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Sleep disturbance and delusion severity in schizophrenia spectrum disorders: protocol for a cross-sectional observational study

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ABSTRACT

Background: Sleep disturbance is highly prevalent in schizophrenia spectrum disorders and has been increasingly proposed as a contributory, modifiable cause of psychotic experiences rather than a simple epiphenomenon. Evidence linking poor sleep with delusions comes mainly from general-population surveys, non-clinical paranoia studies and early-psychosis cohorts. Few clinical studies have examined multidimensional delusion severity while accounting for depression and antipsychotic dose, and data from South Asian services are scarce. This study will estimate the association between subjective sleep quality and delusion severity in adults with schizophrenia spectrum disorders. **Methods and analysis:** A cross-sectional observational study will recruit 160 consecutive adults (18–65 years) with a DSM-5-TR diagnosis of schizophrenia, schizoaffective disorder, schizophreniform disorder or delusional disorder and a current delusion (PANSS item P1 ≥ 3) from psychiatric services at Gulbarga Institute of Medical Sciences, Kalaburagi, Karnataka, India. The primary exposure is the Pittsburgh Sleep Quality Index (PSQI) global score; the primary outcome is the Psychotic Symptom Rating Scales delusions subscale (PSYRATS-D). Secondary sleep measures are the Insomnia Severity Index, sleep onset latency and total sleep time; secondary symptom outcomes are PANSS P1 and the PANSS positive and negative subscales. Covariates include age, sex, chlorpromazine-equivalent antipsychotic dose and depression (Calgary Depression Scale for Schizophrenia). The primary analysis is a multiple linear regression of PSYRATS-D on PSQI adjusted for these covariates. Secondary analyses comprise zero-order and depression-adjusted partial correlations and a Welch t-test comparing poor (PSQI > 5) and good sleepers. A sample of 134 complete cases provides 90% power to detect a partial f^2 of 0.08 at $\alpha = 0.05$; 160 participants will be recruited to allow for incomplete data.

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INTRODUCTION

Delusions, defined as fixed beliefs that are not amenable to change in light of conflicting evidence, are a core feature of schizophrenia spectrum and other psychotic disorders [1]. Persecutory delusions are the most common type. They are frequently persistent despite antipsychotic treatment and are associated with marked distress, social withdrawal, self-harm and hospital admission [2,3]. Identifying factors that maintain delusions, particularly factors that can be treated, is therefore a clinical priority.

Sleep disturbance is one such candidate. Disrupted sleep is common across psychiatric disorders and is increasingly understood as a transdiagnostic contributor to psychopathology rather than a secondary symptom [4]. In schizophrenia, insomnia, delayed or irregular sleep–wake patterns, and hypersomnia associated with sedating medication are all common [5,6]. Diagnosable sleep disorders were present in a large majority of patients with early psychosis in one systematic assessment [7]. Comparable rates of subjective sleep disturbance are reported in schizophrenia and bipolar disorder [8]. Insomnia is independently associated with poorer quality of life in schizophrenia [9]. Objective studies have documented prolonged sleep latency, reduced total sleep time and reduced sleep efficiency even in antipsychotic-naive patients [10], marked circadian misalignment [11], and deficits in sleep spindles that may index thalamocortical dysfunction [12,13]. Despite this, sleep problems in psychosis have historically been regarded as an epiphenomenon of the illness and have received little routine clinical attention [14].

A growing body of evidence suggests that sleep disturbance may contribute causally to delusional ideation. Systematic reviews of clinical and non-clinical samples have found consistent associations between poor sleep and both paranoia and hallucinations [15,16]. In general-population and survey data, insomnia is associated with a higher prevalence and greater persistence of persecutory ideation [17–20], and insomnia prospectively predicts the onset of several mental disorders [21]. Experience-sampling studies in schizophrenia and across the paranoia continuum show that a poor night's sleep predicts more severe psychotic symptoms and paranoia the following day [22,23]. Experimental sleep restriction or deprivation increases paranoia and psychosis-like experiences in healthy volunteers [24,25]. Most persuasively, randomised trials of cognitive behavioural therapy for insomnia have reduced paranoia and hallucinations in student and clinical populations, with the effects mediated by improved sleep [26–28]. Disturbed sleep is also among the most frequently reported factors in the weeks preceding the onset of persecutory delusions [29].

