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Testing the mediating role of negative affect, affect intolerance, and dissociation in the relationship between sleep loss and psychotic experiences

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ABSTRACT

Background and hypothesis: Sleep disturbances are known to causally contribute to psychotic experiences, with negative affect indicated as a mediator in previous research. However, other plausible related mechanisms such as affect intolerance and dissociative experiences have not been tested. We hypothesised that sleep restriction would increase psychotic experiences, with effects mediated by increases in negative affect, affect intolerance and dissociative experiences.

Study design: A within-subjects, cross-over experimental study was conducted with 58 undergraduate participants, comparing sleep restriction (≤4 h sleep for one night) to standard sleep.

Study results: In comparison to standard sleep, one night of sleep restriction resulted in significant increases in all psychotic experiences except grandiosity, as well as significant increases in all proposed mediators. Dissociative experiences (46.2%), and combined mediators (89.1%), substantially mediated the effect of reduced sleep on paranoia. Dissociative experiences also partially mediated the effect of reduced sleep on hallucinations (38.5%). No significant mediation effects were identified for cognitive disorganisation, anhedonia or distress from psychotic experiences.

Conclusions: These findings extend the causal relationship between sleep loss and psychotic experiences as occurring after only one night of sleep restriction. They also identify a novel mechanism – dissociative experiences – as contributing towards both paranoia and hallucinations. Negative affect was supported as a mediating mechanism for paranoia, in line with existing research, however there was limited identification of mediating mechanisms for other psychotic experiences. The results emphasise the importance of further experimental research investigating the relationship between affective processes, particularly dissociative experiences, and specific symptoms of psychosis.

1. Introduction

Sleep disturbances are elevated in psychosis patients relative to the general population (Cederlöf et al., 2022; Waite et al., 2020), with individuals reporting poor sleep quality across clinical stages (Bagautdinova et al., 2023). While historically viewed as a secondary consequence of psychosis, it is increasingly recognised that sleep difficulties precede and predict subsequent psychotic symptoms and are associated with symptom severity (Davies et al., 2017; Reeve et al., 2015). The association between sleep disturbance and psychotic experiences can be understood within theoretical models of psychosis, many of which posit that delusional beliefs and anomalous experiences develop as a consequence of precipitating stressors including sleep dysfunction (Freeman et al., 2009; Freeman et al., 2017; Garety et al., 2001;Garety et al., 2007; Moritz et al., 2016). Altogether this underscores the need for further understanding of how sleep contributes to the development and maintenance of psychosis by clarifying causal mechanisms.

Negative affect is a well-established mechanism linking insomnia to psychotic experiences. Research in non-clinical populations has demonstrated that negative affect mediates the influence of sleep quality on measures of paranoid thinking (Bagrowska et al., 2022; Rehman

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et al., 2018; Scott et al., 2017), and this finding has also been shown using experience sampling methods with both clinical and non-clinical samples (Kasanova et al., 2020). Sleep deprivation and sleep restriction studies in non-clinical samples generally support the role of negative affect in contributing psychotic experiences (Reeve et al., 2018a; Hurdiel et al., 2015; Kahn-Greene et al., 2007; Petrovsky et al., 2014).

However other affect-related processes may also be implicated in this relationship. One candidate is affect intolerance, characterised by beliefs about emotions being threatening alongside attempts to avoid or suppress them (Stapinski et al., 2014). Affective dysregulation also serves as a common moderator across models of delusion aetiology (Denecke et al., 2024), suggesting that reliance on maladaptive emotion regulation strategies may contribute to positive symptom development by increasing the likelihood that anomalous experiences are interpreted as threatening. Individuals with psychosis report stronger beliefs in the uncontrollability of emotions, in association with the elevated use of maladaptive emotion regulation strategies (e.g. suppression) and more severe negative symptoms (Berglund et al., 2023). The use of experiential avoidance is also linked to positive symptoms, including paranoia (Castilho et al., 2017; Moritz et al., 2016; Udachina et al., 2014), hallucinations (Goldstone et al., 2012), and distress related to delusions and voice-hearing (Goldstone et al., 2011; Varese et al., 2016). Similarly, individuals with insomnia and those undergoing sleep deprivation are more inclined towards experiential avoidance and emotional dysregulation (Campbell et al., 2022; Zakiei et al., 2020). These shared processes suggest a potential mediating role for affect intolerance in linking sleep loss and psychosis.

