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Research Methods, Protocols, Procedures

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Dynamic impacts of sleep disruption on ecologically assessed affective, behavioral, and cognitive risk factors for suicide: a study protocol

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Abstract

Diminished sleep health is a known warning sign for suicide. However, the contexts and time periods within which diminished sleep elevates suicide risk are unknown. Modeling the complex process by which diminished sleep health impacts daily functioning and establishing proximal suicide risk factors can aid in addressing these important knowledge gaps. This paper describes the methods and research protocol for a study that aims to elucidate the nature of the sleep–suicide relationship and develop an integrated model of proximal suicide risk. Participants will be 200 adults at high risk for suicide recruited from a psychiatric inpatient unit. They will complete a baseline assessment including clinical interviews and self-reports, and laboratory tasks with concurrent electroencephalography to phenotype-relevant risk processes. This baseline assessment will be followed by 4 weeks of ecological momentary assessment and digital phenotyping, coupled with assessments of sleep via a wearable used to generate a minute-by-minute metric of cognitive effectiveness using the Sleep Activity, Fatigue, and Task Effectiveness algorithm index. Follow-up assessments will be conducted 1-, 3-, and 6-months post-hospital discharge to determine how the developed proximal model of risk prospectively predicts suicidal ideation and behavior. The results of this study have the potential to greatly enhance understanding of how and why diminished sleep health is related to real-world fluctuations in suicide risk, knowledge that can inform efforts to better prevent, and intervene to reduce suicides.

Key words: sleep; suicide; EEG; EMA; SAFTE

Statement of Significance

Diminished sleep health is a known warning sign for suicide. However, the contexts and time periods within which diminished sleep elevates suicide risk are unknown, limiting the clinical utility of this warning sign. The study described in this paper will address these important knowledge gaps by using a combination of experimental, psychophysiological, and intensive longitudinal assessment methods. The results of this study have the potential to greatly enhance understanding of how and why diminished sleep health is related to real-world fluctuations in suicide risk, knowledge that can inform efforts to better prevent, and intervene to reduce suicides.

Suicide is a significant public health problem, resulting in the deaths of approximately 800 000 people worldwide every year [1]. As patients are 134–213 times more likely to die by suicide in the month following discharge from a psychiatric inpatient unit,

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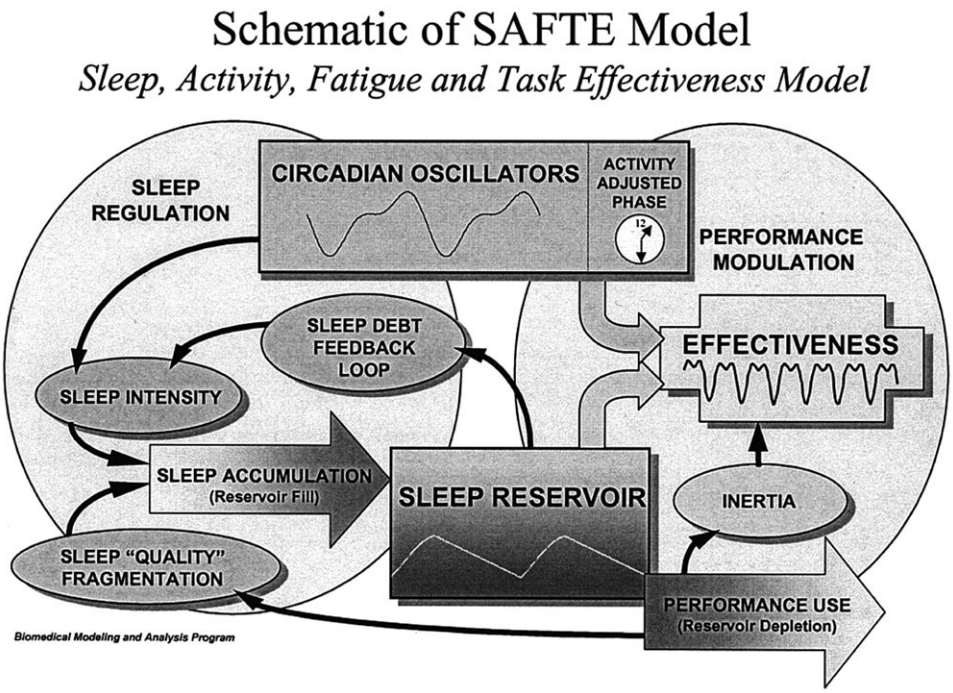

Figure 1. Schematic of SAFTE Model. Reproduced from Hursh et al. [21] with permission from Aerospace Medical Association.

critical transitions in care are a time period of particularly elevated risk [2–4]. Unfortunately, although there are evidence-based treatments for suicide risk [5, 6], there are currently no reliable strategies for delivering appropriate and timely interventions to alleviate an escalation to a suicidal crisis (i.e. intensification of suicidal ideation [SI] or behavior [SB]), making suicide prevention during this time a notable challenge. In support of these efforts, multiple theoretical, conceptual, and statistical models have been developed to anticipate suicide risk to aid in targeted intervention delivery [7]. However, these models generally rely on distal risk factors, that, while effective at identifying general groups of at-risk individuals, cannot identify specifically who is at risk of engaging in SB, and when those risk periods will occur [8]. Thus, there is a pressing need to understand the processes that may indicate proximal risk for suicide that can be implemented in clinical settings in support of broader intervention and prevention strategies.

Diminished sleep health [9] (for our purposes, short sleep time, poor sleep quality, irregular sleep patterns, and/or circadian misalignment) is a known warning sign for SI and SB [10] and thus may be a risk factor that could inform the development of proximal models including interventions to reduce SI and SB [11]. Short or poor quality sleep [12], nightmares [13], and insomnia symptoms [14] can predict SI 1–7 days later. Difficulty sleeping can also predict death by suicide a week later [15], even *after* controlling for depression. Critically, some studies find that atypical sleep precedes increases in suicide risk, but not vice versa [12, 14]. These findings collectively implicate atypical sleep as a proximal risk factor for SI and SB. However, current models lack understanding of the nature of this sleep–suicide risk relationship. Our proposed study aims to address this gap.

As described above, sleep health is an intrinsically multidimensional construct [9]. Yet, existing studies tend to examine single aspects of atypical sleep in relation to suicide risk. Such markers include both perceived disruption (e.g. insomnia symptoms or subjective complaints) and manifest changes to sleep itself (e.g. decreased duration, fragmentation, elongated sleep onset, etc.) [16–18] Regarding sleep timing, epidemiological research implicates eveningness (e.g. the behavioral tendency or subjective preference for going to bed and rising late) in more violent SB as well as more irregular circadian patterns linking to greater SI/SB [19]. Yet, surprisingly few studies have systematically examined the role of sleep timing in suicide risk which given the role of circadian rhythms and sleep timing in mental health writ large [20], may be a major oversight. Taken together, while each of the studies implicates a single aspect of sleep, the lack of a structured multidimensional approach consistent with a sleep health framework intrinsically limits the applicability of this data to understanding the heterogeneous and temporally structured patterns of SI/SB known to be at work [21].

