Psychosis and hopelessness mediate the relationship between reduced sleep and suicidal ideation in schizophrenia spectrum disorders.

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Abstract

Objective: Suicide is a major cause of death amongst individuals with schizophrenia spectrum disorders (SSD). Despite numerous risk factors being identified, accurate prediction of suicidality and provision of tailored and effective treatment is difficult. One factor that may warrant particular attention as a contributor to increased psychopathology and suicidality in SSD is disturbed sleep. Sleep disturbances have been reliably linked to greater levels of suicidal ideation and are highly prevalent amongst individuals with SSD. This study aimed to examine if reduced sleep duration and psychopathology are associated with increased suicidal ideation.

Method: One-hundred and eighteen adults with chronic SSD living within the community participated in this cross-sectional study. Psychosis symptoms were assessed using the Positive and Negative Syndrome Scale. Items 4 and 10 from the Montgomery-Asperg Depression Rating Scale and Item 2 from the Calgary Depression Scale for Schizophrenia were used to assess reduced sleep duration, current suicidal ideation, and hopelessness, respectively. All measures were rated concurrently.

Results: A hierarchical logistic regression revealed that greater acute sleep disturbances were associated with increased suicidal ideation and this relationship was found to be uniquely mediated by both positive symptom severity and hopelessness.

Conclusion: These results suggest that individuals with SSD who exhibited disrupted or disordered sleep, positive symptoms and/or hopelessness should be routinely screened for suicidal thinking. Furthermore, interventions that effectively target sleep disruptions may provide much-needed action against suicidal ideation.

Keywords: schizoaffective disorder, suicide, suicidality, self-harm, insomnia, hallucinations, delusions

Highlights

- Reduced sleep found to be associated with increased suicidal ideation
- This was uniquely mediated by both hopelessness and positive symptoms
- More regular screening of sleep problems in schizophrenia is needed

1. Introduction

Suicide has a devastating impact on individuals, families and communities. People with schizophrenia spectrum disorders (SSD) are at particular risk of suicidal ideation and behaviours. Compared to the general population, people with SSD have 12 times the risk of death by suicide (Saha et al., 2007) and approximately 5% of individuals with SSD die by suicide (Hor & Taylor, 2010a; Palmer et al., 2005). A further 32% either attempt suicide (Lu et al., 2019) or report expressing suicidal thoughts (Chapman et al., 2015). Longitudinal data has shown that suicide risk is greatest early in the illness course and lessens but persists over time (Bertelsen et al., 2007). Early in the illness, suicidal ideation is caused by distress related to emerging psychotic experiences, the potential for stigma, shame and fear about the implications of living with SSD, and the potential for adverse mental health service contact that impacts willingness to access future care (Ventriglio et al., 2016). Across the lifespan, a vast array of risk factors has been uncovered, making identification and treatment of the causes of suicidality in individuals challenging.

1.1. Risk factors and prediction of suicide in SSD

Several reviews have comprehensively examined risk factors for suicide in people with SSD. Carlborg et al. (2010), Hor and Taylor (2010a) and Cassidy et al. (2018) typically distinguished between clinical (e.g. illness awareness, positive symptoms, hopelessness) and biosocial (e.g. male sex, higher education, family history) risk factors; with history of suicide attempts or suicidal ideation consistently identified as the strongest risk factors for suicide. Despite the known risk factors for suicide in SSD, accurate prediction of suicidality and provision of tailored and effective treatment has still not been achieved. Indeed, accuracy of risk prediction was shown to be inadequate in a case-controlled study of people with SSD who did (n = 71) or did not (n = 355) die by suicide (Lopez-Morinigo et al., 2016). Apart

from suicidal history, suicidal ideation and recent discharge from hospital, no other characteristic risk factor was over-represented in people who died by suicide. In addition, only 44% of people who died by suicide had undergone a full risk assessment, highlighting that clinical staff had not identified these individuals as being at high risk of suicide.

A complex pattern of associations among the many known risk factors likely contributes to an individual with SSD engaging in suicidal behaviours. The model for suicide developed by Turecki and Brent (2016) may provide a useful theoretical framework to examine this elaborate pattern. The complete model distinguishes between population-based factors (e.g. media reports, lack of social cohesion) and "proximal or precipitating" (e.g. life events), "developmental or mediating" (e.g. aggressive personality) and "distal or predisposing" (e.g. family history) individual-based risk factors. These population- and individual-based risk factors are said to interact and jointly contribute to the development of suicidal behaviours. Proximal individual risk factors, such as acute substance use, mental illness and suicidal ideation are said to be temporally associated with suicidal behaviours, and thus act as their precipitants. More specifically, it is proposed that biological factors interact with current psychopathology, hopelessness and depressed mood to engender thoughts about suicide within an individual. Suicidal thoughts are said to then interact with other proximal risk factors, such as acute substance misuse and impulsivity, as well as developmental or mediating risk factors involving anxiety and neurocognitive impairment to contribute to suicidal behaviours. Substance use (Hunt et al., 2018), impulsivity (Ouzir, 2013), anxiety (Braga et al., 2013) and neurocognitive impairments (Carruthers et al., 2019) are all co-occurring features of SSD, highlighting the ease by which suicidal thoughts can quickly transition to suicidal behaviours amongst this population. As such, examining the interplay between easily assessable proximal risk factors which are theorised to contribute to suicidal

ideation may help identify targets that can effectively reduce the risk of suicide amongst individuals with SSD.

