c |
| *Mood symptoms* | | | | | |
| BDI-SF score | 11.09 (5.99) | 7.53 (4.54) | 4.64 (4.02) | 14.70*** | a>b > c |
| *Circadian measures* | | | | | |
| MEQ score | 34.62 (5.99) | 42.58 (8.21) | 45.13 (10.51) | 19.34*** | b>a, c>a |
| sMSFsc, hh:mm | 06:46 (01:25) | 05:42 (01:27) | 05:29 (01:25) | 8.38*** | a>b, a>c |
| oMSFsc, hh:mm | 06:54 (01:28) | 05:42 (01:23) | 05:31 (01:29) | 8.78*** | a>b, a>c |
| Time of DLMO, hh:mm | 00:43 (01:29) | 23:10 (01:41) | 22:37 (01:28) | 18.16*** | a>b, a>c |
| *Subjective sleep parameters as measured by sleep diary* | | | | | |
| TIB, min | 480.91 (56.38) | 482.74 (52.49) | 468.75 (59.25) | 0.68 | – |
| TST, min | 404.00 (58.79) | 410.37 (61.02) | 436.47 (56.58) | 3.32* | – |
| SOL, min | 36.75 (21.91) | 39.70 (34.45) | 10.31 (8.43) | 32.66*** | a>c, b > c |
| WASO, min | 6.91 (12.23) | 9.97 (12.04) | 3.50 (7.41) | 4.34* | b > c |
| SE, % | 83.94 (7.12) | 85.00 (10.06) | 93.05 (4.52) | 25.87*** | c>a, c > b |
| SON, hh:mm | 03:20 (01:14) | 02:01 (01:10) | 01:50 (01:23) | 15.13*** | a>b, a>c |
| SOFF, hh:mm | 10:11 (01:32) | 09:01 (01:20) | 09:10 (01:31) | 6.51** | a>b, a>c |
| BT, hh:mm | 01:49 (01:31) | 00:52 (01:11) | 01:16 (01:25) | 4.32* | a>b |
| RT, hh:mm | 10:44 (01:34) | 09:23 (01:16) | 09:28 (01:33) | 8.95*** | a>b, a>c |
| *Objective sleep parameters as measured by actigraphy* | | | | | |
| TIB, min | 433.47 (55.71) | 433.06 (54.07) | 433.07 (49.62) | 0.00 | – |
| TST, min | 405.86 (52.43) | 400.20 (51.32) | 403.92 (51.18) | 0.11 | – |
| SOL, min | 26.43 (20.11) | 27.96 (24.05) | 13.98 (11.22) | 8.48*** | a>c, b > c |

(continued on next page)

**Table 1** (*continued*)

| | *DSWPD (a) n = 34* | *ID (b) n = 40* | *Control (c) n = 39* | *F test/$\chi^2$* | *Post hoc* |
|---|---|---|---|---|---|
| WASO, min | 26.51 (9.82) | 29.76 (16.92) | 26.47 (12.03) | 0.60 | – |
| SE, % | 84.32 (4.47) | 84.07 (6.85) | 87.03 (4.96) | 3.65* | – |
| SON, hh:mm | 03:13 (01:17) | 01:49 (01:11) | 01:55 (01:26) | 12.51*** | a>b, a>c |
| SOFF, hh:mm | 10:27 (01:36) | 09:02 (01:13) | 09:08 (01:28) | 8.99*** | a>b, a>c |

*Notes.* Data were presented as means (SD) for variables that were not specified. [a] Psychiatric history included both current diagnosis or past history of psychiatric disorders. [b] Anxiety disorders included generalized anxiety disorder, panic disorder, social anxiety disorder, specific phobias, and separation anxiety disorder. [c] Presence of specific insomnia symptom for at least three nights per week in the past three months. *$p < .05$, **$p < .01$, ***$p < .001$.
*Abbreviations*. DSWPD = delayed sleep-wake phase disorder; ID = insomnia disorder. DIS = difficulty initiating sleep; DMS = difficulty maintaining sleep; EMA = early morning awakening. OCD = obsessive compulsive disorder. sMSFsc = midpoint of sleep on free days corrected for sleep debt on work days as measured by sleep diary; oMSFsc = midpoint of sleep on free days corrected for sleep debt on work days as measured by actigraphy. TIB = time in bed; TST = total sleep time; SOL = sleep onset latency; WASO = wake after sleep onset; SE = sleep efficiency; SON = sleep onset; SOFF = sleep offset; BT = bedtime; RT = risetime.

