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Review Article

Sleep problems and suicide risk in youth: A systematic review, developmental framework, and implications for hospital treatment☆

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ABSTRACT

Objective: Sleep problems are transdiagnostic symptoms that confer significant risk for suicidal thoughts and behaviors (STBs) in adults. However, less is known about the sleep-STB association in adolescence—a developmental period when rates of STBs increase drastically, and sleep problems may be particularly pernicious. This article provides a systematic review of research on the sleep-STB association in youth, an overview of changes in sleep regulation during adolescence that may make sleep problems particularly detrimental for youth, and a discussion of the clinical implications of the sleep-STB association for hospitalized youth.

Method: The systematic review included all longitudinal studies in which sleep problems were examined as prospective predictors of STBs in adolescents (aged 10–24 years). The search was conducted on December 1, 2017 using PsychINFO, PubMed, and Web of Science databases.

Results: Ten studies qualified for inclusion in this review. Of these, seven studies found at least one type of sleep problem significantly predicted a STB outcome.

Conclusions: Although findings are mixed, growing research suggests that sleep problems may be a unique risk factor for STBs in youth. Sleep problems may be particularly important intervention target because they are easily assessed across healthcare settings and are amenable to treatment.

1. Introduction

The suicide rate in youth has continued to rise over the past decade and suicide is currently the second leading cause of death among youth aged 10–24 years in the United States [1]. Rates of suicidal thoughts and non-fatal suicidal behaviors are even more prevalent in this age group. Each year, approximately 17.7% of high school aged youth report suicide ideation (SI; i.e., thoughts of engaging in behavior to end one's life) and 8.6% report at least one suicide attempt (SA; i.e., engaging in self-injurious behavior with some intent to die) [2]. High rates of suicidal thoughts and behaviors (STBs) in youth are alarming given the risk they confer for suicide death [3], the significant functional impairment associated with STBs [4], and the burden STBs place on the healthcare system [5].

Despite decades of research on risk factors for STBs, the field's ability to effectively predict [6,7] and prevent youth suicide [8,9] has not significantly improved. This slow progress may be attributed, in part, to the types and ways risk factors have been examined in prior research. Specifically, previous studies have focused primarily on risk factors that are diagnostic (e.g., psychiatric disorders), distal from STBs (e.g., early childhood adverse events), time-invariant (e.g., demographic variables), and subjectively reported (e.g., self-report questionnaires) [7]. Although useful for identifying groups of individuals at higher lifetime risk for STBs, these risk factors are less useful for short-term risk detection and prediction [7], and some identified risk factors (specifically distal and time-invariant factors) are less amenable to intervention.

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1.1. Sleep problems: a promising risk factor for suicidal thoughts and behaviors

Sleep problems may be one promising category of risk factors for STBs that address some of the aforementioned limitations. Specifically, sleep problems are transdiagnostic and present among a range of psychiatric disorders linked to STBs (e.g., mood disorders) [10], proximal to STBs [11], time-varying over short [12] and long time periods [13], and can be objectively assessed using laboratory-based polysomnography (PSG) or mobile wearable devices (e.g., wrist actigraphy) [14]. Moreover, a growing literature has revealed significant associations between a range of sleep problems and STBs, particularly in adults. Sleep problems (e.g., insomnia) have been linked to STBs as a cross-sectional correlate [15,16] and prospective predictor in adults [17–19]. Importantly, sleep problems appear to precede STBs, rather than STBs leading to sleep problems [12]. Sleep problems may also be a unique predictor of STBs, over and above other STB risk factors (e.g., depression) [18].

To date, eleven prior reviews have been published on the relationship between sleep problems and STBs across the lifespan: [20–30] five were narrative reviews, four were systematic reviews, and two were meta-analytic reviews. Of the previous reviews, eight focused on all age groups, two focused on adults, and only one (narrative review) focused specifically on youth [25]. Although there are notable strengths of these prior reviews, three limitations are important to address. First, most studies included in prior reviews were cross-sectional, which lack the necessary temporal precedence to classify sleep problems as *risk factors* for STBs [31]. Second, prior reviews offered limited discussion of the mechanisms by which sleep problems may increase risk for STBs, which is important for informing STB treatments [32]. Third, most existing research has focused on the sleep-STB link in adults and much less is known about this association in youth. Given the significant changes in sleep architecture during adolescence [33], and the deleterious effects of sleep problems on cognition, interpersonal functioning, and physical health [34], it is important to examine how the sleep-STB association may be particularly robust in youth.

Cross-sectional research in youth has indicated an association between a range of sleep problems and STBs in community [35–38] and clinical samples [39–41]. The types of sleep problems associated with STBs in youth include: insomnia [42], hypersomnia [39], long (i.e., $\geq$10 h) and short (i.e., $\leq$5 h) total sleep time [35], behaviorally-induced insufficient sleep syndrome [36], nightmare disorder [41] as well as sleep disturbances (i.e., broad term encompassing a variety of medically or behaviorally based sleep problems) [39]. The range of sleep problems have been linked to SI [35], suicide planning [35], SA [35], and suicide death [39]. Although the sleep-STB association in youth has been found consistently in cross-sectional research, much less is known about sleep problems as a prospective risk factor for STBs.

Over a decade ago, Liu and Buysse [25] wrote the only review focused on the sleep-STB association in youth. The review concluded that sleep problems are likely to signal increased risk for future STBs in adolescents, but the existing evidence base was limited. At that time only a few studies in small psychiatric samples had examined the sleep-STB association in youth, which limited generalizability of findings and insights into mechanisms. The authors called for large-scale prospective and neurobiological studies of the sleep-STB link, several of which have been conducted since their review was published in 2006. Thus, an updated review is warranted.

