The Journal of Child Psychology and Psychiatry

*Journal of Child Psychology and Psychiatry* 61:10 (2020), pp 1116–1125 doi:10.1111/jcpp.13234

# Within-person fluctuations in stressful life events, sleep, and anxiety and depression symptoms during adolescence: a multiwave prospective study

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**Background:** Adolescence is characterized by substantial changes in sleep behavior, heightened exposure to stressful life events (SLEs), and elevated risk for internalizing problems like anxiety and depression. Although SLEs are consistently associated with the onset of internalizing psychopathology, the mechanisms underlying this relationship remain poorly understood, especially at the within-person level. Here, we leverage a high-frequency longitudinal design to examine sleep as a potential mechanism linking SLEs to increases in anxiety and depression symptoms over a one-year period. **Methods:** Thirty female adolescents aged 15–17 years completed 12 monthly in-laboratory assessments of exposure to SLEs and symptoms of anxiety and depression ($n$ = 355 monthly assessments), and wore an actigraphy wristband for continuous monitoring of sleep for the duration of the study ($n$ = 6,824 sleep days). Multilevel models examined concurrent and lagged within-person associations between SLEs, sleep duration and timing regularity, and anxiety and depression symptoms. **Results:** Within-person fluctuations in SLEs were associated with variability in sleep duration both concurrently and prospectively, such that when adolescents experienced greater SLEs than was typical for them, they exhibited more variable sleep duration that same month as well as the following month. In turn, within-person increases in sleep duration variability predicted greater anxiety symptoms in the same month and mediated the association between SLEs and anxiety. **Conclusions:** These findings highlight sleep disruptions as a mechanism underlying the longitudinal associations between SLEs and anxiety symptoms, and suggest that interventions promoting sleep schedule consistency may help mitigate risk for stress-related psychopathology in adolescence. **Keywords:** Adolescence; stress; sleep; actigraphy; depression; anxiety; longitudinal.

## Introduction

Adolescence is characterized by increased vulnerability for the emergence of anxiety and depression (Kessler et al., 2005; Paus, Keshavan, & Giedd, 2008). Stressful life events (SLEs) predict the onset of internalizing symptoms (Hammen, 2005; Kendler, Hettema, Butera, Gardner, & Prescott, 2003; McLaughlin et al., 2012), and this link is particularly strong during adolescence (Grant et al., 2003; Larson & Ham, 1993), including at the within-person level (Jenness, Peverill, King, Hankin, & McLaughlin, 2019). However, remarkably little is known about the mechanisms that underlie this relationship. In the current intensive longitudinal study, we examine sleep behavior as a potential mechanism underlying the association between exposure to SLEs and symptoms of anxiety and depression in adolescence.

Sleep plays a critical role in supporting cognitive functioning, including attention, memory consolidation, and emotion regulation (Killgore, 2010), yet most adolescents sleep less than the 8–10 hr per night recommended for this developmental stage (Wheaton, Jones, Cooper, & Croft, 2018). Puberty-driven changes in circadian and homeostatic systems, decreased parental supervision, and increased academic and social demands all gradually shift adolescents to later bedtimes, while early school times restrict adolescents' sleep on weekdays (Hummer & Lee, 2016). Importantly, heightened exposure and vulnerability to stressors can further disrupt adolescent sleep. SLEs often trigger worry and rumination, resulting in trouble initiating and maintaining sleep (Kim & Dimsdale, 2007; Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013; Sadeh & Gruber, 2002). Although most research examining SLEs and sleep has been conducted in adults, several studies in college students have linked stress with shorter and more variable sleep (Bernert, Merrill, Braithwaite, Van Orden, & Joiner, 2007; Doane & Thurston, 2014; Veeramachaneni, Slavish, Dietch, Kelly, & Taylor, 2019), suggesting that these associations also exist in youth.

Sleep disturbances have been consistently associated with internalizing psychopathology (Murphy & Peterson, 2015; Weiner, Elkins, Pincus, & Comer, 2015). Short sleep duration and insomnia are common among youth with anxiety and depression (Weiner et al., 2015; Zhang et al., 2017), and work with adults has found an association between irregular sleep schedules (e.g., due to shift work and time zone changes) and mood disorders (McClung, 2013),

Conflict of interest statement: No conflicts declared.


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suggesting that both sleep duration and sleep timing can contribute to internalizing problems (Czeisler, 2015). An emergent body of longitudinal research with adolescents suggests that sleep-related problems are both predicted by and predict increases in anxiety and depression symptoms months or years later (Alvaro, Roberts, Harris, & Bruni, 2017; Shanahan, Copeland, Angold, Bondy, & Costello, 2014; Wilson, Carpenter, & Hickie, 2019). However, these bidirectional associations have not always been found, with some studies reporting that short sleep duration predicted increased depression and anxiety symptoms a year later, but not vice versa (Conklin, Yao, & Richardson, 2018; Roberts & Duong, 2017).