Dissociation is another candidate mechanism linking sleep and psychosis. While definitions of dissociation vary (Spitzer et al., 2006), dissociation is broadly understood as feeling 'unreal' or detached from one's sense of self, experiences, or surroundings (American Psychiatric Association, 2013). Network analyses reveal strong connections between dissociation, negative affect, and psychotic experiences (Černis et al., 2021b) and robust links to positive psychotic symptoms in both clinical and non-clinical samples (Longden et al., 2020; Renard et al., 2017). Furthermore, both dissociation and elements of affect intolerance predict auditory hallucinations in clinical samples (Varese et al., 2011), with some researchers proposing that auditory hallucinations are dissociative in nature (Longden et al., 2012; Moskowitz et al., 2009). Experimental studies show that sleep loss increases dissociative experiences in non-clinical participants (Giesbrecht et al., 2013; Menicucci et al., 2022; Selvi et al., 2015; van Heugten–van der Kloet et al., 2015), however, to our knowledge whether dissociation mediates the sleep loss – psychosis relationship has not been experimentally tested.

This study investigates the impact of sleep loss on subclinical psychotic experiences using experimental sleep manipulation in a non-clinical sample with a sleep-restriction protocol based on a previous study (Reeve et al., 2018a), applied here for one night rather than three. To investigate causal mechanisms, it examines the potential mediating roles of negative affect, affect intolerance, and dissociative experiences. The following hypotheses were tested:

1. Sleep loss will cause an increase in subclinical psychotic experiences (outcomes).
2. Sleep loss will cause an increase in negative affect, affect intolerance and dissociative experiences (proposed mediators).
3. Increases in psychotic experiences will be mediated by negative affect, affect intolerance and dissociative experiences.

2. Methods

2.1. Study design

A within-subjects cross-over experimental design was employed, with two counterbalanced conditions: one night of standard sleep (control condition, ≥7 h) and one night of sleep loss (sleep restriction condition, <4 h).

2.2. Participants

Participants were 58 undergraduate psychology students, with good quality sleep and no current or prior mental health concerns (see supplementary material S.1 for detailed eligibility criteria, screening process, and exclusion rationale.

All participants gave written informed consent and were granted course credits in return for participation. Ethical approval for this study was granted by the University of East Anglia FMH S-REC (ETH2324-0223) and the study was pre-registered on OSF (osf.io/dnju5).

2.3. Measures

During the study, participants completed assessments via Qualtrics (2024) between the hours of 6pm-midnight before and after both sleep conditions (four timepoints in total), to ensure consistency in circadian phase and reduce the contribution of cyclic daily fluctuations in mood and cognition (Bu et al., 2025). All questionnaire timescales were adjusted to capture beliefs and experiences over the last 24 h to allow for repeated administration.

2.3.1. Demographics

Participants were asked to give their age and self-identify their gender, ethnicity and nationality.

2.3.2. Sleep diary and sleep length recording

Following both nights of sleep, participants completed a sleep diary based on the Consensus Sleep Diary (Carney et al., 2012) which recorded subjective sleep and wake times, hours slept, night-time awakenings, daytime naps and consumption of substances (alcohol, caffeine and nicotine) that influence sleep and wakefulness. Sleep duration in both conditions was objectively recorded using a smartwatch or smartphone app, which estimates sleep periods by monitoring motion, heart rate, and device activity. These methods have been shown to achieve an 85.9% accuracy rate in detecting sleep and wake states (Bhat et al., 2015). This data was used to check adherence to sleep conditions as a requirement for inclusion in analysis.

2.3.3. Proposed mediators

Depression Anxiety Stress Scale (DASS) short form (Lovibond and Lovibond, 1995)**:** a 21-item questionnaire that measures negative affect. There are three subscales: depression, anxiety and stress. Responses are given to each statement on a 4-point scale of agreement, with higher scores reflecting greater negative affect. This measure has demonstrated acceptable-to-good internal consistency (α = .81-.91 for subscales in original sample and α = .69-.86 for subscales across conditions in current sample), as well as good convergent and discriminant validity in non-clinical samples (Lovibond and Lovibond, 1995).

Affect Intolerance Scale (AIS) (Stapinski et al., 2014**):** a 30-item questionnaire that measures appraisals of emotions as threatening and attempts to avoid or suppress unpleasant emotions. Responses are given to each statement on a 6-point scale of agreement, with higher scores reflecting greater affect intolerance. This scale has demonstrated excellent internal consistency (α = .94 in original sample and α = .94-.97 in current sample), as well as good test-retest reliability (r = .86), convergent and criterion validity in an undergraduate sample **(**Stapinski et al., 2014).

