Sleep problems and attenuated psychotic symptoms in youth at clinical high-risk for psychosis

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ABSTRACT

There has been growing interest on the effect of sleep problems on psychotic and prodromal symptoms. The current study investigated cross-sectional relations between sleep problems and attenuated psychotic symptoms in a large sample of 740 youth at Clinical High Risk (CHR) for psychosis in an attempt to replicate previous findings and assess whether findings from general population samples and psychotic samples extend to this CHR sample. Sleep problems were found to be significantly positively associated with attenuated psychotic symptom severity. Sleep problems were also found to be more closely associated with certain specific prodromal symptoms (e.g., suspiciousness and perceptual abnormalities) than other attenuated psychotic symptoms. Further, we found that depression mediated the cross-sectional association between sleep problems and paranoid symptoms only. This adds to a growing body of evidence suggesting the mediation role of depression is more pronounced for paranoid-type psychotic symptoms as compared to other psychotic symptoms (e.g., hallucinations).

1. Introduction

There is a growing interest and recognition of the role sleep problems play in the pathophysiology of psychiatric illness, including psychosis. Sleep problems are common in psychotic disorders with between 30 and 80% of patients with schizophrenia reporting significant sleep disturbance (Cohrs, 2008). In patients with schizophrenia, sleep disruption has been found to precede relapse (Benson, 2008). Retrospective studies of those with a first episode of psychosis show that sleep disturbance was among the most common prodromal symptoms of the disorder (Yung and McGorry, 1996). Recent prospective studies also suggest that sleep disturbance is common among individuals at clinical-high risk (CHR) for psychosis and is associated with more severe attenuated psychotic symptoms (e.g., sub-

clinical hallucinations, delusions, grandiosity) (Poe et al., 2017; Lunsford-Avery et al., 2017). Additionally, there is preliminary evidence that certain aspects of sleep disturbance (e.g., specific kinds of circadian rhythm disruptions measured through actigraphy, and reduced sleep duration) may be associated with worsening prodromal symptoms over time (Lunsford-Avery et al., 2017, 2015; Reeve et al., 2018). These findings point to a potential role of sleep dysfunction in the pathophysiology of psychosis-spectrum disorders and highlight the need to better understand this relationship in CHR samples.

There is also evidence that sleep problems may be associated with specific types of psychotic-like symptoms. For example, epidemiological surveys have found that a diagnosis of insomnia is associated with a five times greater risk of paranoid thoughts (e.g., "a group of people are plotting to harm me"), and a four to five times greater likelihood of

transient hallucinatory experiences (e.g., "hearing name being called when no-one was around") (Freeman et al., 2012). In addition, sleep deprivation studies utilizing experimental and observational designs have documented associations between acute sleep loss and psychotic experiences. For example, Hurdiel et al. (2014) observed ultra-marathon runners who maintained wakefulness for approximately 46 h and found that 4 of the 17 (24%) participants experienced hallucinatory experiences during their sleep deprived state. Sleep deprivation has also been associated with increased paranoid thoughts and perceptual distortions in experimental studies with healthy adults (Reeve et al., 2017; Petrovsky et al., 2014). Taken together, the evidence from general population studies suggests that sleep disturbance may be specifically associated with transient suspiciousness/paranoid-type thoughts and perceptual abnormalities/hallucinations in otherwise healthy individuals. Given the idea that psychotic symptoms may exist on a continuum (Van Os et al., 2009), we might expect to see the same pattern of psychotic-like symptoms affected by sleep problems in clinical samples as well is seen in healthy samples. Indeed, one recent study of 160 individuals at high risk for psychosis revealed that shorter sleep duration was associated with more severe delusional/persecutory ideas and hallucinations (Reeve et al., 2018). Together, this suggests that the same specificity of sleep and psychotic-like symptoms exists that exists in healthy populations also extends to those with the more enduring attenuated psychotic symptoms observed in CHR and patient populations.