### *3.3. Sleep variability in relation to circadian measures and depressive symptoms across the full sample*

The results of multiple regressions are summarized in Table 3. Results of regressions were controlled for age, sex, ISI scores and respective mean of sleep parameters. Later DLMO timing was significantly associated with greater subjective vTIB ($p < .001$), vTST ($p < .001$), and vSOFF ($p = .007$), as well as greater objective vTIB ($p = .001$) and vTST ($p = .006$). Higher levels of depressive symptoms were significantly associated with greater subjective vSOFF ($p = .009$), and vRT ($p = .003$), as well as greater objective vSOL ($p = .031$), vWASO ($p = .015$), and vSOFF ($p = .039$). Diagnostic group (with the control group as the reference) was not associated with sleep variability, except for variability in subjective vSOL (DSWPD vs control, $p = .027$).

## 4. Discussion

The present study compared sleep variability in youths with DSWPD, youths with insomnia disorder and their healthy counterparts. As a secondary exploration, it also tested the associations of sleep variability with an intrinsic circadian phase and depressive symptoms across the full sample. Relative to youths with insomnia disorder, those with DSWPD showed greater variability in self-reported BT, TIB, and TST. Compared with healthy sleepers, the DSWPD group also demonstrated greater variability in self-reported SOL and actigraphy-derived WASO compared to healthy sleepers. Youths with insomnia disorder showed greater variability in self-reported SOL compared to healthy sleepers. Greater sleep variability was associated with a more delayed circadian rhythm as reflected by later DLMO timing and with higher levels of depressive symptoms, after controlling for insomnia symptoms and irrespective of diagnostic group.

### *4.1. Sleep variability in DSWPD*

The greater variability in diary-derived TST, SOL, and actigraphy-derived WASO observed in youths with DSWPD compared to healthy controls was consistent with previous studies based on both youth and adult samples. For example, previous research has consistently shown that individuals with DSWPD exhibit greater TST variability than controls, whether measured by sleep diaries or wrist actigraphy, in youths [61] and adults [38]. It is also plausible that these youths had difficulty in identifying their optimal sleep times. This was evident by our findings of greater variability in BT and RT among youths with DSWPD compared to those with insomnia and healthy sleepers, respectively. Youths with DSWPD may struggle to perceive sleepiness around sleep onset [62], or they may delay their sleep by engaging in other activities [63]. The use of digital devices, a common activity before bedtime in young people, has been found to be more prevalent among those with eveningness and is associated with adverse effects on sleep and mental health [64]. On the other hand, the variabilities in BT and TIB in youths with insomnia were lower than those of youths with DSWPD. These differences in BT and TIB may reflect a more consistent approach to sleep schedule for those with insomnia. Despite these behavioral differences, both groups showed higher SOL variability compared to controls but there was no significant difference between the two clinical groups. Increased sleep variability among individuals with DSWPD has been linked to poorer daytime functioning and greater sleep-related impairment [61], suggesting the need for enhanced clinical attention. Although

**Table 2**
Comparisons of subjectively and objectively measured sleep variability.

| | *DSWPD (a)* | *ID (b)* | *Control (c)* | *F/$\chi^2$* | *Post hoc* |
|---|---|---|---|---|---|
| *Intraindividual sleep variability of subjective parameters* | | | | | |
| vTIB | 148.08 (86.11) | 95.79 (38.33) | 110.65 (62.82) | 4.92** | a>b |
| vTST | 147.12 (65.97) | 105.14 (41.99) | 111.65 (59.10) | 5.36** | a>b, a>c |
| vSOL | 35.68 (32.36) | 28.17 (27.03) | 8.77 (11.72) | 10.27*** | a>c, b > c |
| vWASO | 16.31 (36.41) | 14.86 (17.55) | 6.41 (11.23) | 2.10 | – |
| vSON | 2.12 (1.77) | 1.22 (0.94) | 1.35 (0.71) | 1.64 | – |
| vSOFF | 2.21 (1.37) | 1.74 (0.97) | 1.58 (0.75) | 1.68 | – |
| vBT | 2.10 (1.58) | 1.08 (0.84) | 1.36 (0.73) | 5.06** | a>b |
| vRT | 2.24 (1.32) | 1.76 (0.90) | 1.59 (0.89) | 1.26 | – |
| *Intraindividual sleep variability of objective parameters* | | | | | |
| vTIB | 145.92 (81.25) | 111.55 (46.89) | 115.65 (66.37) | 2.63 | – |
| vTST | 138.93 (76.31) | 103.05 (44.03) | 115.40 (64.83) | 2.57 | – |
| vSOL | 33.08 (23.14) | 35.49 (35.70) | 17.57 (17.15) | 2.36 | – |
| vWASO | 21.09 (17.14) | 20.94 (17.81) | 14.34 (8.61) | 3.30* | a>c |
| vSON | 2.00 (1.53) | 1.29 (0.91) | 1.37 (0.73) | 1.10 | – |
| vSOFF | 2.16 (1.16) | 1.80 (0.89) | 1.56 (0.91) | 1.54 | – |

