Chapter title: Schizophrenia spectrum disorders

(schizophrenia, schizoaffective disorder, short-term psychotic episodes)

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Key Points:

- Insomnia and nightmare disorders are the most commonly presenting sleep disorders in patients with non-affective psychosis.
- Co-occurrence of different sleep disorders is most typical for patients experiencing non-affective psychosis.
- Longitudinal, experimental, and treatment studies indicate that insomnia is a contributory causal factor in the occurrence of delusions and hallucinations.
- CBT treatments, suitably adapted and delivered in one-to-one sessions with a therapist, targeting insomnia and nightmares lead to large effect size improvements in sleep for patients with schizophrenia.
- Patients with psychosis want their sleep difficulties treated: uptake, completion, and satisfaction with sleep therapy is high.

Learning Objectives:

- To appreciate the prevalence and co-occurrence of sleep disorders in patients with schizophrenia and related diagnoses.
- To understand the nature of the relationship between individual sleep disorders and psychotic experiences.
- To be familiar with adaptations to traditional psychological sleep treatment protocols needed for patients with psychosis.

Summary:

Insomnia and nightmares have been recognised as part of the presentation of schizophrenia for a century, since the early descriptions by Emil Kraepelin. Yet this sleep disruption has historically been viewed as either a secondary consequence of psychosis or an epiphenomenon. This view is undergoing significant revision. Over the past ten years, research has supported the view that insomnia is actually causally related to the occurrence of psychotic symptoms. Sleep disorders are common, for example over half of patients with non-affective psychosis have insomnia and approximately half also experience regular nightmares. Cognitive Behavioural Therapy (CBT) for insomnia and nightmares have both been adapted to optimise outcomes for patents with co-occurring psychotic symptoms and tested in pilot randomised controlled trials with highly promising effects on sleep and the potential to also improve psychotic symptoms. A notable finding across trials is the very high level of uptake of psychological sleep treatments: patients are eager for help with their sleep. CBT for insomnia leads to small improvements in paranoia and hallucinations and initial evidence suggests that treating nightmares might lead to moderate improvements in paranoia. The CBT interventions follow core learnings from sleep science but with adaptations to account for the impact of psychotic symptoms and associated lifestyle factors on the sleep system. This chapter shares the evidence linking sleep disorders to psychotic phenomena and the treatment of sleep disorders in this group.

Words: 230 (limit = 250)

Key words:

Insomnia; nightmares; psychosis; hallucination; delusion; cognitive behaviour therapy.

A. Epidemiology of schizophrenia spectrum disorders and sleep disorders

Schizophrenia spectrum disorders (SSDs) cover a range of diagnoses where psychotic symptoms are prominent for example schizophrenia, schizo-affective disorder and delusional disorder. Schizophrenia (the most common of SSDs) is characterised by the presence of psychotic symptoms including delusions, hallucinations and disorganised speech (World Health Organisation, 2016) and has a lifetime prevalence rate of 4 in every 1,000 people (McGrath, Saha, Chant, & Welham, 2008). Whilst it is a low-prevalence disorder, it is ranked 15th in the global burden of disease, owing to its high severity and persistence over time (GBD 2016 Disease and Injury Incidence and Prevalence Collaborators, 2017). For a list of terminology used within this chapter see table 1.

The diagnostic label of schizophrenia is however one of the most contested of psychiatric diagnoses. There is increasing recognition that it comprises multiple different and independent psychotic experiences which have differing levels of heritability (Zavos et al., 2014) and environmental and genetic aetiology (Shakoor et al., 2016). The patient description of individual symptoms supports the view that they are distinct experiences:

"a lot of my voices will come at me like someone is coming with a gun or someone is coming with a knife. As well as having a go at me. [The voices sound like] just a normal person's voice for me, an angry person but a normal person. Which is why it's so confusing. [..]"

'Vicki', who hears threatening voices. From Sheaves et al., (under review)

Sophie (who held the grandiose belief that she was a demigod and could therefore fly): "Trying to fly off various heighted objects"; "[I] stepped off things and expected to fly." Interviewer: "What's the highest thing you've stepped off?" Sophie: [deep exhale, 10s pause]"I can't entirely remember. And I don't want to remember if that makes sense."

'Sophie', who held a grandiose belief. From Isham et al., (2019)

"I had gotten so sort of paranoid of people around me that I was convinced that I was going to have acid thrown in my face. And I was walking around with like my hood up to cover the sides of my face and things like that and I just wasn't really talking with my housemates about it. [..] I had gone into the police because I was convinced that there were gangs who were out to get me and these were the guys who wanted to throw acid in my face".

'Dean', who held a persecutory belief. From Sheaves et al., (under review)

Paranoia, grandiosity and hallucinatory experiences are not experienced exclusively by those with psychiatric disorders. Around one in five people in the general population experience paranoid thoughts (Freeman et al., 2011) and 1 in 20 report hallucinatory experiences (Johns et al., 2004). Instead, psychotic experiences exist on a spectra of severity: general population data illustrates that paranoia for example follows a continuous distribution with an exponential curve (Bebbington et al., 2013). Importantly, there is no clear cut off to distinguish clinical and non-clinical participants. The same distribution has been observed for hallucinatory experiences, albeit typically with a higher degree of skew (Ronald et al., 2014). This provides an opportunity for research to draw on large scale general population datasets to study the association between sleep and psychotic experiences.

Table 1 about here

Sleep disruption has been recognised as a problem for people with psychosis since the early descriptions of 'dementia praecox' (now termed schizophrenia) by Emil Kraepelin. He described *"They complain of sleeplessness, nightmares, oppression in their head"* and recommended: *"Rest in bed, supervision, care for sleep and food, are here the most important requisites"* (Kraepelin, 1919). Yet it is only around a century later that sustained research attention has been given to sleep and psychosis.

Studies using polysomnography have uncovered that patients with schizophrenia have a lower total sleep time, longer sleep onset latency, increased wake after sleep onset, lower sleep efficiency, decreased slow wave sleep and decreased duration and latency of rapid eye movement (REM) sleep (Chan, Chung, Yung, & Yeung, 2017). Yet there is significant heterogeneity across studies and further research is needed to investigate whether these findings are associated with psychotic symptoms themselves or are rather attributable to medication side effects (Chan et al., 2017).

The most common sleep disorders are i) insomnia, present in over half of patients with non-affective psychosis (Freeman, Taylor, Molodynski, & Waite, 2019) and ii) nightmares which occur in approximately half of patients (Reeve, Sheaves, & Freeman, 2018; Sheaves, Onwumere, Keen, Stahl, & Kuipers, 2015). Other sleep disorders such as sleep apnoea likely have an increased prevalence rate in psychosis compared to the general population (Myles et al., 2016), though the prevalence rate has been less studied than that of insomnia and nightmares (Table 2). Individual sleep disorders do not occur in isolation in psychosis samples. In the study by Reeve et al. (2018), patients had an average of three sleep disorders. Co-occurrence between insomnia and nightmares is particularly high; in a pilot trial recruiting patients with nightmares and persecutory delusions, all but one of the 24 participants also had insomnia at baseline (Sheaves et al., 2019). This comorbidity of sleep disorders, alongside psychotic experiences and very often depression, anxiety and suicidal ideation leaves a complex clinical picture. Focusing on one tractable problem at a time (e.g. sleep disruption) is a highly promising treatment approach that may manage this complexity (Freeman et al., 2016).