Several studies have investigated potential mediators of the relationship between sleep problems and psychotic symptoms. A systematic review linking sleep dysfunction and psychotic experiences found that depression was the most commonly tested mediator and that it partially mediated the relationship between sleep dysfunction and psychotic experiences in a number of studies (Reeve et al., 2015). The role of depression in the relationship between sleep and psychotic symptoms is consistent with cognitive models of persecutory delusions (Freeman et al., 2002) and hallucinatory experiences (Waters et al., 2012), in which negative affect (encompassing symptoms of depression and anxiety) is posited to play an important role in symptom formation. These models posit that negative beliefs about the self and the world, as well as negative affective states, result in certain biases when selecting explanations for and making meaning from anomalous internal experiences (e.g., perceptual abnormalities) and recent external experiences (e.g., ambiguous interpersonal interactions). Thus, ambiguous interpersonal interactions may be interpreted as threatening, fueling paranoia, and perceptual abnormalities are interpreted as malevolent (e.g., hearing voices saying personally insulting things). The role of depression in these associations is also relevant given the high rates of co-morbid depression observed in psychosis-spectrum populations. Between 40–50% of CHR and schizophrenia patients also meet criteria for a depressive disorder (Buckley et al., 2008). Thus, clarifying whether depression mediates the relationship between sleep and psychotic symptoms will have important implications for treatment approaches and targets with CHR individuals.

The current study investigated the associations between sleep disturbance, attenuated psychotic symptoms, and clinical outcomes in a large sample of CHR individuals drawn from the North American Prodrome Longitudinal Study (NAPLS-II). The aims were to (1) determine whether CHR individuals report greater sleep disturbance relative to healthy controls, (2) examine whether sleep problems are associated with specific types of attenuated psychotic symptoms, (3) test whether depression mediates the relationship between sleep problem and attenuated psychotic symptoms, and (4) explore whether sleep disturbance predicts subsequent worsening of prodromal psychotic symptoms and/or conversion to a psychotic illness.

Based on previous research in CHR samples (i.e., Poe et al., 2017; Lunsford-Avery et al., 2017), we predicted that sleep disturbance would be more severe in the CHR group in comparison to the healthy control (HC) group. Within the CHR group, we hypothesized that sleep disturbance would be positively associated with the severity of attenuated psychotic symptoms at baseline, and that these associations would be strongest for paranoia/suspiciousness and perceptual abnormalities symptoms. We also predicted that sleep disturbance would have an indirect effect on total attenuated psychotic symptoms through depressive symptoms. Finally, based on previous findings that sleep disturbance predicts worsening of psychotic symptoms over time (Lunsford-Avery et al., 2017), we hypothesized that sleep disruption reported at baseline would predict progression of the psychosis risk syndrome and/or conversion to a psychotic disorder at subsequent time points.

2. Methods

2.1. Participants

See Table 2. for detailed demographic and sample characteristics. The total sample included 1020 individuals between 12–30 years of age. Of the 1020 participants 740 (72.5%) met clinical-high risk (CHR) criteria for psychosis; 280 (27.5%) were healthy controls. All participants were recruited as part of the North American Prodrome Longitudinal Study (NAPLS-2). Specific details about ascertainment, inclusion, and exclusion criteria have been described in detail elsewhere (Addington et al., 2012).

2.2. Assessments

2.2.1. CHR status

Clinical-high risk (CHR) designation was based on the *Structured Interview for Psychosis-Risk Syndromes* (SIPS) (McGlashan et al., 2010). This semi-structured interview assesses prodromal symptoms across 4 symptom domains: (1.) *Positive* (e.g., unusual thought content/ideas, suspiciousness/persecutory ideas, grandiosity, perceptual abnormalities, and disorganized communication); (2.) *Negative* (e.g., social isolation, avolition, decreased expression of emotion, decreased experience of emotions and self, decreased ideational richness, and deterioration of role functioning), (3.) *Disorganized* (e.g., odd behavior of appearance, bizarre thinking, trouble with focus and attention, and impairment in personal hygiene or social attentiveness), and (4.) *General* (e.g., difficulty related to sleep disturbance, dysphoric mood, motor disturbances, and impaired tolerance to normal stress). After administration of the SIPS, each participant's their attenuated psychotic symptoms are then rated on a 0–6 scale on the Scale of Prodromal Symptoms (SOPS). CHR status is based on the severity of the positive attenuated psychotic symptoms (see Addington et al., 2012 for details). The SIPS was administered at each study time point (i.e., 6 m, 12 m, 18 m, 24 m) to assess severity of prodromal symptoms and current clinic state.

