Dynamics of despair: examining suicidal ideation using real-time methodologies

Kivelä, L.M.M.

CHAPTER 05:

Sleep

Sleep, Hopelessness and Suicidal Ideation: An Actigraphy and Sleep Diary Study

Published as:

Kivelä, L., van der Does, W., & Antypa, N. (2024). Sleep, Hopelessness and Suicidal Ideation: An Actigraphy and Sleep Diary Study. *Journal of Psychiatric Research, 177,* 46-52. https://doi.org/10.1016/j.jpsychires.2024.06.039

Abstract

Background: Recent research shows that sleep disturbances are linked to increased suicidal ideation. In the present longitudinal cohort study, we used subjective (ecological momentary assessment, EMA) and objective (actigraphy) measures to examine the effects of sleep parameters on next-day suicidal ideation. Further, we examined hopelessness as a mediator between insufficient sleep and increased suicidal ideation. **Methods:** Individuals with current suicidal ideation (N= 82) completed 21 days of EMA and actigraphy to estimate suicidal ideation, hopelessness and sleep parameters. Multilevel linear-mixed models were used to examine the effects of sleep parameters on next-day suicidal ideation, as well as for the mediating effect of hopelessness (in the morning) on the association between previous night's sleep and suicidal ideation levels the next day. **Results:** Significant concordance existed between subjective and objective sleep measures, with moderate-to-large correlations (r= .44 – .58). Lower subjective sleep quality and efficiency, shorter total sleep time and increased time awake after sleep onset were significantly associated with increased next-day suicidal ideation (controlling for previous-day suicidal ideation). Actigraphy-measured sleep fragmentation was also a significant predictor of next-day ideation. Hopelessness mediated the effects of the subjective sleep parameters on suicidal ideation, but did not account for the association with sleep fragmentation. **Conclusions:** Individuals' psychological complaints (hopelessness, suicidal ideation) were better predicted by subjective sleep complaints than by objective sleep indices. Increased hopelessness following from perceived insufficient sleep appears an important explanatory factor when considering the link between sleep disturbances and suicidal ideation.

Introduction

Sleep has broad implications not only for physical, but also psychological health (Robotham, 2011). Sleep disturbances are implicated in many mental disorders, including depression (Baglioni et al., 2011) and anxiety disorders (Staner, 2003). Sleep disturbances also represent a significant risk factor for all aspects of suicidality (incl. suicidal ideation (SI), behavior and mortality) (Pigeon et al., 2012). Based on a meta-analysis of 42 longitudinal studies, insomnia has been found to confer the most risk for SI, whereas nightmares are most strongly associated with suicide attempts (Harris et al., 2020). Further, insomnia predicts the persistence of SI above and beyond general depressive symptom severity (Kivelä et al., 2019) and other mental health problems (Batterham et al., 2021; Geoffroy et al., 2021; Simmons et al., 2020). This highlights the important role of disturbed sleep for suicidal outcomes.

Sleep disturbances in patients experiencing SI have primarily been assessed with subjective measures. A meta-analysis of 41 such longitudinal studies (Liu et al., 2020) reported a small-to-medium effect size of sleep disturbances on SI. For insomnia specifically, a small-to-medium effect size was reported, while hypersomnia yielded a non-significant negligible effect size. Nightmares were also associated with subsequent SI, with a small-to-medium effect size. In comparison, a recent meta-analysis of studies using objective measures identified only 11 studies – seven of which used actigraphy, six polysomnography and two electroencephalogram (EEG). It was concluded that short sleep duration had a small, significant association with current SI, but no associations were found for other potential markers (such as sleep efficiency or percentage of rapid eye movement (REM) sleep) (Romier et al., 2023). Subjective sleep disturbance therefore appears to be more strongly associated with SI than objective sleep indices, although the literature employing objective measures is still limited and mainly reliant on retrospective reports of SI.

While more studies are needed on the discrepancies between subjective and objective sleep measures, one of the current limitations in the field is the lack of studies examining short-term risk (Liu et al., 2020). A meta-analysis found follow-up length to be a significant moderator in the association between sleep disturbances and SI, with studies employing shorter follow-ups (a few weeks or months) yielding larger effect sizes (Liu et al., 2020). This indicates that sleep disturbances are an imminent and possibly potent risk factor for SI. However, few studies have examined the *immediate* effects of sleep on SI. Both subjective and objective sleep duration, but only subjective sleep quality predicted next-day SI in a 7-day actigraphy and ecological momentary assessment (EMA) study in adults (Littlewood 2019). In a 28-day actigraphy and EMA study in adolescents recently discharged from acute psychiatric care following a suicidal crisis, longer (subjective) sleep onset latency, nightmares and *higher* sleep quality related to greater next-day SI (Glenn et al., 2021). Meanwhile, only objective wake after sleep onset (WASO) was related to next-day SI, whereby, surprisingly, *less* WASO was related to *more* SI (Glenn et al., 2021).