Identification of sleep disorders within clinical services is crucial in order to provide effective interventions. Yet recent data from patients experiencing early psychosis indicates that just half of all sleep disorders experienced by patients had been discussed with a clinician (Reeve et al., 2018). These discussions rely of course on the patient feeling able to share their sleep experiences, but also the topics clinicians choose to cover within an assessment. Recent survey data showed that only a minority (20%) of clinicians routinely carried out sleep assessments with their patients, and most relied on informally asking about sleep rather than using validated assessments (Rehman et al., 2016). Given the complex array of symptoms that patients with psychosis present with, this is understandable. However, given that the majority of psychosis patients have at least one sleep disorder the complexity of patients' sleep presentation is likely to be missed through brief discussion. Hence the opportunity for evidence-based treatment of the sleep disorder is also missed. Increasing the awareness of sleep disruption and its relation to psychotic symptoms is clearly warranted, alongside training opportunities to enable clinicians to feel adequately skilled to assess sleep, which has of course historically been overlooked in psychiatric services.

Table 2 about here

B. The course of psychosis and sleep disruption (1500 words)

Sleep disruption has historically been viewed as either i) a consequence of psychotic symptoms which fuel hyperarousal prior to sleep or ii) an epiphenomenon (a non-specific marker of distress). This may explain why sleep is given a low priority in psychosis service. The clinical implication being that treating psychosis is the priority, and this should alleviate sleep disruption too. However recent empirical studies turn this traditional view on its head. This evidence instead suggests that sleep disruption is a contributory cause of psychotic experiences (Freeman et al., 2017). Of course sleep disorders should be treated irrespective of whether they also improve psychotic experiences, but this recent research adds even greater weight to the argument that they should be given greater priority in psychiatric services (Freeman, Pugh, Vorontsova, & Southgate, 2009).

Research assessing the relationship between sleep and psychotic experiences across the course of psychosis, and across the spectrum of severity of psychotic symptoms, is reviewed in this section.

The course of psychosis and sleep disruption

The onset of psychosis typically falls around late adolescence and early adulthood. Sleep disruption commonly occurs prior to the first episode of psychosis (Yung & McGorry, 1996), during 'ultra-high risk' states (Goines et al., 2019; Poe et al., 2017), and is predictive of later psychotic symptoms (Reeve et al., 2019) and transition to a psychotic disorder (Ruhrmann et al., 2010). In a study of 100 patients, three quarters reported insomnia and half reported nightmares in the weeks leading up the onset of their persecutory delusion (Freeman, Morrison, et al., 2019). Hence temporally it is clear that sleep disruption commonly precedes the onset of psychotic disorder.

Insomnia and psychotic experiences

The co-occurrence of insomnia and psychotic experiences is robustly supported by multiple empirical studies (Reeve, Sheaves, & Freeman, 2015). For example in a study of 267,691 participants, the presence of insomnia symptoms was associated with over a doubling of odds of also reporting a delusional idea or hallucinatory experience (Koyanagi & Stickley, 2015). Longitudinal data is consistent with a causal relationship between insomnia and later paranoia (Bird, Waite, Rowsell, Fergusson, & Freeman, 2017; Freeman et al., 2012; Reeve, Nickless, Sheaves, & Freeman, 2018) and hallucinations (Reeve, Nickless, et al., 2018; Sheaves et al., 2016). In a clinical sample insomnia was a stronger predictor of later hallucinatory experiences than the reverse relationship, however the relationship was bidirectional between insomnia and paranoia (Reeve, Nickless, et al., 2018).

Manipulation studies are the strongest design to investigate a potential causal relationship between insomnia and psychotic experiences. Two studies have assessed the causal relationship between insomnia and psychotic experiences, by both increasing and decreasing sleep. The OASIS trial (Freeman et al., 2017) recruited 3,755 students experiencing insomnia and randomly allocated them to receive digital CBT for insomnia (the Sleepio™ programme) or standard care. Psychotic experiences were assessed as dimensional traits that occur on a spectrum of severity across the general population. The treatment resulted in large effect size improvements in insomnia compared to standard care. Small but highly significant improvements in paranoia and hallucinatory experiences were also found following sleep treatment, compared with treatment as usual. Mediation analysis demonstrated that around two thirds of the change in psychotic experiences after treatment was attributable to the change in insomnia, with little evidence for reverse causation. This is the most convincing evidence yet that insomnia is one contributory causal factor in the occurrence of psychotic experiences.

The reverse manipulation simulated insomnia by restricting sleep duration to four hours across three nights in a non-clinical sample. This sleep restriction condition was compared with normal sleep using a within-subjects cross-over design (Reeve, Emsley, Sheaves, & Freeman, 2017). Sleep was successfully restricted and significant temporary increases in sub-clinical paranoia, hallucinatory experiences and cognitive disorganisation were found. However, there was no increase in grandiosity. This indicates that there is some differentiation of the effect of insomnia on individual psychotic experiences. This is supported by research reporting that genetic and environmental influences on paranoia, hallucinations and cognitive disorganisation show overlap with that of insomnia, but this same overlap was not found for grandiosity (Taylor, Gregory, Freeman, & Ronald, 2015).

The Reeve et al. (2017) study also assessed potential psychological mechanisms linking sleep loss and psychotic experiences. Negative affect almost entirely mediated the association between sleep loss and paranoia (proportion mediated all > 91%), but only partly mediated the association with hallucinatory experiences (proportion mediated: 12-43%) and cognitive disorganisation (proportion mediated: 26-39%). This suggests that there are likely other, yet to be identified mechanisms linking sleep loss to hallucinatory experiences and cognitive disorganisation.

The finding of increased paranoia following sleep restriction has not been replicated in other smaller controlled studies testing one night of total sleep deprivation (Meyhöfer, Kumari, Hill, Petrovsky, & Ettinger, 2017; Petrovsky et al., 2014). However these same studies do still report an increase in

perceptual distortions and cognitive disorganisation. This difference between the total sleep deprivation studies and the sleep restriction manipulation in Reeve et al. (2017) are likely attributable to methodological differences (e.g. different experimental manipulations, different measurement scales or sample sizes).

Patients have clear insight into the relationship between insomnia and voice hearing. For example patients clearly report voices delaying sleep onset *'"Trying to sleep and [the voices] distract me and they keep me awake" (5)'* (p. 6., Waite et al., 2015) and triggering wakefulness after sleep onset '*"suddenly they woke me up, [the voices] need to interrogate me" (V4)*' (p. Sheaves et al., under review). Subsequently, sleeplessness can result in voices becoming louder '*They would be a lot louder and I would hear them more." (V13)*' (p. Sheaves et al., under review) and lack of sleep also impacts the patient's ability to question distressing voice content *'"it's not so much the voices are any different to any other day it's my ability to rationalise them." (V5)*' (p. Sheaves et al., under review). Patient accounts clearly note the link with paranoia and delayed sleep onset and the impact that sleep dysfunction has on next day functioning, for example: *"I kept thinking they would come in when I was sleeping to take me [..] I checked everywhere even the spaces where a person couldn't fit just so that I knew I was alone and even then I couldn't sleep. The lack of sleep made it even worse to function"* ('G', Freeman, Freeman & Garety, 2006). Patients often describe that it is more challenging to cope with threatening experiences after a night of poor sleep, and are clear about the link between insomnia and next day negative affect: *"I am more nervous and worried if I don't get sleep" (7)'* (p. 6., Waite et al., 2015).