1.2. Sleep and suicide in SSD

Disturbed sleep is a biologically-based proximal risk factor which is likely to interact with psychopathology among individuals with SSD to engender thoughts about suicide. Sleep disturbances, such as poor quality, efficiency and duration have been reliably linked to greater levels of suicidality (Bernert & Joiner, 2007; Bernert & Nadorff, 2015) and are commonly recorded amongst individuals with SSD (Chan et al., 2017; Waite et al., 2019). A meta-analysis of 19 studies examining the relationship between sleep disturbance and suicidal behaviours in psychiatric patients, found an overall doubling (odds ratio = 1.99) of the rate of suicidal behaviours in patients with versus without sleep disturbance (Malik et al., 2014). The increased risk of suicidal behaviours with sleep disturbance was far higher in people with SSD; with a more than 12 times higher risk of suicidal behaviours for people with SSD and sleep disturbance versus people with SSD and no sleep disturbance.

Sleep disturbances have also been consistently found cross-sectionally to co-occur with more severe psychotic symptoms (Reeve et al., 2015), with emerging trial-based evidence suggesting that improving sleep in patients with SSD may reduce positive symptom severity (Myers et al., 2011; Tek et al., 2014); however, more sufficiently powered research is needed to adequately establish this connection (see Freeman et al., 2015; Kantrowitz et al., 2010). Disruptions to sleep are also commonly said to elicit despairing thoughts about sleep amongst individuals with SSD (Chiu et al., 2016; Faulkner & Bee, 2016), and may also worsen the ability to cope with encountered stressors, thus increasing a predisposition towards general hopelessness and suicidal ideation (Hofstetter et al., 2005; Sio et al., 2013; Woosley et al., 2014). Both positive symptom severity and hopeless thinking are noted as

important risk factors for suicide amongst individuals with SSD (Carlborg et al., 2010). As such, it is probable that sleep disturbances amongst individuals with SSD may exacerbate positive symptom severity and hopelessness, which then give rise to increasing thoughts about suicide.

1.3. The current study

Despite the theoretical links between sleep disturbance, psychopathology, and suicidal ideation in SSD, rarely have the inter-relationships among these factors been explored (e.g. Kilicaslan et al., 2017; Miller et al., 2019). More specifically, the interplay of reductions in sleep, symptom severity and hopeless thinking has not yet been investigated within the context of suicidal ideation in SSD. As such, this study aimed to examine whether reduced sleep and psychopathology is associated with increased suicidal ideation in a community sample of adults with chronic SSD. It was firstly hypothesised that greater self-reported reductions in sleep, depressed mood, positive symptoms and hopeless thinking would be associated with increased suicidal ideation. Outside of positive symptoms, few studies have investigated the relationship between symptom severity and suicidal ideation, with no clear consensus emerging (see Carlborg et al., 2010). As such, the current study sought to explore any associations between negative and disorganised symptoms with suicidal ideation. Finally, in line with interplay of proximal individual risk factors proposed by Turecki and Brent (2016), it was also hypothesised that the relationship between reduced sleep and current suicidal ideation would be influenced by positive symptom severity and hopelessness.

2. Materials and methods

2.1. Participants

Data from people with chronic schizophrenia (*n*=80) or schizoaffective disorder (*n*=38; *N*=118) were obtained from the Cognitive and Genetic Explanations of Mental Illnesses (CAGEMIS) bio-databank; in accordance with a Swinburne University ethics committee approved protocol (SUHREC: 2018/192 & 20190397-305). All participants had given prior informed consent for the analysis of stored data and were recruited from metropolitan mental health services in Melbourne, Australia. Individuals were included in the current analysis if they had valid responses to the primary study measures (detailed below), and were aged 18-65 years, fluent in English, and had an estimated premorbid intelligence quotient (IQ) >70 confirmed by the Wechsler Test of Adult Reading (Wechsler, 2001). SSD diagnoses were confirmed using the MINI-International Neuropsychiatric Interview Version 5 (Sheehan et al., 1998). Participants with significant visual or auditory impairments, known neurological disorders or current substance abuse/dependence were not eligible for participation.

2.2. Measures

Suicidal ideation was measured using item 10 (Suicidal Thinking) from the Montgomery-Åsberg Depression Rating Scale (MADRS; Montgomery & Åsberg, 1979). A score of ≥2 was used to categorise participants with current suicidal ideation (Ballard et al., 2015). Item 4 was used to measure reduced sleep (using a scale of 0 = "Sleeps as usual" to 6 = "Less than 2-3 hours sleep"). Hopelessness was measured via item 2 of the observer-rated Calgary Depression Scale for Schizophrenia (CDSS; Addington et al., 1990; using a scale of 0 = absent to 3 = severe). Psychotic symptom severity was measured with the observer-rated 30-item Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987). Scoring was conducted in accordance with the five-factor solution recommended by van der Gaag et al. (2006), which generates summed scores for the following domains: positive symptoms,

negative symptoms, disorganisation, excitement and emotional distress. For the purpose of the current study, excitement and emotional distress will not be examined. Due to the use of individual MADRS and CDSS items in the planned analyses, PANSS item 6 was used as a measure of overall depression. Additional demographic and clinical variables were collected including sex, education, age, primary diagnosis, years since illness diagnosis and current medication.

2.3. Statistical analysis

IBM SPSS *v.*26.0 was used for the statistical analyses. To measure whether people with or without current suicidal ideation differed in relevant demographic and clinical variables, independent-measures *t*-tests were conducted with continuous variables and chi-square tests of independence were conducted with categorical variables. For continuous variables with unequal variances between groups, Welch's *t*-test was used. Cohen's *d* was reported as an effect size for *t*-tests and Cramer's *V* for categorical variables. To examine inter-correlations among sleep, psychopathology and current suicidal ideation, partial correlations adjusting for sex (a significant correlate of suicidal ideation status) were used. False discovery rate adjustment (Benjamin-Hochberg method with an accepted false discovery rate = 0.10) was used to protect against family-wise error (Pike, 2011).