2.2.2. Current clinical state

Current Clinical State was determined at each subsequent visit (6 months, 12 months, 18 months, 24 months) using symptom ratings from the SOPS. CHR participants are classified either as 'symptomatic' (i.e., scores between 3 and 5 on the SOPS positive symptoms with no recent changes in severity), 'prodromal progression' (i.e., scores between 3 and 5 on the SOPS positive symptoms with a recent increase in severity) 'converted' (i.e., currently meeting criteria for a psychotic disorder), or 'in remission' (i.e., scores of 2 or less on each of the SOPS positive symptoms). The last available clinical state classification was used as the outcome measure.
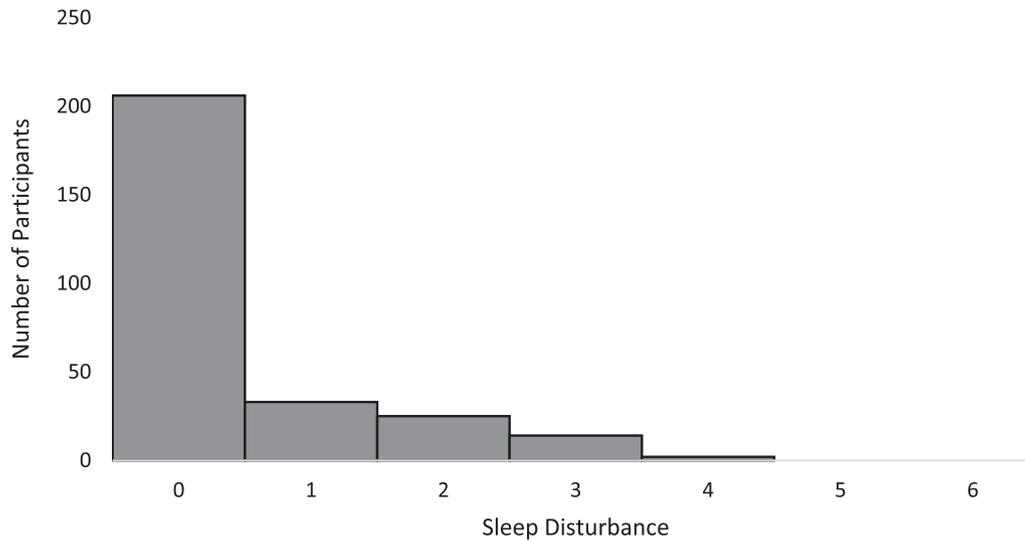
2.2.3. Sleep disturbance

Measures of sleep disturbance were obtained the general symptoms section of the SOPS, which includes an item assessing sleep patterns and concerns (G1). The Sleep Disturbance Item is rated from 0–6 based on the severity of the disturbance. The ratings are as follows: 0 – Absent/

Table 1

G1 SIPS sleep disturbance symptom, based on the SIPS (McGlashan et al., 2010).

Symptom scale	Descriptive anchors*
0 (Absent)	
1 (Questionably present)	Restless sleep.
2 (Mild)	Some mild difficulty falling asleep or getting back to sleep.
3 (Moderate)	Daytime fatigue resulting from difficulty falling asleep at night or early awakening. Sleeping more than considered average.
4 (Moderately severe)	Sleep pattern significantly disrupted and has intruded on other aspects of functioning (e.g. trouble getting up for school or work). Difficult to awaken for appointments. Spending a large part of the day asleep.
5 (Severe)	Significant difficult falling asleep or awakening early on most nights. May have day/night reversal. Usually not getting to scheduled activities at all.
6 (Extreme)	Unable to sleep at all for over 48 h.

* Anchors in each scale are intended to provide guidelines and examples of signs for every symptom observed. It is not necessary to meet every criterion in any one anchor to assign a particular rating. Basis for ratings includes both interviewer observations and patient reports.

