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Are sleep disturbances causally linked to the presence and severity of psychotic-like, dissociative and hypomanic experiences in non-clinical populations? A Systematic Review

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Highlights:

- Sleep disturbances are associated with all experiences reviewed.
- Paucity of research examining circadian rhythms in psychotic-like experiences.
- Limited research addressing causal ordering between sleep and experiences studied.

Abstract

The present review aimed to 1) identify what sleep disturbances co-occur alongside psychotic-like, dissociative and hypomanic experiences; 2) assess the strength of potential associations between the severity of sleep disturbances and of the experiences studied; and 3) appraise evidence for a causal link. MedLine and PsycInfo were searched and 44 studies were deemed eligible.

Results showed that insomnia was associated with all individual psychotic-like, dissociative and hypomanic experiences reviewed (effect size range: small-to-large). Parasomnias were associated with all psychotic-like experiences; however, there was evidence of variation in magnitude between individual experiences. An eveningness chronotype was associated with dissociative and hypomanic experiences, and circadian dysrhythmia was found alongside hypomania but not the other experiences reviewed.

Finally, experimental sleep manipulation studies revealed a potential causal link between sleep loss and psychotic-like and dissociative experiences with a large effect size. However, this was not the case for experiences such as paranoia. Future research, using experimental manipulations of sleep to address putative mechanisms, will enable questions of causality to be answered with more confidence.

Keywords: psychotic-like, sleep, review, causal

Introduction

Sleep is critical for healthy neural, social, emotional and cognitive functioning (Cappuccio et al., 2010; Kripke et al., 2002). Sleep disturbances are present alongside numerous mental disorders such as schizophrenia and bipolar disorder (Baglioni et al., 2016) and there is considerable overlap in the types of sleep disturbances experienced across disorders (Harvey, 2011). Disturbed sleep may refer to the presence of insomnia or parasomnias (e.g. sleepwalking, nightmares or sleep paralysis) and these may act as transdiagnostic markers of, and risk factor for, psychiatric illness (Harvey, 2011; Wulff et al., 2012). There is mounting evidence to suggest that sleep disturbances even occur prior to the

development of a mental disorder such as psychosis, dissociation or mania (Reeve et al., 2015; Van der Kloet et al., 2013; Harvey et al., 2011).

Psychotic experiences occur along a continuum from non-clinical to clinical, and are not uncommon within healthy populations (Yung et al., 2009; Dolphin, 2015). Psychotic-like and psychosis experiences share similar genetic (Kelleher & Cannon, 2010), social (Shakoor et al., 2016), and environmental risk factors (Zavos et al., 2014). In addition, the presence of individual psychotic-like experiences, such as hallucinations or paranoia, is a risk factor for the future development of a psychotic disorder (Van Os et al., 2009). For this review, psychotic-like experiences are referred to as attenuated psychosis symptoms (e.g., hallucinations, delusions and paranoia) which occur in the absence of a reported illness (Meehl et al., 1962; Chapman & Chapman, 1980; Yung et al., 2009).

Sheaves and colleagues (2016) explored the link between insomnia, chronotype, and nightmares with the risk for psychopathology in a non-clinical student population. They found that by grouping non-clinical populations into low, medium and high risk for psychopathology, sleep disturbances increased in a linear fashion from the low to high risk populations. When examined at the symptom specific level, the severity of psychotic-like experiences such as hallucinations and paranoia, and hypomania, were found to significantly correlate with insomnia and nightmare frequency.

Longitudinal evidence also suggests a role for insomnia in predicting future experiences of hallucinations in non-clinical samples (Sheaves et al., 2016b). This longitudinal effect of baseline insomnia on follow-up hallucinations remained significant, although reduced, after controlling for the presence of depression, anxiety and paranoia. Therefore, insomnia may have some predictive value for subsequent psychotic-like experiences in non-clinical and clinical samples (Mulligan et al., 2016).

Within studies examining hypomania in non-clinical samples, there is evidence of increased variability in typical sleep measures, as assessed by actigraphy, rather than an overall reduction in total sleep time (Ankers & Jones, 2009; Rock et al., 2014). Non-clinical hypomanic experiences are defined as attenuated manic experiences in the absence of a reported clinical diagnosis (Depue et al., 1981). Non-clinical individuals with hypomanic experiences has also been shown to be at a

heightened risk for developing mood disorders such as bipolar disorder (Blechert & Meyer, 2005) and hypomanic experiences correlate with psychotic-like experiences in non-clinical samples (Hosang et al., 2017).

Despite psychotic and mood disorders being divided into distinct categories (American Psychological Association, 2013), periods of psychosis are characteristic of a manic or depressive episode in those with a diagnosis of bipolar disorder (Laursen et al., 2009). Differentiation between these patients clinically can be difficult (Maier et al., 2006) and the creation of new, controversial, diagnostic categories such as schizoaffective disorder has only marginally alleviated this uncertainty and overlap (Cardno & Owen, 2014). Moreover, evidence from gene linkage, neural, and cognitive studies supports a diagnostic and biological overlap between these two (Forstner et al., 2017; Moller, 2003; Murray et al., 2004).

A similar argument has been made for a relationship between a diagnosis of a dissociative disorder (DD) and schizophrenia spectrum disorders (SSD). Dissociation is defined in the literature as 'lack of normal integration of thoughts, feelings and experiences into the stream of consciousness and memory' (Bernstein & Putnam, 1986). There has been considerable debate over whether psychosis and dissociation are distinct constructs (Luhmann, 2017); however, evidence supports an overlap between these experiences in clinical (Moskowitz & Corstens, 2008; Renard et al., 2017) and non-clinical samples (Alderson-Day et al., 2014; Cole et al., 2016; Moskowitz et al., 2005; Humpston et al., 2016). The overlap in symptoms and aetiology of psychotic-like, dissociative, and hypomanic experiences highlights the importance of understanding how sleep disturbances are related across all of these experiences.

The study of non-clinical populations removes the uncertain impact of medication and institutionalisation effects. This is an important step to understand the psychological and neural mechanisms by which disturbed sleep might influence vulnerability to psychotic experiences. It is therefore useful to understand the link between sleep and psychosis in the absence of antipsychotic medication where their influence is uncertain and complex (Monti et al., 2016). An understanding of

the type and effect size of the association between sleep disturbances and psychotic-like, dissociative, and hypomanic experiences in non-clinical samples is currently missing.

There is also growing interest in the causal association between sleep disturbances and psychopathology, and this is evidenced by an increase in trials aimed at treating sleep to improve the symptoms of psychosis, psychotic-like experiences and mania (Freeman et al., 2017; Kaplan & Harvey, 2013). However, the causal nature of sleep disturbances and these experiences are not currently known. This is despite multiple plausible theoretical routes through which sleep disturbances may exacerbate or induce psychotic-like, dissociative, and hypomanic experiences (Harvey et al., 2008; Yates, 2016; Pociavsek & Rowland, 2017). Sleep disturbances have been shown to negatively impact emotion regulation (Beattie et al., 2015), executive functioning (Tucker et al., 2010), increase threat anticipation at a behavioural (Kyle et al., 2014) and cortical level (Yoo et al., 2007; Baranger et al., 2017), and increase functional levels of dopamine (Yates, 2016). Furthermore, there is also considerable overlap in brain regions negatively influenced by experimental sleep deprivation and those shown to be altered in psychotic-like (Yates, 2016; Pociavsek & Rowland, 2017) and hypomanic (McKenna & Eyler, 2012) experiences. This suggests that sleep disturbances may mimic some of the cortical changes associated with psychosis, dissociation, and hypomania.

It is not currently known whether the relationship between sleep disturbance and these experiences is bi-directional but recent studies hint at this (Sheaves et al., 2016). However, the focus on whether sleep disturbances are causally linked to psychopathology raises the possibility of intervening before a diagnosis is received or symptoms become distressing.