1.2. Current review

The goals of this review were: [1] to systematically examine if sleep problems increase risk for STBs in youth (focusing exclusively on longitudinal research in which sleep problems were examined as prospective predictors of youth STBs), [2] to use a developmental framework to explore the substantial changes in sleep during adolescence and to offer suggestions for how these changes may increase risk for youth STBs, and [3] to discuss why assessment and treatment of sleep problems may be important for youth with STBs presenting to the hospital.

2. Method

2.1. Objective

We followed PRISMA guidelines [43] when conducting and reporting the results of this systematic review.

2.2. Search strategy

On December 1, 2017, we conducted a systematic search of the following databases: PsycINFO, PubMed, and Web of Science. Search terms were drawn from previous systematic reviews examining risk factors for STBs that had a focus on longitudinal research [7], sleep problems [28], or youth [44] (full search terms listed in footnote 2[2]). To ensure all relevant articles were included, reference lists of sleep-related review articles and all articles that met inclusion criteria for our review were reviewed. This search resulted in 172 unique abstracts for potential inclusion.

2.3. Study selection

Inclusion criteria were as follows: (1) The study population was aged 10–24 years at the initial assessment (consistent with epidemiological definitions of "youth" [1] and research indicating that neural development continues into the mid-20's [45]); (2) The study used a prospective design to examine the sleep-STB association; (3) At least one sleep-related measure was included at the initial assessment; (4) At least one type of STB outcome was measured at a follow-up assessment; and (5) The study statistically examined the magnitude of association between the sleep problem at initial assessment and STB outcome at a follow-up assessment.

Studies that only included nonsuicidal self-injury (i.e., direct and deliberate destruction of body tissue without any intent to die) [46] or deliberate self-harm (i.e., any type of self-injurious behavior regardless of suicide intent) [47] were excluded, given that these behaviors are distinct from STBs [48,49]. Two authors reviewed all abstracts and then the full articles to determine eligibility for this review. When there was reviewer disagreement during the full article review phase, the article was reviewed by four authors to reach a consensus decision. Based on this study selection procedure, 10 studies met inclusion criteria for this review (see Fig. 1; PRISMA diagram).

2.4. Data extraction and synthesis

The following information was extracted from all included articles: sample size, age of sample, setting of sample recruitment, sleep-related predictors and measures (see Table 1 and Buysse [50] for definitions of sleep-related measures), STB outcomes and measures, duration of follow-up between assessment of the sleep predictor and the STB outcome, covariates examined, findings describing all sleep-STB associations, and the magnitude of the sleep-STB association when available (see Table 2).

[2] The search terms for this review were: (sleep OR "sleep disturbance" OR "sleep disorder" OR nightmare OR dream OR insomnia OR "sleep apnea" OR "sleep disordered breathing" OR "sleep initiation disorder" OR "sleep maintenance disorder" OR "sleep psychology" OR "sleep epidemiology") AND (suicid* OR suicidality OR "suicidal behavior" OR "suicide attempt" OR "suicide death" OR "suicide plan" OR "suicide thoughts" OR "suicide ideation" OR "suicide gesture" OR "suicide threat") AND (longitudinal OR longitudinally OR predicts OR prediction OR prospective OR prospectively OR future OR later) AND (youth OR adolescen* OR teen* OR child*).

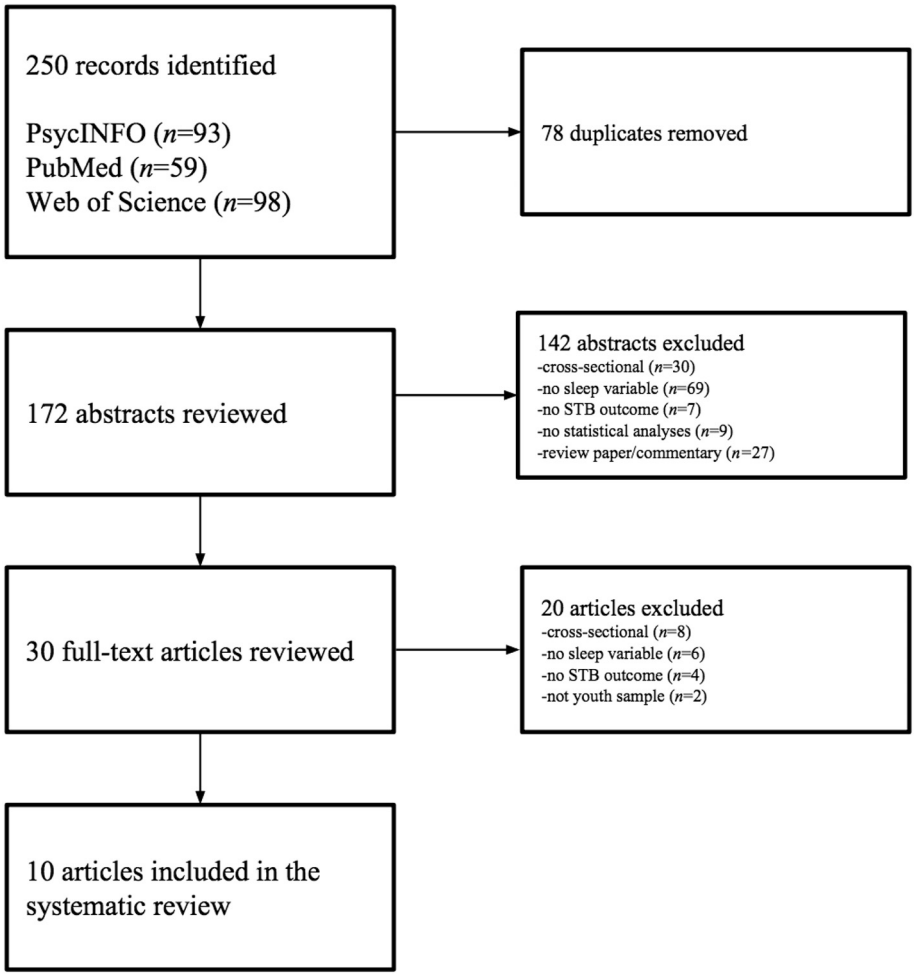

Fig. 1. PRISMA diagram.
STB = suicidal thoughts and behaviors.
Note: No statistical analyses = no statistic comparing sleep problems to STBs.