Fig. 1. Count data showing the distribution of healthy control participants across each level of sleep disturbance (0 = no sleep disturbance, 6 = extreme sleep disturbance).
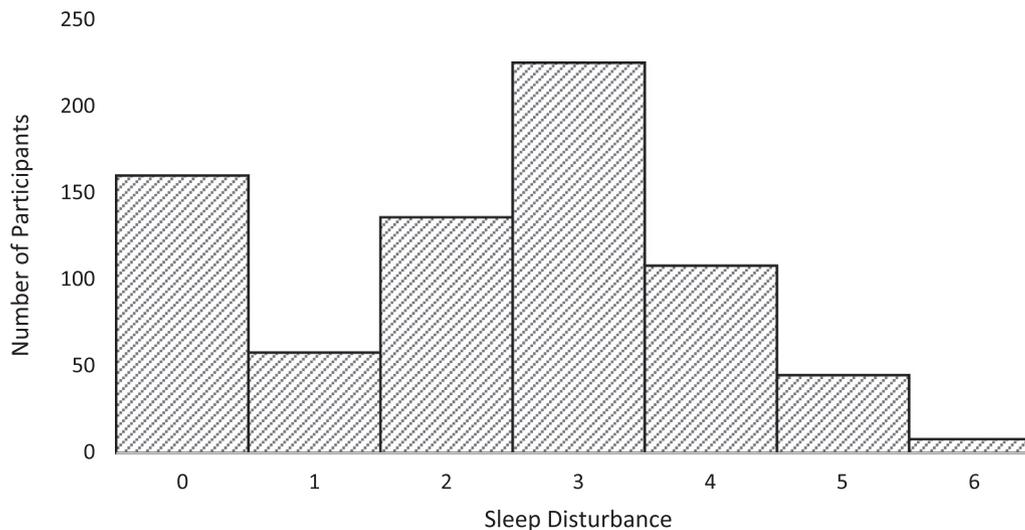
Fig. 2. Count data showing the distribution of clinical high risk participants across each level of sleep disturbance (0 = no sleep disturbance, 6 = extreme sleep disturbance).

No sleep disturbance, 1 – Questionably Present (e.g., restless sleep), 2 – Mild (e.g., some difficulty falling asleep), 3 – Moderate (e.g., daytime fatigue due to difficulty sleeping), 4 – Moderately Severe (e.g., sleep pattern interfering with other aspects of functioning), 5 – Severe (e.g., day/night reversal and missing activities due to sleep problems), 6 – Extreme (e.g., unable to sleep for over 48 h) Table 1. Unlike the positive attenuated psychotic symptoms, there were no restrictions on the sleep item in terms of qualifying as CHR. Thus, scores on the sleep variable were allowed to vary across the full range (from 0–6) for all participants across the entire sample Figs. 1–2.

2.2.4. Depression symptoms

The *Calgary Depression Scale for Schizophrenia* – The CDSS (Addington et al., 1993) was designed to measure depressive symptoms in patients with schizophrenia and shows good reliability and validity for measuring depressive symptoms in those at CHR of psychosis (Addington et al., 2014). It is administered by an experienced rater in the context of a clinical interview and assesses nine symptoms of depression that are summed to create a total score indicating severity of depressive symptoms.

2.3. Statistical analyses

All analyses were performed using SPSS version 25. Analysis of covariance (ANCOVA) was conducted to compare severity of sleep disturbance between groups (CHR versus HC). All subsequent analyses were performed only in the CHR group. Linear regression was used to examine the associations between baseline sleep disturbance and total positive symptom severity. A series of MANCOVA and ANCOVAs were used to examine the associations between sleep disturbance and specific attenuated psychotic symptoms (i.e., delusions, suspiciousness/paranoia, grandiosity, perceptual abnormalities, disorganized communication). To test for mediation, both direct and indirect effects were examined using a bootstrapping approach with the PROCESS macro for SPSS (Hayes, 2013). Confidence intervals that do not include zero indicate a significant effect. Finally, an ANCOVA was conducted to test whether the 4 clinical state outcome groups (remission, symptomatic, prodromal progression, and converted) differed on baseline sleep disturbance. All participants were coded as belonging to one of the 4 outcome groups using the last observation carried forward (LOCF) method.