Present Review

Previous reviews have identified the presence of specific sleep disturbances in first-episode psychosis (Davies et al., 2016) or have focused on specific sleep methodologies (Baglioni et al., 2016; Chan et al., 2016) but not non-clinical populations. One previous systematic review has examined a symptom specific approach to understanding the association between sleep and psychosis experiences (Reeve et al., 2015). The aim of this review is to systematically synthesise research into individual psychotic-

like, dissociative, and hypomanic experiences specifically within non-clinical populations and their association with sleep. Previous reviews have not considered schizotypy, dissociative or hypomanic experiences and have sparingly used effect sizes to support their conclusions (Reeve et al., 2015). Therefore, we have estimated effect sizes pertaining to the associations between specific sleep disturbances and non-clinical experience measures to appraise the magnitude of these associations in the available literature.

The principle questions posed by this review are: (1) What are the types of sleep disturbance (e.g., presence of insomnia, sleep stage disturbances, and nightmares) associated with individual psychotic-like experiences (paranoia, delusions, hallucinations, negative schizotypy, and positive schizotypy), dissociation and hypomania in non-clinical populations? (2) Is the magnitude of sleep disturbances associated with the magnitude of psychotic-like, dissociative, and hypomanic experiences in non-clinical populations? (3) Is there evidence of a causal link between sleep disturbance with individual psychotic-like experiences, dissociation, and hypomania in non-clinical populations?

Method

This systematic review was carried out according to the PRISMA guidelines (Liberati et al., 2009). The search was carried out on MedLine and PsycInfo using search terms informed by those used in previous reviews which have explored psychotic-like experiences, dissociation or hypomania and / or sleep (Davies et al., 2016; Mulligan et al., 2016; Baglioni et al., 2014). The search terms used were: (polysomnogra* OR circadian OR actigraphy OR actigrap* OR actimet* OR actograp* OR actomet* OR accelerometer OR sleep OR “sleep spindle” OR insomnia OR hypersomnia OR “sleep recordings” OR “sleep architecture” OR insomnia or hypersomnia or sleep diary OR subjective sleep*) and (hallucination* OR delusion* OR schizotyp* OR psychosis risk OR magical ideation OR anhedonia OR psychotic OR psychosis proneness OR paranoia OR bipolar risk OR hypomania OR hypomanic personality OR persecutory* OR grandiose* OR dissociation OR unusual experiences OR cognitive disorganisation). Searches were initially carried out in September 2016 and updated in August 2017. The results of the search were then screened by the lead author, JB, and extracted for

analysis based on pre-defined points of interest. Reference lists of the full text studies and their citations were hand-searched, and previous reviews were also searched for additional eligible studies.

Study eligibility was determined based on these criteria: (1) All original research articles written in English; (2) Conducted on human participants without a reported mental disorder; (3) Included an objective or subjective measure of sleep disturbance or circadian functioning (e.g., actigraphy, polysomnography or questionnaire); (4) Included a validated measure of psychotic-like, dissociative, hypomanic experiences; (5) Tested an association between sleep disturbance and individual psychotic-like, dissociative, or hypomanic experiences using appropriate statistical analyses; (6) The studies employed the following designs: cross-sectional, case-control, cohort, longitudinal, and experimental/interventional.

Exclusion criteria included: (1) The study was reported as a review article, case study, conference abstract, thesis, letter, book chapter, a non-peer-reviewed manuscript, or case study; (2) The study examined participants with a reported psychiatric diagnosis; (3) The study only examined hypnopompic or hypnogogic hallucinations.

Quality Assessment

To assess the quality of included studies, an adapted version of the Newcastle Ottawa Scale (Wells et al., 2000) was used. An adapted version was created to attempt to limit the poor specificity and validity of previous quality assessment measures. This adapted quality assessment identifies eight criteria to assess study quality overall: two on the design (e.g., clarity of research aim and hypothesis; validation of measures in non-clinical populations); three on the selection of the sample (e.g., whether sufficient detail for sampling method is provided; whether the sample is representative of the target population; and whether there is a justification for sample size such as a power calculation); one on the procedure; and two on the outcomes (e.g., was appropriate statistical analysis used and is there any missing information; whether the study controlled for confounding variables).

All criteria for each study was rated as either “weak”, “moderate”, or “strong” depending on scores across the eight criteria. This rating was dependent on the extent to which the study met each criterion (see Appendix C).

An overall study rating of “weak” was given when more than one “weak” score was given across the eight criteria; “moderate” when only one “weak” rating was given; and “strong” when no “weak” ratings were given. All studies were quality assessed by JB, and a proportion of these ($k = 18$) were assessed by an independent reviewer (IB). The intra-class correlation (ICC) indicated a high degree of reliability between raters ($ICC=.90$)

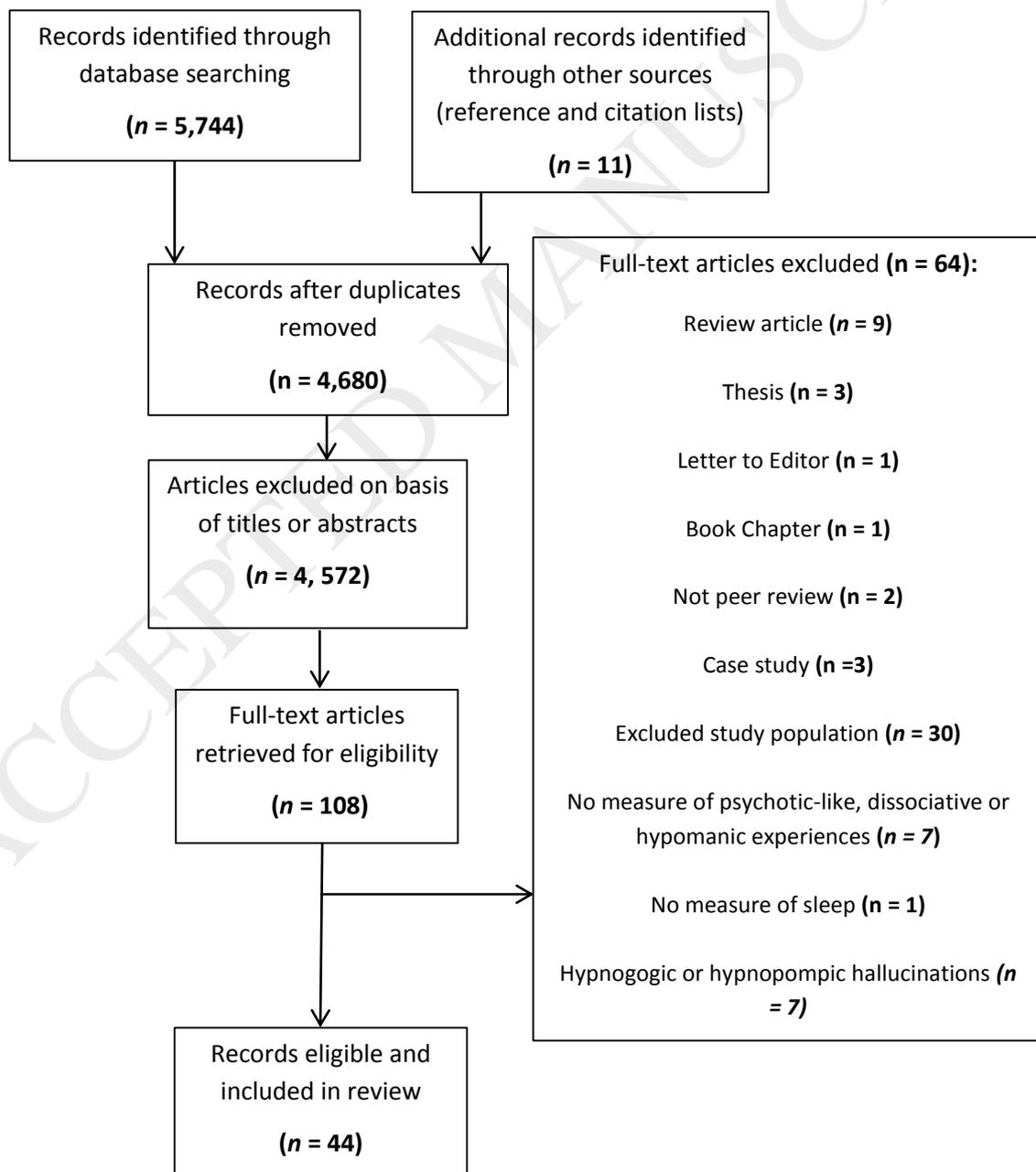


Figure 1. PRISMA flow chart of how studies were selected.