Despite growing insights into the associations between SLEs, sleep, and internalizing symptoms, existing research has several limitations. Most studies rely on self-reported sleep behavior, which is vulnerable to recall biases (Biddle, Robillard, Hermens, Hickie, & Glozier, 2015; Silva et al., 2007). Actigraphy, in the form of wristbands and other wearables, is a promising tool to measure sleep naturalistically overtime, but few studies have used this approach in youth, and typically for very short periods. Furthermore, most studies have focused on clinical diagnoses, but continuous metrics of sleep behavior and internalizing symptoms allow for finer-grained investigation of their fluctuations and associations, even in the absence of a diagnosable disorder (Markon, Chmielewski, & Miller, 2011). Finally, prior work has examined how short-term averages of sleep behavior relate to internalizing disorders months or years later, thus missing the intraindividual variability in these metrics over days, weeks, and months (Becker, Sidol, Van Dyk, Epstein, & Beebe, 2017). Longitudinal research examining within-person fluctuations in SLEs, sleep, and internalizing symptoms is clearly needed.

The present study used an intensive longitudinal approach and leveraged technological advances in objective measurement of sleep to: (a) characterize within-person fluctuations in exposure to SLEs, sleep behaviors, and internalizing symptoms; (b) examine the bidirectional associations between sleep and internalizing symptoms over time; and (c) explore the role of sleep as a mechanism underlying the association between SLEs and internalizing symptoms. We hypothesized that within-person increases in SLEs would predict decreases in sleep duration and sleep timing regularity, and that these changes in sleep would, in turn, predict increases in anxiety and depression and mediate the relationship between SLEs and internalizing symptoms.

## Methods

### Participants

Thirty adolescent females aged 15–17 years ($M$ = 16.36; $SD$ = 0.75) completed 12 monthly study visits ($n$ = 355 out of 360 possible assessments; 99% retention rate). Because our focus was on within-person effects, we chose to reduce interindividual variability in sex and age. Females in this age range were chosen given their elevated risk for stress-related internalizing symptoms (Leadbeater, Blatt, & Quinlan, 1995). Participants were recruited from school-related and public spaces in Seattle, WA, between 2016 and 2018. A total of 60% of the sample met criteria for a depressive or anxiety disorder in their lifetime, and 40% within the past year, as assessed by the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS) interview (Kaufman et al., 1997) administered at the first and last study visits. Written informed consent was obtained from legal guardians, and children provided written assent. Study procedures were approved by the Institutional Review Board at the University of Washington. See Appendix S1 for more details.

### Measures

*Exposure to stressors.* Stressful life events occurring in the past month were assessed at each study visit using the UCLA Life Stress Interview (Hammen, 1988), a semistructured interview designed to objectively measure the impact of life events. Structured prompts query numerous life domains, including peer relationships, academics, and health. Research staff coded the severity of each experience for a child of that age and sex on a scale from 1 (none) to 5 (extremely severe), including half-points (transformed to a 0–8 integer scale for analyses). Following prior work, a monthly total stress score was computed by summing severity scores across all reported events (Hammen, Henry, & Daley, 2000). This interview has been extensively validated, adapted for adolescents, and considered the gold standard for assessing SLEs (Daley et al., 1997). See Appendix S1 for more details.

### Anxiety

Anxiety symptoms were measured monthly with the Generalized Anxiety Disorder 7-item scale (GAD-7), which queries symptoms occurring in the last two weeks. Items are scored on a Likert scale ranging from 0 to 3 and summed to create a symptom severity score. The GAD-7 has good reliability and validity (Spitzer, Kroenke, Williams, & Löwe, 2006), and had good internal consistency across all months in the current study ($\alpha$ = .80–.90).

### Depression

Depression symptoms were measured monthly with the Patient Health Questionnaire 9-item scale (PHQ-9), which assesses depression symptoms occurring in the last two weeks. Items are scored on a Likert scale ranging from 0 to 3 and summed to create a symptom severity score. The PHQ-9 has good reliability and validity (Kroenke, Spitzer, & Williams, 2001), and had good internal consistency across all months in the current study ($\alpha$ = .76–.90).

### Sleep

We monitored sleep passively via an actigraphy wristband worn continuously for the duration of the study. The original device (Microsoft Band 2) was discontinued early in the study, after which we switched all participants to a Fitbit Charge 2. Sleep onset and offset estimates were highly similar between the two devices (Cronbach's $\alpha$ = .80), and data from both devices were used for the present analyses (see Appendix S1 for details). The wristbands used accelerometer data collected in 1-minute epochs and proprietary algorithms to detect sleep and awake states. No changes were made to individual records nor the devices' default algorithms for the detection of sleep. These devices have been validated in healthy adults against polysomnography and EEG, with excellent sensitivity (i.e., ability to detect true sleep) and adequate specificity (i.e., ability

to detect true wake; Liang, Alberto, & Martell, 2018; de Zambotti, Goldstone, Claudatos, Colrain, & Baker, 2018). We collected a total of 6,824 daily sleep observations (see Appendix S1 and Figures S1 and S2 for information on missing data).

*Sleep duration.* We computed daily sleep duration in hours, aggregating over potentially multiple sleep events in the same day (e.g., naps, fragmented night sleep), and not including awake time between sleep events.

*Regularity of sleep timing.* We used minute-level actigraphy data to compute a modified version of the Sleep Regularity Index developed by (Phillips et al., 2017). For each study day *i*, we computed the percentage of total minutes in the day at which each participant was in the same state (asleep vs. awake) as in their average sleep day. An individual who sleeps and wakes up at exactly the same time on both day *i* and on their average sleep day would get a score of 100 for day *i*. See Appendix S1 for more details.