Prior to analyses, we tested whether baseline medication status (psychotropic and sleep medications), cannabis use, and alcohol use were related to the sleep variable. We found no significant associations with any medication class or with alcohol use (see Table 3) and medication or alcohol use status was therefore not included as a covariate in the analyses. The lack of association between sleep disturbance and use of sleep medication likely indicates that the use of sleep medication is effective at reducing sleep disturbance. Lifetime cannabis use was significantly associated with the sleep variable [t(722) = −2.386, $p = 0.017$] such that those who reported any cannabis use had higher mean scores on the sleep variable, indicating poorer sleep ($n = 399$, $M = 2.43$) and those who denied ever using cannabis had lower mean scores on the sleep variable, indicating better sleep ($n = 325$, $M = 2.15$). Thus, cannabis use was included as a covariate in all analyses.

3. Results

Demographic and symptom characteristics of the sample are shown in Table 2. The CHR group was younger ($t = 3.759$, $p < 0.001$) and had a larger proportion of males compared to the HC group (chi = 3.960, $p = 0.047$). There were no group differences in race or ethnicity. Age, sex, and cannabis use were included as covariates in analyses comparing CHR and HC differences. However, neither age nor sex were significantly associated with severity of sleep problems within the CHR group. CHR individuals reported higher levels of sleep disturbance [F (1995) = 306.975, $p < 0.001$] compared to controls.

3.1. Sleep disturbance and attenuated psychotic symptoms

In the CHR group, sleep disturbance was positively associated with total positive symptom severity ($R^2 = 0.032$ $F(2720) = 11.714$, $p < 0.01$, sleep disturbance $\beta = 0.160$). Post-hoc analyses were conducted to determine whether this association differed between CHR individuals with a clinically significant sleep disturbance (i.e., rated as a 3 or above on the SOPS) from those without (i.e., rated as 0–2 on the

Table 2
Sample characteristics.

	CHR	Control
N	740	280
*Age, years (mean ± SD)***	18.5 (± 4.26)	19.7 (± 4.67)
*Sex, n (%)**		
Males	424 (57.3%)	141 (50.4%)
Females	316 (42.7%)	139 (49.6%)
Race, n (%)		
White	426 (57.6%)	152 (54.3%)
Black	111 (15%)	49 (17.5%)
Interracial	94 (12.7%)	29 (10.4%)
Central/South American	32 (4.3%)	13 (4.6%)
East Asian	19 (2.6%)	15 (5.4%)
South Asian	20 (2.7%)	8 (2.9%)
Southeast Asian	15 (2.0%)	7 (2.5%)
First Nations	13 (1.8%)	4 (1.4%)
West/Central Asia and Middle East	6 (0.8%)	2 (0.7%)
Native Hawaiian or Pacific Islander	3 (0.4%)	1 (0.4%)
Ethnicity (Hispanic/Lantinx), n (%)	136 (18.4%)	50 (17.9%)
*Attenuated psychotic symptoms, mean (SD)***	11.86 (3.832)	1.07 (1.67)
*Negative symptoms, mean (SD)***	11.89 (6.067)	1.46 (2.23)
*Sleep disturbance, mean (SD)***	2.31 (1.568)	0.48 (0.904)

Percentages add up to > 100% because one participant can score multiple items.

* $p < 0.05$.
** $p < 0.01$.

SOPS). There was a group difference (clinical vs. nonclinical sleep disturbance) in the severity of total attenuated psychotic symptoms [F (1, 737) = 12.365, $p < 0.001$], such that the clinical sleep disturbance group had higher levels of total attenuated psychotic symptoms. Follow-up analyses of specific attenuated psychotic symptoms showed significant group differences in severity of suspiciousness/paranoia [F (1720) = 7.54, $p = 0.006$), perceptual abnormalities/hallucinations [F (1720) = 10.719, $p = 0.001$], and disorganized communication [F (1720) = 4.696, $p = 0.031$] Table 4. No significant group differences were observed in the severity of unusual thought content/delusions or grandiosity symptoms. After applying the Bonferroni method to correct for multiple comparisons, only the association between sleep problems and suspiciousness/paranoia and the association between sleep problems and perceptual abnormalities/hallucinations remained significant. We then tested for an indirect effect of depression in the association between sleep disturbance and total attenuated psychotic symptoms, and we found no significant indirect effect (Fig. 3). This was repeated for each specific attenuated psychotic symptom associated with sleep disturbance (i.e., suspiciousness, perceptual abnormalities, disorganized communication) (Figs. 4–6). There was an indirect effect of sleep disturbance only on suspiciousness/persecutory symptoms through depression ($b = 0.0537$, CI (95%) = 0.0319–0.0787), but no significant indirect effect was found between sleep disturbance and perceptual abnormalities or disorganized communication.

