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Pathways among sleep onset latency, relationship functioning, and negative affect differentiate patients with suicide attempt history from patients with suicidal ideation☆

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ABSTRACT

Depression, anxiety, sleep disturbances and poor relationship functioning often co-occur with the confluence of these factors contributing to greater suicide risk. This study investigated whether the pathways between depression, anxiety, sleep disturbances, and relationship functioning differentiated patients with suicide attempt history from those with suicidal ideation history. Patients seeking partial hospital treatment for severe psychiatric symptoms ($N = 180$) completed interviews assessing psychiatric and suicidal symptom histories, and self-report measures of sleep behaviors, anxiety, depression, and relationship functioning. Multiple sleep behaviors were examined: duration, sleep onset latency, and bedtime. Bias-corrected bootstrap mediation and moderated mediation analyses with suicide attempt as the moderator were used to evaluate pathways between variables. Among patients with ideation and attempt history, (1) sleep onset latency significantly mediated the association between depression and relationship functioning and that between anxiety and relationship functioning; (2) relationship functioning significantly mediated the association between depression and sleep onset latency and that between anxiety and sleep onset latency. These pathways were not significant among patients with suicidal ideation only. No other sleep behaviors were related to study variables. The reciprocal relationship between disrupted sleep onset latency and poor relationship functioning was specifically linked to more severe psychiatric symptoms among acute patients with suicide attempt histories.

1. Introduction

Suicide accounts for more than 40,000 deaths in the United States annually (World Health Organization, 2016). For every suicide death, it is estimated that 25 individuals attempt suicide and many more individuals think about suicide each year (CDC, 2016). Thus, it is vital to better understand the factors that contribute to suicidal thoughts and suicide attempts. To date, much of existing work has focused on risk factors for suicidal thoughts and not suicide attempts (Franklin et al., 2017) and there is a need to better distinguish pathways contributing to suicidal thoughts from those that contribute to attempts.

Depression and anxiety (i.e., negative affect), sleep disturbances, and impairments in relationship functioning are three, often co-occurring, risk factors for suicidal thoughts and suicide attempts across diverse samples (e.g., Chu et al., 2017a; Hall et al., 1999; May and Klonsky, 2016; Nadorff et al., 2013; Phillips et al., 2002). Greater severity of insomnia, nightmares, trouble sleeping, and poorer subjective sleep quality have all been reported among individuals at risk for suicide (Nadorff et al., 2013; for review, see Bernert et al., 2015). Relationship conflicts and impairments in social functioning are also linked to increased suicide risk (Holt-Lunstad et al., 2010); multiple empirically supported theories of suicidal behavior have described social impairments as precursors to suicide (e.g., Joiner, 2005; Klonsky and May, 2015; O'Connor, 2011).

Studies across the general population have found that the comorbid presentation of sleep disturbances, mood and anxiety disorders is associated with poorer self-reported quality of life and greater functional and social impairments, relative to those presenting with negative affect and no sleep difficulties (Nyer et al., 2013; Soehner and Harvey, 2012). In one sample of undergraduates, Nadorff et al. (2014) found that sleep disturbances (i.e., nightmares, insomnia) were significantly associated with lower interpersonal connectedness and greater suicide

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risk, and the association between sleep disturbances and suicide risk was no longer significant after accounting for interpersonal connectedness and/or depression symptoms. This suggests that interpersonal connectedness and depression may in part account for the relationship between sleep disturbances and suicide risk. Similarly, in Sarchiapone et al.'s (2014) study of adolescents, sleep disturbances (i.e., decreased sleep duration) were primarily associated with increased emotional difficulties, anxiety symptoms, peer-related problems, and suicidal ideation. Thus, these factors often co-occur, and the confluence of these variables may be associated with greater suicide risk. However, little is known about whether such paths may distinguish risk for suicidal thoughts from risk for attempts.

The most consistently reported link is that between depression and anxiety and sleep disturbances. In particular, sleep disturbance is a diagnostic feature shared by major depression and generalized anxiety disorder (APA, 2013; Cox and Olatunji, 2016). The severity of depression and anxiety has been found to have a unique relationship with sleep disturbances, beyond robust correlates, such as psychiatric comorbidity, health-related factors, and sociodemographic variables (e.g., Hom et al., 2016; Taylor et al., 2005). There is also considerable converging evidence suggesting that these variables are bidirectionally related (Alvaro et al., 2013) with numerous studies finding that depression and anxiety increase risk for future sleep disturbances (Jacobs et al., 2006; Johnson et al., 2006; Ruscio and Khazanov, 2017), and sleep disturbances are risk factors for future incidences of anxiety (Batterham et al., 2012; Cox and Olatunji, 2016; Neckelmann et al., 2007) and depression (Franzen and Buysse, 2008; Lee et al., 2013; Roberts et al., 2000).

In addition to depression and anxiety, sleep disturbances are also associated with greater impairments in interpersonal and relational functioning (see Shekleton et al., 2010; Shochat et al., 2014, for review) as well as greater loneliness and feelings that one does not belong (Hom et al., 2017; Matthews et al., 2017; Zeitlhofer et al., 2000). Studies have suggested that this may be due to extended periods of being awake and alone while others are asleep (Chu et al., 2017b; Hom et al., 2017), and disrupted daytime focus, decision-making, and emotion processing and regulation (Walker, 2009). Others have found that sleep disturbances may increase reactivity to daily stressors (Morin et al., 2003) and bias when perceiving social cues, and decrease empathic accuracy (Gordon and Chen, 2014; see Gordon et al., 2017 for review). These factors may all contribute to difficulties resolving social conflicts, forming meaningful connections and employing effective interpersonal skills (Gordon et al., 2017; Hom et al., 2017). Indeed, sleep has been shown to impact how individuals process and respond to social stressors (Gordon et al., 2017); these findings all highlight the importance of sleep in social functioning. Preliminarily, evidence suggests that these variables are also bidirectionally linked. For example, one recent study of young adults at elevated suicide risk found that sleep problems at baseline were associated with greater loneliness at 5-week, and 1- and 6-month follow-ups (Hom et al., 2017). Conversely, in another longitudinal study, older adults who feel lonelier at baseline reported greater sleep disturbances at the 7-year follow-up (Jacobs et al., 2006).