Černis Felt Sense of Anomaly Scale (ČEFSA) (Černis et al., 2021a)**:** a 35-item questionnaire that measures dissociative experiences reflected as anomalous sensations, perceptions or cognitions that feel unusual or misaligned with one's typical sense of reality. Responses are given to each statement on a 5-point scale of agreement, with higher scores reflecting greater dissociative experiences. This measure has demonstrated excellent internal consistency (α = .98 in original sample and

α = .95-.97 in current sample) and test-retest reliability (ICC = .92), and good convergent validity in non-clinical samples, as well as adequate model fit in non-clinical and psychosis samples (Černis et al., 2021a).

2.3.4. Outcome variables

Specific Psychotic Experiences Questionnaire (SPEQ) (Ronald et al., 2014)**:** a questionnaire that measures various psychotic experiences. There are six subscales: paranoia (15 items rated on a 6-point scale of agreement), hallucinations (9 items rated on a 6-point scale of agreement), cognitive disorganisation (11 items rated as a yes or no agreement), grandiosity (8 items rated on a 4-point scale of agreement), anhedonia (10 items rated on a 6-point scale of agreement) and observer-ratings (removed for this study due to not being suitable for self-report). Higher scores in all scales reflect greater psychotic experiences. Distress is also rated for each subscale on a 4-point scale. This measure has demonstrated acceptable-to-excellent internal consistency (α = .77-.93 in original sample and α = .68-.92 in current sample) and test-retest reliability (*r* = .65-.74) (Ronald et al., 2014), as well as sensitivity to changes in a non-clinical sample following sleep restriction (Reeve et al., 2018a).

2.4. Procedure

Participants completed the sleep loss condition (≤4 h sleep) or a night of standard sleep (≥7 h sleep) in the first week, followed by the alternate condition the following week to allow time to return to their baseline sleeping pattern. For the sleep loss condition participants were asked to keep their wake time constant and delay the time they went to bed.

Participants were randomly assigned to their first sleep condition using an online true random number generator, with the allocation list being fixed prior to recruitment. Participants completed the sleep manipulation between a Monday-Thursday evening of their choice, with both conditions being on the same day of the week to maintain consistency in daily schedules. Participants completed the study remotely and were encouraged to follow their usual routines as much as possible. No adverse events from sleep loss were reported over the course of the study, and participants were signposted to resources for sleep and mental health on study debrief. A diagram of the study procedure is shown in Fig. 1.

2.5. Statistical analysis

Data were cleaned, adherence to sleep conditions was verified before analysis, and descriptive statistics were calculated. Where there were discrepancies between sleep length reported by sleep diary and objective recording the latter was chosen. A threshold of <20% for imputation of missing data was chosen, and missing values replaced with the mean item score from the associated subscale. One SPEQ Anhedonia subscale for a single participant exceeded this threshold and therefore was not included in analysis. Paired t-tests were conducted to check for similarity in scores on subscales for data collected at baseline on the evenings before both sleep conditions. Paired t-tests were also conducted to check adherence to sleep diary estimates of sleep, as well as to compare caffeine, alcohol and nicotine consumption across conditions.

Data normality was assessed via Shapiro-Wilk test (see Supplementary Material S.2), and non-parametric measures used where necessary. To investigate whether scores in proposed mediating and outcome variables were correlated, bivariate Pearson or Spearman's rho correlation analyses for both sleep conditions were performed using IBM Statistical Package for the Social Sciences (SPSS; IBM Corp, 2023).

Repeated measures mediation analyses were performed using the MEMORE macro tool for SPSS, which is based on the within-subject mediation approach described by Judd et al. (2001) and extended by Montoya and Hayes (2017) into a path-analytic framework that supports the parallel mediation model employed in the present study. Total, direct and indirect effects of the sleep condition on outcome variables were calculated using percentile bootstrapping with 5000 generated samples to produce standard errors and 95% confidence intervals. Percentile bootstrapping is currently recommended over other approaches that result in increased Type I error (Fritz and MacKinnon, 2007; Tibbe and Montoya, 2022). Mediators were tested in a combined model for each psychotic experience outcome variable to examine the contribution of each mediator while accounting for shared variance among them.