Another target for further investigation are the causal mechanisms tying sleep disturbances to SI. Here, we examine hopelessness as a potential mediator, in line with our previous findings that hopelessness reactivity (i.e., the tendency to experience hopelessness in response to low mood) mediated the effect of insomnia on persistent SI over 9-years (Kivelä et al., 2019). Similar findings were reported in a cross-sectional study of 766 community adults, where hopelessness was found to mediate the association between insomnia and SI (Woosley et al., 2014). In another study, feelings of defeat, entrapment and hopelessness mediated the association between nightmares and SI (Littlewood et al., 2016). However, no previous study has examined whether this relationship also exists on a more immediate, night-to-day basis.

Theoretically, we propose that insufficient sleep may worsen affect (Medic et al., 2017), consequently leading to increased negative emotionality, including pessimism and hopelessness (McCall and Black, 2013), as lack of sleep may have a detrimental effect on one's ability to contain hopeless thoughts. Insomnia may also be more directly associated with hopeless cognitions about the effects of poor sleep, such as expectations of reduced daytime functioning and the persistence of sleep problems over time (McCall & Black, 2013). Such hopelessness about sleep has been referred to as insomnia catastrophizing, whereby individuals specifically ruminate on the worst-case consequences of poor sleep (Jansson-Fröjmark et al., 2020; Winsper and Tang, 2014). Hopelessness, in turn, is a well-established risk factor for both suicidal ideation and behavior (Kuo et al., 2004; Zhang et al., 2011).

The aim of the present study was to examine how subjective and objective sleep parameters relate to next-day SI in a cohort of participants with current suicidal ideation who were monitored for 21 days with EMA and actigraphy. We also explored feelings of hopelessness as a mediator in the association between sleep and suicidal ideation the next day.

Methods

Ethics

All procedures were conducted in accordance with the Helsinki Declaration of 1964 and its later amendments. The study was approved by the Medical Ethics Committee

– Leiden, Den Haag, Delft (METC-LDD) on 24.4.2020 with dossier number NL71510.058.19. All participants provided written informed consent.

Sample

The sample (*N* = 82) was derived from the SAFE study, a longitudinal cohort study in adults with a past-year history of a suicide attempt and/or active SI (as indicated by a score of >= 3 on the Columbia Suicide Severity Rating Scale (CSSRS) (Posner et al., 2011), or a score of >= 2 if symptoms were present in the past two months). Exclusion criteria included a current diagnosis of bipolar disorder, a psychotic disorder, severe substance dependence, or any physical or intellectual impairment that would meaningfully hinder the individual's participation in the study. More details about the SAFE study are reported elsewhere (Kivelä et al., 2023).

Procedure

Participants were recruited through social media and community advertisements, as well as referral from treatment providers in the surrounding areas. Participants attended an (in-person or online) intake interview where, after receiving study information and signing informed consent, the participant's history of SI was assessed via an adapted version of the CSSRS (composed of the first five questions on past-year SI, and additional questions on lifetime history of suicide attempts). Current diagnoses were established via the M.I.N.I. PLUS Neuropsychiatric interview (version 5.0) (Sheehan et al., 1998). Following diagnostics but prior to receiving study instructions, personalized safety plans were created for each participant. Participants then received instructions for the EMA and actigraphy (see *Instruments* below). Following the 21-day assessment period, another meeting was scheduled where participants returned the study materials and received a summary report of their data.

Instruments

Baseline Sociodemographics (age, gender), current medical diagnoses and medication use were collected via a custom semi-structured interview. Current depressive symptoms were assessed with the Beck Depression Inventory (BDI-I) (Beck, 1961), which includes 21 questions on depressive symptoms as present in the past week. Current SI was estimated with the Beck Scale for Suicide Ideation (BSSI) (Beck et al., 1979), which includes 21 items on past-week SI. Insomnia symptoms were established with the Insomnia Severity Index (ISI) (Morin et al., 2011), which includes seven items on sleep complaints in the past two weeks. For the ISI, scoring guidelines indicate 0-7 to reflect the absence of (clinically significant) insomnia, 8-14 subthreshold insomnia, 15-21 moderate, and 22-28 severe insomnia.