Computation of Effect Sizes

Effect sizes were computed using ‘Comprehensive Meta-Analysis V2’ (Borenstein et al. 2015), and are used for comparison of the magnitude of effects detected across studies. Pearson’s r was chosen as the effect size of choice as the majority of studies used a correlational design and analysis when examining the relationship between sleep and psychotic-like, dissociative and hypomanic experiences (see Table 1). All effect sizes where data were available for calculation are included. Effect sizes are referred to as either small ($r=.1$), medium ($r=.3$) or large ($r=.5$) (Cohen, 1988).

Results

Study Characteristics

Overall, 15 studies assessed paranoia; 13 assessed hallucinations; 5 assessed delusions; 8 assessed positive schizotypy; 8 assessed negative schizotypy; 14 assessed dissociation; and 13 assessed hypomania proneness. Studies took place in a variety of sites worldwide: 1 in Canada, 13 in the UK, 1 in Japan, 5 in the Netherlands, 1 in France, 8 in America, 1 in South Korea, 2 in Switzerland, 7 in Germany, 2 in Israel, 1 in Iran, 1 in Turkey, and 3 were International. Of the included studies: 38 employed a cross-sectional design, 12 employed a longitudinal design, 9 employed a sleep manipulation design and 1 study utilised a sleep intervention. See Appendix A, in supplementary material, for a full summary of included studies.

Quality Assessment

A full table of quality assessments can be found in Appendix B. The main reason for why studies were not rated as 'strong' was due to the absence of an a priori power calculation and poor control for known confounding variables.

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Table 1. Effect Sizes (Range) for associations between individual psychotic-like, dissociative, and hypomanic experiences and sleep variables

Psychotic-like, Dissociative, or Hypomanic Experience (Non-clinical)	Insomnia	Parasomnia (e.g. sleep paralysis)	Sleep Deprivation	Sleep Intervention	Nightmare Frequency	Nightmare Distress	Chronotype	Unusual Sleep Experiences (e.g. non-REM parasomnias)	Sleep Spindle Density	Circadian Rhythm Disturbances
Paranoia	$r=.04-.41$ (k=7)	$r=.13$ (k=1)	$r=-.12-.26$ (*k=5)	$r=.07$ (k=1)	$r=.20$ (k=1)	$r=.21-.52$ (k=2)	$r=.06$ (k=1)	-	-	-
Delusions	$r=.23-.28$ (k=5)	-	$r=-.26-.01$ (*k=4)	-	-	-	-	-	-	-
Hallucinations	$r=.14-.37$ (k=6)	-	$r=.19-.52$ (*k=6)	$r=0.06$ (k=1)	$r=.20$ (k=1)	$r=.21-.49$ (k=2)	$r=.03$ (k=1)	-	-	-
Positive Schizotypy	$r=.24$ (k=1)	-	-	-	$r=.13-.40$ (k=3)	$r=.39-.51$ (k=2)	-	$r=.33-.45$ (k=2)	$r=-.64$ (k=1)	-
Negative Schizotypy	$r=.09-.17$ (k=2)	-	$r=.13-.53$ (k=3)	-	$r=-.08$ (k=2)	$r=.38$ (k=1)	-	-	-	-
Dissociation	$r=.39-.55$ (k=1)	-	$r=.25-.71$ (*k=3)	-	$r=.32$ (k=1)	-	$r=-.12--.05$ (k=2)	$r=.33-.55$ (k=7)	-	-
Hypomania	$r=.04-.63$ (k=10)	-	-	$r=-.15$ (k=1)	$r=.16$ (k=1)	$r=.09$ (k=1)	$r=.11$ (k=2)	-	-	$r=.14-.24$ (k=3)

*Effect sizes for all studies could not be calculated based on available data.

What is the type and severity of sleep disturbance within individual psychotic-like, dissociative, and hypomanic experiences? Is there evidence of a causal association?

Paranoia

Fifteen studies explored the link between paranoia in healthy populations and sleep disturbances. These studies examined associations between paranoid ideation and a range of sleep disturbances, including self-reported insomnia (k = 7; Freeman et al., 2009; Freeman et al., 2010; Freeman et al., 2011; Freeman et al., 2012; Hennig & Lincoln, 2017; Sheaves et al., 2016; Taylor et al., 2015); sleep paralysis (k = 1; Fukuda & Miyasita, 1991); experimentally manipulated sleep deprivation (k = 5; Kahn-Greene et al., 2007; Meyhofer et al., 2017; Meyhofer et al., 2017b; Petrovsky et al., 2014; Reeve et al., 2017); nightmare frequency (k = 2; Rek et al., 2017; Sheaves et al., 2016); nightmare distress (k = 1; Sheaves et al., 2016); chronotype (k = 1; Sheaves et al., 2016); and one study improved sleep in a large randomised controlled trial (k=1; Freeman et al., 2017). The samples included in these investigations considered healthy university students (Freeman et al., 2017; Fukuda & Miyasita, 1991; Sheaves et al., 2016), large scale surveys of general public (Freeman et al., 2009; 2010; 2011; 2012) military volunteers (Kahn-Greene et al., 2007) and comparisons between non-clinical identical and fraternal twins (Taylor et al., 2015). Nine studies were cross-sectional (Freeman et al., 2009; 2010; 2011; 2012; Fukuda et al., 1991; Hennig & Lincoln, 2017; Rek et al., 2017; Taylor et al., 2015), one was longitudinal (Freeman et al., 2012), five experimentally manipulated sleep (Kahn-Greene et al., 2007; Meyhofer et al., 2017; Meyhofer et al., 2017b; Petrovsky et al., 2014; Reeve et al., 2017) and one used a sleep intervention (Freeman et al., 2017).

Insomnia

Insomnia was assessed using the Clinical Interview Schedule Revised (CIS-R; Lewis et al., 1992) in three papers (Freeman et al., 2010; Freeman et al., 2011; Freeman et al., 2012); the Insomnia Severity Index (ISI; Morin, 1993) in two studies (Freeman et al., 2009; Taylor et al., 2015); the Sleep-50 (SP-50; Spoomaker et al. 2005) in one study (Freeman et al., 2009); the Pittsburgh Sleep Quality Index (PSQI; Buysse et al. 1989) in one study (Taylor et al., 2015); the Sleep Condition Indicator (SCI;

Espie et al., 2012) in one study (Sheaves et al., 2016); and one study objectively measured sleep using wrist-worn actigraphy (Hennig & Lincoln, 2017). All of the papers found a significant association between insomnia and paranoia scores, with a small to medium effect size ($r=.11-.41$). One study examined whether baseline insomnia could predict the future occurrence and persistence of paranoia (Freeman et al., 2012), and six studies explored cross-sectional correlations between insomnia and paranoia scores (Freeman et al., 2009; Freeman et al. 2010; Freeman et al., 2011; Freeman et al., 2012; Sheaves et al., 2016; Taylor et al., 2015).

There was a consistent significant association between self-reported insomnia and paranoid ideation with a small (Freeman et al., 2009; Freeman et al., 2011; Sheaves et al., 2016) to medium (Freeman et al., 2010; Freeman et al., 2011; Taylor et al., 2015) effect size ($r=.04-.41$). When studies controlled for the possible confounding effect of negative affect (depression, anxiety, worry and irritability), this effect reduced in magnitude ($r=.16$) for Taylor et al. (2015) and became non-significant ($r=.01$) for Freeman et al. (2009). One study found that negative affect was a significant mediator between insomnia and paranoia (Freeman et al., 2010) which supports the reduction in variance reported above. Only one study tested whether baseline insomnia could predict the occurrence of paranoid ideation using a longitudinal design (Freeman et al., 2012). Baseline self-report insomnia was predictive of the development and persistence of paranoia after 18 months with a very small effect size ($r=.11$ and $r=.05$ respectively).

