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Eternal Fallacies of the Sleepless Mind: Investigating the Relationship Between Sleep Loss and Emotion Regulation in the Development of Psychotic Experiences

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Thesis Portfolio Abstract

Background: Sleep disturbances and emotion regulation difficulties are both linked to psychotic experiences through previous research. Given sleep's impact on emotional functioning, regulatory processes may mediate the relationship between sleep loss and psychosis, however this remains largely unexplored.

Methods: A systematic review was conducted to evaluate whether emotion regulation bridges the impact of sleep loss on psychotic experiences. This was done through a synthesis of studies assessing emotion regulation changes that i) experimentally manipulated sleep and ii) targeted emotion regulation for improving psychosis symptoms. Following this, a within-subjects crossover experimental study (N = 58) investigated whether one night of sleep restriction (≤ 4 hours) increased psychotic experiences relative to standard sleep in a non-clinical sample, with mediation by negative affect, affect intolerance, and dissociative experiences.

Results: The systematic review of 15 studies found little support for the influence of sleep loss on emotion regulation. While evidence linked emotion regulation difficulties to the severity of psychotic experiences, the absence of direct tests of association by studies and heterogeneity of measures used limited the ability to draw robust conclusions about a causal mechanism. The empirical study found that sleep loss significantly impacted emotion regulation reflected by elevated affect intolerance, and significantly increased next day negative affect, dissociative experiences, paranoia, hallucinations, cognitive disorganisation, anhedonia and distress. Mediation analyses showed that combined mediators (negative affect, affect intolerance and dissociation) accounted for 89.1% of the association of sleep loss with paranoia. Additionally, dissociative experiences significantly mediated the effect of sleep loss on paranoia (46.2%) and partially mediated the effect on hallucinations (38.5%).

Conclusions: Findings support a causal role of sleep loss in the aetiology of psychosis, with mediation by dissociation and affective processes. Theoretical and clinical implications are considered, as well as recommendations for future research to further develop our understanding of this mechanism.

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List of Contents

List of Tables

Systematic Review

Empirical Paper

List of Figures

Systematic Review

Empirical Paper

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Chapter 1

Introduction to Thesis Portfolio

We spend around one third of our lives sleeping, and far from being a passive state, our brains and bodies use this time to conduct a multitude of biological processes that are essential for our daily functioning. When we don't get enough sleep, we risk short- and long-term consequences to our development, immune functioning, cognition, memory, disease risk and overall wellbeing (Medic et al., 2017; Zielinski et al., 2016). Recent research has found that 43% of adults are regularly getting less than the seven to nine hours recommended by the National Health Service (NHS), and strikingly 9% are getting less than four hours of sleep a night (NHS, 2024; The Sleep Charity, 2024). Insomnia is a sleep disorder defined by persistent sleep difficulties, including difficulties falling asleep, waking several times in the night, or lying awake at night (NHS, 2024). Some researchers believe that the prevalence of Insomnia in the UK is rising (Calem et al., 2012), and recent findings estimate that 31% of UK adults are currently experiencing the most common symptoms of insomnia (The Sleep Charity, 2024).

Sleep loss

Researchers have been able to study the impacts of sleep loss experimentally by requiring participants to deliberately restrict the amount of sleep they have over a set period or even go without sleep entirely. The earliest published sleep deprivation study dates back to the 19th century (Patrick & Gilbert, 1896), and since then, such research has provided valuable insights into the impact of insufficient sleep on human health, cognitive function, behaviour and mood. Sleep loss is known to impair daytime functioning by affecting cognitive and executive functions essential for a variety of practical and interpersonal tasks (Killgore, 2010). Notably, a meta-analysis examining the effects of sleep deprivation on functioning found that mood was significantly more impaired than cognitive or motor performance (Pilcher & Huffcutt, 1996). This conclusion is supported by findings demonstrating that insufficient sleep can result in exaggerated reactivity to emotional stimuli (Gujar et al., 2011), elevated negative emotions in response to stressors (Minkel et al., 2012), interrupted processing of emotional memories (Tempesta et al., 2015) and generally experiencing more negative and less positive emotional states (Palmer et al., 2024; Tomaso et al., 2020). Consequently, good sleep is rated by UK adults as the most important factor for ensuring wellbeing (Oxford Economics, 2018), which may explain why disrupted sleep has been suggested as a causal factor for the development of psychiatric conditions (Freeman et al., 2020).