Ecological Momentary Assessment (EMA) EMA was used to assess SI, hopelessness and subjective sleep parameters over 21 days. Participants downloaded a mobile phone app produced by Ethica (a.k.a. Avicenna), and received alerts for four questionnaires per day. The questionnaires were released on a pseudo-random schedule between the hours of 7am and 10pm. Additionally, participants could self-initiate additional EMA at any time. Questions on SI were presented at all EMAs per day, and each assessment included three questions (*"At the moment... how strong is your desire to live?", "... how strong is your desire to die, or go to sleep and not wake up?", "... do you actually have thoughts of killing yourself?"*) rated on a scale from 0 (none/not at all) to 10 (very strong/very well). The positively worded item (desire to live) was reverse coded and a daily mean score of SI was calculated.

Hopelessness was estimated each morning with the question *"At the moment... how hopeless do you feel?"* rated on a scale from 0 (not at all) to 10 (very much).

Questions on subjective sleep were presented each morning (adapted from the Consensus Sleep Diary – Morning section) (CSD-M; Carney et al., 2012) and included subjective sleep quality (SSQ) (*"How did you sleep last night?"* from 0 (very poorly) to 10 (very well)), timing of sleep (*"What time did you try to get to sleep?", "What time did you wake up for the day?", "What time did you get out of bed?"*), time to fall asleep (*"How long did it take you to fall asleep (in minutes)?"*), night-time awakenings (*"Did you wake up during the night?" yes/no* And if *'yes'*: *"How long were you awake (in minutes)?"*) and nightmares (*"Did you have any nightmares?" yes/no*). Sleep parameters derived from EMA included SSQ, total sleep time (TST; defined as the time spent asleep between initiating sleep and awakening in the morning), sleep efficiency (SE; a percentage calculated by dividing TST with the overall time spent in bed, multiplied by 100), sleep onset latency (SOL; time between initiating sleep and actually falling asleep), wake after sleep onset (WASO; time spent awake between falling asleep at night and waking up in the morning) and nightmares.

Actigraphy Objective sleep data was collected with the MotionWatch 8 (CamnTech, Cambridge, UK). The watch includes a tri-axial accelerometer that samples activity in 30 second epochs, as well as a light sensor (data not reported). Participants were instructed to press a button on the watch when attempting to sleep at night, and when waking up in the morning. The data were uploaded into the MotionWare program

(CamnTech, Cambridge, UK), which produces estimates on sleep parameters based on algorithms that transform the activity data collected by the accelerometer. Parameters used in the present study included the fragmentation index (FI; a percentage reflecting the proportion of mobile/immobile epochs during the sleep period to estimate restlessness during the night) (Shrivastava et al., 2014), SE, TST, SOL and WASO. When event markers were missing, participants' EMA entries and visual inspection of the data were used to mark sleep periods; when both were missing, or event markers and self-reports deviated greatly, visual inspection (based on activity cessation and light data) was used to determine sleep periods. These pre-processing steps are in line with other studies using actigraphy (Falck et al., 2020) Bernert et al., 2017). The MotionWatch 8 has been validated for use with 85% per-epoch agreement of sleep/wake when compared to PSG (O'Hare et al., 2015), with a minimum of 14 nights of measurement recommended to establish reliable estimates (Ancoli-Israel et al., 2015).

Statistical Analysis

All analyses were conducted in IBM SPSS Statistics (version 29.0). Multilevel regression analyses (linear-mixed models) were used to examine the (main) effects of subjective and objective sleep measures on next-day suicidal ideation. Prior to the analyses, all continuous variables (predictors and outcome) were person-mean centered by subtracting a participant's mean score from each individual observation, in order to examine within-person effects. We considered all predictors as fixed effects, and specified a 2-level random intercept model whereby observations were nested within individuals. For repeated effects, we specified a first-order autoregressive covariance structure, which takes into account temporal dependencies and assumes higher correlations between two adjacent time points, with decreasing correlations between observations with increasing distance. Separate multilevel regression analyses were ran for all sleep parameters, as the assumption of no multicollinearity was violated.[1] Finally, we examined the mediating effect of hopelessness (in the morning) on the associations between the sleep parameters and next-day suicidal ideation, in accordance with the steps specified by Baron & Kenny (1986). All models were controlled for previous-day suicidal ideation. Significance was determined at $p < .05 / 11 = .005$ for all multilevel analyses, corrected by the total number of subjective and objective sleep parameters examined.