Finally, impairments in relationship functioning have also been robustly linked to depression and anxiety. In one study, the co-occurrence of depression and anxiety symptoms was associated with more interpersonal difficulties and a greater likelihood of a future suicide attempt in the period following discharge from psychiatric hospitalization (McKenzie and Wurr, 2001). Negative interactions with a spouse or partner have also been associated with increased likelihood of depression, anxiety, and suicidal ideation (Santini et al., 2015). Further, the relationships between impaired social functioning and depression and anxiety have been described as bidirectional (e.g., Verboom et al., 2014). In one study, patients who were depressed at baseline had poorer social functioning at both baseline and at follow-up, 7–10 months later, despite reductions in depressive symptoms at follow-up (Gotlib and Lee, 1989); conversely, poorer peer relationships at baseline predicted increases in depression symptoms at follow-up (Prinstein et al., 2005). Likewise, in one study of adolescents, more severe anxiety symptoms at baseline predicted poorer relationship functioning across the four-year follow-up period (van Eijick et al., 2012) and in another, researchers found that insecure attachment relationships in preadolescence predicted greater anxiety symptoms at follow-up in adolescence (Bosquet and Egeland, 2006).

Although there is extensive research indicating bidirectional associations between depression, anxiety, sleep disturbances, and impaired relationship functioning, no research has examined whether the pathways between these suicide risk factors differentiate those who only think about suicide from those who think about and attempt suicide. This is a key question of interest given that an understanding of the pathways between risk factors may allow us to identify the most effective treatment target for individuals with a heightened risk for future suicide attempts.

1.1. The present study

This study explored the pathways linking depression and anxiety, sleep disturbances, and impaired relationship functioning among individuals with a history of suicidal ideation. First, given evidence for bidirectional associations between these variables, we tested all permutations of mediation models characterizing the pathways between depression, sleep disturbances, and impaired relationship functioning. Subsequently, we similarly evaluated all permutations of mediation models of anxiety, sleep disturbances, and impaired relationship functioning. Anxiety and depression were modeled separately in this study given that there is also substantial evidence that these emotional states are distinct in multiple ways despite significant overlap (see Eysenck and Fajkowska, 2017 for review). Second, to identify the pathways that contribute to suicidal behavior for individuals who report suicidal ideation and also a history of suicide attempts, we examined suicide attempt status as a moderator in all mediation models. Of note, we aimed to evaluate the direction of the associations between these variables; however, these data were cross-sectional and we were unable to determine causality.

This research builds on existing work in at least two ways. First, recent work suggests that disruptions in different parts of this biological network may have distinct impacts on reported or observed sleep behaviors (Nota and Coles, 2015; Nota et al., 2015) and more emphasis on specific sleep parameters, such as total sleep time (i.e., time spent asleep at night), sleep onset latency (i.e., time required to fall asleep), sleep timing (i.e., bed and wake times), and wake after sleep onset (i.e., time awake after sleep is initiated; Cox and Olatunji, 2016), is needed. In response to calls for a more nuanced approach to operationalizing sleep disturbances (Alvaro et al., 2013), we explored the relationships between specific sleep behaviors, including total sleep time, sleep onset latency, and bedtime, and depression, anxiety, and relationship functioning; only the sleep behaviors related to depression, anxiety, and relationship functioning were included in the aforementioned mediation models.

Second, existing work has primarily examined patients with mild to moderate psychiatric symptom and suicide risk severity (e.g., undergraduate students; Chu et al., 2017b). Thus, we build on existing research by examining our aims in a sample of individuals with a history of suicidal ideation who are presenting for treatment of severe psychiatric symptoms at a partial hospital program. This sample is particularly well-suited to examine these aims given that many patients have recently been discharged from inpatient hospitalization and present with severe depression and anxiety, and a history of suicidal ideation. Furthermore, over 25% of this sample reported one or more prior suicide attempts, allowing us to identify the pathways that may distinguish those who think about suicide from those who think about and have attempted suicide. Given that risk for a suicide attempt is greatest in the days and weeks following discharge from psychiatric hospitalization

(e.g., McKenzie and Wurr, 2001), it is particularly important to understand how psychiatric symptoms, sleep disturbances, and relationships functioning are associated in this population.

2. Method

2.1. Participants

A total of 298 patients completed the study measures as part of their baseline clinical assessment in an intensive treatment at a partial hospital program in a private psychiatric hospital in New England from November 2016 to March 2017. The partial hospital program treats adult (age 18+) with a wide range of psychiatric disorders (primarily mood, anxiety, personality, and psychotic disorders). Approximately half of the patients were referred from the community by outpatient treatment providers and half transition from higher levels of care, such as inpatient hospitalizations. For a detailed description of the partial hospital program, see Björgvinsson et al. (2014). All individuals who completed this baseline assessment and reported past or current suicidal ideation on the Columbia Suicide Severity Rating Scale (Posner et al., 2011) were eligible for inclusion in this study. Of the 298 eligible patients, 73 patients who did not complete the semi-structured clinical interviews, 19 who had missing self-report data, and 26 who denied any lifetime suicidal ideation were all excluded.

This resulted in a final sample of 180 individuals who reported past or current, passive and/or active suicidal ideation. In the final sample, the average age of patients was 33.7 years (SD = 14.7 years) and 49.4% identified as male (n = 89) and 88.9% as White/Caucasian (n = 160). Major depressive disorder (38.9%, n = 70) and generalized anxiety disorder (14.4%, n = 26) were the most common primary diagnosis. Regarding suicidal symptom history, 46.7% of the patients reported lifetime passive suicidal ideation (n = 84; e.g., thoughts about death, a non-specific desire to die, and/or identification of suicide methods), and 53.3% reported a lifetime history of active suicidal ideation (n = 96; e.g., intent to engage in suicidal behavior and/or specific suicide plans). Furthermore, over half of the sample (56.7%, n = 102) reported engaging in self-injurious behaviors with the intent to die in their lifetime (e.g., potentially lethal suicide attempt, interrupted attempt) while 26.7% reported a lifetime suicide attempt (n = 48). See Table 1 for demographic and diagnostic characteristics.

Table 1
Sample characteristics, n (%).