Emotion regulation

Beyond its effects on emotion generation and processing, insufficient sleep also impacts emotion regulation (see review by Palmer & Alfano, 2017). Emotion regulation refers to "the processes by which individuals influence which emotions they have, when they have them and how they experience and express them" (Gross, 1998, p. 275). These strategies and mechanisms can be automatic or deliberate, employed consciously or unconsciously, and directed internally or externally (Gross & Thompson, 2007). Individuals choose to modify the intensity, duration, or expression of their emotions as a way of adapting to environmental challenges and accomplishing goals (Aldao & Plate, 2018). Emotion regulation strategies are generally considered adaptive if they improve psychological outcomes (e.g. cognitive reappraisal or problem-solving) and maladaptive if they increase or maintain distressing emotional states (e.g. rumination and avoidance) (Aldao et al., 2010). However, this classification, as well as individual preferences for strategies, is influenced by social and cultural context (Song et al., 2024). For instance, emotional suppression is usually considered to be maladaptive, however there may be social or cultural circumstances where it is adaptive or appropriate (Murata et al., 2013). Therefore, flexibility in emotion regulation strategy choice and use is crucial for optimal emotional functioning.

Competing models of emotion regulation have been developed; however, the most generally accepted model is the Gross 'extended process model of emotion regulation' (1998, 2001, 2002, 2015). This model posits that emotions can be regulated using various strategies at five different stages: situation selection (e.g. avoid or approach), situation modification (e.g. problem-solving or social support), attentional deployment (e.g. mindfulness or rumination), cognitive change (e.g. acceptance or reappraisal), and response modulation (e.g. help seeking or substance use). Responses at the fifth stage occur after the emotional experience has taken place, and the former stages encompass 'antecedent-focused' strategies that are employed before or during emotional events. This model offers a framework for understanding differences in the timing, type, and method by which emotion regulation strategies are used.

Variations in emotion regulation strategies selected at different stages of the extended process model of emotion regulation can help to explain maintaining factors across various clinical presentations (see review by Sheppes et al., 2015), and the use of maladaptive strategies has been identified as a transdiagnostic vulnerability factor for the development of psychopathology (Aldao et al., 2010; Lincoln et al., 2022). Research indicates that individuals with psychotic experiences report greater difficulties with emotion regulation compared to non-clinical

samples, and that the use of maladaptive strategies by this population is associated with psychological outcomes (Igra et al., 2023). This is further supported by studies showing a heightened habitual use of maladaptive strategies such as suppression, rumination and self-blaming, over adaptive strategies such as acceptance and reappraisal in psychosis samples (Kimhy et al., 2012; Lawlor et al., 2019; Livingstone et al., 2009; Ludwig et al., 2019; Pan et al., 2020; Perry et al., 2011; Ward et al., 2020).

Psychosis

Psychosis is estimated to affect 7.49 per 1000 people in their lifetime (Moreno-Küstner et al., 2018), and is a state characterised by a loss of contact with reality, often manifesting through hallucinations, delusional thinking (e.g. paranoid beliefs), and disorganised thinking and speech (e.g. frequent derailment or incoherence). These are all considered 'positive' symptoms (due to the *addition* of experiences) while 'negative' symptoms include *diminished* emotional expression, energy, interest or difficulties with concentration. Several conditions are associated with these experiences and are therefore classed as 'schizophrenia spectrum and other psychotic disorders' including schizophrenia, schizoaffective disorder, schizophreniform disorder, delusional disorder, brief psychotic disorder and psychotic disorders that are substance-induced or due to another medical condition (American Psychiatric Association, 2013).