[1] No multicollinearity was observed between hopelessness and the sleep parameters (VIF = 1.42).

Table 1. Sociodemographic and clinical characteristics of the sample

	EMA $N = 82$	Actigraphy $n = 61$
Age (*M, SD*)	27 (8.6)	28 (8.6)
Gender - Female (*N*, %)	63 (77%)	45 (74%)
Depressive symptom severity (BDI) (*M, SD*)	25.5 (9.6)	25.3 (10.2)
Suicidal ideation severity (BSSI) (*M, SD*)	15.3 (8.6)	15.4 (8.7)
Suicidal ideation (EMA) (*M, SD*)	3.1 (2.0)	2.9 (2.0)
Current diagnoses (*N*, %)		
Depressive disorder	63 (77%)	48 (79%)
Anxiety disorder	47 (57%)	33 (54%)
PTSD	18 (22%)	10 (16%)
OCD	7 (9%)	6 (10%)
ADHD	10 (12%)	6 (10%)
Medication (*N*, %)		
Sedatives	20 (24%)	16 (26%)
Stimulants	10 (12%)	6 (10%)
Antidepressants	33 (40%)	27 (44%)
Concurrent medical diagnosis (*N*, %)	35 (43%)	28 (46%)
Non-psychoactive medication	26 (32%)	19 (31%)
Insomnia severity (ISI) (*M, SD*)	11.8 (5.0)	11.8 (5.4)

Note: EMA = Ecological Momentary Assessment, BDI = Beck Depression Inventory, BSSI = Beck Scale for Suicide Ideation, PTSD = Post-traumatic stress disorder, OCD = Obsessive compulsive disorder, ADHD = Attention deficit hyperactivity disorder, ISI = Insomnia Severity Index

Results

Sample Description

Participants ($N = 82$) provided self-report EMA data for $K = 16$ nights on average, resulting in $k = 1{,}304$ unique observations. For objective sleep parameters, 21 participants (26%) had no actigraphy data available. Approximately half of the missingness was attributable to participants not returning their watches, or watches getting lost in the mail ($n = 9$). Other participants had completely missing data due to either unknown technical issues or user error resulting in no data being recorded by the watch ($n = 12$). The remaining participants ($n = 61$) provided actigraphy data for $K = 18$ nights on average, resulting in $k = 1{,}114$ unique observations. No significant differences emerged between those with and without actigraphy data (see Appendix, Table S1). Sociodemographic and clinical characteristics of the sample are presented in Table 1. More detailed descriptions of the characteristics of the sample may be found in Kivelä et al. (2023).

Table 2. Intra-Individual Means and Standard Deviations of Subjective and Objective Sleep Parameters

	EMA $N = 82$	Actigraphy $n = 61$	Pearson r	t-test (df)	p-value
Subjective sleep quality (SSQ)					
M (SD)	5.6 (1.2)	-	-	-	-
Range	2.0–8.7	-			
Fragmentation index (FI)					
M (SD)	-	26.2 (9.1)	-	-	-
Range	-	9.2–54.4			
Sleep efficiency (SE)					
M (SD)	84% (7.4%)	77% (7.2%)	.51	6.58	.001
Range	56–97%	54–90%		(59)	
Total sleep time (TST)					
M (SD)	416 min (55 min)	395 min (52 min)	.58	2.87	.006
Range	255–527 min	201–473 min		(59)	
Sleep onset latency (SOL)					
M (SD)	23 min (18 min)	18 min (9 min)	.57	2.90	.005
Range	4–92 min	4–43 min		(59)	
Wake after sleep onset (WASO)					
M (SD)	20 min (18 min)	60 min (26 min)	.44	−12.00	< .001
Range	0–83 min	23–132 min		(60)	
Nightmares					
Percentage (%)	23%	-		-	-
Range	0–95%	-			

Note: EMA = Ecological Momentary Assessment; all correlation coefficients were significant with $p < .001$