Hierarchical logistic regression was then used to examine incremental contributions to current suicidal ideation risk, informed by the causal steps proposed in Turecki and Brent (2016); that proximal biological risk factors (e.g. sex, sleep) interact with psychopathology and hopelessness to modulate suicidal thinking. Step 1 included sex and reduced sleep; step 2 added clinical symptoms detected as having a significant association with suicidal ideation; and step 3 added hopelessness. If evidence emerged to suggest possible mediated relationships, this was examined using the PROCESS procedure for SPSS Version 3.3, either

using Model 4 for simple mediation or Model 6 for serial mediation relationships (Hayes, 2018). For all models, coefficients with 95% bias-corrected bootstrapped confidence intervals (CI) were calculated (using 5000 sample) for the indirect path with significant mediation concluded if the range did not span zero.

In addition to the primary analysis outlined above, two supplementary analyses were conducted. Firstly, as the CDSS includes items pertaining to terminal insomnia (item 7, Early Wakening) and suicidal thoughts and behaviours (item 8, Suicide), the above analyses were repeated using these CDSS items in lieu of MADRS items 4 and 10, respectively (see Supplementary Section 1). Secondly, whilst the current study aims to investigate hopelessness and its effects on sleep disturbances and suicidal thoughts, the effect of overall depression severity should be considered. As such, the primary analysis was repeated with PANSS item G6 (depression) entered the hierarchical logistic regression as a fourth step (see Supplementary Section 2).

- Insert Table 1 here -

3. Results

Table 1 shows mean (SD) values for continuous variables alongside frequency values for categorical variables separately shown for participants with (*n*=20; 16.9%) or without (*n*=98; 83.1%) current suicidal ideation. Participants with current suicidal ideation were more likely to be female and displayed significantly higher positive and negative symptom scores, reduced sleep, hopelessness and suicide ideation than participants without current suicide ideation. No other significant differences were found.

- Insert Table 2 here -

Table 2 shows the partial correlation *r*-values (adjusting for sex due to its relationship with current suicidal ideation) between psychopathology, sleep reduction and current suicidal ideation. Increased positive and negative symptoms, hopelessness and reduced sleep were significant correlates of greater suicidal ideation. Greater reductions in sleep was also associated with significantly more severe psychopathology across all domains.

- Insert Table 3 here -

Table 3 shows the results from the three-step hierarchical logistic regression analysis. Each step made a significant additive contribution to the prediction of risk of current suicidal ideation. Both female sex and reduced sleep were significant correlates in step 1, but reduced sleep became non-significant in step 2 after the addition of positive and negative symptoms. Female sex and positive and negative symptoms remained as significant multivariable covariates in step 2. After the addition of hopelessness in step 3, only hopelessness and positive symptoms remained as significant multivariable covariates.

- Insert Table 4 here –

As the coefficient for reduced sleep was reduced in both steps 2 and 3, there is evidence that positive symptoms and hopelessness mediate its relationship with suicidal ideation. Given that both positive symptoms and hopelessness remained significant in the final model, Figure 1 shows the results of a serial mediation (PROCESS model 6). Independent significant mediated pathways were evident: reduced sleep – positive symptoms – suicidal ideation (CI: 0.02 to 0.45); and reduced sleep – hopelessness – suicidal ideation

(CI: 0.00 to 0.55). Significant serial mediation through positive symptoms to hopelessness was not found. Further probing via PROCESS model 4 of a potential mediated relationship between negative symptoms and suicidal ideation was also warranted. As seen in Figure 1, hopelessness was found to significantly mediate this relationship (CI: 0.03 to 0.19).

- Insert Figure 1 here -

4. Discussion

Understanding the interplay of risk factors that contribute to suicidal behaviours amongst individuals with SSD is of vital importance. In consideration of this, the current study examined the relationship between reduced sleep, psychopathology and suicidal ideation in a sample of community-based adults with chronic SSD. It was initially hypothesised that greater reductions in sleep, positive symptoms and hopeless thinking would be associated with increased suicidal ideation, which was supported by the results. Furthermore, negative symptom severity was found to be associated with increased suicidal ideation, however no such relationship was detected with disorganised symptoms. Finally, the results supported our hypothesis that the relationship between reduced sleep and suicidal ideation would be mediated by positive symptom severity and hopelessness.

4.1.1. Sleep, psychopathology and suicidal ideation

Reduced sleep was found to be associated with greater suicidal ideation. Disruptions to sleep have been consistently identified as risk factors for suicide; with the presence and severity of psychopathology previously suggested as an important moderator in this relationship (Bernert & Joiner, 2007; Bernert & Nadorff, 2015). Sleep plays an important role in regulating emotional reactivity. Sleep deprivation enhances sensitivity to aversive or

pleasurable stimuli, partly through reducing connectivity between pre-frontal cortical regions and the amygdala, and can amplify anticipation of aversive and reward stimuli (Goldstein & Walker, 2014). During rapid-eye movement (REM) sleep, cortical networks involved in emotional memory formation are also reactivated, but without the arousal that often accompanies emotion processing during wakening. This can reduce the intensity of experiencing recalled emotional memories. When REM sleep is repeatedly disturbed, this can impact the restoration of adaptive emotional responses, predisposing the person to greater anxiety and anticipation of harm.