Nightmares, dreams and psychotic experiences

The relationship between nightmares, dreams and psychotic experiences has received comparatively less scientific attention than that of insomnia. The first clinical study to identify nightmares as an important clinical issue for people with established psychosis was that of Sheaves et al. (2015). A large significant correlation was found between nightmare related distress and delusion severity, and a medium correlation was found between nightmare distress and the severity of voices, though fell short of significance in the sample of 40 patients. Patient accounts of the content of nightmares indicate that nightmares may be particularly distressing because they relate to daytime paranoia and threats made by voices, for example:

Interviewer: *"I am just wondering the sort of things that you are stressed out about during the day…"* **patient**: *"people coming to get me, torture me and kill me"* **Interviewer:** *Is that the same sort of thing that happens in your nightmares?"* **patient**: *"Yes, I have been chased, yeah. [..] Sometimes they catch me"*

(patient quote from Sheaves et al., 2019)

"Being told [by voices] that your children are going to be taken away from you and then dreaming that your children are going to be taken away from you, that's obviously linked in some way"

(patient quote from Sheaves et al., 2019)

Larger datasets have confirmed the cross-sectional association between nightmares and both paranoid thoughts and hallucinatory experiences (Sheaves et al., 2016). One explanation for the link between nightmares and psychotic symptoms is via post-traumatic stress disorder (PTSD), given the elevated prevalence of PTSD in this group and that nightmares are a hallmark of PTSD. From clinical experience nightmares result from PTSD for only a small sub-sample of patients. For the majority nightmares occur outside of the context of PTSD. This is supported by empirical evidence. Sheaves et al. (2015) found that nightmares were not associated with PTSD symptom severity in a clinical sample, and a large general population survey found that there was still a direct association between nightmares and both paranoia and hallucinatory experiences, even when PTSD symptoms were controlled for (Rek, Sheaves, & Freeman, 2017). Instead it appears that distressing psychotic experiences are depicted within nightmare content and that PTSD (for the majority at least) is not the key driver.

Whilst there is a group of patients who report chronic nightmares since childhood, others report the onset to be more closely tied to periods of stress, including the onset of psychosis. A cross-sectional study with 10 year old children (n=814) reported a three-fold increase in the likelihood of reporting

psychotic experiences with maternal endorsement of nightmares (Koopman-Verhoeff et al., 2019). Longitudinal studies support the hypothesis that nightmares might be an early vulnerability factor for the later development of psychotic experiences. Childhood nightmares have been associated with later psychotic experiences aged 12 (Thompson et al., 2015) and 18 (Thompson et al., 2015) in a birth cohort. The mechanisms linking nightmares and later psychotic experiences have not been investigated from a psychological perspective, but at a molecular genetic level greater polygenic risk of schizophrenia has been associated with an increased risk of nightmares in early life (Reed, Jones, Hemani, Zammit, & Davis, 2019).

Other sleep disorders

One problem recognised very much by patients is that of oversleeping: '*"I don't want nine hours' sleep. Your life's boring enough without sleeping too much." (r04)'* (p. 10., Faulkner & Bee, 2017). Sleeping too much has been associated with poorer processing speed and inhibition in psychosis (Laskemoen et al., 2019) and often presents alongside insomnia (Chiu et al., 2018; Sheaves et al., 2018). However to our knowledge no study has assessed the association with psychotic experiences. Whether the presentation of oversleeping reflects a true hypersomnia disorder in this group remains to be established. Data suggest that it is unlikely to be attributable to narcolepsy (Sansa et al., 2016) but it is plausible that it is secondary to medication side effects, lack of circadian zeitgebers to drive arousal (e.g. light and activity) or using sleep to escape from psychotic symptoms (Faulkner & Bee, 2017; Waite, Evans, et al., 2015).

Whilst sleep apnoea is common, to our knowledge there are no studies reporting the association between sleep apnoea symptoms and psychotic symptoms. However apnoea has been associated with other health indexes in patients with psychosis for example body mass index and diabetes (Annamalai, Palmese, Chwastiak, Srihari, & Tek, 2015). Weight gain is a common side effect of several anti-psychotics (Tek et al., 2016), and may also result from the sedentary lifestyle which is common for people experiencing severe mental illness (Vancampfort et al., 2017). However it is also a risk factor for apnoea, indicating that this specific sleep disorder may follow the onset of psychosis and initiation of pharmacological treatment.

Circadian dysregulation is commonly recognised by clinicians. Many for example recognise that a morning appointment will not be feasible for some clients to attend. Whether these shifted sleep timings reflect true circadian rhythm dysfunction, or rather are a consequence of hyperarousal delaying sleep onset (shifting the sleep window later) requires careful assessment. There is initial evidence that for a sub-sample of patients the delayed sleep phase might be attributable to a later peak of melatonin (mean = 8-9am rather than 4-5am for healthy controls), though the difference fell short of significance and requires testing in a larger sample (Wulff, Dijk, Middleton, Foster, & Joyce, 2012). Delayed sleep phase is associated with increased negative (but not positive) symptoms of psychosis (Poon, Kan, Yeung, & Chung, 2018).

It is notable that some symptoms of narcolepsy such as excessive daytime sleeping, hypnagogic and hypnopompic hallucinations and sleep paralysis are more common in patients experiencing psychosis (Reeve, Sheaves, et al., 2018). Empirical studies assessing narcolepsy in psychosis samples are lacking and there are challenges to typical assessments for narcolepsy in this group: the effects of psychiatric medications on REM sleep and daytime somnolence requires consideration when conducting polysomnography (PSG) or multiple sleep latency tests (MSLT). One study screened 366 patients with schizophrenia spectrum disorders without relying on a PSG or MSLT and found no patients with a clear pattern of symptoms indicative of narcolepsy (Sansa et al., 2016). Nevertheless, symptoms such as night time hallucinations and sleep paralysis can be distressing and hence warrant attention in sleep interventions.

How do psychotic symptoms and lifestyle factors impact on the sleep system?

It is well established that the two key processes which promote sleep are the circadian drive for arousal and homeostatic sleep pressure (Borbély, Daan, Wirz-Justice, & Deboer, 2016). Hyperarousal is

additionally crucial for its ability to over-ride the sleep promoting system in order to remain alert (rather than sleepy) if under threat. There are several factors related to the experience of psychosis which impact on these three interacting systems (see Figure 1). First, there are often reduced circadian zeitgebers to regulate the circadian rhythm. For example, the majority of patients are unemployed because of their difficulties, symptoms can very often lead to social withdrawal and activity levels are markedly decreased. Spending substantial time indoors, or a period in hospital reduces exposure to natural light which is a key circadian regulator. Several factors reduce the opportunity for optimal levels of homeostatic sleep pressure at the desired nocturnal sleep time. The sedative effects of medication can lead to early bed times and later wake times. Inactivity and fatigue (resulting from medication, managing voices, depression or sleeplessness) increase the opportunity for naps, and in the daytime sleep can be used by some patients as a means of escaping from distressing voices, but this napping in turn disrupts nocturnal sleep. Lastly, there are factors which can result in acute pre-sleep hyperarousal in psychosis patients. Night time can be seen as a time of increased vulnerability from a persecutor, and voices are more likely to occur and be listened to when it is quiet (as it often is at night time). These can trigger anxiety and frustration which delays sleep onset and reduces the quality of sleep. These factors each require consideration in adapting sleep treatments for patients with psychosis.

Figure 1 about here

C. Treating psychosis: what is the effect on sleep? (100 words)

The first-line recommended treatment for psychosis in UK guidelines is anti-psychotic medication (National Collaborating Centre for Mental Health, 2014). For patients with schizophrenia presenting with comorbid sleep disturbance the choice of anti-psychotic is often determined by sedating properties. Yet there is a scarcity of controlled trials testing the effects of anti-psychotics on the treatment of sleep disorders (Wilson et al., 2010). In general antipsychotic medications show some beneficial effect on total sleep time, sleep efficiency (Cohrs, 2008) and sleep continuity (Chan et al., 2017). These effects are well recognised by patients: *"I take it [oral anti-psychotic] at night, because it's like your sleeping tablets sort of thing as well…" (r10)'* (p.9., Faulkner & Bee, 2017). However they can also alter sleep architecture (for example by decreasing REM sleep duration and latency) (Chan et al., 2017) and can lead to significant daytime sedation (Fang, Sun, Wang, Ren, & Calabrese, 2016). The sedating effects vary by medication type: clozapine for example is classified as a high somnolence medication, olanzapine and risperidone lead to moderate somnolence and aripiprazole and haloperidol are examples of low somnolence antipsychotics (Fang et al., 2016). It is therefore common in clinical practice that patients report difficulties getting up in the morning, daytime naps (which result in subsequent insomnia) or shifted sleep timing which can result in subsequent sleep disruption. Indeed the majority of patients report significant sleep disturbance despite the sedating effects of medication (70%; Waters, Faulkner, Naik, & Rock, 2012). The sleep promoting effects of antipsychotic medications are therefore not an adequate substitute for targeted sleep interventions.