3.2. Sleep disturbance and clinical outcome

There were no significant differences in baseline sleep disturbance among the four clinical outcome groups (remission, symptomatic, prodromal progression, conversion). This result remained both when controlling for cannabis use and without controlling for cannabis use.

4. Discussion

The current study examined the associations between sleep disturbance, attenuated psychotic symptoms and clinical outcomes in a large sample of CHR individuals. Consistent with our hypotheses, we found that CHR individuals reported greater sleep disturbance than healthy controls and that the severity of sleep disturbance was positively associated with concurrent severity of attenuated psychotic

Table 3
Results of correlations and t-tests comparing sleep disturbance rating in CHR participants with and without medication or alcohol use reported at baseline.

Type of medication	Mean sleep disturbance rating		Correlation (r_{pb})	t - value	df	p - value
	Substance use (n)	No substance use (n)				
Sleep medication	2.63 (42)	2.29 (722)	0.050	− 1.359	738	0.174
Antipsychotic	2.20 (213)	2.36 (551)	− 0.046	1.239	738	0.216
Antidepressant	2.39 (324)	2.25 (440)	0.044	− 1.192	738	0.234
Mood stabilizer	2.26 (77)	2.32 (687)	− 0.011	0.302	738	0.763
Stimulant	2.20 (166)	2.34 (598)	− 0.037	1.015	738	0.311
Benzodiazepine	2.58 (93)	2.27 (671)	0.065	− 1.778	738	0.076
Any psychotropic	2.32 (464)	2.29 (300)	0.011	− 0.287	738	0.774
Alcohol	2.33 (292)	2.30 (444)	0.009	− 0.235	721	0.815

symptoms. We advance the current literature by demonstrating that these sleep disturbance and positive symptom associations are specific to suspiciousness (precursor of paranoia), perceptual abnormalities (precursor of hallucinations), and disorganized speech (indicator/precursor of thought disorder). Contrary to our hypothesis, depression did not mediate the association between sleep disturbance and total attenuated psychotic symptoms; however, it did mediate the association with suspiciousness. Additionally, baseline sleep disturbances did not differ among clinical outcomes groups.
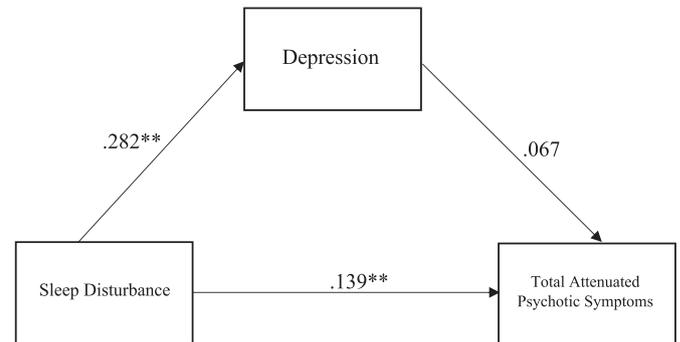
Our finding that greater sleep disturbance is associated with more severe attenuated psychotic symptoms in CHR individuals is consistent with studies in the general population (Freeman et al., 2012) as well as those in at risk samples (Poe et al., 2017; Lunsford-Avery et al., 2017; Reeve et al., 2018). Specific associations between sleep disturbance and suspiciousness and between sleep disturbance and perceptual abnormalities have also been reported in previous literature and were replicated in the current study (Freeman et al., 2012; Reeve et al., 2018). We did not originally hypothesize that disorganized speech would be specifically associated with sleep disturbance, as this was not a consistent finding in the previous literature. However, two experimental studies separately found cognitive disorganization was one of the psychotic-like symptoms elicited by sleep deprivation in healthy subjects (Petrovsky et al., 2014; Reeve et al., 2017). In the current study, the association between sleep disturbance and disorganized speech did not remain significant after correcting for multiple comparisons. This fact combined with the generally sparse previous evidence of association suggests that if sleep problems and cognitive disorganization are indeed associated in healthy samples and/or CHR samples, the relationship is relatively weaker than the relationship between sleep disturbance and suspiciousness or perceptual abnormalities. Overall, when comparing the current findings with previous literature on healthy samples, it appears that the same pattern of association between sleep disturbance and psychotic-like symptoms is observed in both CHR individuals and healthy participants under conditions of sleep deprivation.