Reduced sleep was subsequently found to be indirectly related to increased suicidal ideation through greater positive symptoms and hopelessness. Positive symptoms have been reliably reported as co-occurring with disturbed sleep (Reeve et al., 2015) and thought to arise as a consequence of the increases in negative affect which accompany sleep disturbances in SSD (Kasanova et al., 2020; Reeve et al., 2018). However, they do not appear to influence suicidal thoughts following early wakening. It was revealed in a supplementary analysis that early wakening was indirectly related to increased suicidal thinking and behaviours through greater hopelessness (see Supplementary Section 1), positive symptom severity had no such influence. Positive symptoms, such as voice hearing and persecutory delusions have been reported by patients to worsen before bed (Chiu et al., 2016) and intensify in response to greater disturbances to normal sleep architecture throughout the night (Reeve et al., 2015), More time spent awake at night also enables greater engagement with active positive symptoms to occur, potentially leading to increased thoughts about suicide. Although positive symptoms have been found to intensify with greater sleep disturbances, they are also likely to normalise following a period of sleep (Waters et al., 2018). As such, positive symptoms are likely to be less prevalent and less severe in the morning following sleep and therefore less likely to lead to thoughts about suicide following early wakening. In

contrast, dysfunctional thought processes such as worry, rumination and unrealistic expectations about sleep can occur both throughout the night and morning following early wakening. This can eventually lead to low confidence in one's capacity for optimal sleep and greater sleep-related hopelessness and thus increased thoughts about suicide (Chang et al., 2020; Huthwaite et al., 2014; Kaplan et al., 2009; Woosley et al., 2014). Being awake during the biological night, as well as early wakening with fewer distractions also increases time to ruminate on negative beliefs and hopeless thoughts which can lead to greater suicidal thinking. With friends or family usually asleep, the reduction in available social support likely provides more opportunity to engage in suicidal behaviours (Littlewood et al., 2016).

The findings that positive symptoms and hopelessness are associated with increased suicidal thinking is aligned with a large body of literature which reliably report these symptoms as prominent risk factors for suicidality in SSD (Carlborg et al., 2010; Cassidy et al., 2018; Hor & Taylor, 2010b). Conversely, the field lacks a clear consensus regarding the role of negative symptoms in suicidal ideation in SSD (Cassidy et al., 2018; Hor & Taylor, 2010b). The current study was able to detect a positive association between negative symptoms and suicidal thinking, which appears to be influenced by the severity of hopeless thoughts. The observation that hopelessness mediates the relationship between negative symptoms and current suicidal ideation implies that simply experiencing negative symptoms is not sufficient to arouse thoughts about suicide. This was previously shown in a study in 162 adults with SSD (Jahn et al., 2016), where poorer social role functioning was associated with greater suicidal ideation in patients with a greater desire for social closeness and low impairment in motivation and pleasure-related negative symptoms. This suggested that vulnerability to suicide ideation was greatest for patients who desired but were not experiencing social connection, or more pertinently, were distressed by their lack of pleasure or motivation to pursue meaningful activity. In a longitudinal study of 68 adults with SSD,

negative appraisal and beliefs about controllability of symptoms and disability was also found to mediate the relationship of psychotic symptoms with concurrent and longitudinal suicidal ideation (Hutton et al., 2019). Beliefs about negative symptoms, their impact and the ability to make changes if so desired, are therefore likely to be more impactful on thoughts of hopelessness and associated suicidal ideation more than simply experiencing negative symptoms themselves. Enquiring about such reappraisals as part of suicide risk assessment could enhance the accuracy of risk prediction (Hutton et al., 2019).

One risk factor for suicidal thoughts and behaviours in SSD which doesn't lack a clear consensus is depression. Depressed mood has been reliably identified as an important risk factor for suicide in SSD (Carlborg et al., 2010). It has also been proposed as another individual proximal risk factor that interacts with both biological and psychopathological proximal factors to contribute to suicidal ideation (Turecki & Brent, 2016). And although the current study aimed to focus on the more cognitive-based symptom of hopeless thinking, the influence of depression on the above model was supplementarily examined. It was revealed that once depression severity was considered, the influence of hopelessness on the relationship between reduced sleep and suicidal thinking became mute. Depression has been reliably linked to both sleep disturbances (Lustberg & Reynolds, 2000) and suicidal ideation (Ribeiro et al. 2018), with the current results indicating that greater reductions in sleep appear to intensify feelings of sadness, helplessness, pessimism, and likely hopelessness, which then lead to increased thoughts about suicide. Notwithstanding the importance of screening for depression as part of a suicide risk assessment, hopeless thinking should still be regarded as an important feature of the relationship between sleep disturbances and suicidal ideation considering its documented importance as a risk factor for suicide devoid of concurrent depression (Carlborg et al. 2010; Ribeiro et al. 2018).

4.2. Limitations.

Several limitations must be acknowledged. Firstly, there were several sampling issues which limit the generalisability of the current findings. Foremost was the lack of severe reductions in sleep within the sample and the modest sample size. An average rating of 2 out of 6 (i.e., slight difficulty dropping off to sleep or slightly reduced, light or fitful sleep) was recorded for the suicide ideation group and whilst this was found to be significantly worse than the no suicide ideation group, with moderate effect, it does not necessarily reflect clinically problematic disruptions to sleep. Furthermore, only 20 participants were classified as having current suicidal ideation, half of which were diagnosed with schizoaffective disorder. The higher proportion of individuals with schizoaffective disorder in the suicidal ideation group may have been an influential factor in uncovering the association with reduced sleep. Schizoaffective disorder is characterised by prominent mood symptoms which one would expect to influence both sleep and suicidal thoughts. Without detailed information regarding mood symptoms, no definitive commentary on their influence could be made. Future studies would benefit from examining this relationship in more detail, specifically focussing on participants with schizoaffective disorder using a comprehensive assessment of mood symptoms. Furthermore, 70% of the suicidal ideation group (n=14) were female, compared to 38% (n=37) in the no suicidal ideation group. Although most studies report that males are at a higher risk for suicide than females in schizophrenia (Carlborg et al., 2010), females are generally more prone to suicidal ideation (Turecki & Brent, 2016). Unaccounted psychosocial factors, such as domestic abuse or being a single-mother may have been adversely influencing the prevalence of both sleep disturbances and suicidal ideation in this group. Future research would benefit from a more homogenous sample (e.g. female only participants) or record more detailed information concerning psychosocial factors known to influence both sleep and suicidal ideation. The current sample was comprised of individuals