Characteristics	*n* (%)
Sex	
Male	89 (49.4)
Female	91 (50.6)
Sexual orientation	
Bisexual	20 (11.1)
Gay/lesbian	16 (8.9)
Heterosexual	136 (75.6)
Queer	3 (1.7)
Other	5 (2.8)
Race	
American Indian or Alaskan Native	2 (1.1)
Asian	17 (9.4)
Black	3 (1.7)
Native Hawaiian or Pacific Islander	3 (1.7)
White	160 (88.9)
Other or unknown	6 (3.4)
Education	
High School Graduate/GED	12 (6.7)
Some College/Associates Degree/Trade School	80 (44.4)
Four-year College Graduate	42 (23.3)
Post-Graduate	46 (25.6)
Marital status	
Never married	119 (66.1)
Separated/divorced	10 (5.6)
Widowed	3 (1.7)
Married	44 (24.4)
Living with partner	4 (2.2)
Employment	
Not employed	45 (25.0)
Employed part-time	32 (17.8)
Employed full-time	56 (31.1)
Student	47 (26.1)
Primary diagnosis	
Bipolar disorder	16 (8.9)
Bipolar disorder with psychotic features	8 (4.4)
Major depressive disorder	70 (38.9)
Major depressive disorder with psychotic features	4 (2.2)
Other specified mood	1 (0.6)
Generalized anxiety disorder	26 (14.4)
Panic disorder	1 (0.6)
Panic disorder w/agoraphobia	1 (0.6)
Social anxiety disorder	3 (1.7)
Obsessive-compulsive disorder	15 (8.3)
Posttraumatic stress disorder	6 (3.3)
Other specified anxiety	3 (1.7)
Body dysmorphic disorder	1 (0.6)
Alcohol use disorder	3 (1.7)
Substance use disorder	4 (2.2)
Other	18 (10.0)
Comorbid diagnosis	155 (75.2)

2.2. Measures

2.2.1. Sleep

In this study, we assessed sleep behaviors in the past month using three items that were similar to those used in longitudinal sleep diary studies (Grandner et al., 2006) and the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989). Specifically, participants were asked to respond to the following three questions regarding the past month: (1) "When have you usually gone to bed at night?", (2) "How long (in minutes) has it usually taken you to fall asleep each night?", and (3) "When have you usually gotten up in the morning?". This approach has been supported by prior studies showing that self-reported sleep behavior from longitudinal sleep diaries is highly correlated with those obtained from the PSQI (Grandner et al., 2006). Based on participant responses, patients' average hours of sleep per night, usual bedtime, and sleep onset latency during the past month were calculated. Specifically, the average hours of sleep per night was calculated as the period between the time an individual fell asleep and when they woke up and the average sleep onset latency was calculated as the difference between the time an individual went to bed and when they fell asleep.

2.2.2. Patient Health Questionnaire-9 (PHQ-9; Kroenke and Spitzer, 2002)

The PHQ-9 is a 9-item self-report measure of the severity of both neurovegetative and cognitive/affective depression symptoms over the past two weeks. Items were rated on a 4-point Likert-type scale from 0 (*not at all*) to 3 (*nearly all the time*) and total scores ranged from 0 to 27, with higher scores indicating greater severity of depression symptoms. In this study, total PHQ-9 score was calculated using all items, including item 3, which measures global sleep difficulties (i.e., trouble falling or staying asleep, or sleeping too much). Item 3 was not significantly correlated with any of the PSQI-based sleep behavior items (rs = −0.26 to 0.35, all ps > 0.05) and the pattern of results did not change when item 3 was excluded. The PHQ-9 has previously demonstrated adequate validity and internal consistency in other samples of psychiatric patients (Beard et al., 2016). In this sample, the PHQ-9 also exhibited good internal consistency (α = 0.83).

2.2.3. Generalized Anxiety Disorder Scale-7 (GAD-7; Spitzer et al., 2006)

The GAD-7 is a seven-item self-report measure of general anxiety symptoms (e.g., "trouble relaxing") during the past two weeks. Items were rated on a four-point Likert-type scale that ranged from 0 (*not at all*) to 3 (*nearly every day*); total scores ranged from 0 to 21, with higher scores indicating more severe anxiety symptoms. The GAD-7 has demonstrated good reliability and construct validity as a measure of

general anxiety in psychiatric samples (Beard and Björgvinsson, 2014). In this sample, the GAD-7 demonstrated good internal consistency (α = 0.87).

2.2.4. Behavior and Symptom Identification Scale-24 (BASIS-24; Cameron et al., 2007)

The BASIS-24 is a 24-item measure that assesses symptoms and functioning over the past week. In this study, the 5-item relationship functioning subscale of the BASIS-24 was used to evaluate patients' relationship functioning, such as severity of interpersonal difficulties (e.g., "Get along with people in your family?"). Items were rated on a 5-point Likert type scale ranging from 0 (*none of the time*) to 4 (*all of the time*) and the total subscale score ranged from 0 to 20. Items were reverse-coded, with higher scores indicating poorer relationship functioning. In past research, the BASIS-24 and its subscales have demonstrated good reliability and validity as a measure of mental well-being and functioning (Cameron et al., 2007). In this sample, internal consistency of the relationship functioning subscale was good (α = 0.81).

2.3. Clinical interviews

2.3.1. Columbia Suicide Severity Rating Scale (C-SSRS; Posner et al., 2011)

The C-SSRS is a structured interview that was used to assess patients' lifetime and past month histories of suicidal ideation and behavior (i.e., number of suicide attempts, aborted attempts, and interrupted attempts). In this sample, the C-SSRS was used to assess for lifetime history of suicidal ideation and suicide attempts. Prior research has found that the C-SSRS has demonstrated robust psychometric properties as a suicide risk assessment tool in psychiatric samples (Madan et al., 2016) with adequate interrater reliability (Posner et al., 2011). Given this interview was administered as part of routine clinical care, we were unable to obtain interrater reliability indices.

2.3.2. Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998)

The MINI is a structured diagnostic interview that was used to evaluate criteria for DSM-IV Axis I diagnoses. The MINI was used to characterize the psychiatric diagnoses of patients in this sample. This interview has demonstrated strong reliability and validity in relation to the Structured Clinical Interview for DSM-IV (Sheehan et al., 1998).