Table 1
Definitions of sleep parameters.

Chronotype	How an individual's endogenous circadian clock entrains to a 24-hour day.
NREM sleep	Non-rapid eye movement sleep; ranges from light sleep (stage N1 and stage N2) to deep or slow wave sleep (stage N3).
Number of awakenings	Total number of awakenings during the night lasting at least 15 s (excluding the final awakening).
REM latency	Number of minutes from sleep onset to the first occurrence of REM sleep.
REM sleep	Rapid eye movement sleep.
Sleep continuity	Measures of sleep that can be derived from self-report sleep diaries, from actigraphy or from polysomnography. These include sleep latency, wake after sleep onset, total time in bed, total sleep time, number of awakenings, and sleep efficiency.
Sleep latency	Number of minutes to fall asleep, starting from intention to fall asleep. May also be called sleep onset latency.
Sleep stages	Scored stages of sleep derived from polysomnography. These include Wake, Stage N1, Stage N2, Stage N3 and Stage REM.
Slow wave activity	A finer estimation of slow wave sleep that utilizes power spectral analysis to determine the total amount of slow waves during a sleep period regardless of sleep stage.
Slow wave sleep	Stage N3 sleep; may also be called deep sleep.
Sleep efficiency	Total sleep time divided by total time in bed (including the time trying to fall asleep to the final awakening) multiplied by 100% as it is typically reported as a percentage from 0 to 100%.
Total sleep time	Total time asleep during a sleep period.
Wake after sleep onset	Total minutes of time awake during the night after sleep initiation (does not include minutes of sleep latency).

Note: Definitions of sleep parameters are from Buysse et al., 2006 [50].

3. Results

3.1. Study characteristics

For the 10 studies included in this review, samples ranged in size from 50 to 6504 adolescents and included community only (n = 6), clinical only (n = 1), and combined community/clinical samples (n = 3). The mean age at the initial (baseline) assessment ranged from 13 to 19 years-old. The duration of follow-up ranged from 7 days to 10 years. The STB outcomes examined were SI (n = 3), SA (n = 6), or "suicidality" (i.e., combined SI and SA into one variable; n = 2); no studies examined suicide death. With the exception of one study [11], STBs were measured with 1–2 items per STB. It is important to note that the majority of publications that met inclusion criteria for our review

Table 2
Summary of prospective studies examining sleep and suicidal thoughts and behaviors.

Study	*N*	Age[a]	Setting	Follow-up length	Sleep-related measure	STB measure (STB type)	Covariates	General finding	Effect sizes
Bernert, Hom, Iwata, & Joiner, 2017	50	Range = 18–23 years	College campus	7 and 21 days	Disturbing Dreams and Nightmare Severity Index (nightmare frequency and severity); Insomnia Severity Index (insomnia); wrist actigraphy (sleep efficiency, sleep-onset latency, sleep variability, total sleep time, wake after sleep onset)	Beck Scale for Suicide Ideation (SI)	Depression symptoms	Insomnia and nightmare severity each significantly predicted residual change in SI over follow-up. Among actigraphy measures, only variability in sleep timing (i.e., standard deviations of daily sleep onsets and offsets) predicted residual change in SI over follow-up.	(Insufficient information to calculate individual effect sizes as Cohen's d)
Coplan et al., 2000	77	Depression-free normal group (n = 34), M(SD) = 15.4 (1.4) Lifetime depression group (n = 43), M(SD) = 14.4 (2)	Community & clinical (depression sample from outpatient clinic)[b]	9–10 years	Blood (hGH secretion); PSG (amount of delta sleep, delta sleep latency, sleep latency)	SADS-LA rating of ≥4 (suicidality; i.e., SI or SA)	Age	Certain hGH variables (e.g., earlier onset of sleep-related hGH secretion) significantly related to SA over follow-up in the lifetime depression group. No significant associations with PSG variables measured.	Cohen's ds = 0.10–1.12
Goetz, Wolk, Coplan, Ryan, & Weissman, 2001	70	Depression-free normal group (n = 17), M(SD) = 15.1 (1.4) Lifetime depression group (n = 53), M(SD) = 14.6 (1.8)	Community & clinical (depression sample from outpatient clinic)[b]	9–10 years	PSG (REM density, REM latency, REM sleep, sleep efficiency, sleep latency, SWS)	SADS-LA rating of ≥4 (suicidality; i.e., SI or SA)	Age	No significant sleep-STB associations. However, marginal differences shown for first period REM density and sleep period time in lifetime depression group which predicted lifetime suicidality.	Cohen's ds = 0.06–0.62
Mathew et al., 2003	77	Depression-free normal group (n = 34), M = 15.4 (1.4) Lifetime depression group (n = 43), M = 14.4 (2)	Community & clinical (depression sample from outpatient clinic)[b]	9–10 years	Blood (24-hour cortisol secretion)	SADS-LA rating of ≥4 (suicidality; i.e., SI or SA)	Age, gender	Participants in the lifetime depression group with significantly higher levels of cortisol secretion during the period prior to sleep onset predicted SA over follow-up.	(Insufficient information to calculate individual effect sizes as Cohen's d)
McGlinchey & Harvey, 2015	3843	M = 16	Community[c]	6 years	ADD Health Survey (bedtime)	ADD Health Survey (suicidality; i.e., SI or SA)	Age, gender, race/ethnicity, prior suicidality, delinquent peers, parental attachment, receipt of public assistance	A late bedtime did not predict suicidality. However, there was marginal significance for a dose effect, indicating the later the bedtime, the greater suicidality over follow-up when bedtime was coded on continuous scale.	Cohen's d = 0.12
Nrugham, Larsson, & Sund, 2008	2432	M(SD) = 13.7 (0.5)	Community	7 years	K-SADS-PL (circadian reversal, fatigue, hypersomnia, initial insomnia, middle insomnia, non-restorative sleep, terminal insomnia)	K-SADS-PL & Young in Norway Survey (SA)	None	Initial insomnia, hypersomnia, and fatigue predicted SA over follow-up.	Cohen's ds = 0.12–0.78
Rao et al., 1996	19	Range = 12–18	Clinical (depression sample from outpatient clinic)	7 years	Blood (24-h cortisol secretion); PSG (REM density, REM latency, sleep onset)	SADS-L rating of ≥4 (suicidality; i.e., SI or SA)	None	No significant sleep-STB associations.	(Insufficient information to calculate individual effect sizes as Cohen's d)
Roane & Taylor, 2008	3582	M(SD) = 15.83 (1.46)	Community[c]	6-7 years	ADD Health Survey (insomnia)	ADD Health Survey (SA, SI)	Gender	Insomnia symptoms significantly predicted SA, but not SI, over follow-up.	Cohen's d = 0.58
Wong, Brower, & Zucker, 2011	392	Range = 12–14	Community, including high-risk subsample (i.e.,	3 years	Youth Self Report (feeling overtired, nightmares, trouble sleeping)	Youth Self Report (SI)	Age, gender, prior SI, depression symptoms, aggressive behavior, substance use problems,	Having trouble sleeping predicted SI over follow-up. Feeling overtired and nightmares did not predict SI.	Cohen's ds = 0.05–0.49