Cognitive behavioural therapy for psychosis (CBTp) is recommended as an adjunct to medication in UK treatment guidelines (National Collaborating Centre for Mental Health, 2014) and does not traditionally include any focus on sleep. Meta-analyses indicate that CBTp leads to small to moderate effect size improvements in psychotic symptoms (Van der Gaag, Valmaggia, & Smit, 2014). However to our knowledge no trial has assessed the impact of CBTp on insomnia or nightmares. Of course given the small treatment effects of CBT on psychotic symptoms, any such trial would require a very large sample to detect a potential knock on effect on sleep.

D. Treatment of sleep disorders: what is the effect on sleep and psychosis (2000 words)

Hypnotic medication

A randomised controlled trial of eszopiclone for patients with schizophrenia indicated that it was superior to placebo at treating sleep after eight weeks (mean between group difference in ISI score = 3.8) (Tek et al., 2014). There was no between group difference for PANSS rated symptoms of schizophrenia, though the study was underpowered to detect small effects (n=36) and participants were not selected for having active symptoms of psychosis. Hypnotics are commonly prescribed, and particularly during an acute phase of insomnia which is common on inpatient wards. A qualitative study revealed that whilst patients are aware of the effectiveness of hypnotics for improving sleep, their drawbacks often outweigh their benefits, e.g.: *"They say about sleeping tablets, but I wouldn't want them, because you rely on them then to get sleep, and that's a false sleep then isn't it?" (r10)'* (p.8., Faulkner & Bee, 2017).

The psychological treatment of sleep disorders: the effect on sleep and psychosis.

It has been established in a definitive trial that successful treatment of insomnia leads to small but significant reductions in paranoia and hallucinations (Freeman et al., 2017). However this was conducted in a large student sample, with participants experiencing sub-clinical psychotic experiences. This section therefore addresses whether sleep disorders can successfully be treated in samples with more severe levels of psychotic experiences.

Three pilot randomised controlled trials have been conducted testing interventions for sleep disorders in clinical populations. The Better Sleep Trial (BeST; Freeman et al., 2015) was the first controlled test of a psychological sleep intervention for patients experiencing psychosis. Importantly, the treatment was popular (96% took up the opportunity for therapy). The eight-session intervention of CBT for sleep problems was compared against treatment as usual (TAU) and resulted in large effect size improvements in insomnia (d=1.9), largely maintained at 24-week follow-up (d=1.2). The effect on psychotic experience was not possible to establish with enough precision, owing to the small sample size (n=50). Indeed 95% confidence intervals covered the range from the CBT both increasing and decreasing delusions (d=0.1, 95% CI=-2.0 to 2.6) and hallucinations (d=-0.2, 95% CI=-6.5 to 2.7). This suggests that if an effect exists in this clinical population, similarly to the OASIS trial it is likely small. Nevertheless, insomnia is important to treat in its own right and this was the first randomised test to demonstrate that CBT is potentially highly efficacious for treating insomnia in patients experiencing persistent delusions or hallucinations.

Following completion of the BeST trial, the treatment was adapted for an acute psychiatric inpatient ward sample (Sheaves, Freeman, et al., 2018). Half of the participants had a schizophrenia spectrum diagnosis. A sleep treatment at acute crisis (STAC) intervention was delivered which comprised CBT for insomnia as the key therapeutic agent, with enhanced light/dark exposure and use of wearable devises to engage patients in their treatment plan. The treatment was offered over two weeks with an average of nine sessions, to ensure that a full treatment dose was received prior to discharge. Treatment uptake was remarkably high in this acutely unwell group (100% completion of therapy in the STAC arm). Whilst the TAU comparison group showed some natural improvement in recovery across the 12-week trial period (as expected in an inpatient sample), the STAC led to quicker and potentially fuller recovery of insomnia (d=0.9 at 2 weeks, d=0.5 at 12 weeks). A small but promising effect was observed on the duration of admission (patients receiving STAC were discharged 8.5 days earlier, d=-0.2, 95% CI=-28.0 to 11.1), and a small effect on PANSS total symptoms of schizophrenia, both favouring the STAC group (d=-0.3, 95% CI=-11.5 to 2.8), albeit with wide confidence intervals which warrant cautious interpretation.

The third randomised trial recruited patients experiencing nightmares and persecutory delusions. A brief CBT for nightmares intervention was delivered over four weeks and tested against TAU (Sheaves et al., 2019). Imagery rehearsal therapy was the key therapeutic technique, but with novel CBT for nightmares targets identified from a general population study (Rek et al., 2017). Large effect size improvements in nightmares (d=1.1) and insomnia (d=1.4) were found at week four favouring the CBT group, and a medium effect on paranoia, albeit with wide confidence intervals (d=0.6, 95% CI=-43.2 to 1.7).

Satisfaction with treatment was high (median 9 out of 10, IQR: 6.75-10) and all participants offered the CBT completed therapy.

A case series test of CBT for sleep disturbance has also shown promise for treating sleep disruption in patients at ultra-high risk of psychosis (Bradley et al., 2018). Treatment was delivered in up to eight weekly treatment sessions. Therapy was based on the BeST trial treatment protocol but with adaptations made for this largely adolescent population. These included i) matching the sleep window to the later circadian preference for adolescents ii) recruiting family and friends to support implementation of the intervention and iii) negotiating use of electronic devices at night owing to the impact of blue light and arousing stimuli on sleep. Large within-subjective effect sizes were found on insomnia (d=6.8) and medium effects on paranoia (d=0.6, 95% CI =-30.8 to 4.1) and hallucinations (d=0.3, 95% CI =-5.6 to 1.7) albeit with wide confidence intervals.

Put together, these trials indicate that CBT targeting sleep disturbance leads to large effect size improvements in sleep. Each of the therapies tested was delivered in one-to-one treatment. It is interesting to note that group CBT may have less effect on sleep (d=0.5) (Hwang, Nam, & Lee, 2019).

CBT for insomnia treatment protocols for patients experiencing psychosis

Traditionally CBT for insomnia has been delivered over four to six weekly sessions (Morin & Benca, 2012). However for people with persistent psychotic symptoms the treatment has been tested in eight sessions over 12 weeks to offer additional flexibility (BeST trial; Freeman, Waite, et al., 2015). Telephone calls and text messages in between sessions offer an opportunity to boost engagement and problem solve difficulties in implementing techniques. For patients in the acute phase of illness, on an inpatient ward the protocol was delivered in nine sessions over a two-week therapy window in order to ensure that all patients received a full dose of treatment before discharge. This intensive approach was well received by inpatients and allowed optimisation of the sleep plan each night, which was particularly important in light of the rapid change in clinical presentation which is common on wards (Sheaves, Isham, et al., 2018).

Adaptations to CBT for insomnia in light of co-occurring psychotic symptoms

Six adaptations to treatment are outlined below and in journal articles describing the trial therapy (Sheaves, Isham, et al., 2018; Waite, Myers, et al., 2015).

i) Assessment

A sleep focused assessment is carried out in the first session. The goal of this session is to assess sleep enough to inform the development of a sleep focused formulation and hence early initiation of treatment techniques. In addition to the typical insomnia assessment points, a typical 24-hour schedule is elicited, paying particular attention to the presence (or not) of circadian zeitgebers (e.g. daytime activity, light exposure and social contacts). To aid in the efficiency of the assessment, a tick list of common insomnia maintenance factors is used as a starting point (see Sheaves, Isham, et al., 2018). This is completed immediately following sleep psychoeducation to empower patients with the rationale for change strategies from the outset.