One study employed an objective measurement of sleep using wrist-worn actigraphy (Hennig & Lincoln, 2017) and found that previous night objective and subjective total sleep time predicted next morning paranoia scores. There was found to be no effect for afternoon paranoia scores. Objective wake after sleep onset and sleep efficiency were not significantly predictive of next day paranoia scores. In addition, this paper examined the mediating effect of negative affect, positive affect, dissociation and inattention on the association between previous night total sleep time and morning paranoia. They found that positive and negative affect, but not dissociation or inattention, partially mediated the link between previous night subjective and objective total sleep time and next morning paranoia.

Furthermore, there was some evidence of a dose-dependent relationship between insomnia chronicity and paranoid ideation (Freeman et al., 2010) which would support a causal link between sleep disturbance and paranoia. Self-reported insomnia (limited to the past month) was associated with paranoid ideation with a medium effect size ($r=.33$) and self-reported chronic insomnia (at least six months duration) with an effect size of $r=.41$.

Parasomnias

Sleep paralysis was examined in one study (Fukuda & Miyasita, 1991), using the Sleep Paralysis (*kanashibari*) Questionnaire (Fukuda et al., 1987) and it was found that episodes of sleep paralysis in university students were positively associated with higher self-report paranoia (Fukuda & Miyasita, 1991). However, the effect size for this association was small in size ($r=.13$).

Nightmare Frequency & Distress

Nightmare frequency and nightmare distress were assessed in a single paper using a nightmare log in a large student sample (Sheaves et al., 2016) and in a single paper using the Nightmare Severity Scale (Sheaves et al., unpublished) in a large student sample (Rek et al., 2017). Sheaves et al., (2016) reported that nightmare frequency and distress were significantly correlated with paranoia scores with small effect sizes ($r=.20$ and $r=.21$ respectively). Rek et al. (2017) reported a higher correlation between paranoia and nightmare severity (composed of nightmare frequency, preoccupation, distress, and impairment in daytime functioning) in their sample ($r=.52$). There was also a significant but very small relationship between paranoia scores and nightmare frequency (quantified as presence or absence of nightmares; $r=.06$) following control for negative affect.

Experimental Sleep Deprivation

Five papers looked at the impact of self-reported paranoia scores following periods of experimental sleep deprivation (Kahn-Greene et al., 2007; Meyhofer et al., 2017; Meyhofer et al., 2017b; Petrovsky et al., 2014; Reeve et al., 2017). Each study used a within-subjects design with a comparable sample size. Kahn-Greene et al. (2007) found evidence of a significant increase in paranoia scores after 56

hours of experimental sleep deprivation in their sample with a small effect size ($r=.26$). More specifically, sleep deprivation lead to an increase in persecution and resentment subscales on the Personality Assessment Inventory (PAI; Morey, 1991) and an increase in hypervigilance approached significance ($r=.18$). All of these individual effects were small in size. Petrovsky et al. (2014) did not replicate this effect and found no significant difference in paranoia scores pre-vs-post a single night of experimental sleep deprivation. Meyhofer et al. (2017) and Meyhofer et al. (2017b), using an identical design to Petrovsky et al. (2014), found no significant increase in paranoia (Psychomimetic states inventory; Mason et al., 2008) following sleep deprivation over one night. Finally, Reeve et al., (2017) used a sleep restriction design and attempted to reduce participants' sleep by 4 hours each day for 3 days. They found a significant increase in paranoia scores from baseline in the sleep deprivation condition which was small in size ($r=.16$). Reeve et al. found an effect size comparable to Kahn-Greene et al. (2007) despite sleep loss in this study being less severe than those studies which used a laboratory-based design. Reeve et al. (2017) was the only study to consider mediators which might explain how experimental sleep loss leads to an increase in paranoia. They found that negative affect (depression, anxiety and stress) as measured with the DASS-21 (Depression, anxiety and stress scales; Lovibund & Lovibund, 1995) explained 91-99% of the variance between sleep loss and paranoia. Once more this supports the role of affect in the link between sleep and paranoia.

The conflicting findings are surprising in light of the consistent cross-sectional link between insomnia and paranoia scores. The magnitude of the sleep deprivation (56 hour versus 24 hours) was greater for Kahn-Greene et al. (2007) than in the Petrovsky et al. (2014) and Meyhofer et al., (2017; 2017b) studies which may partially explain the contrasting findings. However, the cumulative sleep loss for Reeve et al. (2017) was considerably less severe (1 hour 43 minutes per day) and still produced a moderate effect on paranoia which suggests that the magnitude of sleep loss cannot explain this inconsistency.

Chronotype

Sheaves et al. (2016) also examined chronotype using the mid-sleep point on free days (MSF) from the Munich Chronotype Questionnaire (Roenneberg et al., 2003). They found no significant correlation between chronotype and paranoia scores ($r=.06$).

Sleep Intervention

The impact of improving sleep on paranoia experiences was assessed in a large randomised controlled trial of a student population (Freeman et al., 2017). This trial randomised 3755 participants to either an online intervention for insomnia (Sleepio) or usual care and they were followed up at 3, 10 and 22 weeks post intervention or care as usual. It was found that treatment for insomnia was associated with a decrease in paranoia with a small effect size at 3, 10, and 22 weeks ($r=.07$, $r=.09$, $r=.12$). This small improvement at week 10 was partially mediated by improvement in insomnia scores at week 3 (29.5%) and insomnia scores at week 10 (57.8%).

Delusions

Five studies explored the association between sleep disturbances and delusions which were not persecutory in nature. One study assessed whether insomnia was associated with delusional mood or delusions of control ($k=1$; Koyanagi & Stickley, 2015); three studies explored whether a single night of sleep deprivation was able to produce significant increases in delusional thinking ($k=3$; Meyhofer et al., 2017; 2017b; Petrovsky et al., 2014); and a fifth examined whether sleep restriction could produce an reduction in grandiosity ($k=1$; Reeve et al., 2017).

Insomnia

Koyanagi & Stickley (2015) found that self-reported insomnia was associated with delusional mood ($r=.26$), delusions of control ($r=.28$) and delusions of reference and persecution ($r=.23$). These both remained significant after adjustment for anxiety and depression but the effect size diminished by a half ($r=.13$, $r=.14$, $r=.12$ respectively). However, it is not certain how insomnia may contribute to the development or severity of delusions.

Sleep deprivation

A single night of sleep deprivation was not found to produce a significant increase in delusional thinking (Petrovsky et al., 2014; Meyhofer et al., 2017; 2017b) in a student sample. In a home-based sleep restriction design (Reeve et al., 2017) no effect of sleep loss was found for grandiosity ($r=.01$).

Hallucinations

Thirteen studies explored the link between hallucinations and sleep disturbances in healthy populations including: self-reported insomnia (k=5; Koyanagi & Stickley, 2015; Ohayon, 2000; Sheaves et al., 2016; Sheaves et al., 2016b; Taylor et al., 2015); experimentally manipulated sleep deprivation (k=6; Giesbrecht et al., 2007; Hurdiel et al., 2014; Petrovsky et al., 2014; Meyhofer et al., 2017; 2017b Reeve et al., 2017); nightmare frequency (k=2; Rek et al., 2017; Sheaves et al., 2016); nightmare distress (k=2; Rek et al., 2017; Sheaves et al., 2016), chronotype (k=1; Sheaves et al., 2016); and one study improved sleep in a large randomised controlled trial (k=1; Freeman et al., 2017). One study was longitudinal (k=1; Sheaves et al., 2016b), four studies experimentally manipulated sleep (k=4; Freeman et al., 2017; Giesbrecht et al., 2007; Hurdiel et al., 2014; Reeve et al., 2017), six were cross-sectional (k=6; Barnes et al., 2011; Koyanagi & Stickley, 2015; Rek et al., 2017; Sheaves et al., 2016; 2016b; Taylor et al., 2015), and one used a sleep intervention (Freeman et al., 2017).