(*continued on next page*)

Table 2 (*continued*)

Study	*N*	Age[a]	Setting	Follow-up length	Sleep-related measure	STB measure (STB type)	Covariates	General finding	Effect sizes
			parental alcohol abuse)				parental alcohol abuse, parental STB		
Wong & Brower, 2012	6504	*M*(SD) = 15.99 (1.75)	Community[c]	6-7 years	ADD Health Survey (sleep problems– broadly defined)	ADD Health Survey (SA, SI)	Age, gender, race/ethnicity, school grade, depression symptoms, alcohol problems, illicit drug use, chronic health problems, poverty status	Sleep problems predicted SI, but not SA, over follow-up.	Cohen's *d*s = 0.02–0.11

hGH = human growth hormone; K-SADS-PL = Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime version; PSG = polysomnography; REM = rapid eye movement; SADS-LA = Schedule for Affective Disorders and Schizophrenia – Lifetime version; SA = suicide attempt; SI = suicide ideation; STB = suicidal thoughts and behaviors; SWS = slow-wave sleep.

[a] All available age information is presented.

[b] Sample was recruited from a large-scale sleep and neuroendocrine study at the Child and Adolescent Sleep and Neuroendocrine Laboratory (Goetz et al., 1987; Dahl et al., 1992).

[c] Sample was recruited from the National Longitudinal Study of Adolescent Health (ADD Health; Harris et al., 2009).

(*n* = 6) described findings from the same two large-scale longitudinal studies (see Table 2).

3.2. Types of sleep problems examined

Five studies utilized only self-report measures of sleep problems. Each of the following types of sleep problems was examined in one study unless otherwise specified: bedtime, circadian reversal (i.e., inversion of normative sleep-wake tendencies), feeling overtired, hypersomnia, insomnia (*n* = 2), nightmares, non-restorative sleep, sleep problems (broadly defined), trouble falling asleep, and trouble staying asleep. The majority of studies (*n* = 9) measured sleep problems with 1–3 item self-report scales as part of a larger study questionnaire or clinical interview. Only one study used full sleep-specific questionnaires [11]—the Insomnia Severity Index [51] and the Disturbing Dreams and Nightmare Severity Index [52].

Five studies utilized objective methods to assess sleep-related variables: PSG (*n* = 3), 24-hour hormonal processes related to sleep (*n* = 3), and wrist actigraphy (*n* = 1). The specific metrics of sleep using PSG were: amount of delta sleep, delta sleep latency, intermittent awake time, percent rapid eye movement (REM), REM density, REM latency, sleep efficiency, sleep latency, and sleep period time (see Table 1 for definitions). Wrist actigraphy parameters were: sleep efficiency, sleep onset latency, sleep-onset variability, and wake after sleep onset (Table 1). The hormonal processes measured via blood samples were: cortisol and human growth hormone (hGH) secretion over 24 h.

3.3. Sleep problems predicting suicide ideation

Four studies used a sleep-related measure to predict SI. Three studies found a significant relationship between at least one measure of sleep (i.e., general sleep problems, trouble sleeping) and SI over the subsequent 7 days to 5 years. In one study, general sleep problems in early adolescence (12–14 years old) predicted SI five years later, after controlling for other known STB risk factors (e.g., depressive symptoms, alcohol-related problems) [53]. The study did not control for prior STBs. In a second study, trouble sleeping in early adolescence (12–14 years old) predicted SI three years later, above and beyond risk factors such as previous SI and depressive symptoms [54]. One recent study examined the sleep-STB association over a much shorter time period than previous studies. Bernert [11] found that self-reported sleep problems (i.e., sleep variability, insomnia, nightmares) and sleep-onset variability (assessed via wrist actigraphy) predicted residual SI changes over a 7- and 21-day follow-up period, beyond baseline SI and depressive symptoms.

However, not all studies found significant associations. Although Wong [54] found that "trouble sleeping" was predictive of later SI, nightmares and "feeling tired" did not predict SI over the 3-year follow-up period. Another study reported no association between insomnia symptoms in adolescence (12–18 years old) and SI 6–7 years later [55].

Overall, there is mixed evidence for the sleep problems predicting SI in youth. However, the one study that utilized an objective measure of sleep problems (i.e., wrist actigraphy), had a short follow-up period (i.e., 7- and 21-day), and controlled for depression [11] found a significant association, suggesting that specific sleep problems (i.e., sleep-onset variability) may be proximal risk factors for SI in youth (18–23 years old).