Patients are asked about their medication regime and the self-reported impact that their medications have on sleep and wakefulness. For example the sedative effects of medication negating the opportunity to stay awake until the start of the sleep window, or the 'hangover' from sedative medication triggering excessive sleep inertia. Where unusual sleep experiences are part of the clinical presentations (e.g. hypnagogic or hypnopompic hallucinations or sleep paralysis) psychoeducation about these experiences is shared to normalise such experiences.

Whilst a constellation of psychiatric symptoms are very often co-occurring, in this brief intervention they are assessed only to the extent that they impact on the sleep system (for example voices delaying sleep onset). If sleep stabilisation is the agreed priority between patient and clinician, permission is sought to delay the direct treatment of co-occurring symptoms until completion of the sleep treatment in order to

ensure a focused and therefore more effective and efficient delivery of the sleep treatment. This modular treatment approach has been piloted and is acceptable to patients (Freeman et al., 2016).

Widely used questionnaire assessments such as the insomnia severity index (Bastien, Vallières, & Morin, 2001) and the sleep condition indicator (Espie et al., 2014) are used to supplement verbal report and completed at the start of each session to monitor treatment progress. Sleep diaries are also used throughout therapy, however clinicians typically offer text or phone support to aid completion. Actigraphy has been used to assess sleep and wakefulness in patients with psychosis (e.g. Freeman et al., 2015) however the poverty of daytime activity which is common can lead to a less well-defined sleep window and hence concurrent sleep diary information is required to aid interpretation. Other commercially available wearable devices combine heart rate with accelerometer data to infer periods of sleep and wakefulness. Whilst these are typically not validated measures, they have been valued as a therapy tool for prompting discussion about sleep. Even if a watch incorrectly infers sleep (typically due to inactivity) this can be helpful assessment information. For example if a patient explains that they were awake but trying to sleep at the start of their sleep window, stimulus control strategies might be indicated.

ii) Sleep hygiene

A greater emphasis is paid to ensuring that the environment is conducive to sleep. Whilst managing distressing psychotic symptoms it is understandably common for amenities such as curtains and clean sheets to fall down the patient's priority list. Home visits are invaluable for assessing this. The clinician works alongside the patient to enable them to foster a dark, quiet and comforting environment to promote sleep. This may for example involve a practical session ordering bed sheets or setting up blinds. These foundations are important for the success of later techniques.

For patients who hear voices, an entirely silent bedroom environment can increase attentional focus on voices. For example "'*although I am not trying to concentrate on him [the voice], when someone speaks you automatically hear what they are saying [..]"* (10)' (p. 6., Waite et al., 2015). A more flexible approach to sleep hygiene is therefore adopted. Many patients find it beneficial to fall asleep with background sounds from the television or radio. Sleep timers can be set on devices to minimise the impact on subsequent sleep quality.

Given the high levels of fatigue in this population it is understandable that patients use substances such as caffeine and energy drinks to boost daytime arousal. Equally, hyperarousal driven by symptoms can trigger patients to try alternative ways to sedate themselves in preparation for sleep, by for example using alcohol or cannabis. Psychoeducation is used to promote a shared understanding of the link between the specific substance used by the patient and its effect on sleep and wakefulness. Alternative means of generating energy (e.g. activity) and decreasing arousal (e.g. relaxation) are then set as behavioural experiments to enable the patient to learn alternative means of controlling arousal and hence reduce reliance on substances.

i) Stimulus control

Standard stimulus control procedures are typically used and effective in reducing insomnia for patients with psychosis. However there are also occasions when modifications are required. Experiences of trauma (either due to life events or traumatic voice experiences) in the bedroom can lead patients to develop a fear of bed and hence sleep elsewhere. Here bed is associated with anxiety rather than sleep. A typical 15-minute rule approach can therefore inadvertently reinforce the fear by leaving the bedroom at the peak of anxiety. With this presentation, a graded exposure approach can be used prior to commencing typical stimulus control (Waite, Myers, et al., 2015). This involves generating a hierarchy, for example spending time in bed during the day until the fear has habituated and gradually working up to spending time in bed at the start of the agreed sleep window.

In inpatient settings the patient's bedroom is typically their only private space. Simple practical solutions such as introducing a bean bag to the patient's bedroom can enable the patient to spend time in their

room in the daytime, but not in their bed (Sheaves, Isham, et al., 2018). This is equally a comfortable place to spend time when winding down or following the 15-minute rule. If a patient feels unable to follow the 15-minute rule by getting out of bed, which can be the case if motivation is low (e.g. linked to depression), they can be advised to sit up in bed with the light on as an alternative. The emphasis is on creating as much distinction in the way that bed is used when awake (light on, sitting upright, engaging in wind down) versus asleep. Therefore just like traditional stimulus control it draws on the principles of associative learning.

ii) Sleep restriction

Optimising homeostatic sleep pressure across nights through sleep restriction (SR) is a core element of CBT for insomnia protocols. However during administration SR results in a temporary reduction in total sleep time and an increase in daytime somnolence (Kyle, Miller, Rogers, Siriwardena, & Espie, 2014). Given data linking SR with an increase in psychotic like experiences (Reeve et al., 2017) and concerns that such a challenging treatment technique might lead to disengagement from therapy, an adapted SR protocol is adopted which sets a consistent sleep window but without reducing the opportunity for sleep below the current sleep duration.

Rather than reducing the opportunity to sleep so that it is in line with the *current* sleep time, a regular sleep window is set which is in line with the *goal* total sleep time (Therapist asks: *"how much sleep do you need when you are at your best"*). Similar to standard SR the emphasis is on a sleep window that is feasible such that it can be kept consistently (Therapist asks: *"what is a reasonable amount of sleep to aim for every night of the week?")*. This ensures an adequate sleep pressure across nights and avoids periods of oversleep. Greater attention is paid to the timing of this sleep window such that it is matched to the self-reported circadian preference of the patient (e.g. a 'morning person' would set an earlier sleep window than an 'evening person') in order to increase the chances of sleep. In adolescent samples psycho-education about matching the sleep window with the typical phase delay experienced by adolescents is important for both the patient and wider family system (Bradley et al., 2018). Where social demands such as work and school put constraints on the flexibility of the sleep window, this is balanced with the circadian preference for sleep timing and a compromise is sought.

Inactivity is common, can maintain fatigue and sleep inertia and provide an opportunity for naps which reduces sleep pressure. Hence a 24 hour approach is adopted to optimise sleep pressure, which includes planning in daytime activity (Sheaves, Isham, et al., 2018; Waite, Myers, et al., 2015). Behavioural experiments can be used to test the impact of activity and exposure to natural light on energy. Physical activity can be particularly powerful for these tests, and is emphasised given its demonstrated effects on improving sleep quality and sleep onset latency (Lowe et al., 2019). Hence a clinician might take a brisk walk outdoors with the patient during a therapy session and elicit energy ratings before and after. The key learning for the patient is that activity (rather than napping) is most effective at overcoming the effects of fatigue.

Sleep is a strategy used by some patients to provide respite from distressing voices. Whilst this is understandable, this daytime napping can result in insomnia symptoms owing to reduced sleep pressure. In turn, the insomnia can trigger a worsening of voices either at night (whilst trying to sleep) or the subsequent day. This vicious cycle can be formulated with the patient to provide the rationale for change strategies. Brief techniques which enable the individual to manage voices can subsequently be used. For example sharing patients accounts to decatastrophise the experience and reducing triggers for voices (e.g inactivity, low mood, social isolation) (Waite, Myers, et al., 2015).

iii) Reducing hyperarousal: relaxation, wind down and worry reduction

For patients with psychosis pre-sleep hyperarousal can be driven by voice hearing, feeling paranoid or co-morbid manic symptoms (when present). Mania is associated with elevated mood, but also sometimes anger and irritability which are equally unhelpful for sleep.