2.4. Procedure

As part of standard clinical care at the partial hospital program, patients completed a computerized battery of self-reported measures upon admission to the program to track their treatment progress. Measures were administered using Research Electronic Data Capture, a secure, web-based application designed to support data collection for research studies (Harris et al., 2009). The admission (pre-treatment) assessment included the items assessing sleep, the PHQ-9, the GAD-7, and the BASIS-24. On patients' second day in the partial hospital program, the MINI and C-SSRS were administered by patients' therapists. Given that this partial hospital program also serves as a training hospital, all therapists were doctoral clinical psychology interns or practicum students and all were supervised by licensed clinical psychologists. Therapists were trained in the administration of clinical interviews; this included reviewing administration manuals, scoring and reviewing training tapes, and completing mock interviews. All clinicians were required to pass a final training interview with their supervisor prior to interviewing patients at this partial hospital program. All data were initially collected by patients' treatment team to inform treatment and subsequently de-identified for use in this research. As such, this study was deemed exempt by the Partners Health Care and McLean Hospital Institutional Review Board.

2.5. Statistical approach

2.5.1. Preliminary analyses

First, descriptive statistics were evaluated for all study measures. Specifically, the distributions of GAD-7, PHQ-9, BASIS-24 relationship functioning subscale, sleep onset latency, sleep duration, and bedtimes were checked for normality and the presence of outliers. Given the exploratory nature of these analyses, a conservative Bonferroni correction (0.05/15 tests) was used to set the criterion for rejecting the null hypothesis (α = 0.003).

Prior to examining the mediation models, we first examined which specific sleep parameters were associated with depression, anxiety, and relationship functioning by evaluating the direct zero-order relationships between these variables. The purpose was to use greater nuance when approaching the conceptualization of sleep disturbances, consistent with recommendations (Alvaro et al., 2013). Pearson's correlations (two continuous variables), point biserial correlations (continuous and dichotomous variables) or phi coefficients (two dichotomous variables) were calculated to examine the relations between (1) psychiatric symptoms (PHQ-9 depression symptoms, GAD-7 anxiety symptoms) and relationship function (BASIS-24); (2) sleep behavior (sleep onset latency, sleep duration, bedtimes) and psychiatric symptoms (PHQ-9 depression symptoms, GAD-7 anxiety symptoms); (3) sleep behavior (sleep onset latency, sleep duration, bedtimes) and relationship function (BASIS-24); and (4) dichotomously coded, demographic covariates (age, biological sex, ethnicity/race, mood diagnosis, and comorbidity; see below for details).

2.5.2. Mediation analyses

Next, we explored the pathways between these variables. Ordinary least squares path analyses with bias-corrected 95% confidence intervals using 10,000 bootstrap samples were conducted using the PROCESS macro for SPSS (Model 4; Hayes, 2013). Mediation models were built based on significant zero-order correlations between depression, anxiety, relationship functioning, and sleep behaviors. In this study, only sleep onset latency was significantly correlated with all other study variables; thus, only sleep onset latency was examined in the mediation models. Although mediation can occur without significant zero-order correlations (Hayes, 2013), we chose to focus on these significant association to mitigate the Type 1 error risk that would accompany a test of every permutation of the included measures.

The following mediation models were tested: (1) sleep onset latency as a mediator of the association between depression and relationship functioning; (2) sleep onset latency as mediator of the association between relationship functioning and depression; (3) relationship functioning as a mediator of the association between depression and sleep onset latency; (4) relationship functioning as a mediator of the association between sleep onset latency and depression; (5) depression as a mediator of the association between relationship functioning and sleep onset latency; and (6) depression as a mediator of the association between sleep onset latency and relationship functioning. As noted above, given evidence that depression and anxiety are distinct in multiple ways (e.g., Eysenck and Fajkowska, 2017), these emotional states were evaluated in separate mediation models. Thus, we also examined the association between anxiety, sleep onset latency, and relationship functioning; the six mediation models described above were tested with anxiety in the place of depression.

2.5.3. Moderated mediation analyses

Finally, to identify the pathways that contribute to suicidal behavior for individuals who report suicidal ideation and also a history of suicide attempts, we examined suicide attempt status as a moderator in the 12 aforementioned mediation models. Moderated mediation analyses were also conducted using the PROCESS macro for SPSS (Model 14; Hayes, 2013). In line with recommendations (Hayes, 2013), the index of mediation moderation was used to evaluate whether suicide attempt

status significantly moderated the mediation pathways.

For all mediation and moderated mediation models evaluated in this study, we included the following four covariates: age, biological sex (coded: male, female), ethnicity/race (coded: non-Hispanic White, other ethnicity/race), mood diagnosis based on the MINI (coded: mood disorder, no mood disorder), and comorbidity based on the MINI (coded: comorbidity, no comorbidity/only one diagnosis).

2.5.4. Power

Per recommendations by Fritz and MacKinnon (2007), a sample size of 148 is required to detect small-moderate effect sizes for both the α and β paths using a bias-corrected bootstrap mediation approach. Thus, with our sample size of 180, there was adequate statistical power ($1 - \beta > 0.80$) for mediation analyses.

3. Results

3.1. Preliminary analyses

Descriptive statistics, including means, standard deviations, ranges, skew and correlations between key study measures, are listed in Table 2. Sleep onset latency was positively skewed (Skew = 2.23); following a log transformation, skew was within an acceptable range ($-2 <$ Skew $< +2$).[1] Notably, consistent with expectations, the correlation between the depression and anxiety symptom measures suggested there was some overlap; however, the measures were not redundant ($r = 0.64$, $p < .001$). Among the specific sleep parameters, number of hours of sleep was negatively correlated with bedtime ($r = -0.33$, $p < 0.001$) and no other significant correlations emerged between sleep onset latency, sleep duration, and bedtimes. Depression symptoms ($r = 0.28$, $p < 0.001$) and anxiety symptoms ($r = 0.36$, $p < .001$) were significantly correlated with relationship functioning. Of the three sleep parameters assessed, sleep onset latency was the only sleep behavior significantly correlated with any of the other variables, including depression symptoms ($r = 0.26$, $p < 0.001$), anxiety symptoms ($r = 0.29$, $p < 0.001$), and relationship functioning ($r = 0.30$, $p < 0.001$). Thus, only sleep onset latency was examined in mediation and moderated mediation models. The majority of the sample reported a sleep onset latency that was greater than 20 minutes (66.7%, $n = 125$). Scatterplots depicting the zero-order associations between depression, anxiety, relationship functioning, and sleep onset latency are available in Figures S1 through S4 in the Supplementary Materials.