*Notes.* Data were presented as means (SD). Variability of sleep parameters (RMSSD) were ln(x+1) transformed before comparisons. Descriptive tables showed values before transformation. Covariates: respective mean. *$p < .05$, **$p < .01$, ***$p < .001$.
*Abbreviations*. DSWPD = delayed sleep-wake phase disorder; ID = insomnia disorder. vTIB = variability in time in bed; vTST = variability in total sleep time; vSOL = variability in sleep onset latency; vWASO = variability in wake after sleep onset; vSON = variability in sleep onset; vSOFF = variability in sleep offset; vBT = variability in bedtime; vRT = variability in risetime.

**Table 3**
Regression analysis of the association of circadian measures, depressive symptoms, and clinical status with intraindividual sleep variability.

| | Unstandardized | | Standardized | | |
|---|---|---|---|---|---|
| | *b* | *SE* | *β* | *t* | *p* |
| *Intraindividual sleep variability of subjective parameters* | | | | | |
| **vTIB** | | | | | |
| Group | | | | | |
| DSWPD-C | 0.02 | 0.18 | 0.03 | 0.09 | 0.932 |
| ID-C | −0.07 | 0.17 | −0.14 | −0.43 | 0.670 |
| DLMO | 0.13 | 0.03 | 0.42 | 4.01 | **<0.001** |
| BDI | 0.02 | 0.01 | 0.18 | 1.73 | 0.086 |
| **vTST** | | | | | |
| Group | | | | | |
| DSWPD-C | 0.08 | 0.17 | 0.16 | 0.46 | 0.644 |
| ID-C | −0.05 | 0.16 | −0.10 | −0.30 | 0.761 |
| DLMO | 0.11 | 0.03 | 0.39 | 3.64 | **<0.001** |
| BDI | 0.01 | 0.01 | 0.06 | 0.51 | 0.606 |
| **vSOL** | | | | | |
| Group | | | | | |
| DSWPD-C | 0.69 | 0.31 | 0.59 | 2.24 | **0.027** |
| ID-C | 0.33 | 0.29 | 0.28 | 1.14 | 0.256 |
| DLMO | 0.02 | 0.05 | 0.03 | 0.42 | 0.219 |
| BDI | −0.02 | 0.02 | −0.10 | −1.23 | 0.227 |
| **vWASO** | | | | | |
| Group | | | | | |
| DSWPD-C | 0.36 | 0.30 | 0.31 | 1.19 | 0.236 |
| ID-C | 0.51 | 0.28 | 0.44 | 1.84 | 0.442 |
| DLMO | −0.01 | 0.05 | −0.01 | −0.11 | 0.142 |
| BDI | 0.02 | 0.02 | 0.07 | 0.93 | 0.226 |
| **vSON** | | | | | |
| Group | | | | | |
| DSWPD-C | −0.11 | 0.13 | −0.29 | −0.84 | 0.402 |
| ID-C | −0.20 | 0.12 | −0.54 | −1.71 | 0.091 |
| DLMO | 0.00 | 0.03 | 0.00 | 0.03 | 0.980 |
| BDI | 0.00 | 0.01 | 0.04 | 0.40 | 0.690 |
| **vSOFF** | | | | | |
| Group | | | | | |
| DSWPD-C | −0.14 | 0.10 | −0.47 | −1.39 | 0.169 |
| ID-C | −0.09 | 0.10 | −0.28 | −0.89 | 0.376 |
| DLMO | 0.08 | 0.03 | 0.44 | 2.73 | **0.007** |
| BDI | 0.02 | 0.01 | 0.27 | 2.67 | **0.009** |
| **vBT** | | | | | |
| Group | | | | | |
| DSWPD-C | 0.09 | 0.13 | 0.25 | 0.69 | 0.491 |
| ID-C | −0.09 | 0.12 | −0.24 | −0.73 | 0.468 |
| DLMO | 0.05 | 0.03 | 0.24 | 1.74 | 0.085 |
| BDI | 0.00 | 0.01 | 0.04 | 0.34 | 0.735 |