Relaxation targets this pre-sleep hyperarousal. Audios are often particularly valued by voice hearers for offering not only a means of relaxing but also something to focus attention on other than voices. Choosing audios which the patient particularly connects with (and hence are more likely to get immersed in) and audios which avoid long periods of silence can therefore be particularly helpful for voice hearers.

Two thirds of patients with psychosis experience clinical levels of worry commensurate with those observed in generalised anxiety disorder (Freeman et al., 2019). A worry thinking style fuels hyperarousal which triggers insomnia. The content of patients' worries include those that are common in the general population (finances, work and relationships) but also worries related to paranoia, voice content and fear of relapse. Cognitive behavioural therapy for worry is effective for reducing worry in patients with persecutory delusions and also improves paranoia (Freeman, Dunn, et al., 2015). Patients are therefore encouraged to limit worry, including paranoia related worries, to a period in the early evening and subsequently postpone worries that pop up in bed.

For patients experiencing co-morbid mania, which is more common on an inpatient environment, relaxation activities can feel so incongruent with manic hyperarousal that they are perceived as boring or irritating. The challenge here is finding activities that the patient is willing to engage in. A more graded approach is therefore adopted, starting first with an activity that is less arousing than their typical routine, but they are still willing to engage in (e.g. playing chess in a quiet room rather than doing sit ups) and gradually working towards relaxation exercises (Sheaves, Isham, et al., 2018). A dark evening environment is also emphasised during the wind down routine owing to the increased sensitivity to light which has been hypothesised to play a role in mania.

Summary of insomnia treatment for patients with psychosis

The treatment of insomnia for patients with psychosis targets the same mechanisms as traditional CBTi. However adaptations are made to increase the accessibility and effectiveness of techniques. Overall the treatment is a popular approach, indicated by the high treatment completion rates. Sleep is a normalising, safe and inclusive topic to begin therapy. This is particularly important for people with psychosis who experience stigma as a result of diagnoses and whose symptoms can result in difficulties trusting a therapist.

Treatment of nightmares

The treatment protocol for nightmares is delivered over a four-week therapy window in one to one sessions (Sheaves et al., 2019). The primary treatment technique is imagery rehearsal training, which is the recommended treatment for nightmares in clinical guidelines (Aurora et al., 2010; Hansen, Höfling, Kröner-Borowik, Stangier, & Steil, 2013). This is supplemented with novel potential causal factors for nightmares identified in a large non-clinical study (Rek et al., 2017). The key elements of treatment are outlined below and principal treatment targets are outlined in the maintenance model in figure 2.

Figure 2 about here

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i) Targeting beliefs about nightmares

Nightmares often draw on daytime experiences, but the narrative may be bizarre, distorted or involve novel characters. They are often described as seeming very real, as if they are actually happening, and are typically acutely distressing. This combination of factors triggers the patient to make sense of them.

Common appraisals include those related to the self *"they mean I'm a bad person"*, the future *"they are a sign of future events"*, and others *"it's my persecutor implanting them into my mind"*. These appraisals exacerbate distress and keep the content of nightmares in attentional focus across the day and are therefore more likely to trigger a nightmare the subsequent night.

Psychoeducation about common causes of nightmares (e.g. daytime worries, traumatic experiences, period of stress) and facts about REM sleep are shared to gently set up an alternative explanation for nightmares. For example a hypnogram which demonstrates the sleep stages and regular cycles of REM sleep throughout the night can be helpful in explaining why nightmares occur at the same time each night. Information is shared about brain functioning during REM sleep, for example:

Therapist: *"Dreams often contain elements of our waking experience. But the clever parts of our brain, which organise our memories and experiences into logical order, are less active during dreaming sleep. And the areas of the brain which process vision, sound, movement and emotion are very active, just as they are when we are awake. They can therefore feel very real (as if they are actually happening) and very scary, but aren't always logical or well organised."*

This normalises the bizarre yet perceptually real and emotionally intense experience and invites the patient to shift focus away from nightmare content and what it might mean. Instead patients are encouraged to plan in activities which help them to 'bounce back' from a nightmare, using the rationale that *"a positive day more often leads to a positive night"*.

ii) Imagery rehearsal

This treatment protocol for imagery rehearsal is considered a cognitive therapy technique, which is embellished in sensory rich detail through guided imagery. It therefore differs to some degree from treatment protocols which invite participants to change the ending of the nightmare any way they wish, or from those which emphasise the importance of exposure to nightmare related content. The appraisal is considered key and hence careful assessment of this is prioritised before the imagery. Assessing cognitions related to very distressing experiences is a sensitive process, a careful rationale is therefore shared with patients:

Therapist: *"Before we can start to work on improving the nightmares we need to understand what it is that makes them so upsetting. This means it's helpful for me to ask you detailed questions about them. It's a bit like if you had a sore leg – there may be many reasons why the leg feels sore (it might be broken or a strained muscle for example). We need to know which it is to know which treatment is needed. It's the same with nightmares, we need to know why they are upsetting so we can work out the best way to overcome them. But if any of my questions feel too difficult, you don't have to answer them. You're in control."*

A bullet point account of the nightmare narrative is then established. The intention is to assess content enough to inform treatment, but not so much as to re-expose patients to distressing sensory detail. The most distressing part is identified, and the associated appraisal is elicited *("what makes that the worst part")*. In some cases, where the link between the appraisal and associated distress is unclear to the therapist, he/she uses the downward arrowing technique (e.g. *"and if that were true, what would that mean about you / what would be bad about that?")* to elicit the appraisal driving distress, but only if the patient can tolerate this level of probing in the first session.

The change of ending of the nightmare restructures the meaning that is driving distress. For example, if the appraisal is *"I can't do anything to stop them hurting me"* the rescripted ending of the nightmare aims to increase *control* over the situation to stop them being harmed. If the appraisal is *"I'm evil"* the new ending includes evidence of being a *good person*. This might require some light touch cognitive restructuring prior to considering the imagery content. When the new appraisal is clear, the patient chooses the story of events to link the original nightmare contents to the change of ending. This might draw on real events or memories but can also include imaginative characters or fantastical powers (e.g. shrinking an attacker to dust). This change of ending is inserted before the worst moment in the nightmare.

Once the new ending is planned and the patient is comfortable with their choice of narrative, guided imagery is used to elaborate the sensory detail. The patient is encouraged to describe the new narrative with their eyes closed, in the present tense and with lots of sensory detail. The therapist first models this way of describing to the patient (*e.g. "I'm walking down the street, I can see the dark rain clouds, hear the thunder around me and the air feels damp"*). Some patients require little in the way of prompting when describing their rescripted nightmare (particularly after careful planning) and in this case the therapist avoids interrupting the immersive experience. However, if the patient isn't immersed in the imagery, the therapist can summarise the patient's descriptions and prompt for further details (e.g. *"so you're in the castle and you're feeling scared… what can you see around you?"*). Distress is monitored throughout by paying attention to body language, voice tone or simply asking (*"how are you feeling?"*). Where distress is elevated, the patient is prompted onto the next event in the rescript (*"ok, and what do you do next?"*). At the end of the imagery the patient is encouraged to savour the emotion and associated physiological feeling of the new ending, before opening their eyes and returning to the room. Feedback is immediately sought and if the change of ending neutralises the initial negative affect home practice is recommended once per day.

i) Pre-sleep voices

Nightmares can be linked to the content of threatening voices and hence reducing the time spent listening to voices can be helpful. Patients are encouraged to find something calming to listen to before sleep, for example relaxation audios or a bespoke calming music playlist. Voices can also be extremely tiring to manage and some patients therefore head to bed early (before their optimal circadian timing for sleep) in an effort to boost energy. However if they are spending time in bed listening to voices and unable to sleep, pushing the bed time later and planning out some enjoyable evening activities can reduce the time spent listening to voices.

ii) Factors which increase REM sleep

The most common factor which increases the proportion of REM sleep is sleep duration. Longer total sleep time has been linked to an increased occurrence of nightmares in the general population (Rek et al., 2017), likely owing to an increase in REM sleep the longer through the sleep period.