living with long-term SSD (average 15.5 years illness diagnosis). Whilst suicide is relevant across all stages of the illness, the risk for suicide in SSD is greatest in the first few years following psychosis onset (Ventriglio et al., 2016). As such, examination of the association between sleep disturbances and suicidal thoughts in individuals with recent onset psychosis is warranted. Finally. the current sample lacked antipsychotic treatment homogeneity. Overall, antipsychotic medications have been found to improve some facets of sleep amongst individuals with SSD (Monti & Monti, 2004), however not all medications have the same effect. Furthermore, clozapine has known preventative effects against suicide (Meltzer et al., 1995; Meltzer, 1998; Meltzer et al., 2000) and 14 of the 15 participants treated with clozapine were classified as having no suicidal ideation. The disproportionate frequency of participants on clozapine in the no suicidal ideation group may have confounded the current findings. In response, future investigations should aim to examine a sample of more homogenously treated participants and consider investigating the differential effects of being treated/not treated with clozapine on the relationship between sleep disturbances and suicidal ideation.

Secondly, the current study was limited by the use of single item assessments of reductions in sleep, hopelessness and suicidal ideation. Although such single item assessments are commonly employed to assess complex psychological states, such as suicide ideation (e.g. Ballard et al., 2015), and have been shown to be as valid as complete measures (e.g. Desseilles et al., 2012), future research would benefit from more comprehensive assessments. Although a supplementary replication was conducted using items from the CDSS in lieu of the MADRS, this limitation still stands. Both suicide ideation and sleep are complex phenomenon. Thoughts about suicide can involve, but are not limited to, the desire to die, reasons for dying, ideation of previous attempt(s) and plan formation, and can vary in frequency, duration and severity. All of which could be differentially influenced by sleep dysfunction. Likewise, individuals with SSD exhibit a range of sleep disturbances and

disorders that go beyond simply a reduction in sleep and that may differ in importance in relation to suicide ideation. For example, it was recently reported by Miller et al. (2019) that current insomnia was a significant indicator of suicidal ideation in SSD. The current study primarily examined the relationship between sleep reductions and unspecified thoughts about suicide. Future studies should therefore employ more objective sleep assessments which quantify the quantity, quality and efficiency of sleep, as well as more detailed evaluations of suicide ideation to more accurately capture the broad range of disturbances prevalent in SSD. Thirdly, the direction of the relationships between reduced sleep, positive symptoms, hopelessness and suicidal ideation cannot be causally examined via cross-sectional research. Given the previously reviewed evidence, it is distinctly probable that reductions in sleep facilitates an increase in hopeless thinking and a worsening of positive symptom severity, which in turn engenders more intense or frequent thoughts about suicide; however, the reverse may also be true. Furthermore, due to the cross-sectional design, we are unable to comment on the ability of baseline reductions in sleep to predict future suicide ideation. As such, more comprehensive longitudinal research should be considered in the future to address both of these issues. Finally, this study did not have access to participants' history of suicide attempts. This is widely regarded as one of the strongest predictors of suicide amongst individuals with SSD (Carlborg et al., 2010; Hor & Taylor, 2010b). It is important to understand the factors which contribute to the transition from suicidal ideation to behaviour. Neurocognitive impairments, impulsive behaviours and high levels of anxiety are a number of individuals risk factors present in SSD that are said to contribute to this important transition (Turecki & Brent, 2016).

4.3. Concluding remarks

Given that reductions in sleep may interact with psychopathology to increase suicidal ideation, sleep problems should serve as both a potential treatment target and screen for suicidality amongst individuals with SSD. Several pilot trials have reported that a derivative of Cognitive Behavioural Therapy designed for insomnia (CBTi) improves sleep quality amongst SSD individuals with disordered sleep (Freeman et al., 2015; Myers et al., 2011). Whilst suicide ideation was not examined in these studies, the strong improvements to sleep are promising in light of the findings of the current study and others (e.g. Li et al., 2016; Miller et al., 2019). It is therefore recommended that future trials investigating CBTi for SSD consider including a measure of suicide ideation and explore any potential secondary treatment effects which may arise. Also, in light of the current findings clinicians should regularly assess the sleep patterns of clients with SSD, a practice which does not seem to be routinely conducted (Rehman et al., 2017). Clinical workers involved with caring for individuals with SSD should be cognisant of those who exhibit reduced sleep, positive symptoms and/or hopelessness.