The proportion mediated for each mediator was calculated as the

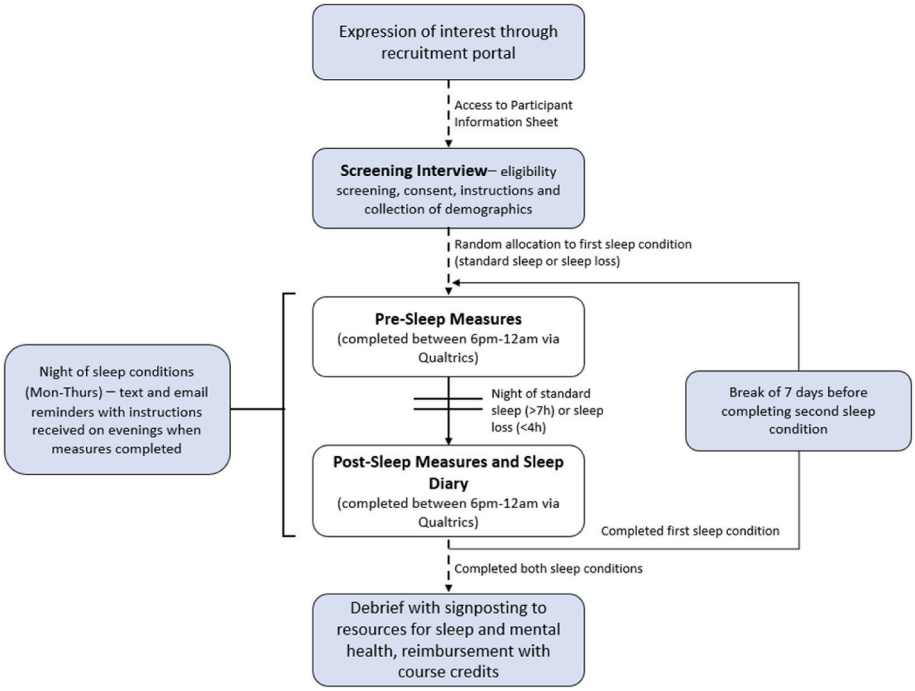

Fig. 1. Flowchart of the study procedure.

percentage of the indirect effect relative to the total effect. This was chosen over other available metrics (Preacher and Kelley, 2011) to allow for comparison with previous studies (Reeve et al., 2018a) and increased interpretability. Effect sizes for candidate mediators were calculated using the pooled standard deviation across conditions as the denominator, which can be interpreted as equivalent to Cohen's *d* (Feingold, 2009). Statistical significance was set at $p < .05$. Multiple comparison corrections were not applied as the comparisons were based on pre-defined hypotheses. A post-hoc power analysis using G*Power (Faul et al., 2009) indicated that the final sample size of N = 58 retained satisfactory power of .75, which indicates that the study was appropriately powered to detect moderate effects.

3. Results

3.1. Demographics

The characteristics of the participants are presented in Table 1. The mean age of participants was typical for an undergraduate sample ($M = 21.02$, $SD = 4.72$). The sample were predominantly female (69.0%, $n = 40$), white (74.1%, $n = 43$) and British (82.8%, $n = 48$). There we no significant differences between condition order groups in any demographic variables.

3.2. Comparison of baseline timepoints

No significant differences were found in any mediator or outcome variable between baseline assessments preceding each sleep condition (see Supplementary Material S.3).

3.3. Adherence to sleep conditions

The mean sleep duration was 8 h and 2 min for the standard sleep condition ($M = 481.7$ min, $SD = 57.3$) and 3 h and 45 min for the sleep loss condition ($M = 225.3$ min, $SD = 29.5$), with a statistically significant reduction in time slept during the night of sleep restriction ($p < .001$). No significant difference in the mean consumption of alcohol (0.48 vs 0.49 units respectively, $p = .910$) or nicotine (0.71 vs 0.81 mg respectively, $p = .910$) over the 24 h before assessment following the nights of sleep was found across conditions. A significant increase in caffeine consumption was recorded for the sleep loss condition (130.84 vs 88.99 mg in the standard sleep condition, $p = .005$).

Table 1
Demographic characteristics of study participants.

	Total Sample	First Week Sleep Restricted	Second Week Sleep Restricted
	$N = 58$	($n = 29$)	($n = 29$)
Mean age (*SD*)	21.02 (4.72)	21.07 (5.22)	20.97 (4.26)
Gender (n, %)			
Male	17 (29.3%)	5 (17.2%)	12 (41.4%)
Female	40 (69.0%)	23 (79.3%)	17 (58.6%)
Non-binary	1 (1.7%)	1 (3.5%)	0 (0.0%)
Ethnicity (n, %)			
White	43 (74.1%)	22 (75.9%)	21 (72.4%)
Asian	6 (10.3%)	2 (6.9%)	4 (13.8%)
Mixed/Multiple ethnic groups	6 (10.3%)	3 (10.3%)	3 (10.3%)
Black/African/ Caribbean	1 (1.7%)	0 (0.0%)	1 (3.5%)
Other	2 (3.5%)	2 (6.9%)	0 (0.0%)
Nationality (n, %)			
British	48 (82.8%)	25 (86.2%)	23 (79.3%)
European	6 (10.3%)	2 (6.9%)	4 (13.8%)
Asian	3 (5.2%)	1 (3.5%)	2 (6.9%)
Other	1 (1.7%)	1 (3.5%)	0 (0.0%)

3.4. Correlation analyses

Correlation coefficients between the variables assessed post-standard sleep and post-sleep loss can be found in Supplementary Material S.4 and S.5.