| **vRT** | | | | | |
| Group | | | | | |
| DSWPD-C | −0.12 | 0.11 | −0.36 | −1.06 | 0.291 |
| ID-C | −0.05 | 0.10 | −0.16 | −0.51 | 0.613 |
| DLMO | 0.06 | 0.03 | 0.31 | 1.88 | 0.062 |
| BDI | 0.02 | 0.01 | 0.31 | 3.01 | **0.003** |
| *Intraindividual sleep variability of objective parameters* | | | | | |
| **vTIB** | | | | | |
| Group | | | | | |
| DSWPD-C | 0.12 | 0.19 | 0.22 | 0.61 | 0.546 |
| ID-C | 0.09 | 0.17 | 0.17 | 0.49 | 0.624 |
| DLMO | 0.11 | 0.03 | 0.36 | 3.32 | **0.001** |
| BDI | 0.01 | 0.01 | 0.07 | 0.64 | 0.523 |
| **vTST** | | | | | |
| Group | | | | | |
| DSWPD-C | 0.13 | 0.19 | 0.26 | 0.69 | 0.494 |
| ID-C | 0.05 | 0.17 | 0.10 | 0.30 | 0.582 |
| DLMO | 0.09 | 0.03 | 0.31 | 2.82 | **0.006** |
| BDI | 0.01 | 0.01 | 0.07 | 0.59 | 0.559 |
| **vSOL** | | | | | |
| Group | | | | | |
| DSWPD-C | 0.05 | 0.20 | 0.06 | 0.24 | 0.812 |
| ID-C | 0.04 | 0.18 | 0.05 | 0.22 | 0.823 |
| DLMO | 0.05 | 0.03 | 0.11 | 1.53 | 0.128 |
| BDI | −0.02 | 0.01 | −0.16 | −2.19 | **0.031** |
| **vWASO** | | | | | |
| Group | | | | | |
| DSWPD-C | −0.01 | 0.18 | −0.02 | −0.07 | 0.948 |
| ID-C | −0.01 | 0.16 | −0.01 | −0.04 | 0.972 |
| DLMO | 0.00 | 0.03 | 0.01 | 0.12 | 0.901 |
| BDI | 0.03 | 0.01 | 0.23 | 2.49 | **0.015** |
| **vSON** | | | | | |
| Group | | | | | |
| DSWPD-C | −0.09 | 0.12 | −0.28 | −0.77 | 0.442 |
| ID-C | −0.16 | 0.11 | −0.48 | −1.44 | 0.181 |
| DLMO | 0.00 | 0.03 | 0.00 | −0.03 | 0.307 |
| BDI | 0.00 | 0.01 | 0.00 | −0.01 | 0.216 |
| **vSOFF** | | | | | |
| Group | | | | | |
| DSWPD-C | −0.10 | 0.11 | −0.31 | −0.90 | 0.370 |
| ID-C | −0.01 | 0.10 | −0.04 | −0.12 | 0.904 |
| DLMO | 0.04 | 0.03 | 0.23 | 1.49 | 0.139 |
| BDI | 0.01 | 0.01 | 0.18 | 2.10 | **0.039** |

*Notes*. Regression models adjusted for age, sex, Insomnia Severity Index (ISI) scores and respective mean of sleep parameters.
*Abbreviations*. DSWPD = delayed sleep-wake phase disorder; ID = insomnia disorder; C = control. vTIB = variability in time in bed; vTST = variability in total sleep time; vSOL = variability in sleep onset latency; vWASO = variability in wake after sleep onset; vSON = variability in sleep onset; vSOFF = variability in sleep offset; vBT = variability in bedtime; vRT = variability in risetime. DLMO = dim light melatonin onset; BDI = Beck Depression Inventory – Short Form.

circadian-based treatments, such as timed melatonin administration [65] and bright light therapy [65,66], have demonstrated efficacy in phase advancing circadian rhythm, there remained limited research to examine their effects on sleep variability outcomes.