Cognitive models of psychosis offer a plausible mechanism. Anomalous experiences, negative affect and reasoning biases are proposed to combine in the formation and maintenance of delusional beliefs [30]. Sleep loss amplifies negative affect, worry and perceptual anomalies, each of which may then feed threat beliefs. Experimental and trial data indicate that negative affect partly mediates the effect of sleep on paranoia [24,26].

Important gaps nonetheless remain. Much of the evidence derives from non-clinical paranoia, early-psychosis or at-risk samples [16,31], and the dimensional severity of established delusions in clinical populations has been less studied. Depression is strongly linked to both poor sleep and paranoia, so it is a key confounder [20]. Antipsychotic dose and sedative-hypnotic use alter sleep [5]. Many clinical studies have adjusted for neither. Delusions are also often indexed by a single rating-scale item, which cannot capture preoccupation, conviction and distress. Finally, almost all of this research comes from high-income Western settings, and data from South Asian psychiatric services, where sleep is rarely assessed routinely, are lacking.

Aims and hypotheses. The primary aim is to estimate the association between subjective sleep quality (PSQI global score) and delusion severity (PSYRATS-D) in adults with schizophrenia spectrum disorders, adjusting for age, sex, antipsychotic dose and depression. We hypothesise that poorer sleep quality will be associated with greater delusion severity after adjustment (H1). Secondary aims are: (a) to examine whether specific sleep parameters (insomnia severity, sleep onset latency and total sleep time) are associated with delusion severity, independent of depression (H2); (b) to compare delusion severity between poor sleepers (PSQI > 5) and good sleepers (H3); and (c) to examine the specificity of associations by comparing delusion outcomes with PANSS positive and negative subscale scores (exploratory).

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Methods and analysis

Study design

This is a single-centre, cross-sectional observational study. The protocol has been written in advance of data collection, and the study will be reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [32].

Setting

Participants will be recruited from the outpatient clinics and inpatient wards of the Department of Psychiatry, Gulbarga Institute of Medical Sciences, Kalaburagi, Karnataka, India, a government tertiary care teaching hospital serving urban, semi-urban and rural populations of the Kalyana-Karnataka region.

Participants

Inclusion criteria: (i) age 18–65 years; (ii) a current DSM-5-TR diagnosis of schizophrenia, schizoaffective disorder, schizophreniform disorder or delusional disorder [1], made by the treating psychiatrist and confirmed with a structured diagnostic interview; (iii) at least one current delusion, operationalised as a PANSS P1 (Delusions) score ≥ 3; (iv) a stable antipsychotic regimen for at least two weeks, or no antipsychotic treatment; and (v) capacity to provide written informed consent and to complete the assessments in Kannada, Hindi or English.

Exclusion criteria: (i) psychosis attributable to a substance or medication, or to a general medical condition; (ii) neurological disorder, significant head injury or intellectual disability; (iii) a previously diagnosed primary sleep disorder other than insomnia (for example narcolepsy or obstructive sleep apnoea); (iv) current night-shift or rotating-shift work, or trans-meridian travel within the past two weeks; (v) pregnancy or the postpartum period (within six months); (vi) electroconvulsive therapy within the past three months; and (vii) acute risk to self or others or behavioural disturbance that precludes assessment. Current substance use will not be an exclusion criterion; it will be recorded and examined in sensitivity analyses.

Recruitment and procedure

Consecutive eligible patients will be identified by treating clinicians and approached by a member of the research team. After an explanation of the study, a participant information sheet in the participant's preferred language and time to consider participation, written informed consent will be obtained. Assessments will take place in a single session of approximately 60–75 minutes, with breaks offered. Sociodemographic and clinical information will be collected by interview and verified from case records. Self-report sleep questionnaires will be administered by a research assistant who does not perform symptom ratings. PANSS and PSYRATS ratings will be completed by a trained clinician who is blind to sleep scores. Reasons for non-participation and the numbers screened, eligible, approached and enrolled will be recorded for a STROBE flow diagram.

Measures

All measures are summarised in Table 1. The variables, coding and permissible ranges were pre-specified in a study codebook and data-entry workbook before recruitment.

Primary exposure: sleep quality. The Pittsburgh Sleep Quality Index (PSQI) is a 19-item self-report measure of sleep quality over the past month [33]. It yields seven component scores and a global score from 0 to 21. A global score > 5 distinguishes poor from good sleepers and was the cut-off reported in the original validation. Sleep onset latency (minutes, item 2) and total sleep time (hours, item 4) will be extracted as continuous secondary exposures.