3.2. Mediation analyses

Mediation models are summarized in Table 3. Among individuals with a history of passive and/or active suicidal ideation seeking treatment at this partial hospital program, all of the tested mediation pathways were significant as the 95% confidence intervals (CI) of the indirect effects did not cross zero. In all models, the direct effects remained significant, suggesting partial mediation. These findings not only indicate bidirectional associations between depression, anxiety, relationship functioning and sleep onset latency, but also interactions between these variables as a network of symptoms. To evaluate the strength of the mediation models, two measures of effect sizes (κ^2, R^2) were examined; however, they produced different conclusions. Broadly, the effect sizes indicated that the mediation models with the strongest indirect effects were similar for depression and anxiety. Based on R^2 values, the effect size estimate most widely used in the literature, the pathway from depression to sleep onset latency through relationship functioning ($R^2 = 0.03$) and that from depression to relationship functioning through sleep onset latency ($R^2 = 0.03$) were the strongest pathways for depression (Fig. 1, A and B). Similarly, for anxiety, the pathway from anxiety to sleep onset latency through relationship functioning ($R^2 = 0.04$) and that from anxiety to relationship functioning through sleep onset latency ($R^2 = 0.04$) were the strongest pathways (Fig. 2, A and B).

3.3. Moderated mediation analyses

Finally, we investigated the pathways between these variables for patients who report suicidal ideation and those who report both suicidal ideation and suicide attempt histories. Moderated mediation models are summarized in Table 4. Findings indicated that only four models—those described above as being the most robust paths—were significantly influenced by patient's history of suicide attempts; for all other models, the indices of moderated mediation were not significant, suggesting that suicide attempt status was not a significant moderator. Specifically, the following four pathways were all significant for patients with a history of suicidal ideation and suicide attempts: (1) indirect effect of depression on relationship functioning through sleep onset latency, (2) indirect effect of depression on sleep onset latency through relationship functioning, (3) indirect effect of anxiety on relationship functioning through sleep onset latency, and (4) indirect effect of anxiety on sleep onset latency through relationship functioning. For those with only a history of suicidal ideation and no prior attempts, there were no significant indirect effects for these four pathways.

4. Discussion

In this sample of 180 patients with a history of suicidal ideation presenting for partial hospital treatment of severe psychiatric symptoms, sleep onset latency was the only sleep behavior that significantly correlated with depression, anxiety, and relationship functioning. All mediation pathways between depression, sleep onset latency, and relationship functioning, as well as all mediation paths between anxiety, sleep onset latency, and relationship functioning were significant. That is, each variable (e.g., anxiety symptoms) partially mediated the association between the other two variables in both directions (e.g., effect of sleep onset latency on relationship functioning and that of relationship functioning on sleep onset latency). Notably, results indicated that the following four pathways distinguished patients with a history of both ideation and attempts from those with a history of ideation and no attempts: (1) the path from anxiety to relationship functioning through sleep onset latency; (2) the path from anxiety to sleep onset latency through relationship functioning; (3) the path from depression to relationship functioning through sleep onset latency; and (4) the path from depression to sleep onset latency through relationship functioning. Several theoretical and clinical implications for assessing and treating psychiatric patients with suicidal symptom histories arise from these findings.

First, in line with existing evidence (Beck, 2010; Chu et al., 2017b; Hom et al., 2017; Renner et al., 2014), our results indicated reciprocal associations between depression, anxiety, relationship functioning, and sleep onset latency in this high-risk sample of acute psychiatric patients with a history of suicidal ideation. Furthermore, each variable mediated the association between the other variables. This suggests that in addition to reciprocal relationships between individual variables, depression, anxiety, relationship and sleep function all interact as a cluster of symptoms, such that increased severity of one of these symptoms will subsequently result in increased severity of the other symptoms. This is especially concerning given that depression, anxiety, and impaired sleep and relationship functioning are, individually, risk factors for

[1] All analyses examining sleep onset latency were conducted with both log-transformed and non-transformed variables; notably, the pattern of results remained unchanged. Thus, for interpretability, the results based on the non-transformed sleep onset latency variable are reported. Results using log-transformed sleep onset latency are available in Tables S1 and S2 in the Supplementary Materials.

Table 2
Zero-order correlations between psychiatric symptoms, relationship functioning, and sleep behaviors.

	1. PHQ9 Depression Sx	2. GAD7 Anxiety Sx	3. BASIS24 Rel Fn	4. Bedtime	5. Sleep Onset	6. Hours Sleep	7. Age	8. Sex	9. Eth/ Race	10. Educ	11. Marital	12. Comorbid Dx	13. Mood Dx
1.	–												
2.	**0.66****	–											
3.	**0.24****	**0.36****	–										
4.	**0.12**	**0.13**	**0.06**	–									
5.	**0.27****	**0.29****	**0.30****	**0.11**	–								
6.	**−0.05**	**−0.10**	**0.18**	**−0.33****	**−0.18**	–							
7.	**−0.02**	**−0.12**	**0.02**	**−0.34****	**−0.12**	**−0.01**	–						
8.	*−0.10*	*−0.14**	*−0.04*	*−0.18**	*−0.02*	*0.03*	*0.10*	–					
9.	*0.04*	*−0.04*	*−0.17**	*0.01*	*−0.27***	*0.13*	*0.03*	0.16*	–				
10.	*0.02*	*−0.06*	*−0.06*	*−0.30*	*−0.05*	*0.07*	*0.43***	−0.17*	0.04	–			
11.	*0.03*	*0.01*	*−0.04*	*−0.29***	*−0.10*	*−0.05*	*0.62***	−0.001	0.18*	0.28**	–		
12.	*−0.32***	*−0.35***	*−0.03*	*−0.17**	*−0.10*	*0.04*	*0.06*	0.14	0.02	0.04	0.06	–	
13.	*0.57***	*0.33***	*0.09*	*0.17**	*0.12*	*−0.04*	*0.03*	−0.07	−0.04	0.06	−0.004	−0.41**	–
Mean	14.4 (5.5)	11.2 (5.3)	1.4 (0.9)	11:22 pm	42 m: 0 s	8 h: 12 m	33.7	0.51	0.79	0.49	0.27	0.25	0.60
SD	5.5	5.3	0.9	1 h: 52 m	37 m: 45 s	1 h: 59 m	14.7	0.50	0.41	0.50	0.44	0.43	0.49
Min.	0	0	0	7:30 pm	0 m: 0 s	2h: 0m	18	0	0	0	0	0	0
Max.	26	21	3.9	5:00 am	240 m: 0 s	14 h: 30 m	78	1	1	1	1	1	1
Skew	−0.28	−0.10	0.75	0.65	2.23	− 0.23	–	–	–	–	–	–	–
α	0.83	0.87	0.81	–	–	–	–	–	–	–	–	–	–