Existing models attempt to explain this link between sleep and suicide via psychological constructs (e.g. sleep-related exhaustion), yet the full cognitive mechanisms involved remain elusive [22]. Thus, with this backdrop of sleep health as a complex determinant of SI/SB, we turn to a core proposal that atypical sleep increases the proximal risk for SI and SB via deleterious impacts on daily functioning. The impact of sleep disruption on waking behavior is well known [21]. For example, even subtle erosion of sleep over multiple days can dramatically impact alertness [23]. Sleep loss alters nearly every higher-order brain system [24]. Sleep disruption itself has even been positioned as a transdiagnostic factor and intervention target across psychiatric conditions [25]. Given that the definition of atypical sleep may vary across studies, quantifying its impairment in a way that integrates disparate multidimensional sleep health components is of paramount interest for optimum risk detection.

To address this conundrum of precise quantification of a multidimensional framework, we propose to leverage a mathematical model that integrates the disparate forces regulating

sleep health: the Sleep Activity, Fatigue, and Task Effectiveness (SAFTE) model (see Figure 1) [21]. SAFTE, a biomathematical model, originates from and is extensively validated by the U.S. Army Research Laboratory. Original SAFTE was conceived to aid in reducing operator errors in mission-critical scenarios (e.g. Department of Transportation, Federal Aviation Administration, U.S. Air Force). SAFTE takes multiple nights of sleep–wake behavioral patterns as input, and multiple underlying processes including sleep homeostasis accounting for accrued "sleep debt" as well as the intrinsic circadian process which together make up the two-process model of sleep–wake regulation [26] while modeling moment-by-moment levels of sleepiness and sleep inertia. By overlaying these processes upon an individual's sleep–wake history, the model estimates moment-to-moment (in 1-minute resolution) fluctuations in cognitive effectiveness [21]. The SAFTE model has been extensively validated using a wide battery of cognitive tests, with results showing that worse SAFTE scores correspond to decrements in performance on a range of tasks, suggesting SAFTE indexes changes in cognitive effectiveness in general [21]. Not only can these estimates be considered retrospectively across a multinight sampling window to probe the cognitive state which coincided with a prior event, but they can be projected into the future to estimate the within-subject consequence of future sleep–wake scheduling over multiple days.

Our study will be the first to apply the SAFTE model framework outside vigilance readiness as a proxy for vulnerability to suicide risk. Our rationale is rooted in a conceptual model that we have previously posited pointing to a cascade of downstream cognitive and affective processes impacted if sleep-dependent vigilance is compromised [27, 28]. Our model presupposes that sleep-related cognitive fatigue impairs cognitive control processes implicated in SI and SB that regulate emotions and determine goal-directed behavior [29]. Thus, we expect that lower SAFTE scores will *dynamically* exacerbate attributes of risk states that intensify proximally to SI/SB, especially within suicide-relevant contexts.

A growing body of evidence supports our claim that sleep disruption increases suicide risk through downstream consequences to cognitive, affective, and social functioning. Shorter sleep duration over multiple consecutive nights predicts impairments in cognitive processes (i.e. cognitive control; attentional biases) [30] that are theorized to proximally increase the risk for SI/SB [31, 32]. Moreover, shorter sleep duration is associated with greater negative affect within stressful contexts [30], noteworthy as there are clear emotional precipitants to increases in SI and SB [33–38]. Longer sleep onset latency, shorter duration, and insomnia have also been linked with increased negative peer and interpersonal relationship perceptions [27, 39] and harmful relational behaviors [30, 40] during stressful social situations. These findings are notable as patient distress due to isolation from social supports and conflict frequently occurs in the days prior to SB [41–43]. Research with high-risk adolescents has integrated these distal and proximal social processes in online social messaging data (i.e. text messages, Facebook, Instagram, and Twitter), with distal childhood maltreatment predicting conflictual messages and sending symptomatic messages on days of SI/SB [44]. However, no study to date has comprehensively examined interrelationships between atypical sleep, these risk processes, and SI and SB. We propose to use SAFTE as a marker of reduced cognitive status resulting from an individual's specific multidimensional sleep–wake patterns which may track not only these proposed downstream cognitive, affective, and social processes but the suicide risk they expose.

If successful, our framework may also uncover particular neurophenotypes particularly prone to both more atypical sleep and greater suicide risk. For example, research indicates that trait-level tendencies toward risky decision-making [45] as well attention and impulsivity [45], and emotional reactivity and regulation [46, 47] may distinguish risk for SI/SB. We propose these trait-level differences contribute to SI/SB by further exacerbating sleep health and, through doing so, altering the sleep–wake behaviors experienced [30, 48] and the vigilance-gated cognitive ability resulting. Using a marker of this sleep–wake-resulting cognitive

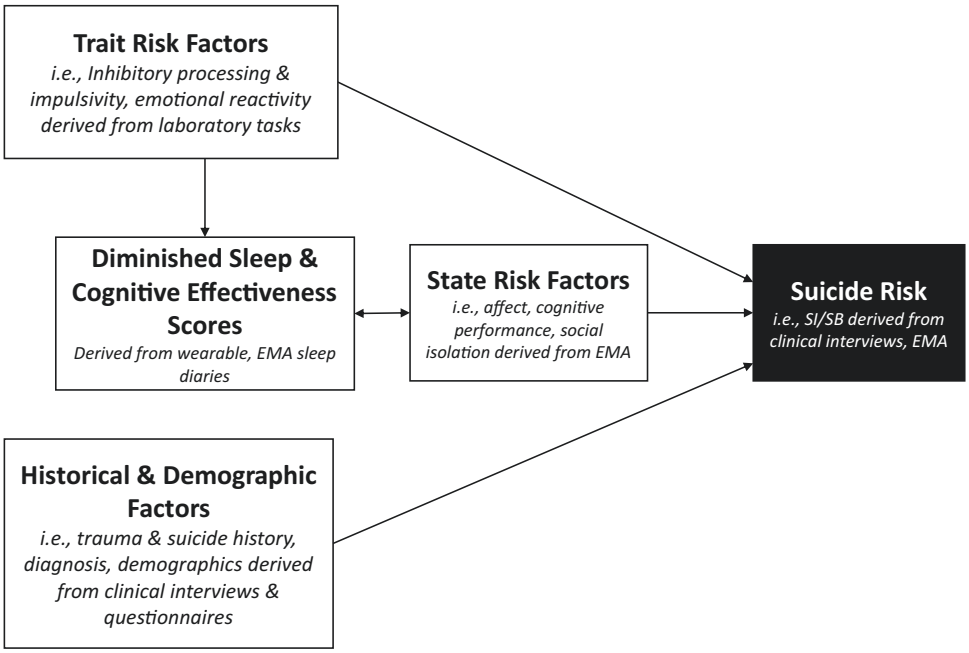

Figure 2. Proposed conceptual model.

state, like SAFTE, to characterize moment-by-moment risk may—in our view—aid in distinguishing these phenotypic differences.

