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Insomnia, negative affect, and psychotic experiences: Modelling pathways over time in a clinical observational study

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ABSTRACT

Insomnia has been shown to contribute to the development of psychotic experiences, predominantly via increasing negative affect. However, the role of insomnia in the persistence of psychotic experiences is yet to be investigated in a clinical population. Furthermore, other plausible influences, such as psychotic experiences contributing to insomnia, remain to be evaluated. This study tests the role of insomnia as a predictor of persistence of psychotic experiences versus other potential causal routes. Twenty-nine patients aged 18–30 with non-affective psychosis completed three assessments over three months of their insomnia, negative affect, and psychotic experiences. Mixed effect models allowed comparisons between hypothesis-based models (comprising insomnia as predictor, negative affect as mediator, and psychotic experiences as outcome) and oppositional models, where relationships were reversed. The results supported the hypothesised mediation model above models where negative affect was primary. Insomnia was also found to be a stronger predictor of later hallucinations than vice versa, although a bidirectional relationship was indicated between insomnia and paranoia. In conclusion, insomnia predicts persistence of psychotic experiences over time to the same or greater extent than psychotic experiences contribute to insomnia. This supports insomnia as a potential intervention target in psychosis.

1. Introduction

Insomnia has traditionally been thought of as a consequence of psychotic symptoms, however recent research indicates that insomnia itself contributes to the development of psychotic experiences (Reeve et al., 2015). For example, an experimental study found that inducing insomnia-like sleep loss in non-clinical volunteers resulted in increased paranoia and hallucinations (Reeve et al., 2018), and a large clinical trial of an online CBT intervention for insomnia in students found that treating insomnia reduced subclinical paranoia and hallucinations (Freeman et al., 2017). Together these findings demonstrate a causal role for insomnia in psychosis, implying that insomnia may represent a novel target for treatment of psychosis.

Sleep disturbance in general has been increasingly associated with psychotic experiences in both clinical and non-clinical populations (Chiu et al., 2016; Davies et al., 2017; Koyanagi and Stickley, 2015) . However, there is a surprising lack of studies investigating the relationship between insomnia (as a specific sleep disorder) and psychosis in individuals with a psychotic disorder (Reeve et al., 2015). Cross-sectional studies indicate that individuals with psychotic disorders and comorbid insomnia have more severe psychotic experiences than those without (Freeman et al., 2009; Xiang et al., 2009). Yet longitudinal research is currently limited to studies utilising experience sampling methods (ESM) to collect high frequency data across a short time period. This technique allows investigation of the interplay between night-time sleep and day-time mental health, with significant relationships reported between lowered sleep quality, efficiency, and duration and increased psychotic experiences the following day (Hennig and Lincoln, 2018; Mulligan et al., 2016; Waters et al., 2011). Notably, in one of these studies shorter sleep was found to predict paranoia (but not the reverse) in a non-clinical adolescent group (Hennig and Lincoln, 2018).

However, there are limitations to the ESM approach. Firstly, although the results are clearly applicable to insomnia (in which sleep efficiency, quality, and duration are lowered), these studies do not measure insomnia symptoms directly. Secondly, as assessments are completed repeatedly within a short time, outcomes are measured using individual items (or a small set of items), instead of a fully validated

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questionnaire or interview assessment. Finally, due to the intensity of the ESM approach the observational period remains short. Therefore, it remains unclear if the day-by-day relationships found in ESM studies can be extrapolated to diagnosable insomnia (which requires a duration of 3 months of symptoms) or psychotic symptoms assessed over longer time periods. This is of particular importance in early psychosis where treatment of predictors of psychotic symptoms could improve later clinical trajectory.

Negative affect – here used as a generic term to refer to depression and anxiety - is often identified as a mediator in the insomnia to psychosis relationship (e.g. Reeve et al., 2018, 2015), as supported by a large literature linking insomnia and affect. Insomnia and depression are strongly related: individuals with insomnia are at higher risk of developing depression (Li et al., 2016), and treating insomnia has also been shown to improve depression (e.g. Christensen et al., 2016). Those with depression are also more likely to develop insomnia (Jansson-Fröjmark and Lindblom, 2008). Insomnia and anxiety have a strong, if less researched relationship. Anxiety is predictive of later insomnia, and insomnia is similarly predictive of later anxiety (Neckelmann et al., 2007). The psychological processes shared between cognitive models of insomnia and anxiety (such as hyperarousal, catastrophising, and intrusive thoughts) also link the phenomena (Espie, 1991; Harvey, 2002). Whilst negative affect has been shown to mediate the relationship between insomnia and psychosis (Hennig and Lincoln, 2018; Mulligan et al., 2016; Reeve et al., 2018), the bidirectional relationship between insomnia and negative affect means that it is equally plausible that insomnia could mediate the relationship between negative affect and psychosis (see Fig. 1a for a diagram of these pathways). No previous study has tested this possibility.