Interestingly, in our analysis of depression as a mediator between sleep disturbance and psychotic symptoms, we found no significant indirect effects on total positive symptom severity. This finding appears at odds with conclusions from a systematic review of the literature (Reeve et al., 2015) which reported that depression is consistently found to be a partial mediator in the relationship between sleep dysfunction and psychotic experiences. However, studies included in this review varied in their definition of 'psychotic symptoms', and those that

Fig. 3. Standardized regression coefficients for the relationship between sleep disturbance and total attenuated psychotic symptoms (controlling for cannabis use). The indirect effect through depression was not significant (unstandardized indirect effect: $b = 0.0467$, 95% CI [− 0.0064, 0.1028]). *$p < 0.05$, **$p < 0.01$.

found a mediating effect of depression tended to focus on paranoid/ suspicious dimension of psychotic symptoms (e.g., Freeman et al., 2009; Freeman et al., 2012; Myers et al., 2011). In addition, two recent investigations have found that depression/negative affect is a more significant mediator of the relation of sleep with paranoia, than with other psychotic symptoms (e.g., hallucinations) (Reeve et al., 2017; Reeve et al., 2018). Thus, our current findings are consistent with growing evidence that the mediation role of depression is more pronounced for paranoid-type psychotic symptoms. Further, our findings also lend support to the idea that sleep problems may be associated with different prodromal symptoms through different mechanisms (Reeve et al., 2018).

As previously mentioned, the cognitive model of persecutory delusions provides a framework for understanding how depression or negative affect may act as a mediator between sleep problems and paranoid thinking. This model posits that certain cognitive biases, often present in those with depression and anxiety, may predispose someone to select a threatening/suspicious explanation for neutral experiences (Freeman et al., 2002). Thus, if sleep disturbance increases depressive/ anxious cognitive biases (as suggested in various studies including Gobin et al., 2015; Alfano et al., 2009), then suspiciousness/paranoid symptoms will also increase in severity. An increase in suspiciousness would, in turn, be expected to result in a state of hypervigilance which would be expected to further interfere with sleep. Preliminary support

Table 4
Summary of univariate analyses for types of attenuated psychotic symptoms.

| Symptom	df	Mean square	F	p - value	Group mean (sleep disturbance)	Group mean (no sleep disturbance)
Unusual thought Content/delusional ideas	1720	2.682	1.529	0.217	3.40	3.26
Suspiciousness/persecutory ideas	1720	16.941	7.540	0.006	2.89	2.57
Grandiose ideas	1720	1.649	.970	0.325	0.94	1.03
Perceptual abnormalities/hallucinations	1720	24.472	10.719	0.001	3.25	2.88
Disorganized communication	1720	10.043	4.696	0.031	1.82	1.58