Whilst nightmares interrupt sleep and cause a fear of sleep, and therefore most patients report insomnia, it is common for patients to catch up on lost hours either by sleeping in the morning or napping. This may not occur every day, hence a sleep diary can map the link between nightmares and total sleep time (adding up day and night time sleep) across the week. Optimising the sleep window to avoid over-sleeping in any 24 hour period, can thus reduce the opportunity for nightmares to occur.

Alcohol use is sometimes used as a strategy to encourage sleep onset. However whilst it suppresses REM sleep early in the sleep period it can trigger nightmares through a REM rebound later in the sleep period. This can be linked to a worsening of nightmares. This psychoeducation is shared with the patient and alternative wind down strategies can be introduced to reduce the reliance on alcohol to initiate sleep.

iii) Pre-sleep worry

Patients describe worrying about the content of nightmares, whether they will have a nightmare that night, as well as other day to day worries (unrelated to nightmares) prior to sleep. For example, a participant in the nightmare intervention study described:

> *"I would be thinking about it all through the day. So, I would be sort of replaying it in my head, I would see the images in my head [..] it's such a worry and then because of what I used to sort of obviously dream about um it was like a constant worry all the time because of the content of it"* (Nm20)

Worry kept the content of the nightmares in mind throughout the day and then at night time this participant described: *"I was kind of dreading if I had one because of what would happen after"*.

Where worry forms part of the nightmare maintenance formulation as it did so clearly for Nm20, it is briefly targeted. Worry strategies (outlined in the CBT for insomnia section) are used. The aim is to limit worry to one brief period in the early evening and then postpone worry at other times, but particularly the pre-sleep period.

Nightmare related worry can also be targeted by increasing the patient's perceived capacity to cope with nightmares. A detailed plan for managing nightmares is devised with the patient. Techniques for managing acute distress are planned in detail. These might include for example: turning the light on to get visual cues of safety, breathing techniques to activate the parasympathetic nervous system, grounding techniques (e.g. keeping a comforting smell by the bed) and writing a *'compassionate message from your daytime self'*. This compassionate message is written by patients and often includes a kind message, reminders of safety and suggestions of how to return to calm, e.g: *"it's so scary but it is just a nightmare, you're safe really. Do your breathing, head to the window to feel the fresh air on your skin and if you can't sleep head to the lounge and cuddle the cats until you feel sleepy again"*

The 15 minute rule is also used to interrupt rumination related to nightmares, to provide an opportunity to decrease arousal and hence improve the chances of returning to good quality (and hence nightmare free) sleep.

Summary of nightmare treatment for patients with psychosis

Imagery rehearsal is the core technique for treating nightmares in patients with psychosis, but other strategies are also chosen in this brief four week intervention on the basis of the maintenance formulation. There is overlap in some treatment techniques between the nightmare and insomnia protocols (e.g. worry reduction, stabilising the sleep window) owing to the shared mechanisms driving these sleep disorders. However the rationale shared with patients differs and is bespoke to their sleep disorder presentation.

Treatment of sleep apnoea

A case series test of continuous positive airway pressure (CPAP) has been completed with eight patients with diagnosed sleep apnoea and schizophrenia (Myles et al., 2019). All were taking clozapine medication, one of the most obesogenic anti-psychotics. The mean BMI was 45 (SD 9.6), indicative of extreme obesity and all had hypertension at baseline. Two participants did not complete CPAP titration due to discomfort and nausea. Six completed titration, and five provided assessment data which indicated descriptive decreases in the apnoea-hypopnoea index, increases in REM and slow wave sleep and decreases in weight (mean weight loss 7.3 ± 9 k at 6 months). Three no longer had diagnosable hypertension following CPAP. Four out of the six participants had adequate adherence (>4 hours per night). Insomnia symptoms improved but not psychiatric symptoms nor overall self-reported sleep quality. Overall, these results indicate promising physical health improvement which warrant replication in a larger controlled trial. However it is also notable from the reported data that the majority of participants who were offered at-home polysomnography assessment for apnoea declined the opportunity. In our experience people with comorbid psychotic symptoms are typically anxious about completing in depth monitoring, or meeting new health professionals. Support from psychiatric services to enable patients to attend respiratory sleep medicine appointments as well as highly transparent descriptions of technology used for health monitoring might increase the accessibility of such appointments for patients experiencing psychosis.

Conclusions:

Sleep disorders are highly prevalent in patients experiencing delusions and/or hallucinations. Almost all of the available evidence, from a range of methodologies suggests that insomnia is a contributory causal factor in the occurrence of hallucinations and paranoia. Initial evidence suggests that nightmares are an early vulnerability factor for the development of later psychotic experiences and might maintain paranoia

by playing out fears in sensory detail. Yet nightmares are rarely discussed in clinic and therefore almost never treated.

Data indicate that there is no reason to expect that CBT for sleep problems does not work as well with patients with schizophrenia as has been found in other patient groups. Indeed psychological sleep treatments lead to large effect size improvements in insomnia and nightmares. Even at the acute phase of illness sleep treatment is accessible and achieves good effects. CBT for insomnia leads to small but significant improvements in paranoid thoughts and hallucinatory experiences, whilst CBT for nightmares has a potentially greater effect on paranoia than hallucinations. One of the most striking results from treatment trials is the very high uptake, completion and satisfaction with therapy. Sleep is a normalising and safe topic for treatment and this is particularly important in a group who experience the effects of stigma and a lack of understanding from others regarding psychotic symptoms. Treating sleep prior to directly targeting psychotic symptoms also brings the benefit of building resilience, hope and engagement for later work. Whilst many patients are still not receiving sleep interventions in clinical practice, there is a rapidly growing interest in sleep from clinicians and an eagerness to receive training in implementing sleep techniques.

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Tables:

Table 1. Definition of terms.

Term	Definition
Cognitive disorganisation	Otherwise termed thought disorder. This is typically observed through speech. The patient may switch from one topic to another (tangential) or appear unrelated to the topic of conversation. In its severest form speech may be incomprehensible (American Psychiatric Association, 2013).
Grandiose delusion	Unfounded beliefs that one has special powers, mission, wealth or identity (Isham et al., 2019).
Hallucination	"Perception like experiences that occur without an external stimulus. They are vivid and clear with the full force and impact of normal perceptions" (American Psychiatric Association, 2013).
Negative symptoms	Describes a range of symptoms including reduced expression of emotion (blunted affect), reduced quantity of speech (alogia) diminished ability to experience pleasant emotions (anhedonia), a reduction in social initiative (asociality) and reduced initiation of goal directed activity (avolition) (Marder & Galderisi, 2017).
Paranoia	An unfounded thought that others are intending to cause harm (Freeman, 2016). Paranoia is exponentially distributed in the general population: many people endorse a few paranoid thoughts and a few people have many (Bebbington et al., 2013).
Persecutory delusion	A paranoid belief that is held with a high degree of certainty (Freeman, 2016). They are one of the most common symptoms of schizophrenia.
Psychosis	Characterised by the presence of delusions or hallucinations.
Schizophrenia spectrum disorders	Include schizophrenia and other psychotic disorders. They are defined by the presence of delusions, hallucinations, cognitive disorganisation, motor abnormalities (e.g. catatonia), and negative symptoms (American Psychiatric Association, 2013).
Schizophrenia	A disorder characterised by at least one out of: delusions, hallucinations or disorganised speech (American Psychiatric Association, 2013).
Voices	A hallucination of spoken word (individual words or full sentences) in the absence of a person generating the sound. They occur in the majority of patients with schizophrenia but also in diagnoses such as bipolar affective disorder, severe depression and post-traumatic stress disorder.