Current Aims

The current paper describes the methods and research protocol for a study that seeks to elucidate the nature of the sleep–suicide relationship and develop an integrated model of proximal suicide risk (see Figure 2) building on the literature described above. Our first aim is to characterize relationships between cognitive effectiveness (operationalized by the SAFTE score derived from actigraphic sleep–wake patterns) and SI over time among psychiatric inpatients admitted for SI or SB following hospital discharge. We expect that poorer cognitive effectiveness (i.e. lower SAFTE scores) will be associated with an increased risk of momentary SI, especially during moments when an individual's cognitive effectiveness is lower than it typically is (H1A). We will also seek to leverage the moment-by-moment resolution of the SAFTE metric to identify the time window within which cognitive effectiveness optimally predicts SI (Exploratory H1B). Finally, we hypothesize that the addition of state-level risk factors will improve the prediction of momentary SI (H1C). Our second aim is to characterize relationships between cognitive effectiveness and state and trait suicide risk factors. We expect that phenotypic trait-risk factors will be associated with lower average levels, greater variability, and lower inertia of cognitive effectiveness scores over time (H2A). We also expect that lower cognitive effectiveness will be associated with worse state suicide risk factors, and vice versa (H2B). Finally, our exploratory third aim is to develop an initial integrated, sleep-based model of suicide risk that can be replicated and validated in future studies. This model will combine cognitive effectiveness scores with measures of state and trait suicide risk factors to develop a model of proximal suicide risk.

Methods and Analyses

Participants

Approximately 240 participants will be recruited for this study, reflecting a target sample size of 200 plus 20% over recruitment to account for expected attrition. Participants will be recruited from the psychiatric inpatient units at Butler and Kent Hospitals in Providence, RI. Participants must have been hospitalized due to SI or SA in the week prior to admission, and physicians must consent for the study team to approach the patient. They must also be aged 18–50 to control for brain differences in aging. They must also be able to speak, read, and understand English well enough to complete study procedures, and be comfortable with the use of smart-device technology. Exclusion criteria include current psychotic symptoms or cognitive impairment severe enough to impair adequate participation in study procedures. Individuals with a Bipolar I diagnosis are also ineligible to participate due to qualitative differences in sleep processes.

Procedures

Study participation will involve the collection of multimethod data across several study appointments. At baseline, we will use laboratory and clinical assessments to phenotype trait-risk factors. We will assess state risk factors and suicide outcomes during a 4-week intensive longitudinal assessment period following discharge from a psychiatric inpatient unit, consisting of ecological momentary assessment (EMA) coupled with digital phenotyping methods and actigraphy using a wearable device. Follow-up assessments will be conducted at 1-, 3-, and 6-month post-discharge to collect suicide outcome data and other measures of interest.

Screening and enrollment.

Following consent from the treating physician, research assistants will screen newly admitted patients' charts using the electronic health record. Screening will be conducted under a Protected Health Information waiver obtained through the Butler Hospital Institutional Review Board. Patients meeting initial inclusion and exclusion criteria will be approached by research staff and provided a description of the study. Research staff will carefully explain all aspects of the study to potential participants, including risks and benefits, its voluntary nature, and the expected duration of participation. Patients who provide written consent will complete additional study screening procedures to confirm eligibility. Suicide-related eligibility criteria will be verified by the Columbia Suicide Severity Rating Scale [49], diagnostic criteria by the Structured Clinical Interview for the DSM-5 [50], and treatment utilization by the Treatment History Interview-4 [51].

Baseline Assessment.

Participants will complete a 3-hour baseline assessment, including clinical interviews and self-reports, and laboratory assessments with concurrent psychophysiology. Assessments will be scheduled 2–3 days post-admission to lessen the impact of distress caused by hospitalization and will occur during periods of inactivity in the inpatient units. At the conclusion of the baseline assessment and post-discharge, participants will be given the wearable device and complete a brief training about how to install and use the mobile application that delivers EMA.

Intensive Longitudinal Assessment Period.

During the 4-week intensive longitudinal assessment period, participants will wear a consumer-grade activity monitor (the Readiband, Fatigue Science, Vancouver, BC, Canada). They will also be administered five brief EMA surveys per day that take approximately 2–5 minutes to complete. Two of these surveys serve as sleep diaries and are administered via morning and evening check-in surveys. The remaining three surveys are prompted at random intervals during the day. Finally, participants will complete an Emotional Go/No-Go (EGNG) task delivered via EMA once per day.

Follow-up Visits.

Follow-up visits will take place at 1-, 3-, and 6-months post-baseline. During these visits, participants will complete a brief battery of self-report and interview assessments (see Table 1). Participants will return the wearable device at the 1-month follow-up either in-person or via mail. All assessments were designed to be able to be administered remotely if needed to accommodate participant needs, and to adhere where appropriate to public health guidelines pertaining to the coronavirus disease 2019 (COVID-19) pandemic.

Description of data collection procedures

Clinical interviews and self-reports.

Participants will complete a series of self-report assessments and clinical interviews at baseline and follow-ups (see Table 1) to assess relevant clinical symptoms, phenotypes, aspects of sleep health, and study outcomes.

Table 1. Self-report and interview assessments

		Time point			
Construct	**Source**	**BL**	**1 m**	**3 m**	**6 m**
Distal risk factors					
Life experiences and sample characteristics					
Prior SI and SB	Columbia Suicide Severity Rating Scale [49], Modified Scale for Suicide Ideation [52]	✔	✔	✔	✔
Psychiatric diagnosis	Structured Clinical Interview for the DSM-5 [50], electronic health record	✔			✔
Trauma history	Childhood Trauma Questionnaire [53]	✔			
Hopelessness	Hopelessness Scale [54]	✔	✔	✔	✔
Borderline traits	McLean Screening Instrument-BPD [55]	✔			
Substance use	Alcohol Use Disorders Identification Test [56], and Drug Use Disorders Identification Tool [57]	✔	✔	✔	✔
Depressed mood	Beck Depression Inventory-II [58]	✔	✔	✔	✔
Suicide risk factors	Interpersonal Needs Questionnaire [59], Acquired Capability for Suicide Scale [60]	✔	✔	✔	✔
Self-report trait phenotypes					
Reward sensitivity	Behavior Inhibition/Behavior Activation Scale [61]	✔			✔
Impulsivity	UPPS-P Impulsive Behavior Scale [62]	✔	✔	✔	✔
Emotion regulation	Difficulties in Emotion Regulation Scale [63]	✔	✔	✔	✔
Sleep					
Sleep disorders	Sleep Disorders Screener [64]	✔			
Perceived sleep disruption	Disturbing Dreams and Nightmare Severity Index [65], Insomnia Severity Index [66]	✔	✔	✔	✔
Sleep phenotypes	Pittsburgh Sleep Quality Index [67], Morningness-Eveningness Questionnaire [68], Munich Chronotype Questionnaire [69]	✔	✔	✔	✔
Suicide and related outcomes					
SI, SB, and NSSI	EMA, Columbia Suicide Severity Rating Scale [49], Self-Injurious Thoughts and Behaviors Interview [70]	✔	✔	✔	✔
Treatment history	Treatment History Interview-4 [51]	✔	✔	✔	✔

BL = Baseline.