### *4.2. Sleep variability in insomnia*

Aligned with previous studies in adults [35,39,67], not only longer SOL but also greater sleep variability in SOL were observed in youths with insomnia disorder compared to healthy controls. However, no significant differences in other sleep variability parameters were found between youths with and without insomnia. Previous evidence regarding variability in other sleep parameters, such as TST and WASO, has been less consistent in adults with insomnia. For example, two studies conducted in older adults found greater variability in TST, SOL, and WASO in those with insomnia compared to controls [35,39], while another study conducted in midlife women found increased SOL variability in individuals with insomnia compared to those without insomnia [67]. The higher SOL variability observed in youths with insomnia may reflect their difficulties in achieving consistent timing in falling asleep. In addition, this variability may be associated with their perceptions of poor sleep quality and sleep loss [31,68]. While there have been limited experimental studies to explore the causal relationship between insomnia and sleep variability, behavioral treatments targeting insomnia have demonstrated associations between reductions in sleep variability and insomnia remission. For example, cognitive behavioral therapy for insomnia (CBT-I), the first-line treatment for insomnia, has been shown to effectively reduce sleep variability across various sleep variables as secondary outcomes (e.g., TST, SOL, WASO, BT, and RT) [28,69,70]. Further research is necessary to elucidate the causal mechanisms underlying the relationship between insomnia and sleep variability, as well as to test whether improving sleep stability could improve both insomnia and mood-related outcomes in youths.

### *4.3. Mood and circadian outcomes linked to sleep variability*

The association between intrinsic circadian phase and sleep variability appeared transdiagnostic: across the full sample, greater variability in diary- and actigraphy-derived TIB and TST, as well as diary-derived SOFF, were found to be associated with a delayed DLMO timing. This finding was consistent with previous studies that relied on subjective circadian measures. For example, a study conducted in first-year college students found that a later chronotype (indicated by a lower MEQ score) correlated with greater variability in TST [41]. Another intervention study reported that increased evening preference, as measured by Morningness-Eveningness Composite Scale (MECS), was associated with greater variability in BT and TIB in adults with insomnia at baseline [28]. Additionally, genetic evidence supports that loci associated with chronotype affect sleep timing but not sleep quality or duration [71]. The current results further supported the relationship between sleep variability and circadian rhythm timing by using DLMO assessment, which is considered the gold standard for determining circadian phase [72]. However, further longitudinal studies are needed to clarify the directionality of this relationship (i.e., whether a delayed circadian phase at baseline predicts greater sleep variability at follow-up or vice versa).

Across the full sample, more severe depressive symptoms were associated with greater variability in sleep schedule (TIB, SOFF, RT) and in sleep parameters associated with insomnia symptoms (SOL, WASO). This observation was consistent with that of prior cross-sectional research conducted in adults [28,31,35]. Several factors may potentially contribute to this observed association. For example, irregular sleep patterns have been found to be linked to insomnia symptoms and stressful events (e.g., exams and heavy workload) [27,73], which are often accompanied by mood disturbances [73]. Longitudinal studies found that variability in total sleep time, wake time, and sleep timing prospectively predicted higher levels of depressive symptoms across diverse populations (e.g. adolescents, young adults, college students [29,32–34]), while the evidence supporting the reverse direction, i.e., depressive symptoms leading to greater sleep variability, has been less consistent [29,74]. An experimental study has further demonstrated that experimentally induced irregular sleep schedules over seven nights (time in bed alternating between 6 h and 9 h for the experimental group vs. fixed time in bed for 7.5 h in the control group) resulted in more negative mood in the evenings in the experimental group [75]. Future longitudinal studies may consider testing the directional nature of the relationship between depressive symptoms and sleep variability. In addition, investigating whether reducing sleep variability can alleviate depression or if improving mood decreases sleep variability may provide valuable insights for clinical practice and inform treatment strategies.