Table 2. The prevalence and treatment of sleep disorders in early psychosis (N=60), adapted from (Reeve, Sheaves, et al., 2018, p. 5)

	Diagnosis	Diagnosis	Treatment received, n (%)		
Sleep disorder	**%**	**n**	**None**	**Recommended**	**Others**
Insomnia	50.0	30	14 (46.7)	6 (20)	10 (33.3)
Nightmare Disorder	48.3	29	20 (71.4)	2 (7.1)	6 (21.4)
Sleep-related hallucinations	41.7	25	n/a	n/a	n/a
Excessive daytime sleepiness	23.3	14	11 (78.6)	-	3 (21.4)
Restless leg syndrome	23.3	14	13 (92.9)	-	1 (7.14)
Periodic limb movement syndrome	20.0	12	n/a	n/a	n/a
Bruxism (teeth grinding)	18.3	11	7 (63.6)	4 (36.4)	-
Sleep paralysis	15.0	9	n/a	n/a	n/a
Night terrors	8.3	5	4 (80.0)	-	1 (20)
Circadian Disruption	8.3	5	5 (100)	-	-
Sleep walking	5.0	3	3 (100)	-	-
REM sleep behaviour disorder	3.3	2	2 (100)	-	-
Enuresis	1.7	1	-	1 (100)	-
Any sleep disorder	80	48			
Total	**-**	**160**	**79 (69.9)**	**13 (11.5)**	**21 (18.6)**

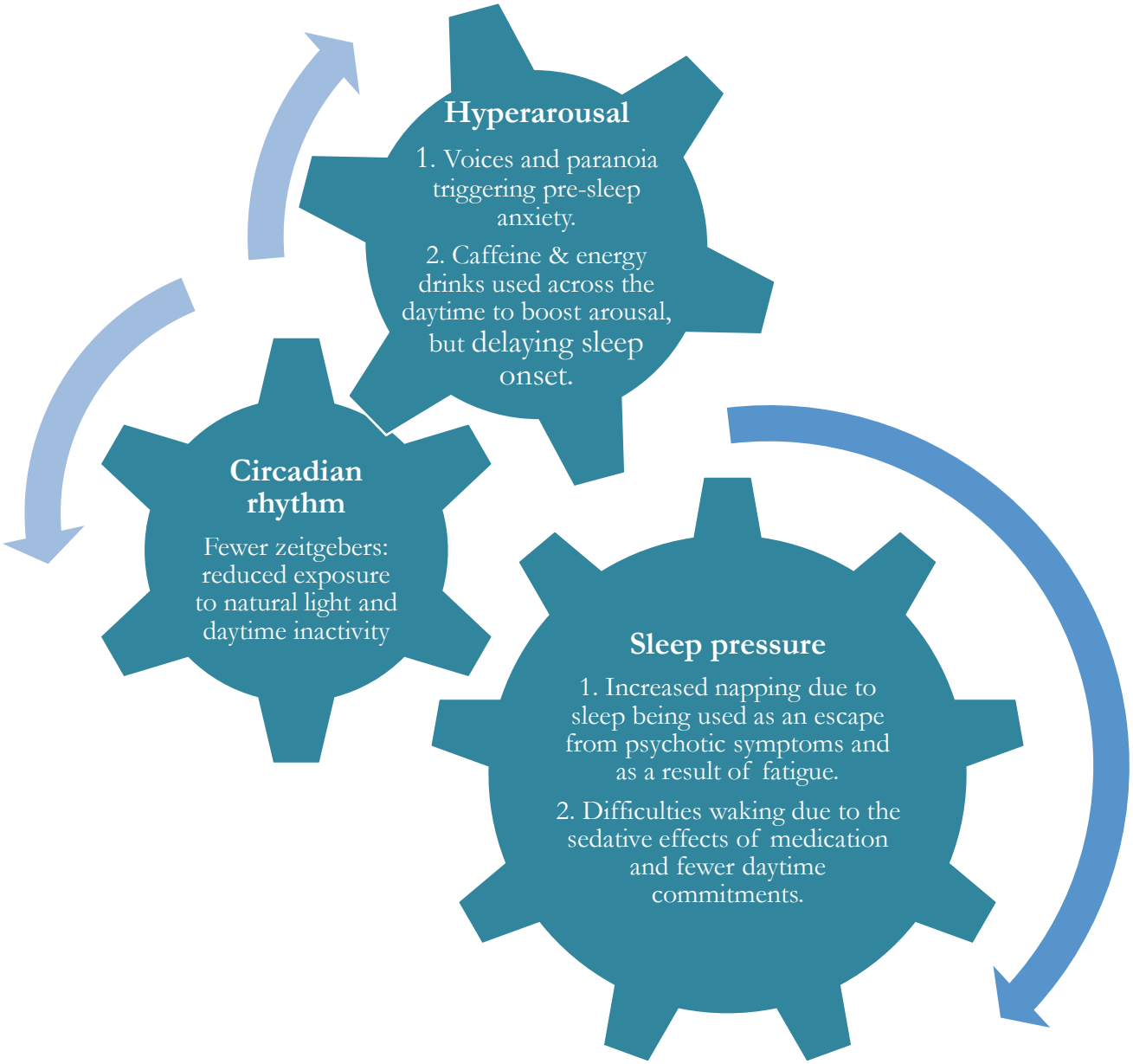

Figure 1: The impact of psychotic symptoms and lifestyle factors on the sleep system.

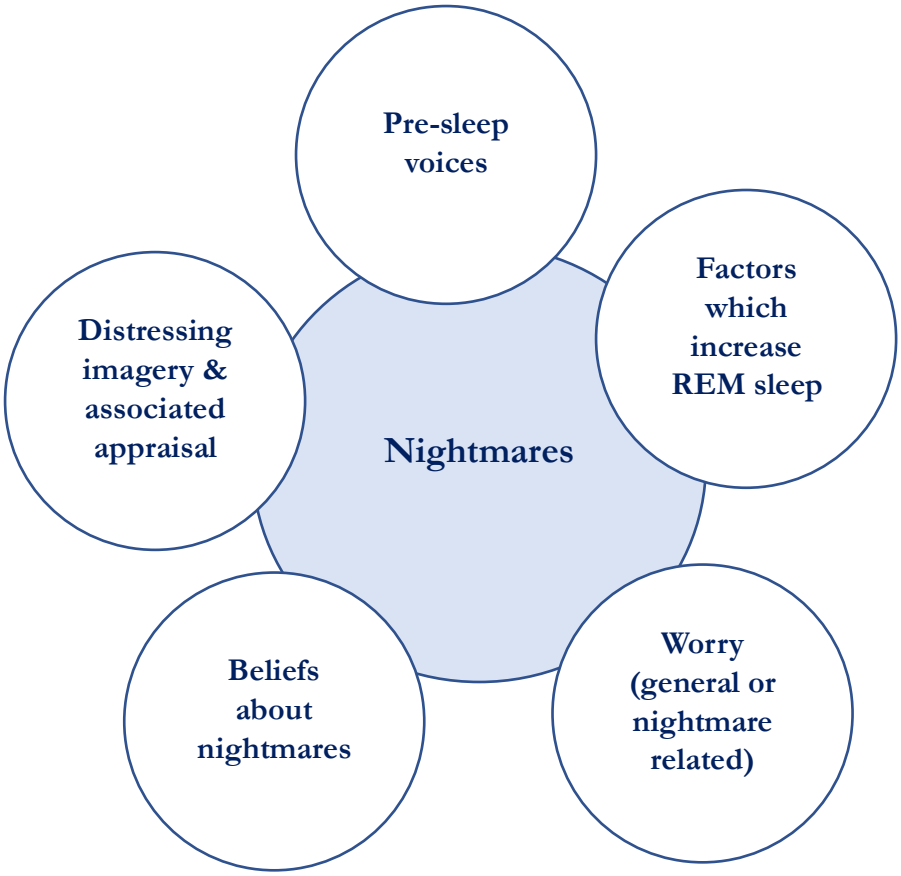

Figure 2: A maintenance model of nightmares for patients experiencing psychosis.