Laboratory-based assessments.

Following clinical interviews and self-reports, participants complete 90 minutes of laboratory-based assessments in a quiet room dedicated to that purpose as part of the baseline assessment to measure trait constructs. We will counterbalance tasks to reduce systematic confounds from task sequencing. Behavioral indices will be calculated from the Iowa Gambling task and the EGNG task. Event-related potentials will be recorded during the EGNG and the Emotional Reactivity and Regulation Task.

Decision-making.

The Iowa Gambling Task [71] is a computer-administered, behavioral measure that will be used to assess risky decision-making. Participants have four decks of cards, are instructed to choose a card from any deck, and that they can switch between decks as often as they like. Participants are given $2000 in-game currency to start and told to maximize their profits over 100 trials by selecting cards from one of four decks [72]. Each card chosen results in either the winning of a hypothetical monetary reward amount or a monetary win followed by a loss. Decks A and B are termed "disadvantageous," while Decks C and D are "advantageous." Outcome measures include (1) total net score (difference between total advantageous and total disadvantageous selections) [71] and (2) total net score on reward and total net score on punishment variants (to identify reward and punishment sensitivity and learning) [73]. The Iowa Gambling Task (IGT) has been used extensively to evaluate decision-making in many neuropsychiatric conditions [74], and in relation to suicide attempts (SAs) [75] and substance use [76, 77].

Attentional Bias and Response Inhibition.

An EGNG task for event-related potential studies [78] will be used to measure attention (e.g. emotional processing) and cognitive control (e.g. response inhibition, conflict detection). The task requires inhibitory control to respond/inhibit responses to word features (normal: Go vs. italicized: No-Go), not emotional content (negative vs. neutral vs. positive). The task includes 32 neutral (e.g. umbrella), 32 negative (e.g. misery), and 32 positive (e.g. applause) words from the Affective Norms for English Words [79]. Conditions are matched on word length and frequency of use in the English language. Negative and positive words are matched on valence and arousal. The task begins with 20 practice trials, and contains 5 blocks for each word category, with 20% No-Go trials to establish a prepotent response. Trials will be presented for 1400 ms, with a 750–1000 ms intertrial interval. Word category sequence will be counterbalanced across participants, and specific No-Go words will differ across blocks. We use words (vs. faces) to include stimuli that are personally salient for participants with histories of SB (to elicit prolonged behavioral effects [78, 80]) that can also be easily administered via EMA. We will

calculate behavioral indices of impulsive responding (percentage of no-go trials with commission errors and per emotional condition), attentional bias (median reaction time to go trials and per emotional condition), and event-related potential indices (described below).

Emotional Reactivity and Regulation.

Participants will complete the Emotional Reactivity and Regulation Task [81], an event-related potentials task that has been used to measure indices of emotional reactivity and regulation in suicidal patients [47]. The task will include 20 neutral, 60 dysphoric negative, and 60 positive color images from the International Affective Picture System [82], with positive and negative images matched on valence and arousal. The task will include five blocks. In the first block, participants passively view neutral, negative, and positive pictures (20 pictures of each valence; emotional reactivity block), with picture type distributed pseudorandomly (with no more than 2 pictures of each valence in a row). The following four blocks will be emotion regulation blocks (40 pictures each). Blocks will contain either positive or dysphoric negative stimuli, with instructions to increase or decrease the intensity of emotions evoked by the image (e.g. increase-positive, increase-negative, decrease-positive, decrease-negative), with the order of emotion regulation blocks counterbalanced across participants. Prior to each block, participants will receive instructions to either decrease or increase the emotional response to pictures viewed (without restricting to specific regulation strategies, but with examples of strategies provided) used in prior research [81]. Block instructions ("view passively," "increase," or "decrease") will be displayed for 1000 ms before each trial. A fixation cross will be presented on the screen for 1000 ms. Images will appear 500 ms after the offset of the fixation cross and remain on the screen for 3000 ms, with a 1750–2250 ms inter-stimulus interval. We will calculate event-related potential indices derived from this task (described below).

Electroencephalography data acquisition and processing

Data acquisition.

Continuous EEG activity will be recorded during the emotional go/no-go and emotional reactivity and regulation tasks. EEG activity will be recorded using the 32-channel Biosemi ActiveTwo System, with active electrodes placed via the International 10–20 system. Data will be referenced to a ground formed from a common mode sense active electrode and driven right leg passive electrode. Electrodes placed lateral to the external canthi will be used to detect horizontal eye movements, and electrodes placed above and below the eyes will be used to detect eye blinks and vertical eye movements. EEG and electro-oculogram data will be low-pass filtered using a fifth-order sinc filter with a half-power cutoff at 204.8 Hz and digitized at 1024 Hz with 24 bits of resolution. Stimuli will be presented on a flat-panel display using E-Prime, with behavioral responses collected with a Psychology Software Tools Chronos response box linked to E-Prime.

Data Processing.

Offline data processing will be performed in Matlab 9.2 (The Mathworks, Inc.) using the EEGLAB [83] and ERPLab [84] Toolboxes. Data will be re-referenced to average mastoids, and bandpass filtered from 0.1 Hz to 30 Hz. Independent component analysis will be performed to identify and remove components associated with eyeblink and eye movement activity, as assessed by visual inspection of waveforms and scalp distributions of components. EEG data will be segmented for each trial using settings specific to each task (emotional go/no-go task: 200 ms before and 800 ms after stimulus response; emotional regulation and reactivity task: 200 ms before and 1700 ms after stimulus onset) with a baseline correction of 200 ms. Segments of data containing artifacts will be removed by means of semi-automated ERPLab algorithms [85].

Event-related potential components.

For the emotional go/no-go task, we will extract the P3a (indexing attention to stimuli valence, and inhibitory control), the average activity at 300–600 ms post-stimuli (at frontal or parietal sites), and the N2 (indexing conflict detection) at 200–350 ms post-stimuli (at frontal sites) [86]. For the emotional regulation and reactivity task, we will extract the Late Positive Potential, the average centroparietal activity between 400 and 1000 ms [87, 88].

Estimates of daily sleep–wake rhythms.