### Statistical analysis

*Actigraphy data processing.* Daily sleep metrics were computed over 24 hr defined from 7 pm to 7 pm and were then aggregated into concurrent, previous, and subsequent sleep relative to the monthly SLEs, anxiety, and depression assessments. Concurrent sleep corresponded to the sleep data in the two weeks prior to the monthly visit for internalizing symptoms and to the full month prior to the monthly visit for SLEs (i.e., the same time period for which symptoms/life events were assessed). For all assessments, previous sleep corresponded to the two weeks prior to the period of concurrent sleep, and subsequent sleep to the two weeks following the monthly visit. For each period, we computed the mean and standard deviation of sleep duration and sleep timing regularity, and used both summary metrics for analysis.

*Power.* Power analyses indicated our design yields greater than 80% statistical power to detect small effects at the within-person level (see Appendix S2 and Figure S3 for details).

*Statistical modeling.* We estimated models to test the following within-person associations over the twelve study timepoints: (a) SLEs and symptoms of anxiety and depression, (b) SLEs and sleep, and (c) and sleep and internalizing symptoms. Finally, we performed a mediation analysis to evaluate whether fluctuations in sleep behavior were a mechanism prospectively linking SLEs with internalizing symptoms.

We conducted Bayesian hierarchical (i.e., multilevel) models specifying a random effect of intercept per subject to account for repeated sampling. To isolate within-person associations, we used a standard centering approach that orthogonalizes variation in a given predictor into between- and within-person variability (Enders & Tofighi, 2007), correcting for the dependent nature of the data within-subjects, and controlling for trait-level characteristics of each predictor. We computed both concurrent and lagged models to assess within-person associations in the same time period and the subsequent time period. In models where sleep was used as predictor, both the mean and standard deviation were used together in the same model. Month of observation (i.e., time) and proportion of school days in the tested time period were included as nuisance covariates. Actigraphy device and lifetime diagnosis of anxiety or depression were examined as a potential moderator of these paths, and no significant interactions emerged (see Appendix S2). Highest posterior density 95% credible intervals (CR) were calculated for all parameters. Parameter estimates were interpreted as statistically significant if the CR did not contain zero. Models were estimated in R 3.6.1 (R Core Team, 2019) using the *Stan* modeling language (Stan Development Team, 2018) and the packages *brms* (v2.10.0; Bürkner, 2017) and *sjstats* (v0.17.6; Lüdecke, 2019). See Appendix S2 and Table S2 for more information on model specification.

Within-person mediation models (i.e., a level 1-1-1 mediation) were estimated when we found significant associations between predictor and putative mediator, and between mediator and outcome, by combining coefficients from two separate Bayesian hierarchical models. The first model, from predictor to mediator, yielded an estimate of the coefficient for the *proximal indirect path* (*a*); the second model, with the predictor and mediator regressed on the dependent variable, yielded coefficients for the *distal indirect path* (*b*) and the *direct path* (*c*′). Coefficients from the *a* and *b* paths were multiplied to calculate the *indirect effect*, and this in turn was divided by the *total effect* (*indirect* + *c*′) to quantify the proportion of variance mediated. 95% CRs were calculated for the indirect effect and proportion of variance mediated. All code and data are posted to Open Science Framework (https://osf.io/nhfsc/).

## Results

### Descriptive statistics

Descriptive statistics for SLEs, internalizing symptoms, and sleep variables are presented in Table 1. On average, participants reported mild-to-moderate levels of anxiety and depression (Figure 1), slept 7.26 hr/day, and had a sleep timing regularity index of 87.98% (i.e., participants were in the same awake/asleep state as in their average sleep day on 21/24 hr; see Figure 2). Pearson correlations between all variables are presented in Table S1.

### Stressful life events and internalizing symptoms

Bayesian hierarchical models showed that at the within-person level, when adolescents experienced higher levels of SLEs than typical for them, they also experienced greater internalizing symptoms. Within-person fluctuations in SLEs were positively associated with increases in anxiety symptoms in the same month (Figure 3A) and increases in depression the following month (Figure 3B). See Table 2 for details.

### Stressful life events and sleep

Within-subjects, higher exposure to SLEs than one's own average was associated with more variable sleep duration, both concurrently and prospectively (Figure 3C). We did not find significant associations of within-person fluctuations in SLEs with sleep timing regularity.

### Sleep and internalizing symptoms

Within-subjects, more variable sleep duration than usual was associated with higher levels of concurrent anxiety (Figure 3D). In addition, higher variability in the sleep timing regularity index predicted higher levels of subsequent anxiety (Figure 3E) and depression (Figure 3F). We did not find significant within-person associations of anxiety or depression with subsequent sleep duration or timing regularity.