3.4. Sleep problems predicting suicide attempts

Six studies used a sleep-related measure to predict SA. Four studies found a significant relationship between sleep (i.e., general sleep problems, insomnia, nocturnal hGH secretion, and 24-hour cortisol secretion) and SA over the subsequent 5–10 years. Self-reported general sleep problems [53] and insomnia symptoms [55] predicted SA over the subsequent 6–7 years, even after controlling for relevant demographic

(e.g., gender) and psychiatric risk factors (e.g., depressive symptoms). Another study found that, in adolescents with depression, insomnia and hypersomnia predicted SA five years later, but the relationship did not hold after controlling for other related risk factors such as hopelessness [56].

Studies using objective sleep-related measures have also produced mixed findings. Two studies found that greater amounts of hGH during the first four hours of sleep [57] (a pattern linked with poorer slow-wave sleep [SWS]) and greater levels of cortisol prior to sleep onset (a pattern linked with sleep disturbance) predicted SA 9–10 years later [58]. Notably, as all adolescents experience dysregulation and increase in hGH during this developmental period and particularly around sleep onset [59], these findings suggest that suicidal adolescents may exhibit even greater dysregulation of hGH near sleep. Activation of the hypothalamic pituitary adrenal (HPA) axis (as indexed by cortisol elevations) prior to sleep onset—a period when the HPA axis is usually inactive—may confer vulnerability for future SA [58], however, the specific mechanism is unknown. Although studies examining sleep-related hormones have been promising, two other studies using PSG, a gold-standard sleep assessment, did not find that objective sleep variables (e.g., REM latency, REM density, SWS) predicted SA 9–10 years later [60,61].

Taken together, this research suggests mixed evidence for the association between sleep problems and SA in youth. The majority of self-report (i.e., subjective) insomnia measures indicated a sleep-STB relationship whereas not all objective sleep measures indicated a sleep-STB relationship. For instance, PSG had null results. This may be due to PSG results attempting to predict SA over a long follow-up periods and therefore objective sleep measures used over short follow-up periods (e.g., as seen in the Bernert [11] study with actigraphy used over a short follow-up period) may be more useful in identifying STB risk factors. Finally, the dysregulation and increase in hGH in adolescence may be a mechanism contributing to decreased SWS (or reduced deep sleep) and consequently, an increase in daytime sleepiness in adolescents [62,63].

3.5. Sleep problems predicting suicidality

Two studies examined sleep-related measures as a predictor of suicidality (a composite category including SI and SA). No sleep-related measure in either study, subjective (late bed time) or objective (REM density, REM latency, sleep latency), predicted suicidality over the subsequent six [64] to ten years [61].

4. Discussion of systematic review

Of the ten studies reviewed, seven studies found that at least one sleep-related measure was a significant predictor of a STB outcome. Significant findings appeared across various sleep predictors (e.g., sleep variability, insomnia symptoms), methodologies (subjective and objective measures), and STB outcomes (SI and SA). However, significant effects were not found across all studies and findings were mixed across the various sleep predictors and STBs examined.

There are six notable findings of this review. First, for SI and SA, sleep problems with greater specificity (e.g., initial insomnia) were more predictive than less specific sleep problems (e.g., feeling tired). Most studies suggested that specific self-reported sleep problems (e.g., insomnia) compared to general sleep problems may be more reliable risk factors for STBs. Second, a range of methods – objective and subjective – have been used to assess sleep problems and when examining within a specific sleep-related method, results were not consistent. Five studies that used only subjective sleep-related measures (i.e., brief self-report questionnaires) [53–56,64] and four studies that used only objective sleep-related measures [57,58,60,61] demonstrated mixed findings. Future research is needed to clarify the optimal method of sleep measurement. Third, studies predicting “suicidality” (combining SI and SA into one variable) were not significant. Previous research suggests the importance of separating suicidal thoughts and behaviors [65,66] and the null suicidality results may be due to different STBs having distinct risk factors. Fourth, the relationship between sleep problems and STBs was demonstrated over both long follow-up periods (i.e., 3–10 years), but more importantly, over relatively short follow-up periods (i.e., 7- and 21-days). However, present findings can only speak to sleep problems as a risk factor for STBs over long intervals, which is a common issue with prospective STB research [7]. The recent work by Bernert [11] suggests that sleep problems may be a promising short-term predictor of SI, thus filling a significant gap in the sleep-STB literature. Fifth, in some of the studies the relationship between sleep problems and STBs held after controlling for a range of known long-term risk factors for suicide, such as sociodemographic characteristics (e.g., age), psychiatric disorders (e.g., depression), and prior SI. This demonstrates that sleep problems may be unique predictor of STBs in youth. Notably, in one study using objective measures (i.e., actigraphy), sleep problems demonstrated unique predictive ability controlling for baseline SI [11]. Sixth, three findings drawn from a single small-scale study ($n = 77$) with a clinical sample suggest a relationship between hormonal processes linked with sleep, such as hGH and cortisol, and future STBs [57,58]. Together, these studies suggest that fluctuations in hormonal processes represent a disruption in a core sleep-onset mechanism (i.e., circadian phase delay), which may increase risk for STBs over the next decade.