Actigraphy will be used in conjunction with EMA-based sleep diaries (described below) to provide an estimate of sleep–wake patterns. Due to our focus on SAFTE as an estimate, we will use the SBV2 ReadiBand (Fatigue Science, Honolulu, HI) wrist-worn actigraph device specifically tuned for implementing this biomathematical model [89]. The ReadiBand will be worn on the nondominant wrist. The ReadiBand contains a 3D accelerometer sampled at 16 Hz. Activity data are collected in 1-minute epochs. Participants receive their watch immediately upon hospital discharge and the data are wirelessly downloaded to an iPad at the 1-month follow-up.

Nonwear windows are detected by the Readiband software and together with sleep diaries used to audit the activity estimates. We will use Fatigue Science's zero-crossing-mode-derived sleep estimation to derive estimates of sleep parameters, which shows 93% accuracy in classifying sleep–wake periods relative to polysomnography [89]. We will use Fatigue Science's detected and diary-verified nonwear windows to estimate sleep–wake patterns in the open-source GGIR R-package [90]. GGIR is a device-agnostic validated workflow for deriving sleep–wake from the z-axis angle of accelerometer data yet recent advances have allowed the import of count data like that derived from Readiband. While GGIR is ideal for its open-science framework of reproducibility, should GGIR's pipeline prove incompatible with Readiband counts, sleep–wake estimates from Fatigue Science's proprietary software will be used in its place [89]. Momentary EMA prompts will be used to assess compliance with wearing the Readiband throughout the day (assessing battery life, technical issues, wristband on/off), and researchers will communicate with participants to rapidly correct any issues that need to be remedied (i.e. battery dies). Participants are given a charger to charge the watch battery and are reminded by staff to charge it weekly during periods of brief inactivity, so as not to impact sleep monitoring at night (i.e. while showering). EMA-based sleep diaries, together with manual inspection of raw accelerometer data from the Readiband, will be used to confirm daytime naps where present and exclude off-wrist windows. Derived variables include estimations of total sleep time, wake after sleep onset (i.e. total number of minutes that a person is awake after initially falling asleep), number of awakenings, number of awakenings per hour, sleep efficiency (i.e. the ratio between the time a person spends asleep and time in bed), sleep onset latency (i.e. the duration in minutes from attempting to fall asleep to actually falling asleep), sleep onset, onset variance, and sleep–wake time. While we focus on traditional sleep–wake estimated variables in this study, we will also

include exploratory measures of rest–activity cycles derived from the count actimetry data itself, including but not limited to interdaily stability, interdaily variability [91], as well as the most and least active 5 hours (M5/L5), and estimates of circadian rhythmicity in activity (e.g. cosinor-fit acrophase, mesor, amplitude).

SAFTE estimation.

From these sleep–wake estimates, we will then derive moment-by-moment SAFTE scores in 1-minute resolution using the recent SAFTEr package in R [92]. SAFTEr is an open-source implementation of the SAFTE model which can be run on any device-estimated multinight sleep–wake pattern [92]. The SAFTE algorithm requires a 3-day "burn-in" period to generate valid estimates [21]. Thus, our data will implement open-source tools throughout its entire processing pipeline to maximize reproducibility. As above, our analytic plan favors open-science solutions wherever possible; however, the proprietary SAFTE estimates from Fatigue Science serve as a fallback approach should unforeseen issues arise.

Ecological momentary assessment

EMA design.

EMA will be administered through Ilumivu's HIPAA-certified mEMA system, which provides a cross-platform (iOS and Android) application for the delivery of multiple simultaneous scheduled EMA protocols. Participants will complete random, event, daily, and cognitive task EMA prompts. Random prompts will be administered at random times, three times a day, at least 2 hours apart, and will be available for 30 minutes. Participants will also be trained to self-initiate event prompts (event-cued assessments) when they experience SI or SB urges or engage in SB. Random and event-cued assessments will be identical to facilitate data harmonization and analysis. Participants also will complete a morning and an evening EMA survey with items largely identical to the random and event surveys, but including additional items needed to compute sleep diary metrics. The mEMA app, installed on participants' own phones, will regularly upload data to the Ilumivu servers using encrypted communications. Uploaded data will be viewable only by the research team.

EMA Items.

The EMA protocol will be informed by our prior EMA studies of suicide and self-harm behavior [34, 93–95]. PANAS-X [96] derived items assess current affect and we will use items derived from the Response Styles Questionnaire [97] to quantify brooding as a metric of repetitive negative thinking. Questions will also assess for substance use, recent stressful situations, and social engagement/isolation. We will also ask questions about napping during the day and consumption of caffeine and other substances (i.e. alcohol, cannabis) that can impact alertness. We will use a series of questions derived from the Modified Scale for Suicide Ideation [52] and the Columbia Suicide Severity Rating Scale [98] to assess for current and recent SI and SB. All items ask about "right now," with the exception of the items that ask about SB, naps, and medication consumption, which ask about the last 2 hours. Our battery includes an integrated safety protocol to direct participants to their treatment team and/or emergency services should we identify suicide risk.

EMA-based Sleep Diary.

One additional morning assessment will serve as a sleep diary, assessing subjective sleep quality, nightmares, medication use, caffeine consumption, and treatment utilization from the prior day. There will also be an evening assessment to ask about behaviors that can impact sleep prior to bed (i.e. substance use, sleep medications, daily naps). During random EMA prompts (described above), we will also ask questions about napping and consumption of caffeine and other substances (i.e. alcohol, cannabis) in the prior 2 hours that can impact alertness.

EMA-based Cognitive task.

We will deliver an adapted, 3-minute version of the EGNG task via the mEMA system once per day. We chose to administer the task once per day to reduce practice effects and improve participant response rates. The task will be administered equally between morning, afternoon, and night periods to capture circadian variation in cognition. Participants will complete three practice trials, followed by a neutral, negative, and positive word 32-trial word block (three blocks). Blocks will be randomly selected from three possible blocks per condition (with the same words, but different words as no-go trials to reduce practice effects), and blocks will be counterbalanced across administrations. Reaction times and commission errors will be calculated overall and within emotional conditions to capture attentional biases and inhibitory control per condition. Consistent with procedures used in studies with EMA tasks [99], after completing the task, we will assess via a single item if participants were interrupted during the task. We will also examine device usage telemetry through mEMA (sensors, mEMA as app focus) and Sochiatrist (incoming and outgoing texts and social media) to identify task distraction and will flag tasks for removal when response times are more than two standard deviations from the participant's mean.

Online social networking and text messaging.