**Table 1** Descriptive statistics and ICC for each dependent variable

| Dependent variable | *N* | *n* | *M* | *SD* | Range | Possible range | ICC |
|---|---|---|---|---|---|---|---|
| Stressful life events (UCLA life stress interview) | 30 | 356 | 2.500 | 3.329 | 0–19 | 0–N/A | 0.246 |
| Generalized anxiety (GAD-7) | 30 | 355 | 5.118 | 3.842 | 0–14 | 0–21 | 0.611 |
| Depression (PHQ-9) | 30 | 355 | 5.454 | 4.102 | 0–17 | 0–27 | 0.620 |
| Daily sleep duration (in hours) | 30 | 6,824 | 7.262 | 1.889 | 0.226–21.945 | 0–N/A | 0.124 |
| School days | 30 | 3,342 | 6.836 | 1.693 | 0.845–21.945 | 0–N/A | 0.191 |
| No-school days | 30 | 3,482 | 7.670 | 1.975 | 0.226–17.867 | 0–N/A | 0.134 |
| Daily sleep timing regularity index | 30 | 6,824 | 87.984 | 10.510 | 14.110–100 | 0–100 | 0.390 |
| School days | 30 | 3,342 | 90.204 | 9.268 | 31.288–99.931 | 0–100 | 0.477 |
| No-school days | 30 | 3,482 | 85.853 | 11.171 | 14.110–100 | 0–100 | 0.405 |
| Sleep duration mean (two-week aggregated, in hours) | 30 | 578 | 7.183 | 1.178 | 1.708–11.783 | 0–N/A | 0.383 |
| Sleep duration variability (two-week aggregated, in hours) | 30 | 567 | 1.561 | 0.805 | 0.024–7.631 | 0–N/A | 0.408 |
| Sleep timing regularity mean (two-week aggregated) | 30 | 578 | 86.673 | 8.907 | 38.021–97.051 | 0–100 | 0.651 |
| Sleep timing regularity variability (two-week aggregated) | 30 | 567 | 6.819 | 4.126 | 0.372–36.068 | 0–N/A | 0.294 |

ICC = intraclass correlation coefficient; *M* = mean; *n* = number of observations across participants; *N* = number of participants; *SD* = standard deviation.

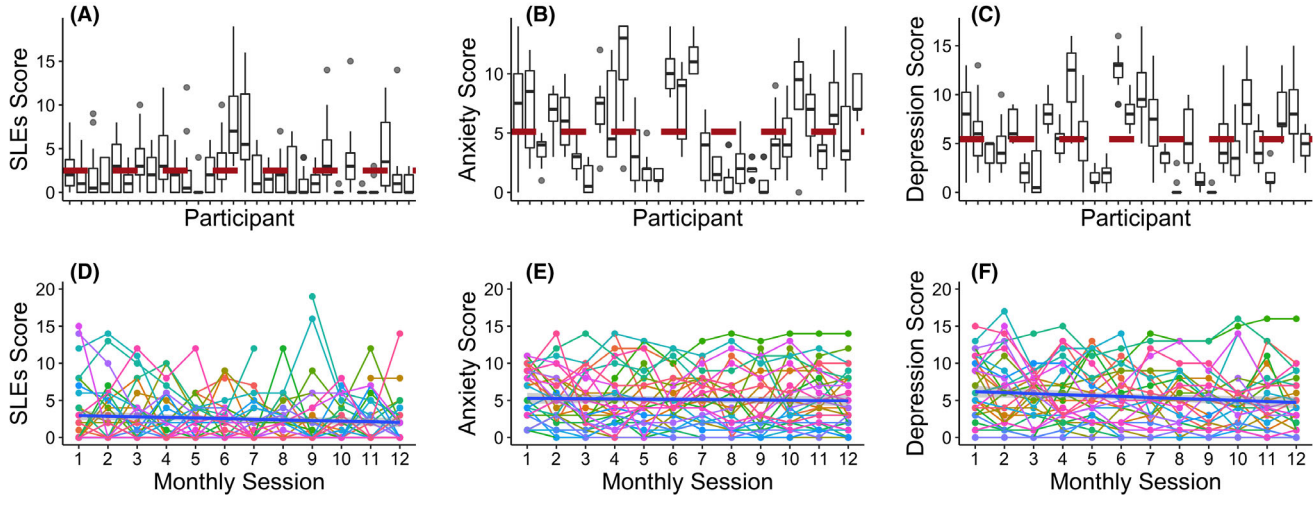


**Figure 1** Boxplots show the distribution of: (A) SLEs, (B) anxiety symptoms, and (C) depression symptoms per subject across monthly sessions. Dashed lines show the group mean. Spaghetti plots show their distribution per subject over time (D-F). Solid lines show linear fit line for the group [Colour figure can be viewed at wileyonlinelibrary.com]

### *Sleep as a mediator linking stressful life events with internalizing symptoms*

There was one possible mediation model for which each arm of the indirect path showed significant associations. Namely, within-person increases in SLEs prospectively predicted more variable sleep duration, and, in turn, more variable sleep duration predicted increases in anxiety symptoms. A within-person mediation model for these associations found that fluctuations in sleep duration variability mediated 28.45% (95% CR: [0.19%, 82.68%]) of the total relationship between SLEs and anxiety (indirect effect median = 0.019, 95% CR: [0.001, 0.042]; Figure 4).

## Discussion

The present multiwave longitudinal study used actigraphy to examine sleep behavior as a potential mechanism linking monthly within-person fluctuations in SLEs and internalizing symptoms among adolescents over a one-year period. Within-person increases in exposure to SLEs were associated with increases in anxiety and depression symptoms and prospectively predicted increased variability in sleep duration. Within-person increases in sleep duration variability, in turn, predicted increases in anxiety symptoms and mediated the association between SLEs and anxiety.