Secondary exposure: insomnia severity. The Insomnia Severity Index (ISI) is a 7-item measure of the nature, severity and impact of insomnia over the past two weeks (range 0–28) [34]. A score ≥ 15 indicates clinical insomnia of at least moderate severity.

Primary outcome: delusion severity. The delusions subscale of the Psychotic Symptom Rating Scales (PSYRATS-D) is a 6-item clinician-rated interview assessing the amount and duration of preoccupation, conviction, the amount and intensity of distress, and disruption to life caused by delusions (range 0–24) [35]. It has good inter-rater reliability and validity in chronic and first-episode psychosis [35,36].

Secondary outcomes. The Positive and Negative Syndrome Scale (PANSS) [37] will be used to rate item P1 (Delusions, 1–7), the positive subscale (P1–P7, range 7–49) and the negative subscale (N1–N7, range 7–49). The negative subscale is included to test whether any association is specific to positive psychotic symptoms.

Covariates. Depression will be rated with the Calgary Depression Scale for Schizophrenia (CDSS), a 9-item interview designed to separate depressive symptoms from negative and extrapyramidal symptoms (range 0–27; ≥ 7 suggests probable depression) [38]. Current antipsychotic doses will be converted to

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chlorpromazine-equivalent mg/day using a published dose-equivalence method [39]. The equivalents table used will be fixed before analysis. Age, sex, years of education, illness duration (years since first psychotic episode), current use of sedative-hypnotics (benzodiazepines, Z-drugs or sedating antihistamines) and current substance use (cannabis or alcohol misuse in the past month) will also be recorded.

Rater training and reliability

Raters will complete standardised PANSS and PSYRATS training with a senior psychiatrist before data collection. Inter-rater reliability will be checked on a random 10% of interviews (audio-recorded with consent or jointly rated). Intraclass correlation coefficients (two-way random, absolute agreement) will be reported, with ICC ≥ 0.75 as the target. Disagreements will be resolved by consensus and recalibration sessions.

Table 1. Study measures, ranges and analytic roles

Domain	Instrument / variable	Range	Role in analysis
Sleep quality	Pittsburgh Sleep Quality Index (PSQI) global score [33]	0–21; > 5 = poor sleeper	Primary exposure
Insomnia	Insomnia Severity Index (ISI) [34]	0–28; ≥ 15 = clinical insomnia	Secondary exposure
Sleep parameters	Sleep onset latency (min) and total sleep time (h), from PSQI items 2 and 4	0–300 min; 0–16 h	Secondary exposures
Delusions	PSYRATS delusions subscale (PSYRATS-D) [35,36]	0–24	Primary outcome
Psychotic symptoms	PANSS P1 Delusions; positive and negative subscales [37]	1–7; 7–49; 7–49	Secondary outcomes
Depression	Calgary Depression Scale for Schizophrenia (CDSS) [38]	0–27; ≥ 7 = probable depression	Covariate (primary model)
Medication	Antipsychotic dose, chlorpromazine-equivalent mg/day [39]	0–3000	Covariate (primary model)
Demographics	Age (years); sex	18–65; male/female	Covariates (primary model)
Other clinical	Diagnosis; education (years); illness duration (years); sedative-hypnotic use; current substance use	As coded	Description; sensitivity analyses

PSQI, Pittsburgh Sleep Quality Index; ISI, Insomnia Severity Index; PSYRATS, Psychotic Symptom Rating Scales; PANSS, Positive and Negative Syndrome Scale; CDSS, Calgary Depression Scale for Schizophrenia.

Sample size

The sample size was calculated for the primary analysis, which tests the increment in explained variance attributable to PSQI in a linear regression model with five predictors (PSQI, age, sex, chlorpromazine-equivalent dose and CDSS). Published clinical and experience-sampling studies report correlations between sleep disturbance and paranoia or delusion severity of roughly 0.2–0.3 [7,15,22]. We therefore powered the study for a partial effect size of $f^2 = 0.08$, a small-to-medium effect corresponding to a partial correlation of about 0.27. With $\alpha = 0.05$ (two-sided), 90% power, one

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tested predictor and five predictors in total, the required sample is 134 participants with complete data (G*Power 3.1, linear multiple regression, R^2 increase [40]; confirmed by a non-central F calculation). Allowing approximately 15% for incomplete assessments, the recruitment target is 160 participants. This sample also provides more than 80% power to detect a zero-order correlation of $r \geq 0.25$ for each secondary sleep measure.