The Sochiatrist, "social psychiatrist," application will be used to collect online social networking and text message data. The application facilitates retrospective data extraction, in this case encompassing messages occurring for the month prior to baseline and throughout the EMA period, and may be used with different device types, operating systems (i.e. iOS, Android, web), and online social networks (e.g. text messages, Facebook, Instagram, Twitter, WhatsApp) [100, 101]. Online social networking data will comprise text-based direct messages, group chats, or the participant's own public posts through SMS/MMS text messages, iMessage, Instagram, Facebook Messenger, SnapChat, Discord, X (formerly known as Twitter), WhatsApp, and posts for those platforms, for example, Tweets or "wall comments." Some data may not be available for participants who do not use a mobile device or a particular service. Sochiatrist extracts data with consented access through the participants' accounts and devices and therefore does not depend on API permissions; upon data collection, it removes nontext content, and replaces all names with de-identified numeric codes. Downloaded data are temporarily stored on the study computer while Sochiatrist strips participant identifiers. The original file will be securely deleted after the de-identified file is created. All data are timestamped for linkage to other data. Sochiatrist data includes (1) sentiment-based features derived from a compound score of sentiment, ranging from +1.00 to −1.00, as calculated via the VADER sentiment lexicon [102]; (2) content-independent features calculated based on messaging metadata (e.g. total messages sent) that do not require the text of a message or information about sender or recipient; and (3) content-dependent features including counts of specific words and phrases.

Compensation.

Participants enrolled in the study will be paid for scheduled assessments. They will receive $50 for the Baseline assessment, $25 for the 1-month assessment, $50 for the 3-month assessment, and $100 for the 6-month assessment. Participants will also receive $1 for the completion of each randomly cued EMA (3/day) as well as $1 for each of the morning and evening sleep assessments. Participants will also be compensated $2 for each EMA cognitive task that they complete (1/day). This results in a total EMA compensation across all 4 weeks of $196. Participants will not be compensated for any event-cued assessments to reduce the incentive to complete assessments for compensation only. We will provide a $54 bonus for participants who complete at least 75% of the total assessments. In total, between scheduled assessments ($225), EMA ($196), and the EMA incentive ($54), participants can earn up to $475 for participation over 6 months, which we believe will aid retention but is not coercive.

Data analysis plan

Data management and confidentiality.

All staff with access to participant data and identifying information will be trained in the management of sensitive clinical information. Data will be stored on secure servers, which undergo daily backups. Participant identifying information will be stored separately from study data in a password-protected database. Any paper records will be stored in a locked file cabinet within a locked office. To ensure the reliability and validity of interview assessments, all interviews, assessments, and sessions will be, with participant consent, audio recorded.

EMA data will be collected via the mEMA mobile application, an HIPAA-compliant platform for the collection of EMA data and secure transmission of those data to a cloud-based central server with dedicated data backup. The app protects participant data by temporarily storing participant responses on the device in an encrypted database. Transfer of data to the cloud is through an encrypted secure socket layer connection that cannot be read even if intercepted by a third party (i.e. a man-in-the-middle attack). Only study personnel will be able to access EMA responses.

As noted above, sleep and activity data will be collected with the Fatigue Science Readiband. There is no personally identifiable information stored on the device. Should a participant's ReadiBand be lost or stolen, data on the device will be inaccessible without the username and password linked to the device. However, if accessed, the only data that could possibly be disclosed would be activity and sleep data, which is the same kind of data any commercial sleep or activity monitor user might produce. Data will be downloaded to a secure tablet device at the end of the study. Data will be downloaded to a secure tablet device at the end of the study.

Several steps will be taken to ensure the confidentiality of online social networking data collected via Sochiatrist, as noted above. We will show the de-identified file to the participant to verify that the data are correct and that they agree to share their data. We will record if participants refuse to share online social networking data as a potential covariate in study models. The original file will be securely deleted after the de-identified file is created.

Primary outcomes.

Momentary SI, measured as a continuous outcome via EMA, with 0 representing no ideation and a value of 1–5 representing an increasing severity of ideation, will be the primary outcome for Aim 1. The primary outcome for Aim 2 will be SAFTE scores, which will be preprocessed and averaged across 1-hour epochs during the participant's wake period. The primary outcome for Exploratory Aim 3 will be a composite score of suicide risk comprised of subscales calculated from the Columbia Suicide Severity Rating Scale [49], including SI intensity and all SBs (i.e. suicide attempts, aborted, interrupted, and preparatory acts), the presence of SI and SB events reported in the weekly EMA assessments, and suicide deaths. This approach facilitates a data integration and reduction approach that captures important aspects of suicide phenomenology and is designed to approximate a cumulative risk for suicide based on several variables, thereby increasing power, and mimicking operationalizations of suicide risk used in the broader literature [103]. A similar approach was previously used in prior research [103], where 21% of participants made an SA at 12-month follow-up, but more than twice that number (46%) reported an event based on a composite measure.

Analytic overview.

Prior to analysis, all data will be checked for errors and examined for statistical assumptions and relevant estimators will be employed as appropriate (i.e. logit, Poisson, zero inflation, etc.). All estimates will be accompanied by 95% confidence intervals (CIs). We will employ MPlus MPlus [104] due to its ability to test and fit complex models using a combination of categorical, count, and continuous variables as well as R and SAS to test models not supported by MPlus. We will adjust for multiple testing to account for false discovery rates using step-down procedures where appropriate [105, 106]. Ambulatory data from the Readiband will be preprocessed, reduced into epochs, and time aligned with EMA survey timestamps to permit time parameterization for statistical models. Psychometric properties (i.e. reliability, variance explained, model fit) of latent constructs will be carefully evaluated and if specific items do not load well on hypothesized constructs, alternative models will be employed using observed indicators. We will determine the reliability of constructs such as affect using multilevel factor analyses at both the state and trait levels. If a construct is not well represented as a single value, we will analyze variables separately. All time-varying variables will be disaggregated into within- and between-persons components to ensure that we are able to adequately separate variance related to stable "trait"-level characteristics (between-person variance) as well as temporal fluctuations and "state"-level characteristics (within-person variance).

Relevant covariates.

Analyses will consider the influence of potentially relevant covariates (i.e. age, gender, treatment adherence, past SA, sleep medication usage, and substance use). We will consider relevant characteristics captured via EMA such as response latency, situational context, day of week/time of day, adherence, etc. For all longitudinal analyses, we will assess for time trends and stationarity, including cyclicity within the day and across the week, as well as time between EMA prompts. As sleep/circadian patterns vary developmentally, we will also stratify analyses by age group where appropriate to examine whether relationships in our study vary as a function of developmental period [107].

Missing Data.

We will closely monitor missing data and study attrition throughout the project. We will apply missing data procedures where appropriate, as Mplus permits both frequentist (full-information

maximum likelihood) and Bayesian estimation for models with missing data. Prior to analyses, all data will be examined for patterns and mechanisms of missingness to ensure missing data approaches are appropriate. Given the high potential for data to be nonrandomly missing (i.e. those experiencing acute distress may be less likely to complete assessments), we will carefully evaluate compliance rates and missing data as a potential predictor of our primary analyses. We will also evaluate whether there are other demographic or key differences among participants with lower compliance and/or among those who drop out early relative to those who don't.

Sample size calculation.

We conduct all power calculations with the assumption that a portion of our full sample size ($N = 240$) may be lost to attrition or other technological complications. Therefore, power was evaluated assuming that we will have data for 200 participants and we will overrecruit.