Note. * < .05, ** = $p \leq .001$. – = not applicable. h = hours. m = minutes. s = seconds. Sx = symptoms. Rel Fn = relationship functioning. Sleep onset = sleep onset latency. Eth/Race = ethnicity/race. Educ = education. Marital = marital status. Dx = diagnosis. SD = standard deviation.
Sleep Onset Latency, items based on the Pittsburgh Sleep Quality Inventory. Depressive Symptoms, PHQ-9 = Patient Health Questionnaire-9. Anxiety Symptoms, GAD-7 = Generalized Anxiety Disorder Scale-7. Relationship Functioning, BASIS24 = Behavior and Symptom Identification Scale-24.
Demographic variables were recoded into dichotomous variables. Sex coded 0 = male, 1 = female. Eth/Race coded 0 = other ethnicity/race, 1 = non-Hispanic White. Education coded 0 = high school/some college, 1 = college grad and above. Marital status 0 = single/widowed/divorced, 1 = married/common-law. Comorbid Dx 0 = no comorbidity, 1 = comorbid diagnoses. Mood Dx 0 = no mood diagnosis, 1 = current mood diagnosis.
Pearson's correlations (two continuous variables; bolded), point biserial correlations (continuous and dichotomous variables; italicized) or phi coefficients (two dichotomous variables) were calculated.

Table 3
Summary of bias-corrected bootstrap mediation analyses.

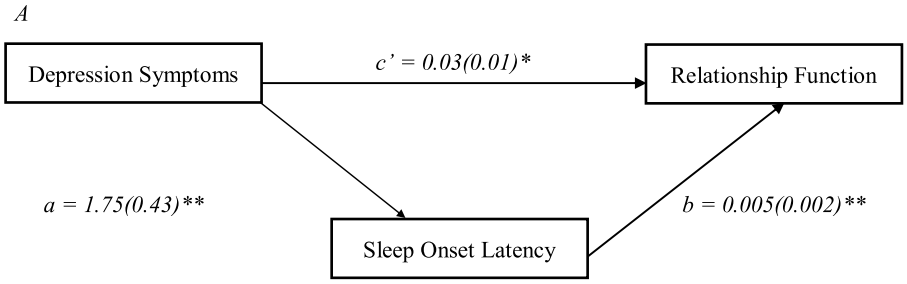
X	M	Y	Indirect effects	95% CI%	Kappa^2	R^2	Direct effects
Depression	Sleep Onset	Relationship	0.0095*	0.0023, 0.0197	0.0594	0.0278	0.0280*
Relationship	Sleep Onset	Depression	0.3818*	0.1304, 0.7554	0.0598	0.0272	1.1189*
Depression	Relationship	Sleep Onset	0.3305*	0.0707, 0.8212	0.0518	0.0280	1.4224*
Sleep Onset	Relationship	Depression	0.0074*	0.0013, 0.0181	0.0475	0.0272	0.0348*
Sleep Onset	Depression	Relationship	0.1104*	0.0232, 0.2543	0.0465	0.0224	0.0054*
Relationship	Depression	Sleep Onset	2.1345*	0.6194, 4.8139	0.0528	0.0270	8.8276*
X	**M**	**Y**	**Indirect effects**	**95% CI**	**Kappa^2**	**R^2**	**Direct effects**
Anxiety	Sleep Onset	Relationship	0.0096*	0.0016, 0.0209	0.0584	0.0388	0.0407*
Relationship	Sleep Onset	Anxiety	0.3830*	0.1290, 0.7452	0.0637	0.0377	1.4780*
Anxiety	Relationship	Sleep Onset	0.4005*	0.0738, 0.9420	0.0596	0.0389	1.6079*
Sleep Onset	Relationship	Anxiety	0.0097*	0.0035, 0.0200	0.0659	0.0376	0.0349*
Sleep Onset	Anxiety	Relationship	0.0018*	0.0008, 0.0033	0.0705	0.0377	0.0048*
Relationship	Anxiety	Sleep Onset	2.9923*	1.2522, 5.7889	0.0725	0.0376	7.9699*

Note. * < .05. X = predictor variable. M = mediator. Y = outcome variable. CI = confidence interval.
Sleep Onset Latency, items based on the Pittsburgh Sleep Quality Inventory. Depressive Symptoms, PHQ-9 = Patient Health Questionnaire-9. Anxiety Symptoms, GAD-7 = Generalized Anxiety Disorder Scale-7. Relationship Functioning, BASIS24 = Behavior and Symptom Identification Scale-24.
For all mediation models, PROCESS macro 4 was used; age, biological sex, ethnicity/race, comorbidity, and mood diagnosis were all included as covariates.

suicidal behavior (Chu et al., 2016, 2017a, 2017b; Hom et al., 2017; Large et al., 2011). Thus, the network of associations between all these symptoms may not only impact patients' symptoms, social, and sleep function, but also exacerbate risk for suicidal behavior.

Second, this study found that among the most severe patients, those with more severe depression and anxiety symptoms were more likely to report more time spent awake prior to falling asleep due in part to experiencing more difficulties with interpersonal functioning in daily life. Additionally, these patients were more likely to experience impairments in their relationship functioning partly due to difficulties falling asleep at night. This cycle of worsening sleep and social functioning occurred primarily among the patients in this sample who presented with a history of not only suicidal ideation, but also suicide attempts. Thus, in contrast to patients who have not acted on their thoughts of suicide, for those who have attempted suicide—and who are therefore at greater risk of attempting suicide again—it is the cycle between sleep disturbances (delayed sleep onset) and disrupted interpersonal relationships that is associated with more severe psychiatric symptoms. Clinically, these findings suggest that targeting both sleep and social functioning may have the greatest impact on depression and anxiety symptoms among the most acute and high-risk patients with suicide attempt histories.