3.5. Effects of sleep loss on psychotic experiences and proposed mediators

The mean scores for variables in both sleep conditions, along with the total effects of sleep loss on proposed mediating variables and psychotic experience outcome variables, are summarised in Table 2.

Sleep loss was associated with significant increases in negative affect ($d = 0.77$), affect intolerance ($d = 0.45$) and dissociative experiences ($d = 0.43$).

Sleep loss was also significantly associated with increased paranoia ($d = 0.44$), hallucinations ($d = 0.55$), cognitive disorganisation ($d = 0.25$), anhedonia ($d = 0.26$), and distress related to experiences ($d = 0.39$). No significant change in grandiosity was reported.

3.6. Mediation analyses

Mediation analyses were conducted to explore the indirect effects of mediating variables on the relationship between sleep loss and psychotic experiences in line with proposed hypotheses (see Table 3).

The relationship between sleep loss and paranoia was mediated by dissociative experiences, which accounted for 46.2% of the association, with the direct effect no longer reaching significance. There was also a significant combined indirect effect of negative affect, affect intolerance and dissociative experiences, accounting for 89.1% of the association between sleep loss and paranoia. No significant indirect effects were found for negative affect or affect intolerance.

Dissociative experiences partially mediated the relationship between sleep loss and hallucinations, accounting for 38.5% of the association, with no other significant mediation effects found.

After accounting for the proposed mediators, the direct effects of sleep loss on both cognitive disorganisation and anhedonia were no longer significant. However, no significant mediation effects were observed for negative affect, affect intolerance, dissociative experiences, or combined mediators in accounting for the associations. Finally, no significant effects of mediation were found for the association between sleep loss and distress related to psychotic experiences.

A summary of the proposed causal pathway in relation to mediation analyses is shown in Fig. 2.

4. Discussion

The present study tested whether a single night of sleep restriction increases subclinical psychotic experiences and sought to identify if this effect is mediated by negative affect, affect intolerance, and dissociative experiences. In line with our first hypothesis, sleep loss resulted in a significant increase in paranoia, hallucinations, cognitive disorganisation, anhedonia, and distress related to these experiences. No significant change in grandiosity was found. Our second hypothesis was also confirmed, as negative affect, affect intolerance, and dissociative experiences were all significantly increased in the sleep loss condition relative to standard sleep. For our third hypothesis, we successfully demonstrated that elevated dissociative experiences, and combined mediators, both substantially mediated increases in paranoia following sleep loss. Additionally, elevated dissociative experiences partially mediated the association between sleep loss and increased hallucinations. The increases in cognitive disorganisation, anhedonia and distress related to psychotic experiences following sleep loss were not

Table 2
Changes in psychotic experiences and proposed mediators following standard sleep vs sleep loss condition.

	Standard Sleep Mean (*SD*)	Sleep Loss Mean (*SD*)	Effect (*SE*), *p*-value	95% *CI*	Effect Size
Psychotic Experiences					
Paranoia (SPEQ)	2.4 (3.7)	4.8 (6.8)	2.38 (0.75), .002	0.88 to 3.88	0.44
Hallucinations (SPEQ)	0.7 (1.4)	2.2 (3.6)	1.48 (0.39), <.001	0.71 to 2.25	0.55
Cognitive Disorganisation (SPEQ)	5.1 (3.8)	6.0 (3.5)	0.97 (0.27), <.001	0.43 to 1.50	0.25
Grandiosity (SPEQ)	2.4 (3.1)	2.2 (2.8)	−0.17 (0.33), .606	−0.84 to .49	−0.07
Anhedonia (SPEQ)	14.6 (9.1)	17.0 (9.7)	2.21 (0.99), .030	0.22 to 4.2	0.26
Distress Related to Experiences (SPEQ)	0.4 (1.0)	0.9 (1.5)	0.45 (0.14), .003	0.16 to 0.73	0.39
Proposed Mediating Variables					
Negative Affect (Total DASS)	5.2 (5.1)	11.3 (10.0)	6.09 (1.02), <.001	4.03 to 8.14	0.77
Affect Intolerance (Total AIS)	83.1 (28.6)	96.4 (30.1)	13.33 (3.01), <.001	7.31 to 19.35	0.45
Dissociative Experiences (Total ČEFSA)	10.8 (14.5)	18.4 (20.1)	7.69 (1.65), <.001	4.38 to 11.00	0.43

Table 3
Mediation analyses for proposed mediators and psychotic experiences.