In Aim 1, the primary outcome (SI) will be measured via EMA on a continuum, with 0 representing no ideation and a value of 1–5 representing an increasing severity of ideation. We will use a general linear model (GLM), which flexibly accounts for repeated measures within person over time. To determine adequate power to test Aim 1 hypotheses, a series of simulations with 5000 replications for our most conservative two-level models were conducted consistent with empirical recommendations [108]. Specifically, the model contained a Level 1 predictor, representing the disaggregated momentary-level SAFTE score (within-person effect), and a Level 2 predictor, representing the person-centered SAFE score (between-person effect). Both predictors were assumed to have a standard normal distribution. The intraclass correlation (ICC) was assumed to be 0.3, the variance of the slope was set to 0.09, and the intercept–slope covariance was set to 0. Simulations also assumed a Type 1 error rate of 0.05 with a Level 1 sample size of at least 28 repeated assessments and a Level 2 sample size of $N = 200$ participants. These sample sizes were chosen because, while Aim 1a/1b will leverage momentary repeated measures within each day (with a max of four surveys per day for 28 days), Aim 1c will be conducted on the day-level aggregated data and thus will have a maximum number of 28 repeated measures per person. Thus, we decided to take a conservative estimate of power assuming we only have 28 repeated measures for 200 people, though we anticipate having a much larger number of repeated measures for Aim 1a/1b, which will provide greater power. Based on these simulations, we are well-powered (80%) to find effects as small as 0.08 (a very small effect) for the within-person standardized effect of SAFTE and an effect as small as 0.20 (a small-moderate effect) for the between-person standardized effect of SAFTE on momentary SI.

For Aim 2, which is concerned with the hourly SAFTE score as the primary outcome, we rely on a recent Monte Carlo (MC) simulation study [109] showing that adequate power (>0.80) for Dynamic Structural Equation Modeling (DSEM) of a single process in which the random mean, autocorrelation, and residual variance are regressed on a predictor (with moderate effects) in a sample of $N = 100$ and as few as 50 repeated measures ($T = 50$). While it is difficult to provide a priori estimates of power for DSEM due to the complexity of dynamic multilevel autoregressive models, in the proposed study, SAFTE scores for $N = 200$ individuals will be assessed continuously each day. We will extract 12 values each day, resulting in a total of $T = 336$ per person. Under the MC simulation-based guidelines and assuming $\alpha = 0.05$ (i.e. Model 6) [109], with $N \times T$ of >67 000 observations, we are very well powered for estimation of the dynamic processes estimated in DSEM. For Aim 2b, models are set up identically to Aim 2a; however, these analyses are conducted at the day level and therefore contain $T = 28$ per person ($N \times T = 5600$). Even with $T = 28$, simulations demonstrate very good model performance for models with $N = 200$ and T as low as 10. We refer readers to Asparouhov et al. [110] for comprehensive coverage of power consideration for DSEM.

The primary outcome for Exploratory Aim 3 will be a composite score of suicide risk (see *Primary Outcomes* section), and power for calculations was based on effects observed in prior research. In one study, attention bias toward suicide-related words accounted for a moderate proportion of variance ($R^2 = 0.18$) in suicide attempts above and beyond common clinical predictors (i.e. history of mood disorder, history of multiple suicide attempt, severity of suicidal thoughts, and both patient and clinician prediction of a future suicide attempt) [111]. To provide conservative estimates, we conducted power for multiple linear regression with a continuous outcome representing our composite indexing suicide risk regressed on 10 predictors (i.e. factor scores extracted from trait and state variables, atypical sleep, and individual life experiences) using G*Power [112]. Assuming $\alpha = 0.05$, with a sample size of 200, we are powered (80%) to detect small to moderate effects (e.g. $f^2 = 0.03$ for individual coefficients or an omnibus $f^2 = 0.08$). This corresponds to the ability to detect as little ~8% total explained variance in the suicide outcome composite.

Data analyses

Our primary outcome for Aim 1 is momentary SI and the goal is to characterize relationships between SAFTE scores and SI over time. To evaluate this aim, we will use GLMs to model the effects of SAFTE scores on SI while accounting for the clustering of repeated measures within individuals during the EMA period. Cognitive effectiveness will be represented via SAFTE scores, which are preprocessed into hourly epochs and time aligned with EMA. SI will be conceptualized as a continuous outcome (with 0 representing no ideation and 1–5 representing increasing severity). Conceptualizing ideation severity rather than simply the presence/absence of ideation will facilitate an outcome with more variability and greater spread. Given that our sample comprises high-risk individuals, we anticipate that most will endorse some degree of SI during the study. All time-varying independent variables, such as SAFTE scores will be disaggregated [113] via grand mean and person mean centering to evaluate *within-person effects* (e.g. how someone looks relative to themselves) while controlling for *between-person effects* (e.g. how someone looks relative to others). For **Aim 1a**, the average SAFTE score during the 2 waking hours preceding the EMA survey will be extracted and aligned with EMA data via the timestamp. The between-/within-components [113] of SAFTE will be entered into the model to assess whether lower within-person SAFTE scores (representing moments when individuals have better/worse cognitive effectiveness than they normally do) are associated with higher SI while controlling for whether individuals who, on average, have lower SAFTE scores relative to others tend to report higher SI. For Aim 1b, to identify the optimal time window for which SAFTE scores are most salient for predicting SI, we will vary the lag used to derive SAFTE scores from 12 to 48 hours prior to the EMA prompt. To ensure that each of the comparisons is conducted on the same amount of data (i.e. lagging variables by 48 hours results in a smaller number of data points to draw from) and because the derivation of reliable SAFTE scores requires a burn-in period

of around 3 days, only data after the third day will be included to ensure that all models cover comparable spans of time. For Aim 1c, we will integrate our within-person state-level variables, into a model predicting day-level SI. This model will also include day-level SAFE score (running average of SAFTE scores aligned with the time of the cognitive assessment). To determine whether adding state-level predictors improves the variance explained in SI, we will use nested model comparison tests for a model that includes all predictors (full model) against a model that only includes SAFTE scores (nested model).

Finally, because of the three-day "burn-in" period to converge on reliable estimates of SAFTE scores, we will also explore whether the relationship between SAFTE scores and SI in Aim 1a changes as a function of time using time-varying effects models (TVEM) [93, 114, 115]. In other words, it may take several days for the SAFTE algorithm to converge on initial estimates and this approach allows us to determine if their relationship strengthens over time. TVEM is a nonparametric regression approach used to estimate time-varying regressions (i.e. regression coefficients are not fixed across time but estimated using splines) without assuming a fixed functional form (i.e. models use splines to determine change trajectory) and can be used to explore the possibilities that relationships among variables fluctuate over time. Consistent with our past TVEM work [93], models will be tested for both between- and within-subjects centering of the data to evaluate the relative strength of each approach.