Insomnia was assessed using the CIS-R in two papers (k=2; Koyanagi & Stickley, 2015; Sheaves et al., 2016b); the ISI in one study (k=1; Taylor et al., 2015); SCI in one study (k=1; Sheaves et al., 2016); Sleep-EVAL in one study (k=1; Ohayon, 1999); and the PSQI in one study (k=1; Taylor et al., 2015). Sleep deprivation was explored in three studies: one which explored self-report hallucinations following an ultra-marathon lasting 27-44 hours (k=1; Hurdiel et al., 2015), one which deprived participants of sleep experimentally over 24 hours (k=1; Giesbrecht et al., 2007), and one which restricted sleep to four hours per day for three days (Reeve et al., 2017). Nightmare frequency and nightmare distress were assessed with a nightmare log (k=1; Sheaves et al., 2016) or the Nightmare Severity Scale (NSS; Sheaves et al., unpublished) in one study (k=1; Rek et al., 2017). Finally,

chronotype and hallucinations were assessed in only one paper which used the Munich Chronotype Questionnaire (k=1; Sheaves et al., 2016)

Insomnia

Five studies explored the association between insomnia and hallucinations in non-clinical populations. Each study found a significant correlation between self-report hallucination proneness and self-reported insomnia scores (Koyanagi & Stickley, 2015; Ohayon, 2000; Sheaves et al., 2016; Sheaves et al., 2016b; Taylor et al., 2015). Self-reported insomnia, in the past 30 days, as assessed with a single question, was significantly associated with hallucinations ($r=.22$; Koyanagi & Stickley, 2015). Sheaves et al. (2016b) examined whether baseline insomnia in two separate British Psychiatric Morbidity Surveys was correlated with hallucinations and whether baseline insomnia was predictive of new hallucinations at an 18 month follow-up. They found that insomnia was significantly associated with hallucinations with a small effect size ($r=.23$). Furthermore, they found evidence of a dose-dependent link between the chronicity of the insomnia and hallucinations. Chronic insomnia was correlated with hallucination proneness with a medium effect size ($r=.31$). A medium effect size between insomnia and hallucination experiences was also found in a study of healthy twins by Taylor et al. (2015) ($r=.32$). Ohayon (2000) also found that hallucinations within the past week were associated with short sleep duration. The effect size for Taylor et al. (2015) reduced by a half when negative affect was controlled for ($r=.16$). Finally, baseline insomnia was found to be predictive of future hallucination experiences in a large sample (N=2406) with a very small effect size ($r=.04$) (Sheaves et al., 2016b). This suggests that sleep problems may precede the emergence of hallucinations in combination with other factors and hints at a causal link between sleep disturbance and hallucinations. However, the very small effect size suggests caution should be exercised.

Nightmare Frequency & Distress

Two studies examined the correlation between nightmare frequency and hallucinations in the general population. Sheaves et al. (2016) found a significant and positive correlation between self-reported hallucination proneness and nightmare frequency with a small effect size ($r=.20$). They also found

that nightmare distress and hallucinations were also significantly positively correlated. Rek et al. (2017) found a significant association between nightmare severity and hallucinations with a medium, approaching large, effect size ($r=.49$). Therefore, as for paranoia, nightmare distress and frequency appears to be significantly associated with hallucinations with similar effect sizes.

Sleep Deprivation

Six studies examined the effect of experimentally manipulating sleep on next day hallucination scores (Giesbrecht et al., 2007; Hurdiel et al., 2014; Reeve et al., 2017). Hurdiel et al. (2015) examined the impact of a long-distance ultra-marathon on self-reported hallucinations following 27-44 hours of wakefulness. In total, 17 participants were followed up after completing the race and were asked to report on hallucinations. Out of these, 4 participants reported visual hallucinations during the race (24%). However, it is impossible to deduce whether the physical intensity of the race or the sleep deprivation was responsible for these changes. Furthermore, no baseline hallucinations scores were reported and so it cannot be certain whether any of these participants had any previous experiences of hallucinations prior to the race. Two studies experimentally manipulated sleep loss on hallucinations scores. One study examined whether 24 hours of sleep deprivation increased self-report hallucination scores (Giesbrecht et al. 2007) using the behavioural 'White Christmas Task' (Merckelbach & van de Ven, 2001). The task assesses the frequency of false perceptions of the lyrics from the song 'White Christmas' during a period of white noise. However, there was no evidence of increases in hallucination frequency on this task following sleep deprivation. Reeve et al. (2017) found an increase in hallucination scores following sleep restriction over three days with a medium effect size ($r=.40$), and this was mediated by changes in stress scores as assessed by the DASS.

Finally, three studies from the same research group (Meyhofer et al., 2017; 2017b; Petrovsky et al., 2014) explored the link between sleep deprivation and hallucinations using a within-subjects design. Petrovsky et al., (2014) found that scores on self-reported perceptual distortion were significantly higher following a single night of sleep deprivation than at baseline with a large effect size ($r=.52$).

Meyhofer et al. (2017; 2017b) also found an increase in self-reported perceptual distortions following one night of sleep loss ($r=.46$).

Chronotype

One study examined chronotype alongside hallucinations but found no association between the mid-sleep point on free days and self-reported hallucination proneness ($r=.03$; Sheaves et al., 2016).

Sleep Intervention

In a large randomised controlled trial (Freeman et al., 2017) it was found that an online intervention for insomnia led to a reduction in hallucinations compared to care as usual in a student sample. The effect of treating insomnia was small on 3, 10, and 22 week post intervention hallucinations scores ($r=.06$, $r=.12$, $r=.11$).

Positive Schizotypy

Eight studies explored an association between positive schizotypy and sleep disturbance. These examined an association between positive schizotypy and insomnia ($k=1$; Barnes et al. 2011); nightmare frequency and positive schizotypy ($k=3$; Levin, 1998; Levin & Raulin, 1991; Levin & Fireman, 2002); nightmare distress and positive schizotypy ($k=2$; Claridge et al., 1997; Levin & Fireman, 2002); sleep spindles ($k=1$; Lustenberger et al., 2015); and sleep experiences ($k=2$; Knox & Lynn, 2014; Watson, 2001). All studies were cross-sectional with the exception of three studies which experimentally manipulated sleep ($k=3$; Meyhofer et al., 2017; 2017b; Petrovsky et al., 2014).

Insomnia

Barnes et al. (2011) conducted a cross-sectional study which examined correlations between PSQI and positive schizotypy scores. An increase in sleep disturbance was associated with an increase in unusual experiences scores with a small effect size ($r=.24$).

Nightmare Frequency & Distress

Levin and Fireman (2002) found that a higher score in all positive schizotypy subscales was associated with increased nightmare frequency. Specifically, they found a significant correlation between nightmare frequency and perceptual aberration ($r=.39$), magical ideation ($r=.37$) and overall PER-MAG ($r=.40$) scores with medium effect sizes. Levin and Raulin (1991) also found evidence of a significant correlation between nightmare frequency and perceptual aberration ($r=.13$), and Levin (1998) provided evidence of a significant positive correlation between nightmare frequency and magical ideation ($r=.29$). Claridge et al. (1997) explored the association between nightmare distress and overall schizotypy scores. They found that nightmare distress was significantly correlated with overall schizotypy scales; participants who reported greater nightmare distress also reporting greater schizotypy scores ($r=.51$). Levin and Fireman (2002) also found that nightmare distress was significantly correlated with increases in perceptual aberration ($r=.44$) and magical ideation ($r=.39$). The effect sizes were medium to large in size for nightmare distress ($r=.39-.54$) and slightly larger than for nightmare frequency ($r=.13-.40$) which highlights that ‘distress’ may be more important when considering positive schizotypy. All of these studies used a cross-sectional and correlation design which limits comments on causal links between nightmare frequency and distress and positive schizotypy to be made. However, the consistency of findings and small to large effect sizes suggest that a potential causal link warrants further examination.

Unusual Sleep Experiences

Two studies explored sleep using the IOWA sleep experiences survey (ISES) subscale, general sleep experiences (ISES-GSE; Watson, 2001). This scale explored specific phenomena which occur during the night such as, “I walk in my sleep”. Knox and Lynn (2014) and Watson (2001) both found that ISES-GES was significantly correlated with both perceptual aberration ($r=.38$) and magical ideation ($r=0.42$) with a medium effect size. However, although this scale provides evidence of sleep-related phenomena which may impact the quality of sleep, its use in positive schizotypy may be harder to interpret and inflate effects due to overlapping questions.