Outcome	Total Effect, Estimate (*SE*), Bootstrap 95% *CI*, *P* value	Mediators Tested	Direct Effect, Estimate (*SE*), Bootstrap 95% *CI*, *p*-value	Indirect Effect, Bootstrap Estimate (SE), Bootstrap 95% *CI*	Proportion of Total Effect Mediated
SPEQ Paranoia Subscale	2.38 (.75), 0.88 to 3.88, .002*	Negative Affect *(Total DASS)*	0.26 (0.98), −1.72 to 2.24, .794	0.55 (0.94), −1.22 to 2.55	23.1%
		Affect Intolerance *(Total AIS)*		0.47 (0.44), −0.18 to 1.64	19.7%
		Dissociative Experiences *(Total ČEFSA)*		1.10 (0.58), 0.04 to 2.39*	46.2%
		Combined *(DASS, AIS and ČEFSA)*		2.12 (0.95), 0.62 to 4.28*	89.1%
SPEQ Hallucinations Subscale	1.48 (0.39), 0.71 to 2.25, <.001*	Negative Affect *(Total DASS)*	1.20 (0.51), 0.18 to 2.23, .023*	−0.34 (0.63), −1.48 to 0.99	−23.0%
		Affect Intolerance *(Total AIS)*		0.05 (0.22), −0.38 to 0.54	3.4%
		Dissociative Experiences *(Total ČEFSA)*		0.57 (0.31), 0.02 to 1.24*	38.5%
		Combined *(DASS, AIS and ČEFSA)*		0.28 (0.51), −0.57 to 1.47	18.9%
SPEQ Cognitive Disorganisation Subscale	0.97 (0.27), 0.43 to 1.50, <.001*	Negative Affect *(Total DASS)*	0.61 (0.40), −0.20 to 1.42, .138	0.15 (0.28), −0.41 to 0.69	15.5%
		Affect Intolerance *(Total AIS)*		−0.08 (0.19), −0.51 to 0.23	−8.2%
		Dissociative Experiences *(Total ČEFSA)*		0.29 (0.21), −0.06 to 0.78	29.9%
		Combined *(DASS, AIS and ČEFSA)*		0.36 (0.27), −0.17 to 0.89	37.1%
SPEQ Anhedonia Subscale	2.21 (0.99), 0.22 to 4.20, .030*	Negative Affect *(Total DASS)*	0.65 (1.30), −1.96 to 3.27, .618	1.70 (1.67), −1.27 to 5.14	76.9%
		Affect Intolerance *(Total AIS)*		−0.65 (0.52), −1.66 to 0.45	−29.4%
		Dissociative Experiences *(Total ČEFSA)*		0.51 (0.84), −1.12 to 2.19	23.1%
		Combined *(DASS, AIS and ČEFSA)*		1.56 (1.31), −0.77 to 4.34	70.6%
SPEQ Distress Subscale	0.45 (0.14), 0.16 to 0.73, .003*	Negative Affect *(Total DASS)*	0.43 (0.20), 0.04 to 0.83, .031*	−0.05 (0.18), −0.38 to 0.33	−11.1%
		Affect Intolerance *(Total AIS)*		−0.07 (0.07), −0.23 to 0.05	−15.6%
		Dissociative Experiences *(Total ČEFSA)*		0.13 (0.18), −0.21 to 0.51	28.9%
		Combined *(DASS, AIS and ČEFSA)*		0.01 (0.25), −0.43 to 0.55	2.2%

Note. *$p < .05$.

significantly mediated by any of the proposed variables. Overall, the results are consistent with a causal role for sleep loss in the development of subclinical psychotic experiences, in some cases via affective processes.