We replicate previous research showing that within-person increases in SLEs were concurrently and prospectively associated with changes in internalizing symptoms (Jenness et al., 2019). Here, within-person increases in SLEs were associated with increased symptoms of anxiety in the same month and with increased symptoms of depression the following month. We are unaware of other work examining the within-person coupling of SLEs and internalizing symptoms at this time scale, and these

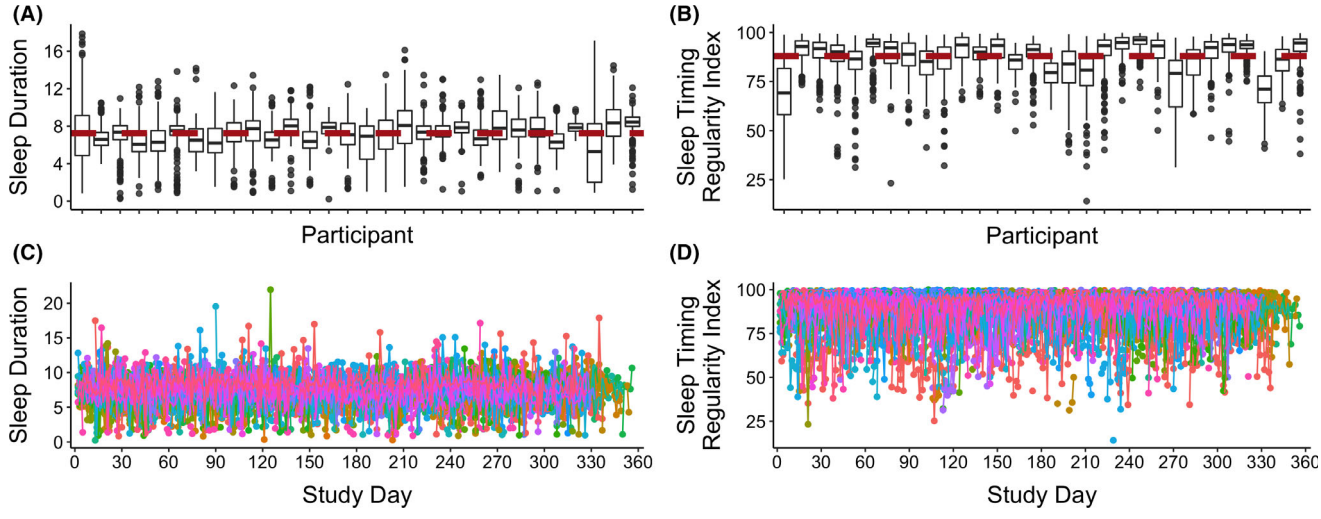


**Figure 2** Boxplots show the distribution of (A) sleep duration in hours and (B) sleep timing regularity per subject across study days. Dashed lines show the group means. Spaghetti plots show the distribution of these sleep variables per subject over time (C, D) [Colour figure can be viewed at wileyonlinelibrary.com]