Third, the results of this study highlight the importance of specificity when operationalizing sleep behaviors to enhance our understanding of the sleep-related processes that are linked to psychiatric symptoms (Alvaro et al., 2013; Mai and Buysse, 2008; Nota and Coles,



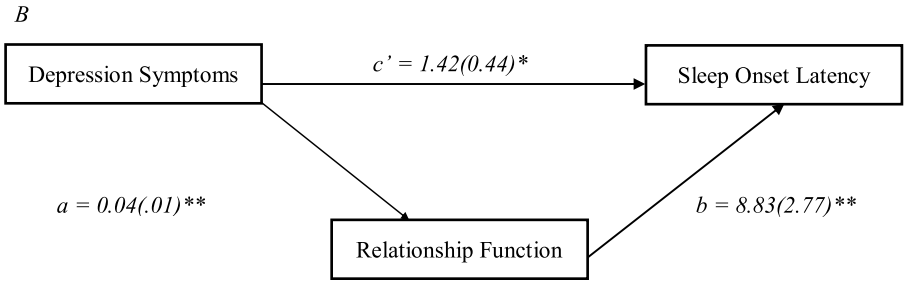
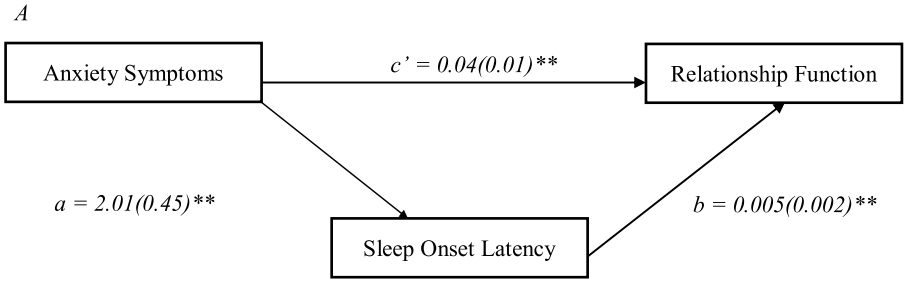


Fig. 1. Mediation models. *$p < .003$ (Bonferroni-corrected), **$p \leq .001$. Unstandardized coefficients (standard error) are shown. 2A: *ab* = indirect effect of depression symptoms on relationship function through sleep onset latency. *c'* = direct effect of depression symptoms on functional outcome. 2B: *ab* = indirect effect of depression symptoms on sleep onset latency through relationship function. *c'* = direct effect of depression symptoms on sleep onset latency.



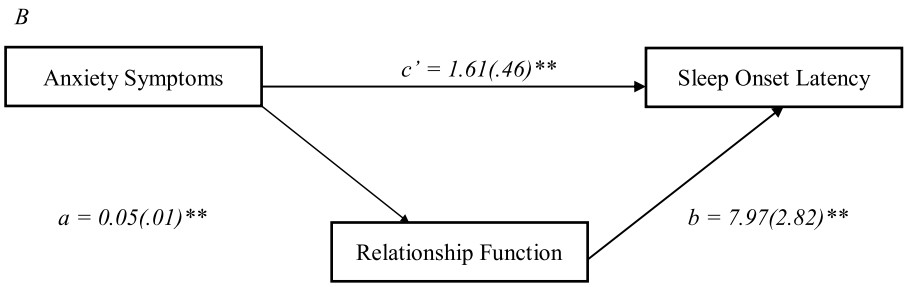


Fig. 2. Mediation models. *$p < 0.003$ (Bonferroni-corrected), **$p \leq 0.001$. Unstandardized coefficients (standard error) are shown. 2A: *ab* = indirect effect of anxiety symptoms on relationship function through sleep onset latency. *c'* = direct effect of anxiety symptoms on functional outcome. 2B: *ab* = indirect effect of anxiety symptoms on sleep onset latency through relationship function. *c'* = direct effect of anxiety symptoms on sleep onset latency.

2015; Nota et al., 2015). In this study, sleep onset latency was implicated as the link between relationship functioning and both depression and anxiety. There were no significant correlations between the severity of depression and anxiety symptoms, and sleep duration and bedtime. It is unclear why sleep onset latency was specifically related to the other study variables. However, recent work has found that longer sleep onset latency is associated with more difficulties disengaging from negative emotional stimuli (Nota and Coles, 2018)—a difficulty that underlies both depression and anxiety (e.g., Koster et al., 2005; Mogg and Bradley, 2005). Greater sleep onset latency may be the result of many factors; however, it is often associated with increased levels of arousal, which may prolong the transition from wakefulness to sleep (Smith and Trinder, 2000; Tang and Harvey, 2004; Zoccola et al., 2009). Indeed, increased arousal has been observed in individuals with acute psychiatric symptoms (e.g., Ribeiro et al., 2014) and it has been suggested that heightened alertness may account for sleep disruptions and the observed differences in attention and response inhibition associated with anxiety (Kalanthroff et al., 2017). If arousal systems are implicated, there may be a feedforward effect: arousal leads to disrupted sleep, which contributes to increased concern about sleep and increased arousal at bedtime, and the cycle repeats. Given that increased sleep onset latency was further associated with poorer relationship functioning, these relationships also may interact as a cycle whereby greater sleep onset latency leads to poorer relationships functioning, poorer functioning leads to more arousal, and more arousal feeds back and maintains greater sleep onset latency. This study did not measure overarousal; however, if greater sleep onset latency is linked to upregulated arousal systems, improvements in sleep may diminish risk for overarousal (cf. agitation)—this is an important future direction as overarousal and agitation have been identified as near-term suicide risk factors (Ribeiro et al., 2014).

Finally, in this psychiatric sample, depression and anxiety symptoms are both similarly connected to difficulties falling asleep and poorer relationship functioning. That the same pattern emerged for depression and anxiety symptoms is not surprising as it is consistent with existing research that identifies sleep as a transdiagnostic variable associated with mood and anxiety symptoms (Fairholme et al., 2013; Harvey et al., 2011). Given that few studies have tested the pathways among these variables in a treatment-seeking, high-risk sample, future work investigating these relationships in other samples will contribute

Table 4
Summary of bias-corrected bootstrap moderated mediation analyses.