Furthermore, while recent research has focused on demonstrating the causal role of insomnia in psychotic experiences (Freeman et al., 2017; Reeve et al., 2018), it also remains likely that psychotic experiences contribute to insomnia (see Fig. 1b). One obvious route would be that distress from psychotic experiences increases arousal and delays sleep onset (Waite et al., 2016a). Other possible factors include lowered daytime activity, which is common in psychosis (Hodgekins et al., 2015; Stubbs et al., 2016), and can cause sleep disturbance by destabilising circadian rhythms (Waite et al., 2016b). Based on these and other factors a bidirectional relationship between insomnia and psychosis has been proposed, but not adequately tested (Harvey and Murray, 2011; Reeve et al., 2015). Whether this relationship is truly bidirectional is important to assess, since it may have clinical implications for prioritising treatment of insomnia versus psychotic experiences.

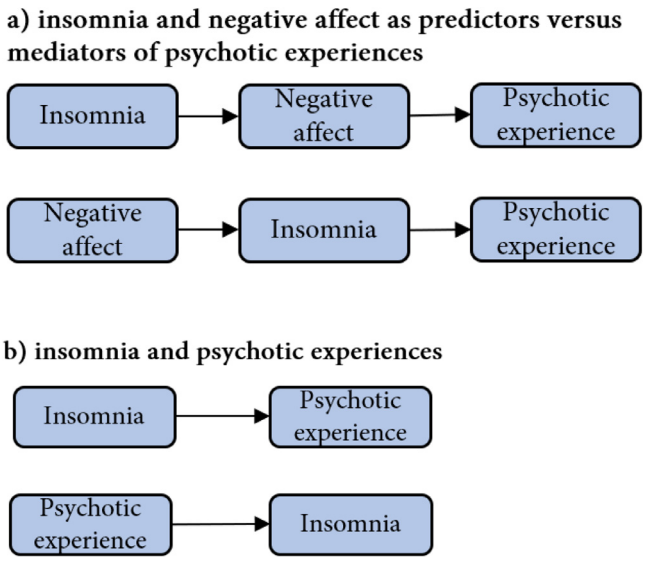

Fig. 1. Possible pathways between insomnia, negative affect, and psychotic experiences.

1.1. The current study

The current study aimed to investigate the interaction between insomnia, negative affect (i.e. depression and anxiety), and psychotic experiences (i.e. paranoia and hallucinations) over several months within a cohort of individuals with early psychosis. The analytical approach was to test the directions of effect between insomnia, negative affect, and psychotic experiences by comparing models derived from key hypotheses (that insomnia predicts later psychotic experiences, with negative affect acting as the key mediator) to oppositional models where key relationships are reversed in order to disentangle the most relevant causal influences between these factors. The hypotheses tested were:

1. Insomnia, negative affect, and psychotic experiences are cross-sectionally associated;
2. Insomnia is predictive of later psychotic experiences;
3. The relationship between insomnia and later psychotic experiences is mediated by negative affect;
4. Psychotic experiences are predictive of later insomnia;
5. Insomnia is more predictive of later psychotic experiences than psychotic experiences are of later insomnia.

2. Method

2.1. Recruitment

Twenty-nine participants were recruited for the current study. The inclusion criteria were: primary diagnosis of non-affective psychotic disorder; outpatient status; and age between 18 and 30. The age range was selected to minimise the effects of long-term antipsychotic medication usage on sleep, and to control for changes in sleep over the lifespan. Exclusion criteria were: primary diagnosis of affective, substance abuse, organic, or neurological disorder; and non-fluency in English. Eligible participants were initially approached by members of their NHS care team and given information regarding the study. Those willing to participate provided written informed consent to take part in the study and received compensation for their time in taking part. The study received approval from an NHS research ethics committee (South West-Frenchay REC reference 15/SW/0291), and local approvals were received for each of the three study sites.

2.2. Design and assessments

In this longitudinal observational study participants were assessed at baseline, one month, and three months. All measures were completed at every time point. For all measures higher scores indicate greater severity of symptomatology.

2.2.1. Insomnia

Insomnia was assessed using the Sleep-50 (Spoormaker et al., 2005), a self-report questionnaire indexing severity of a number of sleep disorders. The total scale is comprised of 50 statements which are rated for agreement over the past month, on a 1 (“Not at all”) to 4 (“Very much”) Likert scale. The subscale for insomnia was used in the current study, which comprises 8 items, with a minimum score of 8 and a maximum score of 32. The insomnia subscale demonstrates high consistency (Cronbach's alpha = 0.85)

2.2.2. Psychotic experiences

Paranoia and hallucinations were assessed using the Specific Psychotic Experiences Questionnaire (SPEQ; Ronald et al., 2014). The SPEQ is a self-report questionnaire with dimensions for individual