Concordance Between Sleep Measures

Means and standard deviations of the subjective and objective sleep parameters, and correlations between them, are presented in Table 2. Subjective and objective estimates of SE, TST, SOL and WASO exhibited moderate-to-large correlations. Actigraphy measures indicated significantly shorter TST and SOL, lower SE and higher WASO compared to self-reports. Baseline insomnia severity (ISI) significantly predicted EMA-measured lower subjective SSQ ($B = -0.10$, $SE = 0.03$, $p < .001$, $R^2 = 0.15$), SE ($B = -0.59$, $SE = 0.17$, $p < .001$, $R^2 = 0.15$) and WASO ($B = 1.78$, $SE = 0.36$, $p < .001$, $R^2 = 0.26$), but not TST ($p = .091$), SOL ($p = .487$), nightmares ($p = .052$), or any of the objective sleep parameters (all p's > .05). Baseline insomnia severity significantly predicted higher EMA-measured SI (B = 0.13, SE = 0.05, p = .005, R^2 = 0.11).

Subjective and Objective Sleep Parameters and Next-Day Suicidal Ideation

Lower subjective SSQ and SE, shorter TST and longer WASO were significantly associated with within-person increases in SI the following day, while SOL and nightmares were not. Out of the objective sleep parameters, only FI was significantly associated with next-day SI after correction for multiple testing (Table 3).

Table 3. Multilevel Regression of Subjective and Objective Sleep Parameters on Next-Day Suicidal Ideation, Controlling for Previous-Day Suicidal Ideation

	B	*SE*	95% CI	*p*-value
EMA				
Subjective sleep quality (SSQ)	−0.090	0.012	[−0.114; −0.066]	< .001
Sleep efficiency (SE)	−0.008	0.002	[−0.012; −0.003]	< .001
Total sleep time (TST)	−0.001	0.001	[−0.002; −0.001]	< .001
Sleep onset latency (SOL)	0.002	0.001	[−0.001; 0.003]	.086
Wake after sleep onset (WASO)	0.003	0.001	[0.001; 0.004]	< .001
Nightmares	0.145	0.056	[−0.035; 0.256]	.010
Actigraphy				
Fragmentation index (FI)	0.007	0.003	[0.002; 0.012]	.004
Sleep efficiency (SE)	−0.003	0.004	[−0.011; 0.004]	.344
Total sleep time (TST)	−0.001	0.001	[−0.001; 0.001]	.829
Sleep onset latency (SOL)	0.002	0.001	[−0.001; 0.005]	.122
Wake after sleep onset (WASO)	0.003	0.001	[0.001; 0.005]	.009

Note: EMA = Ecological Momentary Assessment; significance was determined at p < .005

Suicidal Ideation and Subjective and Objective Sleep Parameters the Following Night

Examining the opposite direction of causality, SI during the day was not significantly associated with any of the subjective or objective sleep parameters the following night (Table S2).

Mediation Analyses: The Role of Hopelessness

Hopelessness significantly mediated the relationship with next-day SI for all subjective sleep parameters (SSQ, SE, TST, WASO), with partial mediation for SSQ and TST, and full mediation for SE and WASO. Hopelessness was not significantly associated with FI (actigraphy) and did not mediate its relation with SI (Table S3).

Discussion

In the present study, we examined the effects of subjective and objective sleep parameters on next-day SI. Overall, subjective sleep estimates appeared more consistently associated with SI than actigraphy measures, with subjective SSQ, SE, TST and WASO all significantly predicting next-day SI, while only actigraphy-measured FI emerged as a significant predictor.

The association between subjective SSQ and next-day SI is in line with prior research (Littlewood et al., 2019). However, while SE (which is often used as an indicator of sleep quality) did not emerge as a significant predictor in prior research (Littlewood et al., 2019), we found that subjective SE (but not objective SE) was associated with next-day SI. Indeed, only one of the objective sleep parameters, FI, remained a significant predictor after correction for multiple testing. FI is a measure of restlessness during the night, and higher values indicate greater sleep disruption (Shrivastava et al., 2014). The FI parameter has not previously been identified as a marker for increased SI. However, our findings are in line with a cross-sectional actigraphy study of 3,045 older adult women from the community, which found depressive symptoms to relate to poor subjective sleep quality and actigraphy-measured increased sleep fragmentation (as indicated by increased WASO) (Maglione et al., 2012). We also found objective WASO to be significantly associated with SI, but only prior to correction. The finding that indicators of sleep fragmentation (i.e., FI, WASO) specifically were highlighted both in our subjective and objective analyses, while SOL was significant in neither, seems to indicate that trouble maintaining sleep (i.e., middle insomnia) is more closely related to increased SI than trouble initiating sleep (i.e., early insomnia), at least in the very short-term. While both sleep deprivation (i.e., insufficient TST) and sleep fragmentation are associated with negative mental and physical health consequences, it has also been demonstrated that the effects of sleep fragmentation are unique and not simply explained by sleep loss (Benkirane et al., 2022; Bonnet and Arand, 2003). Explanations for the deleterious effects of sleep fragmentation include that it may be more detrimental to sleep architecture than short sleep duration in itself, therefore impairing the restorative function of sleep. For example, it has been shown that the increased sleep fragmentation associated with aging is specific to slow wave sleep (SWS) (Varga et al., 2016); this sleep stage is thought to be crucial for restoration and recovery (Roth, 2009).