Data management

Each participant will be assigned a pseudonymised study ID (for example SZ001). Data will be entered into a pre-built, password-protected data-entry workbook whose column structure and coding follow the study codebook (Table 1). Range checks will flag out-of-range entries. A second researcher will independently enter 20% of records, and discrepancies will be resolved against source documents. Identifiable information will be stored separately from research data on secured institutional servers and retained for [five] years after publication in line with institutional policy.

Statistical analysis

Analyses will be conducted in R (version 4.x) or IBM SPSS Statistics, with two-sided $\alpha = 0.05$. The analysis plan is summarised in Table 2.

Descriptive analysis. Continuous variables will be summarised as mean (SD) and median (interquartile range), and categorical variables as n (%). The proportions of poor sleepers (PSQI > 5), participants with clinical insomnia (ISI ≥ 15) and participants with probable depression (CDSS ≥ 7) will be reported with 95% confidence intervals. Distributions will be inspected with histograms, Q–Q plots and the Shapiro–Wilk test.

Primary analysis (H1). A hierarchical multiple linear regression will be fitted with PSYRATS-D as the dependent variable. Model 1 will include PSQI global score alone. Model 2 will add age, sex, chlorpromazine-equivalent dose and CDSS score. The primary estimate is the adjusted unstandardised coefficient (B) for PSQI with its 95% confidence interval, together with the standardised coefficient (β), R^2, adjusted R^2 and the change in R^2 between models. Assumptions will be checked: linearity (residual-versus-fitted plots), normality of residuals, homoscedasticity (Breusch–Pagan test), multicollinearity (variance inflation factor; VIF > 5 flagged) and influential observations (Cook's distance > 4/n). If heteroscedasticity is present, heteroscedasticity-consistent (HC3) standard errors will be reported.

Secondary analyses (H2, H3). Pearson correlations (or Spearman's ρ where distributions are markedly skewed) will be computed among the sleep measures (PSQI, ISI, sleep onset latency, total sleep time), CDSS, PANSS P1 and PSYRATS-D. For each sleep measure, its association with PSYRATS-D will also be estimated as a partial correlation controlling for CDSS. Delusion severity will be compared between poor sleepers (PSQI > 5) and good sleepers using Welch's t-test, which does not assume equal variances or equal group sizes, with Cohen's d and its 95% confidence interval. The Holm–Bonferroni procedure will control the family-wise error rate across the four secondary sleep–delusion associations.

Exploratory and sensitivity analyses. (i) The primary model will be repeated with PANSS P1, the PANSS positive subscale and the PANSS negative subscale as outcomes to assess symptom specificity. (ii) Sedative-hypnotic use, current substance use, illness duration and education will be added to Model 2. (iii) The primary model will be re-estimated excluding participants with delusional disorder and, separately, inpatients, to test robustness to diagnostic and setting heterogeneity. (iv) A PSQI × diagnosis interaction will be tested descriptively. (v) An exploratory mediation model will estimate the indirect effect of PSQI on PSYRATS-D via CDSS using bias-corrected bootstrap confidence intervals (5000 resamples). Because the data are cross-sectional, this model will be interpreted only as hypothesis-generating.

Missing data. The primary analysis will use complete cases. If more than 5% of cases are missing any variable in the primary model, multiple imputation by chained equations (20 imputed datasets, including all analysis variables and auxiliary variables) will be performed as a sensitivity analysis, and results will be pooled using Rubin's rules.

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Table 2. Pre-specified statistical analysis plan

Hypothesis / question	Analysis	Primary estimate
H1: poorer sleep quality is associated with greater delusion severity	Hierarchical linear regression: PSYRATS-D ~ PSQI (Model 1); + age, sex, CPZ-equivalent dose, CDSS (Model 2)	Adjusted B for PSQI (95% CI), β, ΔR^2
H2: specific sleep parameters are associated with delusion severity independent of depression	Pearson/Spearman correlations; partial correlations controlling for CDSS; Holm correction	r and partial r (95% CI) for ISI, sleep onset latency, total sleep time
H3: poor sleepers have more severe delusions than good sleepers	Welch's t-test, PSQI > 5 vs ≤ 5	Mean difference (95% CI); Cohen's d
Specificity (exploratory)	Model 2 repeated with PANSS P1, positive and negative subscales as outcomes	Adjusted B for PSQI per outcome
Robustness	Additional covariates; diagnostic and setting exclusions; multiple imputation	Change in PSQI coefficient
Mechanism (exploratory)	Mediation via CDSS with bootstrap CI	Indirect effect (95% CI)