Fig. 4. Standardized regression coefficients for the direct effect between sleep disturbance and suspiciousness without depression (controlling for cannabis use) (A) and indirect effect between sleep disturbance and suspiciousness through depression (controlling for cannabis use) (B). The indirect effect was statistically significant (unstandardized indirect effect: $b = 0.0537$, 95% CI [.0319, 0.0787]). *$p < 0.05$, **$p < 0.01$.
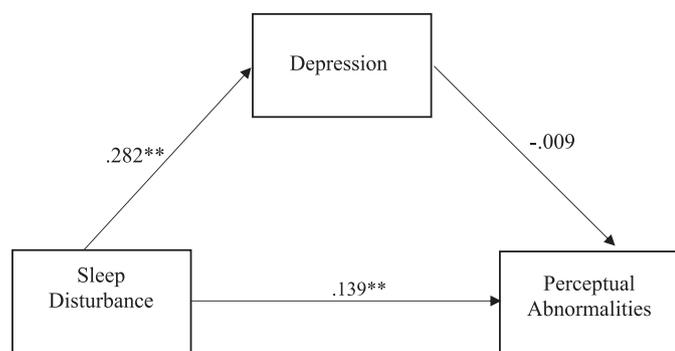
Fig. 5. Standardized regression coefficients for the relationship between sleep disturbance and perceptual abnormalities (controlling for cannabis use). The indirect effect through depression was not significant (unstandardized indirect effect: $b = -0.0026$, 95% CI [-0.0256, 0.0196]). *$p < 0.05$, **$p < 0.01$.
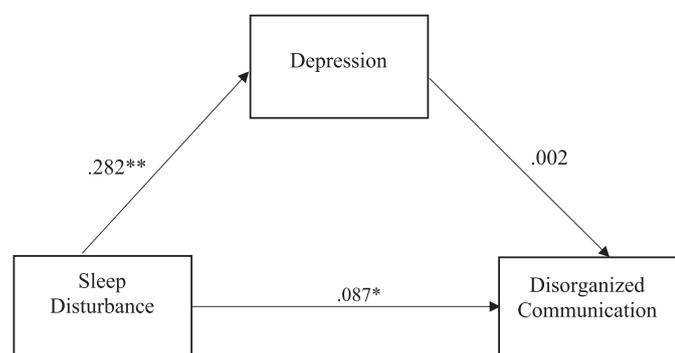
Fig. 6. Standardized regression coefficients for the relationship between sleep disturbance and disorganized communication (controlling for cannabis use). The indirect effect through depression was not significant (unstandardized indirect effect: $b = 0.0005$, 95% CI [-0.0198, 0.0215]). *$p < 0.05$, **$p < 0.01$.

for the bidirectional association between sleep problems and paranoia was provided by a recent longitudinal investigation in 29 patients with psychosis (Reeve et al., 2018). The current study did not assess directionality of associations and focused only on the potential mediational role of depression in the relationship between sleep problems and psychotic symptoms. Future investigations testing different mediators, such as anxiety, and using longitudinal data are needed to better understand the specifics of the association of different psychotic symptoms and sleep problems over time. In addition, models and theories that could explain how sleep, depression and psychotic symptoms may be associated from a biological perspective may also help to clarify and guide future research directions in this area.

Finally, we did not find that baseline sleep disturbance predicted subsequent worsening of the prodromal syndrome and/or conversion to psychotic disorder. In fact, none of the outcome groups (conversion, prodromal progression, symptomatic, remission) differed in severity of baseline sleep disturbance. This null finding should be interpreted with caution given that this was not a longitudinal study. One explanation is that these findings are due to the temporal lag between the baseline assessment of sleep and the subsequent designation of clinical outcome, which could have a duration of up to two years. The adverse effects of sleep disturbance on clinical status may be more proximal. Thus, it may be that sleep problems are related to psychotic symptom severity only contemporaneously, but are not predictive of continued worsening of symptoms over time. This is consistent with findings in the experimental sleep literature, which have found that acute sleep deprivation is associated with transient psychotic symptoms in a state-dependent way, such that psychotic-like symptoms are only present during the night of deprived sleep and dissipate during the day, or when recovery sleep is allowed (van Heugten–van der Kloet et al., 2015). However, this brings into question the nature of the association between sleep problems and psychotic symptoms over time and indicates the need for more detailed longitudinal investigations in a clinical sample. This is especially relevant given the recent interest in sleep problems as a potential target for preventative interventions for psychosis (e.g., Bradley et al., 2018; Waite et al., 2018).

There are several important limitations in the current study that should be noted. First, sleep disturbance was assessed with a single item based largely on self-report, which has several inherent limitations. The sleep disturbance item was also assessed from the same scale used to measure attenuated psychotic symptoms. Future studies would benefit from using more independent and objective measures of sleep (e.g., actigraphy, sleep monitoring devices) that can also assess multiple aspects of sleep. Chronicity of sleep problems was also not assessed in this study, and it may be an important factor to consider when investigating the potential role of sleep problems in psychotic symptom exacerbation over time. For example, the neurological impact of a few bouts of insomnia over a lifetime may be negligible in comparison to long-term chronic insomnia. Finally, our analyses were cross-sectional in nature,

and thus limit our ability to assess directionality of effects as well as our ability to assess true mediational effects over time. Additional longitudinal analyses of sleep and psychotic symptoms will be important to more fully understand the role of sleep problems in the development of psychotic disorders.

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