Nightmares did not emerge as a significant predictor of next-day SI. This is contrary to a prior study that found nightmares to relate to increased next-day SI among adolescents (Glenn et al., 2021). Meanwhile, the general literature indicates that nightmares may be more closely tied to suicidal behavior (i.e., attempts) than ideation (Harris et al., 2020). Findings on the association between nightmares and suicidal ideation therefore appear inconclusive.

We did not find evidence for the opposite direction of causality (i.e., suicidal ideation disrupting sleep). Prior studies with daily measures have also indicated unidirectional effects of sleep on affect (Barber et al., 2023; de Wild-Hartmann et al., 2013; McCrae et al., 2008). One previous EMA study found significant bidirectional effects, but concluded that the effects of sleep on mood were substantially larger than vice versa (Triantafillou et al., 2019).

Hopelessness was a significant mediator when examining all subjective sleep parameters (SSQ, SE, TST, WASO) and their effects on next-day SI. We also previously found hopelessness reactivity to mediate the effect of insomnia on the persistence of suicidal ideation over time, based on an examination of 195 individuals observed over 9 years (Kivelä et al., 2019). Prior cross-sectional studies have also identified hopelessness as a mediator of both insomnia (Woosley et al., 2014) and nightmares (Littlewood et al., 2016). Here, we extend on these findings by indicating that disturbed sleep, through increased hopelessness, may have an immediate worsening effect on suicidal ideation the very next-day. Future research should further aim to examine the roots of hopelessness resulting from poor sleep, whether that be more direct worry about the consequences of a bad night's sleep (Jansson-Fröjmark et al., 2020; McCall and Black, 2013), or more complex mechanisms impacting affective (Groeger et al., 2022; Medic et al., 2017; Ritchie et al., 2018) or cognitive functioning (Alhola and Polo-Kantola, 2007; Holding et al., 2021; Medic et al., 2017). For example, executive dysfunction is observed both among people with insomnia (Bredemeier and Miller, 2015), as well as those at risk of suicide (Ballesio et al., 2019). Sleep fragmentation specifically has been identified as especially deleterious to cognition: in a study utilizing polysomnography, increased sleep fragmentation was associated with worse executive function performance, irrespective of sleep duration (Benkirane et al., 2022). Similar findings have emerged with regard to emotion regulation, whereby maladaptive emotion regulation (i.e., rumination) mediated the association between actigraphy-measured sleep fragmentation and negative affect (Boon et al., 2023). Future research may find it relevant to examine whether executive dysfunction and/or emotion dysregulation may underlie these associations between poor sleep, hopelessness and SI.

Finally, we examined concordance between EMA and actigraphy measures and found moderate-to-large correlations between subjective and objective SE, TST, SOL and WASO. Actigraphy provided estimates that were significantly lower for TST, SE and SOL, but higher for WASO, as compared to EMA. It is well-established that in comparison to PSG, actigraphy tends to overestimate sleep duration and underestimate wakefulness due to its movement-based algorithms that struggle to correctly classify moments of wakefulness in the absence of movement (e.g., when lying still in bed) (Lehrer et al., 2022; O'Hare et al., 2015; Sadeh, 2011). However, self-reports instead may *overestimate* sleep duration (Benz et al., 2023; Lehrer et al., 2022; Littlewood et al., 2019).