This review also identified seven main limitations of prior studies that suggest important directions for future research on sleep problems and STBs in youth. First, the evidence base for the sleep-STB connection in youth is largely based on two samples of youth: the National Longitudinal Study of Adolescent to Adult Health ($n = 3$ studies) [53,55,64] and a PSG and neuroendocrine study ($n = 3$ studies) [57,58,61]. Therefore, replication in additional and more diverse samples is needed. Second, sleep problems and STBs were primarily measured via self-report using only a few items (an exception being the work by Bernert [11]). Given the known limitations and threats to validity of single-item assessments of STBs [67] and sleep problems [68,69], studies reviewed may have either underestimated or overestimated the sleep-STB link. Greater specificity in the assessment of sleep problems contributing to future STBs could aid in the development and implementation of effective sleep interventions [70]. Third, STB studies are limited on which sleep health domains (e.g., duration, regularity) they are examining and are an area of neglected research [71]. Given the growing body of cross-sectional literature on the relationship between sleep duration and STBs [35], there is a need to examine sleep duration longitudinally with objective and subjective measures. There is considerable support that short sleep duration measured objectively in adults with insomnia specifically is associated with a variety of negative consequences [72,73]. Research on sleep duration among adolescents could be extended to clarify whether long sleep duration (i.e., ≥ 10 h of sleep) [35] and/or short total sleep duration (i.e., ≤ 5 h of sleep) [35] predicts short-term risk of STBs in youth. Fourth, STB studies incorporating objective sleep measures are so limited that it is not yet possible to determine whether these measures add predictive value above subjective sleep measures. Objective sleep measures are important because people are inaccurate in reporting their sleep duration and sleep latency [74,75] and objective assessment of these parameters allows for more precise measurement and could enhance prediction [76]. Fifth, for the majority of studies in this review, the follow-up assessment was relatively long and occurred during adulthood. Therefore, as STB risk was ultimately measured in adulthood, these studies tell us less about the prediction of suicide risk in youth. Sixth, the variability in follow-up duration (Range = 7 days-10 years) may have contributed to the mixed results. Significant findings from the only short-term prediction study [11] included in the review as well as numerous significant findings in the adult short-term prediction literature [12,24], suggest that sleep problems have greater utility as a proximal short-term risk factor than a long-term predictor.

Short-term prediction of STBs has important clinical implications [77] and given feasibility and utility of these studies [24], there is a pressing need for more short-term prediction studies in this area. Seventh, and finally, only three of the studies controlled for baseline STBs in the analyses. Controlling for prior STBs aids in understanding the unique contribution of sleep problems in predicting future STBs. Given that prior research in adults has examined the unique contribution of sleep to STBs [27], there is an important issue to address in future research in youth.

Findings from this review indicate that, although prospective research is limited, sleep problems represent a promising risk factor for youth STBs. Based on the limitations noted in prior research, future studies could enhance understanding of this association with study designs that include: validated self-report scales or diagnostic interviews of sleep and STBs, objective measures of sleep, multi-item measures of specific STBs and sleep, assessment of sleep health domains, and examine the sleep-STB relationship over shorter follow-up periods.

One crucial gap in the literature that remains is *how* sleep problems confer risk for STBs in youth. In the next section, we use a developmental framework to explore normative changes in sleep during adolescence that may make sleep problems particularly pernicious for youth.

5. Developmental framework

Adolescence is a developmental period typified by widespread biological, psychological, and social shifts [78,79], including significant changes in the amount and type of sleep obtained [80,81]. The majority of adolescents get less than the recommended 8–10 h of sleep per night and obtain less restorative sleep [33]. Consequently, most adolescents experience daytime fatigue [82].

It is important to note that most suicide research lack of specific terminology when examining sleep-related processes (e.g., broad terms like “sleep problems”). In contrast, the sleep literature employs specific terminology and has focused much of its attention on insufficient sleep, objective measures of sleep (e.g., PSG), and specific sleep disorders (e.g., insomnia). As such, this portion of the review will explore how insufficient sleep (i.e., failing to get adequate sleep) may be caused by sleep-related developmental processes and why these may place youth at greater risk for STBs. This review will focus on normative changes in sleep regulation, sleep architecture, and sleep continuity.

5.1. Sleep regulation, sleep architecture, and sleep continuity

Although the amount of needed sleep does not change from childhood to adolescence, adolescents exhibit a significant decrease in sleep duration and sleep depth compared to children [80,83]. The 10-hour nightly average obtained during childhood decreases to 7.5 h in adolescence [84,85]. There is a notable shift in chronotype (i.e., how an individual's endogenous circadian clock entrains to a 24-hour day) during this developmental period with a phase delay toward a more evening chronotype, leading to adolescents' preference for a later bedtime [84]. This later bedtime is in direct conflict with the early rise times necessitated by school schedules, resulting in overall reductions in nightly sleep duration. In addition, when adolescents do sleep, they experience less slow wave sleep (SWS; i.e., deep sleep/non-rapid eye movement [NREM] sleep, stage N3 sleep that is visually scored), or less restorative sleep, compared to children [33,86]. Taken together, adolescents experience persistent insufficient sleep (duration) and less restorative sleep (depth).

Insufficient and less restorative sleep results from developmental shifts in sleep architecture that may be driven by endogenous changes in both homeostatic drive to sleep and circadian regulatory processes [86]. The two-process model of sleep regulation [87–89] posits that sleep structure and timing are a direct result of the interaction between two systems: homeostatic system (Process S) and circadian system (Process C). Process S is a sleep-wake dependent process that changes as a function of a previous sleep episode, increasing while awake and decreasing while asleep. Process S governs sleep depth and generates homeostatic sleep drive/pressure. Indicators of Process S are time spent in SWS, slow-wave activity in NREM (assessed via power spectral analysis of raw PSG data) [90], and sleep drive/pressure [87]. Process S interacts with Process C, which is the clocklike aspect of sleep regulation that is responsive to the light-dark cycle in a 24-h day. This model has been examined in adolescents to understand how changes in sleep regulation may be unique among this population [33,86]. The developmental shifts in circadian timing and homeostatic drive of sleep during adolescence will be discussed in turn.

5.1.1. Changes in circadian timing of sleep

Research indicates a developmental delay in circadian regulation of sleep (Process C) which results in a preference for eveningness in adolescence or circadian phase delay [91]. Even when environmental factors (e.g., school) are removed, adolescents still demonstrate a delay in preferred activity phase (i.e., preferred time being active and awake) [91]; most adolescents prefer to stay up late and sleep late. Adolescents demonstrate a delay in melatonin secretion (i.e., hormone which begins to increase several hours before sleep onset in preparation for sleep) due to melatonin being sensitive to and suppressed by light [86,92]. Longer internal day length (i.e., circadian period) and light sensitivity are potential mechanisms thought to account for the circadian phase delay.