X	M	Y	V	Indirect effects	95% CI%	Index of moderated mediation
Depression	Sleep Onset	Relationship	SA no	0.0050	−0.002, 0.0146	0.0161*
			SA yes	0.0211*	0.0067, 0.0388	
Relationship	Sleep Onset	Depression	SA no	0.3463*	0.0924, 0.7471	0.1083
			SA yes	0.4545	−0.0598, 1.1715	
Depression	Relationship	Sleep Onset	SA no	0.1585	−0.0486, 0.5515	0.8600*
			SA yes	1.0185*	0.1254, 1.9149	
Sleep Onset	Relationship	Depression	SA no	0.0069*	0.0006, 0.0181	0.0038
			SA yes	0.0107	−0.0031, 0.0318	
Sleep Onset	Depression	Relationship	SA no	0.0011*	0.000, 0.0026	0.0006
			SA yes	0.0016*	0.0001, 0.0039	
Relationship	Depression	Sleep Onset	SA no	1.7414*	0.3289, 4.5528	1.4047
			SA yes	3.1461*	0.4411, 8.4056	
X	**M**	**Y**	**V**	**Indirect effects**	**95% CI**	**Index of moderated mediation**
Anxiety	Sleep Onset	Relationship	SA no	0.0039	−0.0045, 0.0141	0.0198*
			SA yes	0.0237*	0.0071, 0.0421	
Relationship	Sleep Onset	Anxiety	SA no	0.4338*	0.1426, 0.8483	−0.2033
			SA yes	0.2305	−0.1349, 0.8274	
Anxiety	Relationship	Sleep Onset	SA no	0.1812	−0.1186, 0.6144	1.1309*
			SA yes	1.3121*	0.3358, 2.5526	
Sleep Onset	Relationship	Anxiety	SA no	0.0089*	0.0024, 0.0193	0.0050
			SA yes	0.0139*	0.0020, 0.0332	
Sleep Onset	Anxiety	Relationship	SA no	0.0016*	0.0003, 0.0032	0.0009
			SA yes	0.0025*	0.0010, 0.0045	
Relationship	Anxiety	Sleep Onset	SA no	3.0521*	1.1587, 6.2294	−0.3187
			SA yes	2.7335	−0.1094, 7.5546	

Note. * < .05. X = predictor variable. M = mediator. Y = outcome variable. V = moderator, suicide attempt history status. CI = confidence interval. SA = suicide attempt history.
Sleep Onset Latency, items based on the Pittsburgh Sleep Quality Inventory. Depression Symptoms, PHQ-9 = Patient Health Questionnaire-9. Anxiety Symptoms, GAD-7 = Generalized Anxiety Disorder Scale-7. Relationship Functioning, BASIS24 = Behavior and Symptom Identification Scale-24.
For all moderated mediation models, PROCESS macro 14 was used; age, biological sex, ethnicity/race, comorbidity, and mood diagnosis were all included as covariates.

to converging research implicating sleep as a potential transdiagnostic risk factor.

4.1. Clinical implications

The present results suggest that for acute patients with a history of suicidal ideation and attempts and current symptoms of depression or anxiety, sleep and social functioning may also be disrupted. Thus, should these findings be replicated in other samples, sleep functioning and/or social functioning could be important treatment targets. To improve sleep function, psychoeducation regarding sleep hygiene and the potential impact of poor sleep hygiene on relationship functioning is important. Interventions for individuals reporting delayed sleep onset latency may include cognitive-behavioral therapy for insomnia (CBT-I; Edinger and Means, 2005), which has been shown to be efficacious for treating problems with sleep onset latency (Koffel et al., 2015). Other sleep hygiene techniques, such as diminishing intake of caffeine and other stimulants (particularly prior to bed time), engaging in regular daily exercise, limiting the time spent in bed engaging in activities other than sleeping, and practicing mindfulness skills during waking hours, may also be helpful. Patients may also set up a “wind-down” period before bedtime during which individuals engage in relaxing activities before bedtime to reduce arousal and decrease sleep onset latency. It is also important to target relational conflicts in therapy; patients may benefit from skills for improving communication with others, challenging distorted cognitions about relationships and others, and managing intense negative affect during interpersonal conflicts. As shown in prior studies (e.g., Joiner et al., 2009; Trockel et al., 2015), interventions targeting sleep and social functioning may also contribute to decreases in suicide risk.

4.2. Limitations and future directions

Several limitations should be noted. First, this study relied primarily on self-report measures. As such, it is possible that shared-method variance may account for associations reported in this study. Additionally, there is evidence to suggest that relative to actigraphy, daily sleep logs, and other sleep monitoring device, self-report questionnaires are less accurate measures of sleep behaviors (e.g., Lauderdale et al., 2008; Sharwood et al., 2012). Given the limitations of self-report measures, future studies may benefit from the inclusion of multiple self-report and objective measures of sleep (e.g., actigraphy), other study variables (e.g., suicide risk IAT; Nock et al., 2010), and greater frequency of assessments (e.g., ecological momentary assessment). Relatedly, other sleep parameters not available in this study could be included in future research; for example, information about wake after sleep onset is needed to more accurately determine total sleep time. Additional indices of interpersonal function (e.g., loneliness; Hom et al., 2017) beyond self-reported relationship conflicts are also needed because relationship conflicts and, for example, loneliness may differentially relate to sleep and negative affect, warranting different clinical approaches. Second, our sample identified primarily as White/Caucasian, which limits the generalizability of these findings. Nevertheless, this patient sample was heterogeneous with regard to symptom presentation and as such, these findings have significant clinical relevance. It is also important to note that the indirect effects were relatively small and the direct associations remained significant when accounting for the indirect effect, suggesting partial mediation. Thus, other variables not evaluated in this study may be contributing to these results. For example, some studies have reported that the season of data collection may influence sleep quality (Hashizaki et al., 2018) and social belongingness (Van Orden et al., 2008); thus, future studies should account for the potential influence of weather on these variables.

Finally, although multiple mediation models were tested to determine the direction of the pathways between depression, anxiety, relationship functioning, and sleep onset latency, this study used a cross-sectional design. Therefore, future work that employs a longitudinal study design is needed to determine temporal precedence or causality.

4.3. Conclusions

Overall, in this sample of acute patients with a history of suicidal ideation and suicide attempts seeking treatment in a partial hospital setting, more severe depression and anxiety symptoms were associated with poorer relationship functioning, and these associations were in part accounted for by sleep onset latency. More severe depression and anxiety symptoms were also associated with greater sleep onset latency, and these associations were in part accounted for by poorer relationship functioning. These results suggest that for acute patients with suicidal symptom histories and current symptoms of depression and/or anxiety, sleep and social functioning may also be disrupted. This study was the first, to our knowledge, to examine all the pathways between these variables and as such, further work is needed to replicate and extend these findings using longitudinal study designs and objective measures. Future studies that address the limitations of the current investigation are needed to better understand the important roles of sleep and social functioning in the treatment of depression and anxiety symptoms among acute patients at risk for suicide.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at 10.1016/j.psychres.2018.11.014.

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