Sleep Spindles

Finally, one study examined micro-architectural sleep in relation to positive schizotypy. Lustenberger et al. (2015) found that magical ideation (Eckblad & Chapman, 1983) was significantly negatively correlated with sleep spindle density. As magical ideation scores increased the density of sleep spindles decreased over the course of a night with a large effect size ($r=-.64$).

Negative Schizotypy

Eight studies explored the association between sleep disturbance and negative schizotypy: insomnia (k=2; Barnes et al. 2011; Taylor et al., 2015); nightmare frequency (k=2; Levin & Raulin, 1991; Levin, 1998); nightmare distress (k=1; Claridge et al., 1997); and experimental sleep deprivation (k=3; Meyhofer et al., 2017; 2017b; Petrovsky et al., 2014). All studies were cross-sectional with the exception of three studies which experimentally manipulated sleep (k=3; Meyhofer et al., 2017; 2017b; Petrovsky et al., 2014).

Insomnia

Barnes et al. (2011) conducted a cross-sectional study which examined associations between the Pittsburgh Sleep Quality Index (PSQI) and Introvertive Anhedonia (IA) and Impulsive Non-Conformity (IMP) subscales of the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE; Mason et al., 1995). An increase in sleep disturbance was associated with an increase in IA ($r=.31$) scores but not IMP ($r=.13$). Taylor et al., (2015) explored psychotic-like experiences in identical and fraternal twins. They found that insomnia, as measured by the Insomnia Severity Index (ISI; Bastien et al., 2001) and PSQI, was significantly positively correlated with negative symptoms but with a small effect size ($r=.09-.17$). They also found a significant correlation between PSQI scores and physical anhedonia ($r=.09$), but not between ISI and physical anhedonia ($r=.08$). However, when negative affect was controlled for these correlations became non-significant.

Nightmare Frequency & Distress

Levin and Raulin (1991) found that nightmare frequency in a population of university students was significantly correlated with physical anhedonia. However, surprisingly, it was found that a reduced

nightmare frequency was associated with an increased physical anhedonia score ($r=-.08$). Levin (1998) found no evidence of a significant correlation between nightmare frequency and physical anhedonia. Both of these studies found small effect sizes ($r<.10$). Together, this suggests that the evidence of a link between nightmare frequency and negative schizotypy is uncertain or non-existent. Claridge et al. (1997) found that nightmare distress in a sample of healthy individuals was significantly correlated with physical anhedonia scores ($r=.38$). This was a moderate effect size and highlights that distress may be more informative than frequency of nightmares in relation to both positive and negative schizotypy.

Sleep Deprivation

Following a single night of sleep deprivation, there was found to be a significant increase in anhedonia scores with a large effect size ($r=.53$; Meyhofer et al., 2017; Petrovsky et al., 2014). However, a study by Meyhofer et al. (2017b) found no significant increase in anhedonia following a single night of sleep deprivation in controls ($n=19$; $r=.13$) or a high schizotypy group ($n=17$; $r=.25$).

Dissociation

Fourteen studies examined the association between dissociation and sleep disturbances: insomnia ($k=2$; Hennig & Lincoln, 2017; Van Heughten et al., 2015); nightmare frequency ($k=2$; Levin & Fireman, 2002; Rek et al., 2017); sleep deprivation ($k=3$; Giesbrecht et al., 2007; Soffer-Dudek et al., 2017; van Heughten et al., 2015); chronotype ($k=2$; Giesbrecht & Merckelbach, 2004; Selvi et al., 2017); and unusual sleep experiences ($k=7$; Fassler et al., 2006; Giesbrecht & Merckelbach, 2004; Giesbrecht et al., 2006; Knox & Lynn, 2014; Soffer-Dudek et al., 2009; Van Heughten et al., 2014; Watson, 2001). All studies were cross-sectional with the exception of three studies which experimentally manipulated sleep ($k=3$; Giesbrecht et al., 2007; Soffer-Dudek et al., 2017; van Heughten et al., 2015).

Insomnia

Van Heughten et al. (2015) explored the correlation between self-reported dissociation and insomnia as measured with Sleep-50 (Spoormaker, Verbeek, van den Bout, & Klip, 2005). They found that there was a significant positive correlation between SL-50 scores and Dissociation Experiences Scale (DES; Bernstein & Putnam, 1986) with a medium effect size ($r=.45$). Furthermore, a significant correlation, with a medium effect size ($r=.39$), was also found between SL-50 and the Cambridge Depersonalisation Scale (CDS; Sierra & Berrios, 2000). However, in an experience sampling methodology design, sleep measured with actigraphy or sleep diary were not found to be predictive of next morning dissociation (Hennig & Lincoln, 2017).

Nightmare Frequency

One study examined the link between nightmare frequency and self-reported dissociation. They found a significant positive correlation between DES scores and nightmare frequency (Levin & Fireman, 2002) with a medium effect size between high nightmare frequency and low frequency ($r=.36$) and high nightmare frequency and medium frequency ($r=.32$).

Sleep Deprivation

Three studies examined the effect of experimentally manipulated sleep loss on dissociation experiences (Giesbrecht et al., 2007; Soffer-Dudek et al., 2017; van Heughten et al., 2015). Van Heughten et al. (2015) found a significant increase in dissociation scores using the Clinician-Administered Dissociation States Scale (CADDS; Bremner et al., 1998) following a single night of experimentally manipulated sleep deprivation. The effect size for this was large ($r=.71$) which hints at a strong link between dissociation and sleep loss. Giesbrecht et al. (2007) and Soffer-Dudek et al. (2017) also found a significant increase in dissociation using the CADSS following experimentally manipulated sleep deprivation with a smaller effect size ($r=.30$) which supports this finding.

Unusual sleep experiences

A number of studies examined general sleep experiences (e.g. sleep talking, walking or presence in room) of the ISES in dissociation. All studies showed a significant positive correlation between scores on the DES and total ISES scores (Fassler et al. 2006; Giesbrecht & Merckelbach, 2004; Giesbrecht et al., 2006; Knox & Lynn, 2014; Soffer-Dudek et al. 2009; Van Heughten et al., 2014; Watson, 2001), with a medium to large effect size ($r=.33-.55$) for the general sleep experiences scale of the ISES (Fassler et al. 2006; Giesbrecht et al., 2006). This suggests that there is a medium to large effect between dissociation and parasomnias as assessed with the ISES.

Chronotype

One study (Giesbrecht & Merckelbach, 2004) failed to find a difference between the Morning-Eveningness Questionnaire (Horne & Ostberg, 1976) and dissociation scores using the DES. The effect size was very small ($r=-.05$). By contrast, Selvi et al., (2017) found a significant correlation between the DES and MEQ with a small effect ($r=-.12$). Both studies suggest that eveningness may be associated with increases in dissociation scores.

Hypomania

Thirteen studies examined an association between sleep disturbance and hypomania: insomnia (k=9; Ankers & Jones, 2009; Bajoghli et al., 2014; Brand et al., 2010; Brand et al., 2011; Brand et al., 2015; Meyer & Maier, 2006; Monk et al., 2001; Ritter et al., 2015; Sheaves et al., 2016); parasomnias (k=1; Fukuda et al., 1992); objectively measured sleep loss (k=1; Rock et al., 2014); nightmare frequency (k=1; Sheaves et al., 2016), nightmare distress (k=1; Sheaves et al., 2016); circadian rhythm disturbances (k=4; Bae et al., 2014; Brand et al., 2011; Rock et al., 2014; Sheaves et al., 2016); and one study improved sleep in a large randomised controlled trial (k=1; Freeman et al., 2017). All studies were cross-sectional except one (Freeman et al., 2017).