Our study demonstrated that a single night of sleep loss was sufficient to cause a significant increase in paranoia, hallucinations, cognitive disorganisation, anhedonia, and distress associated with these experiences in a non-clinical sample. While prior sleep restriction and total sleep deprivation studies have shown mixed results (Reeve et al., 2018a; Hurdiel et al., 2015; Kahn-Greene et al., 2007; Petrovsky et al., 2014), to our knowledge this is the first study to show that these changes can occur after just one night of reduced sleep. Our effect sizes

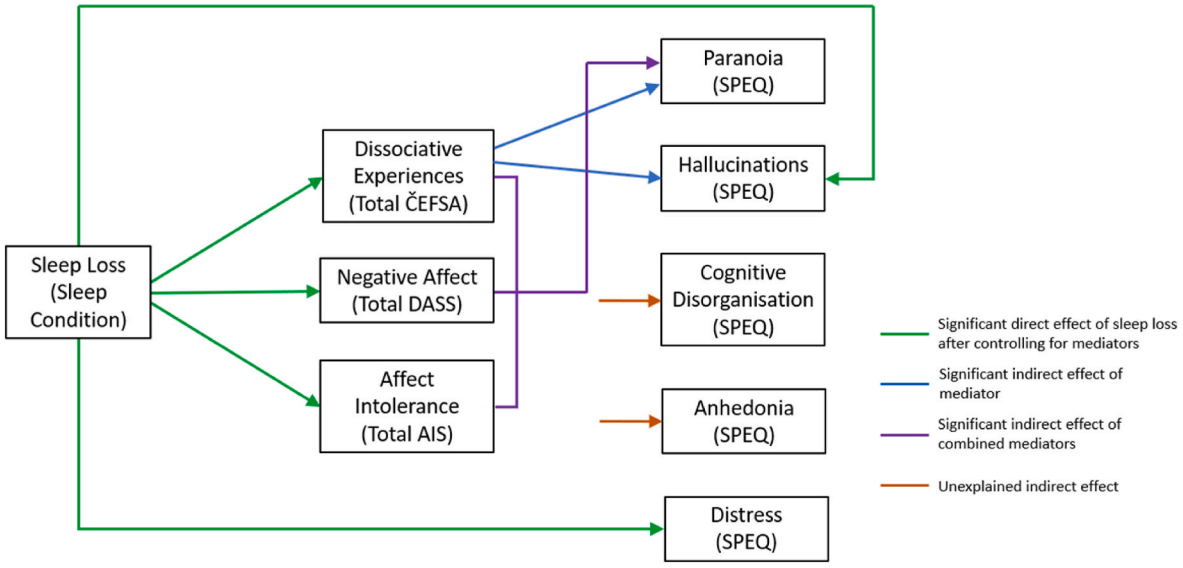

Fig. 2. Proposed causal pathway following mediation analyses.

were smaller than previous studies, as would be expected since the effects of sleep loss are cumulative over time (Van Dongen et al., 2003). However, even these smaller magnitude effects are likely important, given that individuals who experience psychotic symptoms often report prolonged sleep disruption (Cederlöf et al., 2022; Waite et al., 2020). Additionally, our findings are consistent with previous research indicating that sleep loss differentially influences individual psychotic experiences, with a lack of effect on grandiosity, and larger effects on paranoia and hallucinations than on cognitive disorganisation or anhedonia (Reeve et al., 2018a).

One night of sleep restriction significantly increased next day negative affect in line with previous findings (Reeve et al., 2018a; Barber et al., 2023) and in contrast to others (Palmer et al., 2024). Regarding our other proposed mediating variables, results from prior studies are consistent with our findings in showing that sleep disruption leads to an increase in dissociative experiences (Giesbrecht et al., 2013; Menicucci et al., 2022; Selvi et al., 2015; van Heugten–van der Kloet et al., 2015). To our knowledge the causal increase in affect intolerance following sleep restriction is a novel finding, underscoring the role of sleep loss in disrupting affective processes relevant to the development of psychosis.

We found that increases in subclinical paranoia following sleep loss were substantially mediated by the combined effects of negative affect, affect intolerance and dissociative experiences. This aligns with research linking paranoia to both insomnia and the intolerance of distressing emotional states (Brown et al., 2024; Freeman et al., 2009; Moutoussis et al., 2015). Our findings are consistent with evidence from clinical samples showing that maladaptive emotion regulation partially mediates the association between sleep difficulties and paranoid thinking (Grezellschak et al., 2017). No significant mediators were identified for cognitive disorganisation and anhedonia, or for distress from psychotic experiences. This was unexpected given previous research highlighting the key role of emotion regulation processes in influencing distress in clinical samples (Bak et al., 2008), suggesting that other mechanisms, such as thought control strategies (Hartley et al., 2015) or reappraisal (Zhan et al., 2022), may better explain distress in response to psychotic experiences.