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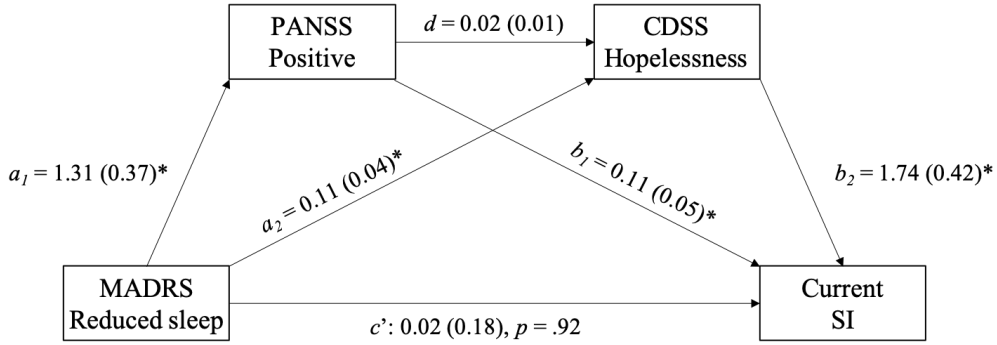
Table 1. Characteristics for patients with or without suicidal ideation.					
Variable	Total Sample (*N*=118)	No suicidal ideation (*n* = 98)	Suicidal ideation (*n* = 20)	*t (df)* or X^2 *(df)*	Cohen's *d* or Cramer's *V*
Age	41.0 (10.0)	41.1 (9.8)	40.6 (11.2)	0.21 (116), $p = .83$	0.05
Female: *n* (%)	51 (43.2%)	37 (37.8%)	14 (70.0%)	7.04 (1), $p = .008$	0.24
Education years	14.1 (3.2	13.8 (3.3)	15.3 (2.0)	1.90 (106), $p = .06$	0.48
Schizoaffective: *n* (%)	38 (32.2%)	28 (28.6%)	10 (50.0%)	3.49 (1), $p = .06$	0.17
Years since diagnosis	15.5 (9.6)	15.3 (9.6)	16.6 (9.7)	0.52 (108), $p = .61$	0.13
PANSS Positive	18.0 (7.7)	16.8 (6.8)	23.8 (9.4)	3.14 (23.2), $p < .001$	0.96
PANSS Negative	14.4 (6.5)	13.7 (6.1)	17.7 (7.2)	2.60 (116), $p = .01$	0.64
PANSS Disorganised	19.5 (6.9)	19.3 (6.7)	20.8 (7.8)	0.87 (116), $p = .39$	0.22
PANSS Depression (G6)	2.6 (1.8)	2.1 (1.4)	5.0 (1.5)	8.56 (116), $p < .001$	2.10
MADRS Reduced Sleep	1.2 (1.8)	1.0 (1.7)	2.3 (2.0)	2.78 (116), $p = .006$	0.74
MADRS Suicidal Thoughts	0.6 (1.4)	0.1 (0.24)	3.3 (1.3)	11.09 (19.27), $p < .001$	5.61
CDSS Hopelessness	0.6 (0.8)	0.3 (0.6)	1.7 (1.0)	5.69 (21.9), $p < .001$	2.05
WTAR Scaled Score	101.9 (12.1)	101.6 (12.0)	103.2 (12.7)	0.53 (116), $p = .60$	0.13
Notes: Values are presented as mean (standard deviation) unless stated otherwise; Education years unavailable for 10 participants; Years since diagnosis unavailable for 8 participants; CDSS = Calgary Depression Scale for Schizophrenia; MADRS = Montgomery-Åsberg Depression Rating Scale; PANSS = Positive and Negative Syndrome Scale; WTAR = Wechsler Test of Adult Reading.					

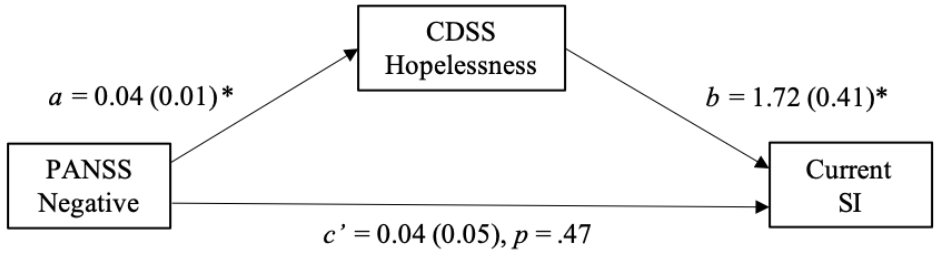
Table 2. Medication details for patients with or without suicidal ideation.					
Variable	Total Sample (*N*=118)	No suicidal ideation (*n* = 98)	Suicidal ideation (*n* = 20)	*t (df)* or X^2 *(df)*	Cohen's *d* or Cramer's *V*
Antipsychotic: *n* (%)				0.60 (2), *p* = .74	0.07
Atypical only	93 (78.7%)	76 (77.6%)	17 (85.0%)		
Typical only	11 (9.3%)	10 (10.2%)	1 (5.0%)		
Atypical and Typical	5 (4.2%)	4 (4.1%)	1 (5.0%)		
Clozapine only	15 (12.7%)	14 (14.3%)	1 (5.0%)		
CPZ equivalence	606.1 (508.5)	598.7 (458.7)	649.94 (554.4)	0.33 (102), *p* = .58	0.11
Typical Antipsychotic					
Chlorpromazine	1 (0.9%)	1 (1.0%)	-		
Flupentixol	4 (3.4%)	4 (4.1%)	-		
Fluphenazine	1 (0.9%)	-	1 (5.0%)		
Haloperidol	2 (1.7%)	2 (2.0%)	-		
Trifluoperazine	1 (0.9%)	1 (1.0%)	-		
Zuclopenthixol	7 (5.9%)	6 (6.1%)	1 (5.0%)		
Atypical Antipsychotic					
Amisulpride	8 (6.7%)	6 (6.1%)	2 (10.0%)		
Aripiprazole	27 (22.9%)	19 (19.4%)	8 (40.0%)		
Asenapine	4 (3.4%)	2 (2.0%)	2 (10.0%)		
Clozapine	25 (21.2%)	22 (22.5%)	3 (15.0%)		
Lurasidone	1 (0.9%)	1 (1.0%)	-		
Olanzapine	18 (15.3%)	15 (15.3%)	3 (15.0%)		
Paliperidone	6 (5.1%)	6 (6.1%)	-		
Quetiapine	21 (17.8%)	16 (16.3%)	5 (25.0%)		
Risperidone	9 (7.6%)	8 (8.2%)	1 (1.0%)		
Ziprasidone	4 (4.2%)	3 (3.1%)	2 (10.0%)		
Notes: CPZ equivalence unavailable for 14 participants; CPZ, chlorpromazine					