Insomnia

A significant positive correlation between self-reported insomnia and hypomania was found for all but one study which examined this association (Bajoghli et al., 2014; Brand et al. 2010; Brand et al.,

2011; Brand et al., 2015; Ritter et al., 2015; Sheaves et al., 2016). More specifically, Brand et al., (2011) compared scores on the risk-taking and active-elevated scales of the Hypomanic Check List-32 (HCL-32; Angst et al., 2005). They found that risk-taking hypomania was significantly associated with insomnia scores ($r=.19$) but not active-elevated hypomania ($r=.05$). No effect sizes between insomnia and hypomania proneness were above small in size. Furthermore, Ritter et al. (2015) identified that insomnia at baseline in healthy adolescents and young adults (age 14-24 years old) was significantly predictive of future risk for developing bipolar disorder ($r=.19$). In addition, this predictive quality of insomnia remained significant after controlling for sex, age, baseline parental mood disorder and baseline substance abuse (alcohol and cannabis) ($r=.15$). A group of short sleepers ($n=12$; duration of less than 6 hours) were found to score significantly higher on hypomania scores than controls ($n=12$; $r=.63$).

Sleep onset latency, that is the time in bed until sleep onset, was found to be significantly reduced in those reporting higher hypomania in three studies (Bajoghli et al., 2014; Brand et al., 2015; Ritter et al., 2015). Reduced sleep duration was shown to be significantly associated with increased hypomania in two studies (Brand et al., 2015; Monk et al., 2001) with a small effect size ($r=-.21$). Meyer & Maier (2006) did not find a significant association between sleep duration and hypomania ($r=.04$) but they found a decrease in the stability of sleep patterns correlated with hypomania scores in a student sample ($r=.26$). Wake after sleep onset was found to be significantly increased in those who reported higher hypomania in two studies (Bajoghli et al., 2014; Brand et al., 2015). Overall sleep quality as assessed by the ISI was also found to be significantly correlated with hypomania with a medium effect size ($r=.31-.39$). Furthermore, Ankers and Jones (2009) showed reduced sleep duration ($r=.37$) in a hypomania proneness group, and greater variation in sleep fragmentation and efficiency compared to controls. Rock et al. (2014) did not find evidence for alterations in sleep continuity using actigraphy between those at phenotypic risk for bipolar disorder compared to controls ($r=.04-.25$).

Parasomnias

One study (Fukuda et al., 1992) examined the association between sleep paralysis and hypomania proneness as assessed by the Maudsley Personality Inventory (MMPI; Jensen, 1958) but found no significant difference between the high and low sleep paralysis groups ($r=.05$).

Nightmare Frequency & Distress

Sheaves et al., (2016) found a positive significant correlation with nightmare frequency ($r=.16$) but no significant correlation between hypomania and nightmare distress ($r=.09$). However, this single study makes it hard to draw definite conclusions about nightmare frequency or distress in hypomania.

Circadian Rhythm Disturbances

Three studies explored circadian rhythm disturbances alongside hypomania. This was the only non-clinical experience (e.g. paranoid ideation, hallucinations, dissociation) studied here which explored circadian variations beyond chronotype. Sheaves et al. (2016) found that hypomania was positively correlated with a later mid-sleep point on free days, or an eveningness preference. The effect was only small ($r=.11$). In contrast, Bae et al. (2014) found that irritable-risk taking component of the HCL-32 was significantly correlated with morningness with a slightly larger effect size ($r=.18$). The same group also found evidence of greater variability in daily rise time, weekday bedtime, weekend rise-time and weekend bedtime in those with higher irritable risk-taking hypomania scores. Again, no effects were larger than small in size (all $r<.24$). However, a study conducted by Rock and colleagues (2014) found that a high hypomania group did not differ from a low hypomania group on chronotype categorisation (e.g., morning, evening, intermediate type). The same study did find evidence of increased L5 activity (lowest 5 hours of activity during the day; $r=.36$) and reduced relative amplitude in the high compared to low hypomania group ($r=.31$).

Sleep Intervention

In a large randomised controlled trial (Freeman et al., 2017) it was found that an online intervention for insomnia led to a small increase in mania scores compared to care as usual in a student sample.

The effect of treating insomnia was small on 10 and 22 week mania scores ($r=-.15$, $r=-.12$). This is somewhat surprising as it suggests that improving sleep is associated with an increase in mania but the authors cite specific items used to assess mania might also have overlapped with general wellbeing which was shown to improve in the treatment group.

Discussion

Summary of results

The aim of this review was to (1) identify and compare the types of sleep disturbances present alongside individual psychotic-like experiences, dissociation and hypomania in non-clinical populations, (2) assess whether the magnitude of sleep disturbances are associated with these experiences and (3) examine whether there any evidence of a causal association between sleep disturbance and these experiences. There are overlaps in the type of sleep disturbances present in individual psychotic-like, dissociative and hypomanic experiences. Nonetheless, no single experience had the same pattern of sleep disturbances as any other experience reviewed here. There was evidence of an increase in the severity of sleep disturbance being associated with increased severity of psychotic-like, dissociative and hypomanic experiences. Our review showed a dose-dependent relationship between sleep disturbance and paranoia, and evidence of an increase in paranoia, positive and negative schizotypy, and dissociation following a period of experimentally controlled sleep deprivation. The reliance on cross-sectional and correlational studies made it hard to make causal inferences. There was partial evidence of a causal link, according to the Bradford-Hill (1965) criteria, but gaps in the literature made it hard to assess the validity or nature of a causal link between sleep disturbance and psychotic-like, dissociative and hypomanic experiences.

Potential mechanisms

This review highlighted that insomnia is associated with all reviewed psychotic-like experiences although the magnitude of this link varies substantially. There's evidence from our review to suggest that anxiety and depression symptoms may mediate this link. Psychological processes such as worry and rumination have been shown to be important in the development and persistence of psychotic

experiences and insomnia (Hartley et al., 2014; Harvey, 2002). These may form an indirect route through which sleep disturbance and psychosis are associated. This would cast doubts on a direct route from sleep disturbance to psychosis experiences. Moreover, poor or inflexible use of emotion regulation which has been shown to be important in psychopathology and sleep in general (Goldstein-Piekarski et al., 2015; Kyle et al., 2014; Watling et al., 2016) and may be useful for understanding sleep disturbances and psychotic-like experiences.

There is also evidence to support the importance of emotion regulation deficits between sleep and psychosis at a neurophysiological level. Research on sleep architecture in psychosis has identified disturbances in sleep continuity, a reduction in slow wave sleep (SWS), and REM density (Baglioni et al., 2016; Chan et al., 2016). REM sleep is associated with consolidation of emotional memories (Groch et al., 2013) and emotion regulation (Gujar et al., 2010). Furthermore, sleep deprivation in healthy individuals is found to increase amygdala reactivity to negative stimuli and is associated with reduced connectivity between the medial prefrontal cortex (mPFC) and the amygdala (McKenna & Eyler, 2012; Yoo et al., 2007). REM sleep is also important for the process of reducing emotional reactivity to negative stimuli from the previous day (van der Helm et al., 2011). Cumulatively, these may provide mechanistic explanations for the reduction in REM sleep and affective regulation deficits found in psychosis (Grezellschak et al., 2016; Strauss et al., 2013) and bipolar disorder (Harvey, 2008). Therefore, a greater understanding of how REM sleep may be implicated in emotion reactivity and regulation in psychosis and psychotic-like experiences is warranted. This could be achieved through selective disruption of REM sleep in non-clinical participants who score high on psychotic-like experiences with clearly defined questions on putative mechanisms. Questions which focus on emotional reactivity, memory and emotion-regulation would have clear transdiagnostic implications beyond psychosis and psychotic-like experiences (Harvey, 2008b).

The lack of studies which have explored circadian rhythms in this review is surprising as circadian rhythm abnormalities (e.g., phase advance, phase delay and free-running rhythms) have been frequently identified within populations with a diagnosis of schizophrenia (Bromundt et al., 2011; Monti et al., 2013; Wulff et al., 2010) and bipolar disorder (Harvey, 2008; Melo et al., 2017). Recent

evidence has also shown that circadian disturbances are predictive of future psychosis symptom severity in groups at risk for bipolar disorder and psychosis (Castro et al., 2015; Lunsford-Avery et al., 2017). More specifically, a recent study with ultra-high risk participants found that increased fragmentation of circadian rest-activity rhythms, reduced daytime activity, increased daytime activity variability, and later nocturnal rest time at baseline were associated with the severity of psychosis symptoms and functioning at a one year follow-up (Lunsford-Avery et al., 2017).