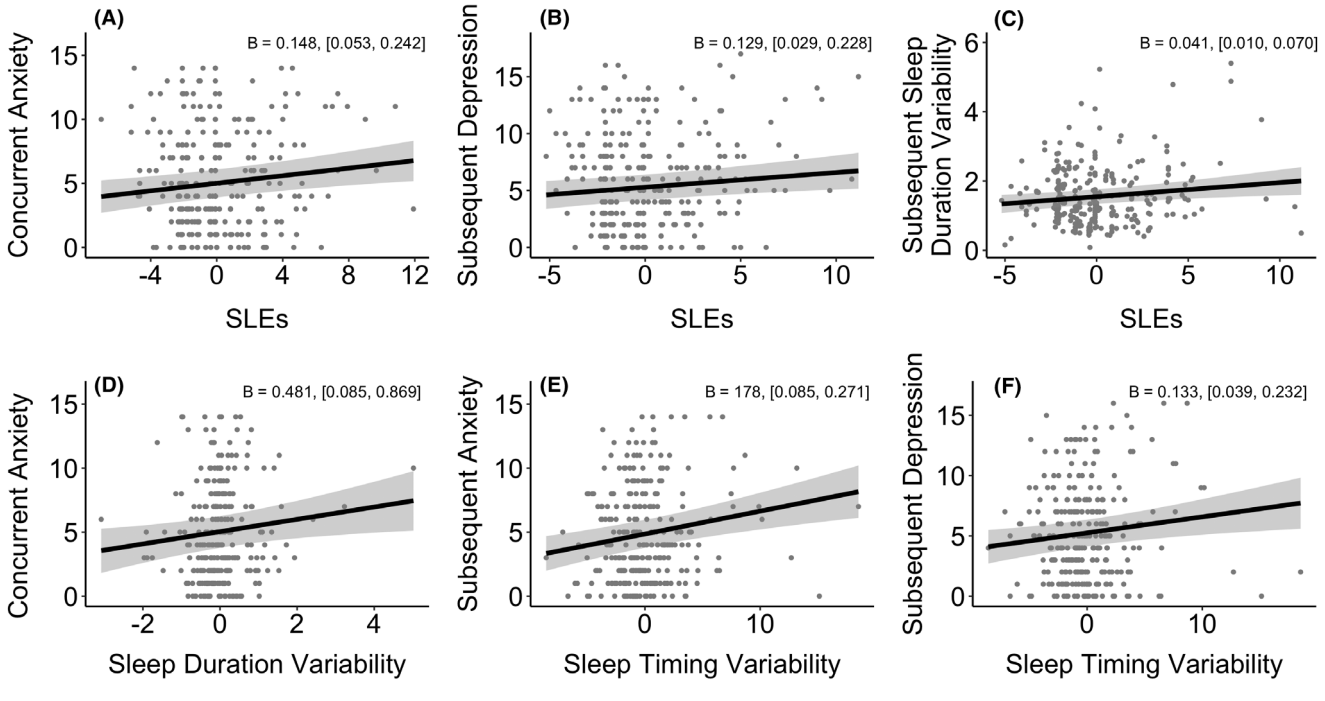


**Figure 3** Within-person fluctuations in SLEs were positively associated with concurrent anxiety symptoms (A), subsequent depression symptoms (B), and subsequent sleep duration variability (C). Within-person fluctuations in sleep duration variability were positively associated with concurrent anxiety symptoms (D). Within-person variability in sleep timing regularity was positively associated with subsequent anxiety symptoms (E) and subsequent depression symptoms (F). *X*-axis reflects within-person mean-centered variables, and *y*-axis reflects raw variables (not mean-centered). Black line with shading indicates estimated effect from Bayesian hierarchical model with 95% CR (15,000 samples)

findings warrant replication in other high-frequency longitudinal designs. Nonetheless, they suggest that within-person increases in SLEs may have more immediate effects on anxiety and that influences on depression emerge more slowly.

Our findings extend prior literature on stress and sleep disturbances. Specifically, we found that within-person increases in SLEs predicted concurrent and subsequent increases in sleep duration variability. Exposure to SLEs might elicit hyperarousal and rumination (Sadeh & Gruber, 2002), and/or behaviors aimed at mitigating the source of stress (e.g., completing schoolwork late at night), both of which can interfere with regular sleep patterns. To compensate, adolescents might sleep longer than usual on subsequent nights, thus leading to inconsistent sleep duration over time. Of note, we did not find a significant association between

**Table 2** Results of Bayesian hierarchical models

| Model | Within-person effects: Concurrent | | | Within-person effects: Subsequent | | |
|---|---|---|---|---|---|---|
| | *B* | *SE* | 95% CR | *B* | *SE* | 95% CR |
| Stressful life events predicting | | | | | | |
| Generalized anxiety (GAD-7) | **0.148** | **0.048** | **[0.053 0.242]** | 0.090 | 0.050 | [−0.006 0.191] |
| Depression (PHQ-9) | 0.090 | 0.050 | [−0.006 0.191] | **0.129** | **0.051** | **[0.029 0.228]** |
| Mean sleep duration | −0.015 | 0.018 | [−0.051 0.021] | −0.003 | 0.021 | [−0.045 0.036] |
| Variability in sleep duration | **0.030** | **0.014** | **[0.002 0.057]** | **0.041** | **0.016** | **[0.010 0.070]** |
| Mean sleep timing regularity index | −0.050 | 0.123 | [−0.292 0.192] | 0.037 | 0.135 | [−0.225 0.307] |
| Variability in sleep timing regularity index | 0.042 | 0.065 | [−0.088 0.166] | −0.005 | 0.075 | [−0.150 0.144] |
| Mean sleep duration predicting | | | | | | |
| Generalized anxiety (GAD-7) | −0.321 | 0.181 | [−0.680 0.029] | 0.095 | 0.170 | [−0.233 0.432] |
| Depression (PHQ-9) | −0.053 | 0.190 | [−0.428 0.321] | 0.036 | 0.178 | [−0.313 0.377] |
| Variability (*SD*) in sleep duration predicting | | | | | | |
| Generalized anxiety (GAD-7) | **0.481** | **0.199** | **[0.085 0.869]** | 0.055 | 0.241 | [−0.413 0.533] |
| Depression (PHQ-9) | −0.022 | 0.210 | [−0.423 0.388] | −0.181 | 0.248 | [−0.674 0.302] |
| Mean sleep regularity timing index predicting | | | | | | |
| Generalized anxiety (GAD-7) | 0.005 | 0.037 | [−0.070 0.075] | 0.059 | 0.032 | [−0.005 0.121] |
| Depression (PHQ-9) | **0.078** | **0.038** | **[0.003 0.153]** | **0.074** | **0.034** | **[ 0.009 0.142]** |
| Variability (*SD*) in sleep timing regularity index predicting | | | | | | |
| Generalized anxiety (GAD-7) | 0.042 | 0.050 | [−0.055 0.143] | **0.178** | **0.047** | **[0.085 0.271]** |
| Depression (PHQ-9) | 0.069 | 0.053 | [−0.037 0.168] | **0.133** | **0.049** | **[0.039 0.232]** |
| Generalized anxiety (GAD-7) predicting | | | | | | |
| Mean sleep duration | −0.045 | 0.023 | [−0.090 −0.000] | 0.005 | 0.023 | [−0.040 0.050] |
| Variability in sleep duration | 0.042 | 0.021 | [0.000 0.081] | 0.030 | 0.017 | [−0.003 0.064] |
| Mean sleep timing regularity index | −0.010 | 0.126 | [−0.260 0.236] | −0.101 | 0.149 | [−0.386 0.193] |
| Variability in sleep timing regularity index | 0.086 | 0.093 | [−0.099 0.269] | 0.036 | 0.083 | [−0.126 0.199] |
| Depression (PHQ-9) predicting | | | | | | |
| Mean sleep duration | −0.009 | 0.023 | [−0.052 0.037] | 0.034 | 0.022 | [−0.009 0.077] |
| Variability in sleep duration | −0.002 | 0.020 | [−0.041 0.038] | 0.019 | 0.016 | [−0.013 0.051] |
| Mean sleep timing regularity index | 0.217 | 0.122 | [−0.020 0.459] | −0.041 | 0.142 | [−0.323 0.233] |
| Variability in sleep timing regularity index | 0.037 | 0.090 | [−0.140 0.213] | 0.078 | 0.078 | [−0.077 0.232] |