Limitations of the present study include more missing data on the objective measures. This may have reduced power in our actigraphy analyses, although our sample is still the largest to date to examine night-to-day associations between actigraphic sleep and SI. The general pattern observed in the present study is also in line with prior literature indicating larger effect sizes for suicide outcomes when self-report measures are used to estimate sleep (Harris et al., 2020). Further, a substantial portion of participants were using either antidepressant (40%), sedative (24%) or stimulant medication (12%). While antidepressants have been associated with side effects of both insomnia and hypersomnia (Wichniak et al., 2017), use of sedatives such as benzodiazepines may increase sleep duration while simultaneously decreasing sleep quality (Holbrook et al., 2001; Manconi et al., 2017). Likewise, stimulants may reduce both sleep quality and quantity through increased alertness (Stein et al., 2012). However, due to our small sample size and heterogeneity in medication usage we were unable to account for these potential confounders in our analyses. Finally, our sample was fairly young, and predominantly female: sleep characteristics may change as a function of age (Li et al., 2018), and gender-related differences in sleep architecture are also observed (Krishnan and Collop, 2006). Hence, our findings may have limited generalizability to older, and male, populations, and replication in corresponding samples is needed.

Our study underscores that sleep disturbances may represent an important warning sign for increased suicide risk. While estimates of sleep disturbances are well-known risk factors for suicidal ideation in longitudinal cohort studies (Harris et al., 2020), much less is known about the immediate effects of disturbed sleep on suicidal ideation in the short-term. Meanwhile, sleep disturbances also represent a risk factor that is readily modifiable through intervention. A number of recent studies have indicated reductions in SI following treatment for sleep disturbances in patients with bipolar disorder (Sylvia et al., 2021), college students with a lifetime history of SI (Crosby and Witte, 2021) and veterans with PTSD (Bishop et al., 2016). In a randomized controlled trial (RCT) of online-based cognitive behavioral therapy for insomnia (CBT-I), the treatment was associated with reduced SI both post-treatment, as well as 1-year follow up (Kalmbach et al., 2022). Similarly, RCTs examining pharmacotherapy for sleep disturbances have also indicated concomitant reductions in SI (see e.g., McCall et al., 2019). Sleep disturbances, if untreated, can persist even at times of remission, and may predispose individuals to both depressive as well as suicidal ideation relapse (Gallo et al., 2020). Further, as our findings indicate that poor sleep may have immediate effects on psychological well-being, improving sleep may be relevant as a crisis management intervention prior to employing more long-term treatments for SI. In addition to sleep interventions (Kalmbach et al., 2022; McCall et al., 2019), chronotherapeutics (i.e., interventions that work to re-synchronize the biological clock) have also been shown to have rapid antidepressant effects, including relief in suicidal symptoms (Sahlem et al., 2014).

In conclusion, we found that subjective sleep estimates (SSQ, SE, TST, WASO) relate to next-day SI, while sleep fragmentation (FI) emerged as the only significant predictor of the objective indices. Interpreting these sleep parameters as a whole, we observe that shorter sleep duration and interrupted sleep during the night pose individuals at increased risk of higher SI the following day. Increased hopelessness following from perceived insufficient sleep is an important explanatory factor when considering the link between sleep disturbances and SI.

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Appendix

Table S1. Sociodemographic and Clinical Characteristics of the Sample

	EMA *N* = 82	Actigraphy *n* = 61	N/A *n* = 21	*t*-test / Chi-Square (df)	*p*-value
Age (M, SD)	27 (8.6)	28 (8.6)	25 (7.8)	−1.59 (80)	.116
Gender - Female (*N*, %)	63 (77%)	45 (74%)	18 (86%)	1.87 (2)	.393
Depressive symptom severity (BDI) (*M, SD*)	25.5 (9.6)	25.3 (10.2)	26.4 (7.6)	0.40 (69)	.917
Suicidal ideation severity (BSSI) (*M, SD*)	15.3 (8.6)	15.4 (8.7)	15.1 (8.7)	−0.10 (69)	.689
Suicidal ideation (EMA) (*M, SD*)	3.1 (2.0)	2.9 (2.0)	3.4 (1.8)	0.86 (80)	.395
Current diagnoses (*N*, %)					
Depressive disorder	63 (77%)	48 (79%)	11 (52%)	1.05 (1)	.313
Anxiety disorder	47 (57%)	33 (54%)	14 (67%)	0.87 (1)	.444
PTSD	18 (22%)	10 (16%)	8 (38%)	4.13 (1)	.065
OCD	7 (9%)	6 (10%)	1 (5%)	0.54 (1)	.670
ADHD	10 (12%)	6 (10%)	4 (19%)	1.18 (1)	.275
Medication (*N*, %)					
Sedatives	20 (24%)	16 (26%)	4 (19%)	0.44 (1)	.509
Stimulants	10 (12%)	6 (10%)	4 (19%)	1.23 (1)	.266
Antidepressants	33 (40%)	27 (44%)	6 (29%)	1.60 (1)	.206
Concurrent medical diagnosis (*N*, %)	35 (43%)	28 (46%)	7 (33%)	1.00 (1)	.315
Non-psychoactive medication	26 (32%)	19 (31%)	7 (33%)	0.03 (1)	.853
Insomnia severity (ISI)	11.8 (5.0)	11.8 (5.4)	11.8 (3.5)	−0.04 (69)	.972