5.1.1.1. Internal day-length and sleep duration. The intrinsic circadian period for adolescents (24.27 h) is significantly longer than the circadian period for adults (24.12 h) [93,94]. As a result, adolescents need more time awake before they are signaled to sleep through Process S. This delays peak melatonin production and sleep onset combined with early required wake times for school ultimately impacts sleep duration [91]. Although more precise measurements across both pubertal development and the intrinsic circadian period are needed, longer internal day-length may play an important role in how insufficient sleep occurs during this developmental period.

5.1.1.2. Light sensitivity, sleep duration, and sleep depth. Light sensitivity may also play an important role in delayed circadian timing. This delay has been examined across different pubertal stages. Melatonin levels are more suppressed at the lowest lux level (i.e., unit of illumination indicating intensity of light) in early pubertal adolescents compared to late pubertal adolescents [95,96] and adults [97], suggesting the circadian timing system in early adolescence is extremely sensitive to light. This is notable because 96% of adolescents report using at least one form of technology that emits a short-wavelength light (i.e., higher concentration of blue than natural light) before bedtime [98]. The best evidence of the effects of short-wavelength light comes from studies in adults. Research in adults indicates that blue-light exposure significantly suppresses the evening rise of melatonin. As a result of melatonin suppression, individuals display heightened evening arousal, take longer to fall asleep, display a decreased average accumulation of REM sleep [99], and demonstrate decreased alertness the next morning [100]. This light sensitivity is believed to produce a similar decrease in sleep depth and increase in sleep delay effects in adolescents [96].

5.1.2. Changes in homeostatic drive for sleep

In addition to shifts in circadian timing, adolescents also display changes in homeostatic drive for sleep that are distinct from childhood and adulthood. Although limited studies have examined age-related changes in adolescents' homeostatic process regulating sleep (Process S), findings have been similar across adolescent age groups regarding their resistance to sleep pressure, sleep duration, and decrease in slow wave sleep [33,101].

5.1.2.1. Sleep pressure and sleep duration. The build-up of sleep pressure is slower in mature adolescents compared to early pubertal adolescents and adults [80]. This suggests that sensitivity to sleep pressure undergoes significant changes during the course of adolescence. The slower accumulation of sleep pressure is thought to allow later sleep onset in more mature adolescents [102]. As a result, adolescents resistance to sleep pressure may not signal a need for sleep when it might be necessary to do so. Resistance to sleep pressure may be a mechanism leading to insufficient sleep observed among adolescents.

5.1.2.2. Slow wave sleep and sleep depth. Adolescents experience not only a significant decrease in total SWS, but also a decrease in time spent in SWS during a single sleep episode [33,101]. The decrease in SWS is paralleled by a decrease in slow-wave activity during NREM and REM sleep and an increase in Stage 2 (N2) sleep (i.e., most common stage of sleep characterized by theta waves) [33]. Consequently, adolescents' sleep becomes less deep and restorative. The benefits of restorative sleep [103,104] (e.g., better cognitive performance) and the negative consequences of insufficient sleep [104,105] (e.g., attention difficulties) have been well-documented in adults. Determining whether adolescents are as sensitive to insufficient sleep as adults remains an open question and important future research direction. The existing evidence in youth suggests a potential pathway linking insufficient sleep to increased health risk behaviors, such as STBs. This pathway will be discussed in detail in the next section.

5.2. Sleep changes and their association with suicide risk in youth

Although two distinct literatures indicate that adolescence is characterized by significant changes in sleep and that sleep problems confer unique risk for STBs, the mechanisms underlying the sleep-STB relationship remain unclear. We propose that executive function (EF) and emotion regulation (ER) deficits may be two potential mediators linking sleep problems to increased risk for youth STBs. Since no research to date has connected sleep problems to STB risk in youth through deficits in EF and ER, evidence will be drawn from their separate literatures (i.e., sleep problems relating to EF and ER difficulties; EF and ER difficulties relating to STBs).

5.2.1. Executive function

Adolescents who experience insufficient sleep display deficits in a range of EFs such as attention, working memory, and processing speed [106–109]. While attention and working memory play a role in higher-order EFs (e.g., decision making, planning), the impact of sleep deprivation on higher-order EFs remain relatively underexplored in adolescents. Drawing from the adult literature, insufficient sleep has been found to impair problem-solving [110] and cognitive flexibility required for decision making [111].

Several of these same EF deficits have also been found to distinguish suicide attempters from individuals who do not attempt suicide. For instance, adult suicide attempters have been found to display deficits in a range of EFs such as attention [112,113], working memory [113], and flexible thinking [114] compared to non-attempters. Suicide attempters have also displayed deficits in in higher-order EFs such as decision making compared to non-attempters [115–117]. Similarly, adolescent suicide attempters display deficits in higher-order EFs such as decision making (specifically risky decision making) [118,119] and problem-solving [120] and young adult suicide attempters display deficits in cognitive flexibility [121].

One study in adults has found that, after accounting for wakefulness, SA occur most frequently in the circadian night [122]. Being awake during the circadian night, when an individual is not biologically predisposed to being awake, may result in impaired EF and place an individual at heightened risk for STBs.

In sum, EF deficits may be one mechanism underlying the sleep-STB relationship in youth, however, research examining this specific link is limited.