Bold denotes statistically significant effect. Where sleep duration and the sleep regularity index are used as predictors, the mean and standard deviation of each of these sleep variables were used within the same model. Additionally, all models were run controlling for subject-level average of the predictor of interest (centered around the sample mean), study visit number (i.e., time), and proportion of days in school over the time period tested.
*B* = unstandardized coefficient; CR = 95% credible interval (15,000 samples); *SD* = standard deviation; *SE* = standard error.

SLEs and sleep timing regularity. One possible explanation is that, due to set school schedules, stress-induced variability in sleep duration was not large enough to result in significant changes in sleep timing.

Moreover, although prior work has demonstrated that adolescents with internalizing disorders experience sleep disturbances (Wilson et al., 2019), we present new evidence at the within-person level that disruptions in sleep may contribute to the emergence of internalizing symptoms. Increased sleep duration variability was concurrently associated with heightened anxiety symptoms, suggesting that getting inconsistent amounts of sleep might have relatively short-term influences on symptoms like worry and nervousness. In fact, laboratory-based studies with adults have found increased self-reported anxiety shortly following sleep deprivation (Sagaspe et al., 2006; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). Furthermore, greater variability in sleep timing regularity predicted increases in anxiety and depression symptoms in the subsequent time period, suggesting

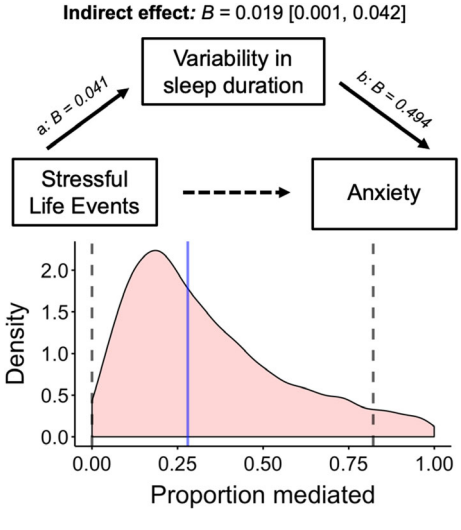


**Figure 4** Within-person variability in sleep duration mediated the prospective association between SLEs and anxiety. Figure shows Bayesian mediation model results and 95% highest posterior density credible interval (CR) displayed in brackets (15,000 samples). Density plot displays the posterior density of the estimated proportion mediated (solid line indicates median estimate; dashed lines indicate 95% CR)

that more dramatic changes in sleep timing, perhaps through disrupted circadian mechanisms, might take a protracted, cumulative toll on internalizing symptoms. These effects could be explained by elevations in negative affect, reduced energy levels, and anhedonia, as found in prior research linking sustained sleep irregularity (e.g., shift work, frequent time zone changes) with the subsequent onset of mood disorders (McClung, 2013).

In contrast, we found no significant associations of anxiety and depression symptoms with subsequent sleep patterns. Prior studies have sometimes (Alvaro et al., 2017; Shanahan et al., 2014), but not always (Roberts & Duong, 2017), observed bidirectional associations between sleep and internalizing disorders, though this work has relied on self-reported sleep behavior and a small number of assessments taken several months apart. It is also possible that these effects might not emerge until internalizing problems become more clinically meaningful than in the current sample, where most adolescents reported mild–moderate internalizing symptoms. Greater high-frequency longitudinal research is needed to better establish the directionality of these associations within individuals.

Bringing these findings together, our mediation analysis provides preliminary evidence for the role of inconsistent sleep duration as a pathway linking SLEs with symptoms of anxiety. We found that intraindividual variability in sleep duration accounts for a significant proportion of the within-person association between exposure to SLEs and heightened anxiety symptoms. In line with previous studies linking poor sleep quality with disruptions in emotion regulation (Palmer & Alfano, 2017), it is possible that adjusting to stress-induced inconsistency in sleep duration might impede effective reappraisal of negative affect and fuel maladaptive strategies like rumination and suppression. Difficulties with emotion regulation are well-established risk factors for internalizing psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Michl et al., 2013). Although future research should investigate in greater detail the cognitive, affective, and biological underpinnings of these associations, this work suggests that passive monitoring of sleep behavior via actigraphy could provide a useful target for interventions aiming to reduce the emergence of anxiety and depression following stress exposure in adolescence (Blake, Latham, Blake, & Allen, 2019).