Our primary outcome for Aim 2 is momentary SAFTE scores and the goal is to characterize dynamic relationships between SAFTE scores and state and trait-risk factors consistent with our hypothetical model (see Figure 2). To evaluate this aim, we will utilize dynamic structural modeling (DSEM) [110, 116, 117], a multilevel extension of structural equation modeling (SEM) and utilizes a vector autoregressive model to account for the underlying time series within the repeated measures [110, 116, 117]. DSEM is implemented using Bayesian estimation and treats missing data within specified intervals as randomly missing across blocks of data that are handled using a Kalman filter (robust to ~80% missingness) [110]. This provides a robust approach for evaluating characteristics of person-level metrics (represented as random effects; Level 2) that represent different aspects of time-based relationships [110, 118]: (1) *intensity* (i.e. random mean = person-centered mean level SAFTE score; higher values represent differences relative to each person's average level) and (2) *variability* (i.e. random variance = person-specific variation surrounding mean level SAFTE score); (3) *inertia* (i.e. random autoregression = the person-specific autocorrelation of SAFTE over time; higher values indicate that individuals are resistant to perturbations). DSEM is also able to integrate a within-person (Level 1) cross-lag model in which associations between bivariate processes are tested to evaluate lagged and contemporaneous relations on a momentary basis. In **Aim 2a**, we focus on the estimation of the person-level (Level 2 random effects) intensity/variability/inertia of SAFTE scores over the course of the EMA. Because the SAFTE metric is continuously generated throughout the day/night, we will reduce the data down to 1-hour epochs during participants' waking hours and derive ~12–18 repeated assessments per person each day. In DSEM, person-level metrics of average/variability/inertia of waking-hours SAFTE scores will be regressed on trait-risk factors assessed at baseline in the Level 2 model of the DSEM (i.e. inhibitory processing, impulsivity, emotional reactivity). Prior to analysis, we will de-trend the data to ensure stationarity and we will account for the potential cyclic nature of SAFTE throughout the day by including relevant contrast-coded covariates indicating those cycles as necessary (e.g. morning vs evening hours). Trait-level risk factors will be entered as person-level predictors of the random effects of the mean, variability, and inertia of SAFTE scores over time. In **Aim 2b**, we will use DSEM to estimate a first-order cross-lagged vector autoregressive model to determine the lagged and contemporaneous associations between SAFE scores and day-level state risk factors (i.e. affect, cognitive performance, social isolation). In this approach, SAFTE scores will be aggregated at the day level in order to facilitate modeling of the within-person (Level 1) cross-lagged effects of state risk factors (which are assessed once per day) on SAFTE scores over the course of 28 days.

Our primary outcome for Exploratory Aim 3 is suicide risk and the goal of this aim is to build an integrated model of proximal suicide risk. For this exploratory aim, consistent with our conceptual model (see Figure 2), we will integrate findings from Aims 1 and 2 to identify significant predictors of risk for suicide at follow-up. We will use SEM to assess the relationship between our hypothesized constructs (i.e. trait and state risk factors, diminished sleep, cognitive effect) and suicide risk at 3 and 6 months in separate models. SEM facilitates the modeling of complex relations among both latent and observed variables as well as the explicit addition of covariances to account for relationships among predictors (i.e. to address potential multicollinearity). While we expect 20%–40% of our sample to report SA at the 6-month follow-up (see Sample Size Calculation above), our primary outcome for this aim will be a composite score of suicide risk computed using data from each study follow-up (3 and 6 months). Therefore, we expect to capture higher rates of risk than those observed for SA alone (see *Primary Outcomes* for more detail). Separate suicide risk composites will be calculated for 3-month and 6-month follow-ups to be evaluated separately.

Because of the large numbers of potential predictor variables across our hypothesized constructs (i.e. Figure 2), we will use data reduction techniques (i.e. factor analysis) to create composites of variables that are highly correlated (such as sleep variables to provide an overall index of atypical sleep) to model associations between these key constructs and suicide risk (see *Analytic Overview* for more detail). As this is an exploratory aim, with the goal of integration and hypothesis generations, variable selection will be guided by major, mechanistic findings from Aims 1 and 2 and models will control for life experiences (i.e. gender, trauma history, past suicide attempts). One of the benefits of SEM is that several competing models can be compared through model fit indices to determine the most parsimonious model that fits the data well. We will compare nested models using chi-squared difference tests and will use information criteria to compare across different models. The best-fitting model from this aim will directly inform the theoretical basis of our future work, in which we hope to build, test, and validate a predictive model of proximal risk that is translatable to clinical and real-world scenarios.

Ethics

All study procedures were approved by the Institutional Review Board at Butler Hospital. All sensitive suicide and interview assessments will be supervised by licensed clinical psychologists trained in the assessment battery and with a human subjects certification, who will also be on-call in the event of participant crises. Participant frustration or distress during laboratory procedures will be closely monitored by trained research staff, and participants will be allowed to discontinue participation at any time,

or to complete the laboratory protocol at another time when they feel less distress. To reduce any distress associated with assessments, we have integrated a relaxation period following the completion of laboratory assessments.

Given the foci of our study, it is probable that some participants may disclose SI or other symptoms necessitating immediate psychiatric hospitalization. Participant affect and suicidal and homicidal ideation will be assessed at each patient contact using the Assessment Session Check-In (a modified version of the UWRAP [119]). We have developed an emergency protocol designed to manage exacerbations of negative affect and urges to self-harm, providing research staff with detailed action plans to manage these situations. Should participants report SI with imminent risk of self-harm during our inpatient procedures, we will coordinate our observations with patients' inpatient treatment team and chart our findings. Outpatient assessments will be conducted at Butler Hospital, with a licensed clinical psychologist available for consultation at all times. Any serious psychiatric symptoms identified at follow-up may be quickly and efficiently managed through consultation with Butler Hospital's Psychiatric Assessment Services, where trained professionals can determine if participants require immediate hospitalization. As some follow-up assessments are conducted by phone, research staff will request information about the participant's location; therefore, if participants require immediate hospitalization, we can provide that information to emergency services.

Participants will be informed that study staff will not be monitoring EMA responses. However, if they endorse items via EMA indicative of current high risk, they will be provided a "we are concerned about you" message, containing contact information for crisis services. They will also have a list of emergency numbers available to them on-demand via the EMA platform that they can access should they experience a clinical crisis.

Summary and implications

Integrated models of proximal risk for SI/SB are critically needed to better understand suicide risk and ultimately prevent death by suicide. In this study, we will characterize temporal relationships between atypical sleep, suicide risk factors, and SI/SB episodes to inform the development of an integrated model of proximal suicide risk. The results of this study will significantly enhance our understanding of atypical sleep-associated suicide phenomenology as it exists in the real world as well as to greatly improve our ability to prevent and treat suicidality using traditional and novel, technology-enhanced, interventions.

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Data availability

The principal investigators will deposit study data in the National Mental Health Data Archive (NDAR) at NIMH Data Archive - Data - Collection (https://nda.nih.gov/edit_collection.html?id=3760) for data sharing.

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