CPZ, chlorpromazine; CDSS, Calgary Depression Scale for Schizophrenia; ISI, Insomnia Severity Index; PSQI, Pittsburgh Sleep Quality Index; PSYRATS-D, Psychotic Symptom Rating Scales delusions subscale.

Patient and public involvement

A plain-language summary of findings will be shared with participating service-user groups.

Ethics and dissemination

The study will be conducted in accordance with the Declaration of Helsinki and the Indian Council of Medical Research National Ethical Guidelines for Biomedical and Health Research Involving Human Participants (2017). Approval will be obtained from the Institutional Ethics Committee, Gulbarga Institute of Medical Sciences, Kalaburagi [reference number], before recruitment begins, and the study will be registered with the Clinical Trials Registry – India.

Capacity to consent will be assessed by the treating clinician and documented. Individuals who lack capacity at the time of approach will not be enrolled but may be re-approached if their capacity improves. Participation is voluntary and will not affect clinical care, and participants may withdraw at any time without giving a reason. Where the ISI indicates severe insomnia (≥ 22), the CDSS indicates probable depression, or suicidal ideation is disclosed, the treating team will be informed with the participant's knowledge so that appropriate assessment and treatment can be offered. Participants will receive.

Findings will be disseminated through publication in a peer-reviewed journal, presentation at national and international psychiatry and sleep-medicine conferences, and feedback sessions for the participating clinical teams. De-identified data and analysis code will be made available on reasonable request to the corresponding author, subject to ethics committee approval.

Discussion

This study will provide a clinically grounded, confounder-adjusted estimate of the association between sleep quality and delusion severity in schizophrenia spectrum disorders. It addresses three limitations of the existing literature: reliance on non-clinical or at-risk samples [16,31], measurement of delusions by single items rather than a multidimensional scale [35], and inadequate adjustment for depression and antipsychotic dose [5,20]. A robust, independent association would add to the case that sleep is a treatable target in established psychosis [14,27] and would support

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routine sleep assessment in psychiatric services where it is rarely performed. A null result after adjustment would also be informative. It would suggest that, in clinical samples, the sleep–delusion relationship is largely accounted for by depression or medication effects.

Several limitations are anticipated. First, the cross-sectional design precludes conclusions about direction or causality. Longitudinal, experience-sampling and interventional evidence suggests that the relationship is likely bidirectional [22–24]. Second, sleep will be measured only by self-report. Self-reported sleep in psychosis may diverge from objective measures, and circadian misalignment [11] will not be captured. Future work should add actigraphy or sleep diaries. Third, antipsychotics and sedatives affect both sleep and symptoms, and statistical adjustment cannot fully remove confounding by indication [5]. Fourth, single-centre recruitment in a tertiary setting may limit generalisability to community or first-episode populations. Finally, the PSQI has not been extensively validated in Indian psychosis samples, and translated versions will be used where required.

If an association is confirmed, these data will inform the design of an adequately powered randomised trial of cognitive behavioural therapy for insomnia adapted for psychosis [26–28] in Indian psychiatric services, with delusion severity as a primary outcome.

Ethics and dissemination: Approval will be obtained from the Institutional Ethics Committee of Gulbarga Institute of Medical Sciences before recruitment. All participants will provide written informed consent after a capacity assessment. Results will be reported according to STROBE and disseminated through peer-reviewed publication, conference presentations and summaries for participating clinical teams.

Strengths and limitations of this study

•The protocol pre-specifies the primary exposure, outcome, covariates and analysis plan, including the effect size and sample-size calculation, before any data are collected.

•Delusion severity is assessed multidimensionally with PSYRATS-D by raters blind to sleep scores, rather than with a single symptom item.

•Depression, antipsychotic dose and sedative use, which are key confounders of the sleep–psychosis association, are measured and adjusted for.

•The cross-sectional design cannot establish temporal order or causality between sleep disturbance and delusions.

•Sleep is measured by self-report and is not corroborated by actigraphy or polysomnography.

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