### 4.4. Study strengths and limitations

To the best of our knowledge, the present study was the first study to compare sleep variability in youths with DSWPD and insomnia disorder. The findings may enhance our understanding of sleep patterns and circadian features associated with DSWPD and insomnia disorder in youths. The present study utilized DLMO, a gold-standard marker of the central circadian clock [72], to objectively measure circadian rhythms. However, the following limitations should be considered when interpreting the results of this study. First, the current sample consisted of more late adolescents, with most of them being university students. Although the sensitivity analyses did not reveal any substantial differences, secondary school students or younger adolescents may face challenges distinct from those experienced by college or university students. Therefore, caution should be exercised when generalizing these findings to younger populations. Second, although significant group differences were found for anxiety disorders, we could not include this factor in the models due to the limited number of cases. Future studies should consider examining the effects of anxiety symptoms/disorders. Third, some discrepancies in the findings of sleep variability based on sleep diary vs. actigraphy were noted. Nonetheless, the trends of the differences were consistent based on these two measures (e.g., highest variability in DSWPD group, lower variability in ID group or control group). Future research could incorporate additional objective sleep measures (e.g., under-mattress sleep sensors [76]), and further explore concordance between measures, as well as exploring sleep misperception in relation to sleep variability in DSWPD and ID. Lastly, the present study used a cross-sectional design and could not draw any bidirectional or causal conclusions. Future studies may consider using a longitudinal or experimental design to further explore the causal relationship between sleep variability and its correlates.

### 4.5. Conclusion and clinical implications

The present study showed greater sleep variability in youths with DSWPD and insomnia disorder. These findings highlight the importance of considering sleep variability for assessing and managing sleep problems in young people, emphasizing the need to consider circadian factors and/or mood symptoms when treating youths with DSWPD or a delayed intrinsic circadian phase. Furthermore, the observed transdiagnostic link between greater sleep variability and a higher level of depressive symptoms suggested the need to address sleep variability in the context of mood symptoms in young people.

## CRediT authorship contribution statement

**Yue Pan:** Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Forrest Tin Wai Cheung:** Writing – review & editing, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Joey Wing-Yan Chan:** Writing – review & editing, Methodology, Conceptualization. **Ngan Yin Chan:** Writing – review & editing, Methodology, Conceptualization. **Xiao Li:** Writing – review & editing, Methodology, Conceptualization. **Amy Wing Yin Ho:** Writing – review & editing, Methodology, Conceptualization. **Chung Shun Ho:** Writing – review & editing, Methodology, Conceptualization. **Michael Gradisar:** Writing – review & editing, Methodology, Conceptualization. **Yun-Kwok Wing:** Writing – review & editing, Methodology. **Shirley Xin Li:** Writing – review & editing, Validation, Supervision, Resources, Methodology, Investigation, Funding acquisition, Conceptualization.

## Data availability statement

The data that support the findings of this study are available from the corresponding author, upon reasonable request and study protocol. The data are not publicly available due to privacy or ethical restrictions.

## Disclosure statement

YKW received personal fees from Eisai Co. For lectures and travel support from Lundbeck HK Limited and Aculys Pharma, Japan. JWYC received personal fees from Eisai Co. MG received consulting fees from Sleep Cycle AB, Sweden, travel support from the Association for Sleep Disorders in Children and Adolescents, and is the CEO of WINK Sleep Pty Ltd. All other authors declare no financial or non-financial competing interests.

## Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Shirley Xin Li reports financial support was provided by University Grants Committee Research Grants Council. Yun Kwok Wing reports a relationship with Eisai Co Ltd that includes: speaking and lecture fees. Yun Kwok Wing reports a relationship with Lundbeck HK Limited that includes: travel reimbursement. Yun Kwok Wing reports a relationship with Aculys Pharma, Japan that includes: travel reimbursement. Joey Wing-Yan Chan reports a relationship with Eisai Co Ltd that includes: travel reimbursement. Michael Gradisar reports a relationship with Sleep Cycle AB, Sweden that includes: consulting or advisory. Michael

Gradisar reports a relationship with Association for Sleep Disorders in Children and Adolescents that includes: travel reimbursement. Michael Gradisar reports a relationship with WINK Sleep Pty Ltd that includes: employment. Given her role as the Field Editor, Joey Wing-Yan Chan had no involvement in the peer review of this article and had no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to another journal editor. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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## Appendix A. Supplementary data

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