Table 3. Partial correlation *r*-values among clinical variables covarying for sex (*N* = 118).						
	2	3	4	5	6	7
1. MADRS Suicidal Thoughts	.35**	.28*	.12	.61**	.23*	.58**
2. PANSS Positive		.23*	.61**	.35**	.31**	.24**
3. PANSS Negative			.34**	.43**	.20*	.34**
4. PANSS Disorganised				.19*	.18*	.21*
5. PANSS Depression (G6)					.48**	.66**
6. MADRS Reduced Sleep						.29**
7. CDSS Hopelessness						
CDSS = Calgary Depression Scale for Schizophrenia; MADRS = Montgomery-Åsberg Depression Rating Scale; PANSS = Positive and Negative Syndrome Scale * *p* significant after applying False Discovery Rate. ** $p < .01$						

Table 4. Hierarchical logistic regression predicting current suicidal ideation (N = 118).				
	Step X^2 change (p)	B (SE)	p	Odds Ratio (95% CI)
Step 1	12.43 (.002)			
Female Sex		1.27 (0.54)	.020	3.55 (1.22-10.30)
MADRS Reduced Sleep		0.29 (0.13)	.020	1.34 (1.05-1.71)
Step 2	14.50 (.001)			
Female Sex		1.72 (0.62)	.006	5.56 (1.64-18.86)
MADRS Reduced Sleep		0.15 (0.14)	.300	1.16 (0.88-1.53)
PANSS Positive		0.11 (0.04)	.008	1.11 (1.03-1.20)
PANSS Negative		0.09 (0.04)	.039	1.09 (1.00-1.18)
Step 3	20.47 (<.001)			
Female Sex		1.38 (0.72)	.055	3.97 (0.97 -16.19)
MADRS Reduced Sleep		0.02 (0.18)	.920	1.02 (0.71-1.46)
PANSS Positive		0.11 (0.05)	.021	1.12 (1.02-1.23)
PANSS Negative		0.03 (0.06)	.630	1.03 (0.92-1.14)
CDSS Hopelessness		1.67 (0.44)	<.001	5.30 (2.25-12.47)
CDSS = Calgary Depression Scale for Schizophrenia; MADRS = Montgomery-Åsberg Depression Rating Scale; PANSS = Positive and Negative Syndrome Scale Step 3: Cox & Snell R^2 = .33; Nagelkerke R^2 = .55; 88% of cases correctly classified				



Log-odds indirect effect (95% bootstrapped CI): 0.37 (0.17 to 0.89)
Mediating through PANSS Positive: 0.15 (0.02 to 0.45)
Mediating through CDSS Hopelessness: 0.18 (0.00 to 0.55)
Mediating through PANSS Positive and CDSS Hopelessness: 0.04 (-0.01 to 0.15)



Log-odds indirect effect (95% bootstrapped CI): 0.08 (0.03 to 0.19)

Figure 1. Effects (bootstrapped standard error in parenthesis) for serial (model 6) and simple (model 4) mediation examining how clinical symptoms were associated with current suicidal ideation after covarying for sex (*N*=118). * $p < 0.05$

1. Supplementary Section 1 – Calgary Depression Scale for Schizophrenia

In the following analysis item 8 of the Calgary Depression Scale for Schizophrenia (CDSS) was used to measure suicide. CDSS item 8 differs from the MADRS item 10, as item 10 of the MADRS pertains only to suicidal thoughts/ideation, whereas the CDSS rating is based on both thinking and behaviour. A score of ≥1 was used to categorise participants with current suicidal ideation. Furthermore, CDSS item 7 (early wakening) was included in lieu of MADRS item 4 (reduced sleep).

Table S1. Partial correlation *r*-values among clinical variables covarying for sex ($N = 118$).

	2	3	4	5	6	7
1. CDSS Suicide	.36**	.25*	.12	.57**	.34*	.58**
2. Positive PANSS		.23*	.61**	.35**	.26**	.24**
3. Negative PANSS			.34**	.43**	.12	.34**
4. Disorganised PANSS				.19	.12	.21
5. PANSS Depression					.45**	.67**
6. CDSS Early Wakening						.36**
7. CDSS Hopelessness						

CDSS = Calgary Depression Scale for Schizophrenia; PANSS = Positive and Negative Syndrome Scale * *p* significant after applying False Discovery Rate. ** $p < .01$

Table S1 shows the partial correlation *r*-values (adjusting for sex due to its relationship with current suicidal ideation) between psychopathology, early wakening and suicidal thinking/behaviour. Increased positive and negative symptoms, depression, early wakening and hopelessness were significant correlates of greater suicidal thinking/behaviour. More frequent early awakenings were also associated with significantly more severe positive symptoms, depression, and hopelessness.