5.2.2. Emotion regulation

A second potential link between insufficient sleep and STBs may be ER difficulties. Adolescents who experience insufficient sleep display increased emotional reactivity [123] and significant difficulties with ER (e.g., use of ineffective strategies such as suppression, avoidance, rumination) [124–126]. A recent study examined the effects of sleep restriction on ER in adolescents and found that sleep restriction not only worsened mood, but also decreased adolescents' ability to regulate their negative emotions [125]. In addition, insufficient sleep has also been linked to facets associated with ER such as negative emotion states (e.g., worsened mood [127], increased anxiety [128]) and greater emotional reactivity to negative stimuli [127]. Similar difficulties with ER have also been found in adolescent suicide attempters [129–131]. In two studies –one on adolescents [129] and one on college-aged adults [132]– multiple suicide attempters (i.e., more than one SA) were distinguished from single attempters by greater difficulties with ER (e.g., inability to access effective regulation strategies). In sum, ER difficulties may be another mechanism linking sleep problems to risk for STBs in youth. However, no research has examined ER difficulties as a potential mediator in the sleep-STB relationship.

5.2.3. Environmental and social factors

A range of environmental and social factors may also have a major influence on the adolescent sleep-STB relation. Academic demands and school schedules are an external environmental factors that require early wake times despite adolescent's natural preference for late bedtimes and wake times [133]. In addition, research indicates an increase in the use of electronic media (e.g., smartphone) from childhood to adolescence [134] and suggests that this usage before bed plays a considerable role in insufficient sleep [135]. Essentially, what is already developmentally occurring within sleep processes (i.e., changes in circadian timing, sensitivity to sleep pressure and sleep architecture) is made worse by adolescents' use of electronics before bed [98,100,135]. Moreover, what adolescents are looking at and actively engaging with on electronic media may increase their emotional and physiological arousal and further shorten sleep duration [136,137]. One study demonstrated that adolescents who spent more time engaging with electronic media reported more mental health problems (e.g., depressive symptoms) compared to adolescents who spent more time engaging in non-electronic activities [138]. As a result, electronics before bed coupled with the biological effects of electronic use on sleep (i.e., blue light exposure suppressing evening rise of melatonin) may exacerbate insufficient sleep. This disruption in sleep, over and above normative sleep architecture changes, may place adolescents at heightened risk for STBs. Overall, findings suggest that developmental changes in sleep processes may be exacerbated by social and environmental factors in adolescents' lives.

6. Implications for hospital treatment

Sleep problems may be a clinically useful target for youth at risk for STBs because they are easily assessed in a variety of settings (including hospitals) and are amenable to treatment.

6.1. Assessment

Sleep problems may signal a need for acute intervention and are therefore important to monitor across healthcare settings. As previously noted, multiple lines of research indicate an important relationship between sleep problems and STBs. Sleep problems may be a short-term risk factor for STBs [11], precede adolescent suicide death in retrospective research [39], and are reported during presentation to emergency services for a SA [139]. Together, this research indicates that if an adolescent is reporting significant sleep problems, assessment of

suicide risk may be indicated. Therefore, it may be important to monitor sleep problem across medical settings.

Questions about sleep problems can be routinely assessed in healthcare settings as part of a standard clinical intake and/or monitored during clinical care. Hospital settings may benefit from using a brief sleep self-report measure for youth (e.g., Sleep Quality Scale) [140], which assesses recent sleep problems. For settings in need of a briefer assessment, adult research has shown that the one-item sleep question on the Patient Health Questionnaire-9 (i.e., "Over the last 2 weeks, how often have you been bothered by trouble falling or staying asleep, or sleeping too much?") [141] is a useful screen in primary care settings to assess hypersomnia and insomnia [142]. This brief screen could hold similar promise in youth as it assesses short and long total sleep time which have both been found to be related to SI in youth [35]. Youth who screen positive on this item could receive further assessment with a validated self-report measure of sleep quality and quantity (see Erwin and Bashore [143] for age-appropriate measures), clinical interview (e.g., Diagnostic Interview for Sleep Patterns and Disorders) [144], or an objective sleep assessment (e.g., PSG).

Sleep problems may be particularly useful to assess in a range of settings because individuals may be more willing to report sleep concerns than other, more stigmatized clinical phenomena such as STBs [145,146]. Moreover, given that self-reports of STBs may be limited by reporting biases (e.g., youth may be likely to deny STBs) [147,148], the ease and willingness to report sleep problems may give providers an additional avenue to investigate youth suicide risk. Although sleep problems are not a suicide specific risk factor, they may indicate more severe psychopathology [124] and signal a need for additional assessment.

6.2. Treatment

The efficacy of treatments for sleep problems in adolescents is also promising (see Blake and colleagues [149] for a review). Although not the only treatment for sleep problems in youth, cognitive behavioral therapy for insomnia (CBT-I) has the strongest evidence base with positive effects across several studies in youth [150,151]. Two randomized controlled trials in adolescents that compared CBT-I to a waitlist control [152] and a sleep hygiene condition [153] found CBT-I to be a superior treatment. CBT-I is advantageous because it is brief (i.e., 6 sessions), can be delivered directly to adolescents with no parental involvement [153], and can improve sleep outcomes (measured by self-report, sleep diaries, and actigraphy) with medium to large effect sizes [150,151,153]. CBT-I has also shown efficacy for even subthreshold insomnia symptoms in college students [154]. As such, CBT-I represents an ideal treatment to use in hospital settings when the presentation is insomnia or subthreshold insomnia. In addition to CBT-I, medication is a common treatment for youth sleep problems. Nightmares may be treated with alpha-blockers (e.g., prazosin) and insomnia may be treated with antihistamines (e.g., hydroxyzine) [155]. There has been an increase in treating adolescent insomnia with hypnotics (e.g., zolpidem) [156]. However, additional research on this treatment in youth is needed given recent work in adults indicating that hypnotics may increase SI [157].

7. Conclusion

A small, but growing prospective literature indicates that sleep problems may confer significant and unique risk for STBs in youth. Normative changes in sleep architecture during adolescence could explain why sleep problems may be particularly pernicious for young people. Although there appears to be a sleep-STB association, additional research is needed to clarify the mechanism linking sleep problems increase risk for youth STBs. Sleep problems, particularly insomnia, may be a promising treatment target for hospitalized youth because they are easily assessed and amenable to treatment.

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