Dissociation was found to substantially mediate elevated paranoia, and partially mediate increased hallucinations, which is consistent with previous findings (Černis et al., 2021b; Longden et al., 2020; Varese et al., 2011; Creatura et al., 2022). Our finding that dissociation mediates hallucinatory experiences supports approaches that posit auditory hallucinations as dissociative in nature, such as dissociated components of the self or distressing past events (Longden et al., 2012; Moskowitz et al., 2009). The relationship between cognitive disinhibition and dissociation has been linked to hallucinations (Varese et al., 2011, 2012), suggesting that cognitive processes may explain the remaining partial mediation of hallucinations after accounting for dissociation (Sheaves et al., 2025). Overall, these findings emphasise the key role of affective processes in the development of psychosis symptoms following sleep loss.

4.1. *Strengths and limitations*

The present study has several strengths in its methodology, as an experimental study both replicating and extending previous findings in the pathway from sleep to psychosis. Mechanisms tested and analysis plan were pre-specified based on empirical and theoretical hypotheses, and relevant controls included counterbalancing sleep conditions to reduce order and practice effects, and including washout periods between conditions to prevent carryover effects following sleep loss. Adherence to sleep conditions was verified by assessment of sleep length recordings, alongside assessment of the potential confounding effect of recently consumed wakefulness substances. An increase in caffeine consumption during the sleep loss condition was found, which amounted to less than the content of a cup of black tea (around 40 mg). While caffeine can have an acute effect on mental health variables (e.g. anxiety) this would generally be expected at higher doses (e.g. 400 mg or more; Liu et al., 2024).

Several limitations should be considered when interpreting the results of this study. Due to time constraints during recruitment and exclusion of participants, the final participant sample was smaller than originally planned. The sample itself was also composed of undergraduate psychology students – while these are in a representative age range for onset of psychosis (Solmi et al., 2022) they are not representative in other important dimensions (e.g. socio-economic status or ethnicity; Kirkbride et al., 2012). The aims and hypotheses of the study were not shared with participants until after they had participated, but it is possible that their familiarity with the topic may have affected their responses. Corrections for multiple comparisons were not applied since analyses were based on pre-specified hypotheses, which may have increased the likelihood of Type I error. In line with previous experimental protocols mediators and outcomes were measured contemporaneously, meaning models could be explored testing reverse pathways (e.

g. sleep to psychosis to mood) – however these have been found to be less empirically supported in prior research (e.g. Reeve et al., 2018b). Finally, the timescale of all measures was adapted to suit the 24-h period between baseline and post-intervention assessments, which may have influenced their validity.

4.2. Future directions and clinical implications

Using a sleep continuity disruption protocol, rather than delaying sleep onset, may produce more robust effects on reported affect or psychotic experiences and provide insights into whether these processes differ for individuals with insomnia characterised by frequent nighttime awakenings (Brederoo et al., 2021; Finan et al., 2015). If negative affect, affect intolerance and dissociation were shown to moderate changes in symptoms along the psychosis continuum, future studies could focus on investigating targeted interventions to determine whether these variables act as mechanisms of change for improving psychotic symptoms. Other potential mediators also require identification and investigation, particularly for hallucinations where a relatively small proportion of the direct effect of sleep restriction was explained (<40%) – these could include for example, stress, inflammation, or source monitoring (Sheaves et al., 2025). While this study is conducted in a non-clinical group, and the psychotic experience changes seen are below clinical thresholds, the results have applicability for ongoing consideration of sleep difficulties and related processes, including emotion regulation, as a target for intervention in psychosis (Freeman et al., 2017; Livingstone et al., 2009).

4.3. Conclusion

Our findings support a model in which sleep difficulties act as a causal factor in the development of psychotic experiences, offering insights into the mechanisms that drive the onset and maintenance of psychosis. Sleep may therefore serve as a protective factor against symptom development, and sleep difficulties should be recognised as a treatment target and early warning sign for clinicians. Additionally, negative affect, affect intolerance and dissociative experiences may be targets for monitoring and intervention in the onset or maintenance of psychosis, with further investigation indicated to explore this possibility.

CRediT authorship contribution statement

Madeleine R. Johnson: Conceptualization, Formal analysis, Investigation, Methodology, Project administration, Writing – original draft, Writing – review & editing. **Joanne L. Bower:** Conceptualization, Formal analysis, Investigation, Methodology, Project administration, Supervision, Writing – original draft, Writing – review & editing. **Sarah Reeve:** Conceptualization, Formal analysis, Investigation, Methodology, Project administration, Supervision, Writing – original draft, Writing – review & editing.

Declaration of competing interest

None to declare.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2026.06.028.

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