Table S2. Hierarchical logistic regression predicting current suicidal thinking/behaviour (N = 118).				
	Step X^2 change (p)	B (SE)	p	Odds Ratio (95% CI)
Step 1	18.24 (<.001)			
Female Sex		1.58 (0.62)	.010	4.88 (1.47-16.16)
CDSS Early Wakening		0.76 (0.23)	.001	2.15 (1.36-3.38)
Step 2	12.57 (.002)			
Female Sex		1.96 (0.69)	.005	7.09 (1.84-27.36)
CDSS Early Wakening		0.59 (0.25)	.017	1.81 (1.11-2.94)
PANSS Positive		0.11 (0.04)	.011	1.11 (1.02-1.21)
PANSS Negative		0.08 (0.05)	.076	1.08 (0.99-1.21)
Step 3	17.67 (<.001)			
Female Sex		1.51 (0.77)	.049	4.52 (1.01 -20.30)
CDSS Early Wakening		0.36 (0.31)	.241	1.43 (0.79-2.61)
PANSS Positive		0.12 (0.05)	.017	1.13 (1.02-1.24)
PANSS Negative		0.01 (0.06)	.885	1.00 (0.90-1.13)
CDSS Hopelessness		1.64 (0.46)	<.001	5.15 (2.09-12.69)
CDSS = Calgary Depression Scale for Schizophrenia; PANSS = Positive and Negative Syndrome Scale; Step 3: Cox & Snell R^2 = .34; Nagelkerke R^2 = .59; 91% of cases correctly classified				

Table S2 shows the results from the three-step hierarchical logistic regression analysis. Each step made a significant additive contribution to the prediction of risk of current suicidal thinking/behaviour. Both female sex and early awakening were significant correlates in step 1 and remain significant in step 2 after the addition of positive and negative symptoms. Female sex, early wakening, and positive symptoms remained as significant multivariable covariates in step 2. After the addition of hopelessness in step 3, female sex, positive symptoms, and hopelessness remained as significant multivariable covariates.

As the coefficient for reduced sleep was reduced in both steps 2 and 3, there is evidence that positive symptoms and hopelessness mediate its relationship with suicidal thinking/behaviour. An independent significant mediated pathway was evident between early wakening – hopelessness – suicide (β = .13, SE = .06, CI: 0.03 to 0.26), however not for early wakening – positive symptoms – suicide (β = 0.03, SE = .03, CI: -0.01 to 0.11) . No

significant serial mediation from early wakening through positive symptoms and hopelessness to suicide was found (β = 0.02, SE = .02, CI: -0.01 to 0.05).

2. **Supplementary Section 2 – Depression**

Table S3. Hierarchical logistic regression predicting current suicidal ideation (N = 118).				
	Step X^2 change (p)	B (SE)	p	Odds Ratio (95% CI)
Step 1	12.43 (.002)			
Female Sex		1.27 (0.54)	.020	3.55 (1.22-10.30)
MADRS Reduced Sleep		0.29 (0.13)	.020	1.34 (1.05-1.71)
Step 2	14.50 (.001)			
Female Sex		1.72 (0.62)	.006	5.56 (1.64-18.86)
MADRS Reduced Sleep		0.15 (0.14)	.300	1.16 (0.88-1.53)
PANSS Positive		0.11 (0.04)	.008	1.11 (1.03-1.20)
PANSS Negative		0.09 (0.04)	.039	1.09 (1.00-1.18)
Step 3	20.47 (<.001)			
Female Sex		1.38 (0.72)	.055	3.97 (0.97-16.19)
MADRS Reduced Sleep		0.02 (0.18)	.920	1.02 (0.71-1.46)
PANSS Positive		0.11 (0.05)	.021	1.12 (1.02-1.23)
PANSS Negative		0.03 (0.06)	.630	1.03 (0.92-1.14)
CDSS Hopelessness		1.67 (0.44)	<.001	5.30 (2.25-12.47)
Step 4	12.32 (<.001)			
Female Sex		1.69 (0.84)	.043	5.41 (1.05-27.80)
MADRS Reduced Sleep		-0.23 (0.22)	.286	0.79 (0.52-1.21)
PANSS Positive		0.12 (0.61)	.045	1.13 (1.00-1.27)
PANSS Negative		0.02 (0.07)	.728	0.98 (0.84-1.12)
CDSS Hopelessness		0.87 (0.51)	.086	2.40 (0.88-6.51)
PANSS Depression		0.99 (0.33)	.003	2.70 (1.40-5.19)
CDSS = Calgary Depression Scale for Schizophrenia; MADRS = Montgomery-Asperg Depression Rating Scale; PANSS = Positive and Negative Syndrome Scale Step 3: Cox & Snell R^2 = .33; Nagelkerke R^2 = .55; 88% of cases correctly classified				

Table S3 shows the results from the four-step hierarchical logistic regression analysis. Each step made a significant additive contribution to the prediction of risk of current suicidal ideation. Both female sex and reduced sleep were significant correlates in step 1, but reduced sleep became non-significant in step 2 after the addition of positive and negative symptoms. Female sex and positive and negative symptoms remained as significant multivariable covariates in step 2. After the addition of hopelessness in step 3, only hopelessness and

positive symptoms remained as significant multivariable covariates. With the addition of depression in step 4, hopelessness was no longer significant, with depression, positive symptoms and female sex remaining as significant multivariable covariates.

As the coefficient for reduced sleep was reduced in steps 2, 3 and 4, there is evidence that positive symptoms and depression mediate its relationship with suicidal ideation. Independent significant mediated pathways were evident: reduced sleep – positive symptoms – suicidal ideation ($\beta = 0.05$, SE = .03, CI: 0.00 to 0.13); and reduced sleep – depression – suicidal ideation ($\beta = 0.26$, SE = .07, CI: 0.12 to 0.41). A significant serial mediation through positive symptoms to depression was found ($\beta = 0.04$, SE = .02, CI: 0.01 to 0.09).