Finally, our study found substantial intraindividual variability in SLEs, sleep behavior, and internalizing symptoms over the course of a year, thus reinforcing the importance of intensive longitudinal research that looks beyond individual- and group-level averages. Indeed, we found that increases in intraindividual variability in sleep behavior had stronger associations with SLEs and internalizing symptoms than fluctuations in mean sleep behavior. In the context of this study, this suggests that inconsistency in an individual's sleep over short periods of time (within two weeks) has stronger associations with anxiety and depression than changes in that individual's overall sleep from month to month. These findings might explain why studies using average sleep metrics and brief study periods have found mixed results, including unidirectional (Roberts & Duong, 2017), bidirectional (Alvaro et al., 2017), or no associations between sleep behavior and internalizing symptoms in adolescence (Cohodes et al., 2019). Ultimately, our findings provide support for interventions that promote consistent sleep schedules among adolescents (Bei, Manber, Allen, Trinder, & Wiley, 2017; Dahl & Harvey, 2007; Hummer & Lee, 2016).

Important limitations should be addressed in future research. First, we used two off-the-shelf actigraphy wristbands. Although sleep estimates were highly reliable between the two devices, we would have ideally used the same device throughout the study. In addition, while these devices have been validated in adults, and similar products have been validated in adolescents (Lee et al., 2019; de Zambotti et al., 2016), a validation of the exact device we used in adolescents is warranted. Lastly, research-grade devices might have further improved sleep estimation accuracy, although this must be balanced with the ability to get adolescents to wear the device over a long time period. Commercial actigraphy wristbands were more appealing to the teenagers in our study than research-grade actigraphs, which we prioritized to increase compliance rates in this intensive year-long study.

Furthermore, although our study design allowed us to establish temporal precedence between our variables of interest (critical for mediation and causal inference), an observational study cannot rule out unmeasured variables underlying shared causes of SLEs, sleep, and internalizing symptoms. Future longitudinal work should attempt to replicate our findings and extend this work by investigating other dimensions of sleep (e.g., sleep fragmentation and subjective sleep satisfaction), sex and developmental effects (e.g., puberty-driven changes in circadian and homeostatic systems underlying fluctuations in sleep and mood across adolescence), and the potential heterogeneity of these associations between and within individuals at varying time windows. For example, while our aggregating approach over two-week and month-long time periods allows for more stable and meaningful estimates of sleep, SLEs, and internalizing symptoms, daily-level analyses combining actigraphy with ecological momentary assessments of affect and stress might inform how these relationships play out more immediately. Moreover, future research could apply clustering techniques to investigate distinct sleep-stress-internalizing symptom phenotypes between individuals and/or idiographic

models to assess changes in these associations within individuals over time.

## Conclusion

Stressful life events are strongly associated with the emergence of anxiety and depression in adolescence, but the mechanisms underlying this relationship remain poorly understood, particularly at the within-person level. Using an intensive longitudinal approach, we found substantial intraindividual variability in adolescents' SLEs, actigraphy-based sleep behavior, and anxiety and depression symptoms over the course of a year. Within-person increases in SLEs were associated with more variable sleep duration, and greater variability in both the duration and timing of sleep predicted heightened internalizing symptoms over time. More variable sleep duration, in turn, significantly mediated the association between SLEs and anxiety symptoms. This work provides new insights into the nature and directionality of associations between SLEs, sleep, and internalizing symptoms over time, and suggests that interventions promoting regular sleep schedules in adolescence may help mitigate risk for stress-related psychopathology.

## Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

**Appendix S1**. Supplementary methods.

**Appendix S2**. Supplementary statistical analysis.

**Table S1**. Results of Bayesian hierarchical models using Beta distribution specification.

**Table S2**. Correlation matrix for all dependent variables.

**Figure S1**. Available actigraphy data.

**Figure S2**. Distribution of missing sleep data by SLEs and anxiety and depression symptoms.

**Figure S3**. Statistical power for within-person effects.

## Acknowledgements

This work was supported by the National Institute of Mental Health (R56-119194) to K.M., a One Mind Foundation Rising Star Award to K.M., and a Jacobs Foundation Early Career Research Fellowship to K.M. The authors are grateful to Debbie Bitran, Lucy Lurie, Elizabeth McNeilly, Zoe Miles, and Azure Reid-Russell for help with participant recruitment and testing, Alex Ramsdell for technical support, Steve Worthington for statistical modeling advice, and the Stress and Development Lab at Harvard University for helpful discussion. The authors have declared that they have no competing or potential conflicts of interest.

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### Key points

- Stressful life events (SLEs) are strongly associated with the emergence of anxiety and depression in adolescence, but the mechanisms remain poorly understood, particularly at the within-person level.
- Our year-long intensive longitudinal study used actigraphy to examine sleep behavior as a potential mechanism linking SLEs and internalizing symptoms in a community sample of adolescents.
- Within-person increases in SLEs were associated with higher anxiety symptoms that same month, higher depression symptoms the following month, and increased variability in sleep duration both concurrently and prospectively.
- In turn, within-person increases in sleep duration variability predicted increases in anxiety symptoms and mediated the association between SLEs and anxiety.
- Interventions promoting regular sleep schedules in adolescence may help mitigate risk for stress-related psychopathology.

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Accepted for publication: 13 February 2020