Only one study explored the association between microstructural alterations in sleep (sleep spindles) and psychotic-like experiences (Lustenberger et al., 2015). In light of sleep spindles' relevance to memory deficits in schizophrenia (Pocivasek & Rowland, 2017) future research would benefit from expanding on the single study (Lustenberger et al., 2015) identified in this review. A greater characterisation of sleep spindles in at-risk populations has been proposed to also have treatment implications for memory consolidation deficits in psychosis (Manoach et al., 2016).

Future research exploring sleep spindles may also identify underlying neural mechanisms between sleep and psychosis experiences. Sleep spindles are generated by the thalamic reticular nucleus (TRN) and the dorsal thalamus (Steriade et al., 1987). A reduction in spindle density would indicate a potential problem in the TRN. Individuals with a diagnosis of schizophrenia are found to have reductions in left mediodorsal thalamic volume compared to controls (Buchmann et al., 2014) and dysfunction of the TRN has been implicated in psychosis – specifically the presence of hallucinations (Behrendt, 2006). Moreover, Lunsford-Avery et al. (2013) showed that ultra-high risk participants had reduced thalamic grey matter volume compared to healthy controls. However, the function of sleep spindles is yet to be discerned despite current excitement for their role in psychosis and memory (Tesler et al., 2015; Pocivasek & Rowland, 2017).

There have been attempts to examine the putative causal link between sleep disturbance and psychosis experiences within the non-clinical literature reviewed. Although all experimental sleep deprivation studies consistently reported a small to large effect size (with the exception of paranoia) the largest effect size was seen for next day dissociation scores ($r=0.71$). This is an interesting finding given the

link between childhood trauma, dissociation and psychosis (Varese et al., 2012). Childhood trauma is linked to sleep disturbances and increased risk of a future diagnosis of schizophrenia and bipolar disorder (Noll et al., 2006; Varese et al., 2012; Watson et al., 2014). One avenue to explore the association between sleep and dissociation further may involve examining the impact of sleep loss on intrusive memories and affect (Kleim et al., 2016; Porcheret et al., 2015; Lerner et al., 2017).

Non-clinical populations are useful to understand causal links between sleep disturbance and psychosis experiences, although this approach is not without limitations. The ability to manipulate sleep in a healthy population strengthens causal and directional inferences between sleep and psychosis experiences. Although the experimental manipulation of sleep is not comparable to insomnia, it does provide a more objective way to test associations between sleep loss and putative mechanisms of psychotic-like, dissociative and hypomanic experiences. However, many of the studies attempting to answer questions of causation relied on self-report outcome data to assess psychotic-like, dissociative, and hypomanic experience changes following experimental sleep loss. One alternative approach, already described, is to explore general affective and cognitive consequences of disrupted sleep which overlap with cognitive-affective deficits seen in psychotic-like experiences (e.g. source monitoring, sensory gating, and reasoning biases; McKenna & Eyler, 2012; Yates, 2016). There is evidence that sensory gating, a candidate mechanism for hallucinations (Freedman et al., 1987; Sanchez-Morla et al., 2008), is diminished following a single night of sleep deprivation (Meyhofer et al., 2017; 2017b; Petrovsky et al., 2014). Moreover, the effects of sleep deprivation on brain connectivity, specifically the prefrontal cortex and amygdala, are strikingly similar to those seen in psychosis (McKenna & Eyler, 2012; Yates, 2016) and bipolar disorder (Foland et al., 2008). Future sleep manipulation studies could incorporate tasks to assess such cognitive and physiological mechanisms specific to psychosis. This approach can start to address questions of causality in more detail and unpick the mechanisms linking sleep and psychosis together.

Strengths and Limitations

This review comprehensively searched the literature for evidence of specific sleep disturbances found within individual psychotic-like, dissociative and hypomanic experiences. Unlike similar reviews, we included effect sizes to identify the magnitude of associations between sleep and non-clinical psychotic-like, dissociative and hypomanic experiences. However, this review only examined articles written in English, and did not search for conference abstracts, theses or unpublished data. Although there is merit in this approach, it potentially creates a positive bias towards studies which may have found an association between specific sleep disturbances and psychotic-like experiences.

Furthermore, this review did not try to include individuals at heightened genetic risk or those deemed at ultra-high risk (Lunsford-Avery et al., 2013) for psychosis. Previous reviews have already touched on ultra-high risk (Davies et al., 2016) and their inclusion was beyond the scope of this review. In addition, we only included studies where participants did not have a reported diagnosis of a psychotic, mood or dissociative disorder. We have outlined the benefits of this approach; however, few studies used clinical interviews to verify the absence of a mental health condition and this is a potential limitation for our review which aimed to examine only non-clinical experiences.

One additional limitation of this review is that its attempt to claim certain sleep disturbances are specific to individual psychopathological experiences must be made with caution. Paranoid ideation, hallucination proneness, and hypomania are all highly correlated in non-clinical populations and the studies in this review did not account for these inter-correlations when assessing sleep disturbances. This makes it difficult to claim whether an observed sleep disturbance is due to the measured psychopathological experience or one which was not included in the analysis.

Conclusion

In conclusion, sleep disturbances are associated with individual psychotic-like, dissociative and hypomanic experiences. All of the experiences studied are associated with increased self-report insomnia with a small to medium effect size. Nightmare frequency is positively correlated with all psychotic-like experiences, except negative schizotypy, and nightmare distress is not significantly associated with hypomania. However, the lack of studies assessing chronotype or stability of circadian

rhythms (e.g., dim light melatonin onset, cortisol and temperature) makes it hard to make comparisons between individual experiences. The paucity of research characterising the circadian profiles of non-clinical populations exhibiting sub-clinical experiences is stark and clear. Research has currently focused on sleep disturbances despite evidence of considerable circadian rhythm abnormalities in activity, cortisol, and sleep in schizophrenia and bipolar populations (Wulff et al., 2010; Harvey, 2008).

There is some evidence for an association between severity of sleep disturbance and psychotic-like, dissociative and hypomanic experiences. Yet, despite plausible mechanisms linking sleep disturbances to the studied experiences, it is not clear yet whether sleep disturbances are causally linked to psychotic-like, dissociative, or hypomanic experiences in non-clinical populations. The evidence reviewed here, from experimental sleep deprivation and intervention studies, supports a causal association between sleep disturbances and the experiences studied. However, small effect sizes, and inconsistent results, raise doubts for a causal role of sleep in experiences such as paranoia.

Future research is needed to address mechanisms linking sleep disturbance to psychotic-like, dissociative, and hypomanic experiences. Well controlled experiments manipulating sleep and measuring specific and general cognitive-affective mechanisms of the experiences studied will prove more informative in understanding causal associations. There are groups who are starting to do this in non-clinical populations (Petrovsky et al., 2014; Reeve et al., 2017). Such studies will identify what specific effects (e.g., deficits in sensory gating) sleep disturbance may have on each experience studied and which are likely to be shared (e.g., mood and emotion regulation deficits) across diagnoses and psychopathology in line with transdiagnostic approaches (Harvey, 2001).

Finally, the existence of sleep disturbances prior to a diagnosis of psychosis, dissociation or mania raises the opportunity to intervene and slow the transition to a clinical diagnosis. Although only a modest percentage with these experiences will go on to develop a mental illness (Yung et al., 2009), the ubiquitous association of insomnia with these experiences is relevant and important for those keen to reduce risk of psychopathology. Insomnia can be effectively treated with CBT (Espie et al., 2012)

and addressing sleep difficulties is appropriate and less stigmatising (Sheaves et al., 2016) for at risk populations. Sleep disturbances are one of many risk factors for the development of psychosis, dissociation or mania and risk-reduction should include sleep improvement alongside other interventions. This review highlights that difficulties in sleep occur prior to clinical diagnoses and, provisionally, sleep loss is a potential causal agent in exacerbating, and inducing, these experiences.

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