Note: EMA = Ecological Momentary Assessment, N/A = actigraphy data not available, BDI = Beck Depression Inventory, BSSI = Beck Scale for Suicide Ideation, PTSD = Post-traumatic stress disorder, OCD = Obsessive compulsive disorder, ADHD = Attention deficit hyperactivity disorder, ISI = Insomnia Severity Index; statistics are reported for group comparisons between those with and without actigraphy data

Table S2. Multilevel Regression of Suicidal Ideation on Subjective and Objective Sleep Parameters the Following-Night

	B	*SE*	95% CI	*p*-value
EMA				
Subjective sleep quality (SSQ)	−0.005	0.047	[−0.098; 0.088]	.923
Sleep efficiency (SE)	−0.298	0.268	[−0.824; 0.228]	.266
Total sleep time (TST)	1.050	2.121	[−3.112; 5.212]	.621
Sleep onset latency (SOL)	1.188	0.673	[−0.132; 2.508]	.078
Wake after sleep onset (WASO)	0.218	0.823	[−1.397; 1.833]	.792
Nightmares*	0.028	0.059	[−0.088; 0.144]	.634
Actigraphy				
Fragmentation index (FI)	0.382	0.284	[−.175; 0.939]	.178
Sleep efficiency (SE)	−0.025	0.210	[−0.437; 0.387]	.905
Total sleep time (TST)	3.362	2.066	[−0.691; 7.415]	.104
Sleep onset latency (SOL)	0.248	0.585	[−0.899; 1.395]	.671
Wake after sleep onset (WASO)	0.126	0.691	[−1.231; 1.482]	.856

Note: Sleep characteristics are the outcome; EMA = Ecological Momentary Assessment; significance was determined at $p < .005$; *based on a multilevel binary logistic regression analysis

Table S3. Multilevel Mediation Analyses of Hopelessness in the Relation Between Subjective and Objective Sleep Parameters and Next-Day Suicidal Ideation, Controlling for Previous-Day Suicidal Ideation

	B	*SE*	95% CI	*p*-value
EMA				
Subjective sleep quality (SSQ)				
Path a	−0.257	0.026	[−0.308; −0.206]	< .001
Path b	0.167	0.012	[0.143; 0.192]	< .001
Path c	−0.090	0.012	[−0.114; −0.066]	< .001
Path c’	−0.047	0.012	[−0.070; −0.023]	< .001
Sleep efficiency (SE)				
Path a	−0.025	0.005	[−0.035; −0.016]	< .001
Path b	0.179	0.013	[0.154; 0.204]	< .001
Path c	−0.008	0.002	[−0.012; −0.003]	< .001
Path c’	−0.003	0.002	[−0.008; −0.001]	.114
Total sleep time (TST)				
Path a	−0.003	0.001	[−0.004; −0.001]	< .001
Path b	0.178	0.012	[0.153; 0.202]	< .001
Path c	−0.001	0.001	[−0.002; −0.001]	< .001
Path c’	−0.001	0.001	[−0.001; −0.001]	.004
Wake after sleep onset (WASO)				
Path a	0.008	0.002	[0.005; 0.011]	< .001
Path b	0.176	0.012	[0.152; 0.200]	< .001
Path c	0.003	0.001	[0.001; 0.004]	< .001
Path c’	0.001	0.001	[−0.001; 0.003]	.037
Fragmentation index (FI)				
Path a	0.009	0.006	[−0.004; 0.021]	.163
Path b	0.176	0.014	[0.001; 0.011]	< .001
Path c	0.007	0.003	[0.002; 0.012]	.004
Path c’	0.006	0.003	[0.001; 0.11]	.013

Note: EMA = Ecological Momentary Assessment; significance was determined at $p < .005$