Psychosis has traditionally been viewed categorically, with only a small minority experiencing severe symptoms while the general population remains unaffected. However, recent research challenges this model by demonstrating that psychotic experiences occur on a spectrum, often referred to as the 'psychosis continuum' (Johns & van Os, 2001). Around 5-8% of the general population are estimated to experience 'psychotic-like experiences' (Allardyce et al., 2007; Van Os et al., 2009), which are subclinical experiences that are likely to occur infrequently and not cause significant distress. Then there are individuals who are at 'clinical high-risk' for developing a psychotic disorder, of which approximately 20-35% will convert to psychosis within two years (Cannon et al., 2016), as well as individuals in a 'prodromal phase' who show early signs and symptoms that gradually increase before the clinical onset of psychosis (Goulding et al., 2013).

Studying psychotic experiences along this continuum allows for a deeper understanding of the mechanisms and risk factors involved in the development of psychosis. For instance, variations in self-reported emotion regulation strategy use have been observed across different stages of the continuum in association with symptom severity (Chapman et al., 2019). Similarly, self-reported sleep disturbances are prevalent in early psychosis and have also been linked to

symptom severity (Davies et al., 2017). Furthermore, between 30-80% of the clinical population experience difficulties initiating or maintaining sleep, depending on the severity of their symptoms (Cohrs, 2008).

Models of the aetiology and maintenance of psychosis

A number of theoretical models have been developed to explain the onset and maintenance of psychosis, with each emphasising different risk factors and contexts due to the complex interaction of genetic and environmental influences (Radua et al., 2018; Stilo & Murray, 2019). A widely used clinical model, particularly for guiding Cognitive Behavioural Therapy for psychosis (CBTp), is the 'cognitive model of positive psychotic symptoms' by Garety et al. (2001). This model outlines that symptoms are influenced by underlying reasoning and attribution biases, dysfunctional beliefs about the self and the world, and experiences of isolation and adversity. Emotional changes are central to the model and represented as interacting with these cognitive processes. Garety et al. (2001) describe how emotional changes occur in response to anomalous experiences, such as paranoid beliefs and hallucinations, and feed back into the processing of these experiences to influence their content. This model aligns with and often informs the delivery of CBTp interventions, which target cognitive and behavioural factors that maintain symptoms. CBTp is one of the most well-supported interventions and is therefore recommended in National Institute for Health and Care Excellence (NICE) clinical guidelines (2014). Findings indicate that the effects of CBTp on psychotic symptoms are small-to-medium (Sitko et al., 2020) and that therapeutic benefits are small or nonsignificant (Laws et al., 2018). CBTp has also been criticised for failing to target emotion directly (Gumley et al., 2013), underscoring the need to explore alternative intervention approaches and targets that may prove more effective.

Garety et al.'s model (2001) also considers biological, psychological, and social factors that contribute to an individual's vulnerability to developing symptoms following triggering events. Another model that highlights the importance of biological factors and environmental stressors in influencing the onset and severity of psychosis symptoms is the 'stress-vulnerability model' (Zubin & Spring, 1977). According to this model people have different levels of vulnerability to developing psychosis due to predisposing genetic and early life risk factors, which then interact with lifestyle choices, substance use or stressful life events. The more vulnerable someone is to developing psychosis, and the greater the presence of psychological and physiological stress, the more severe and distressing their symptoms are likely to be. This model supports a holistic

understanding of individual differences in psychosis risk and can inform early intervention and prevention approaches that target lifestyle changes, stress reduction and healthy coping skills.

The 'cognitive model of positive psychotic symptoms' (Garety et al., 2001) and 'stress-vulnerability model' (Zubin & Spring, 1977) are among the most familiar and accessible to a broad audience, with 2124 and 4645 citations respectively. Therefore, we focus primarily on situating the findings from this portfolio in relation to these established conceptual frameworks to support with the interpretation and contextualisation of findings to applications that typically build on familiar models. However, it is important to acknowledge the range of different models of psychosis that highlight key biopsychosocial factors relevant to aetiology and maintenance. For instance, the model of 'aberrant salience' (Kapur, 2003) posits a central role of dopamine systems in mediating the perceived significance of internal and external experiences that contribute to associated hallucinations and delusional beliefs. Dopaminergic pathways are also central to models that emphasise the role of environmental risk factors, such as the 'social defeat hypothesis', which proposes that long-term exposure to social exclusion can sensitise dopamine systems thereby increasing individuals' vulnerability to developing psychosis (Selten et al., 2013).

The 'cognitive model of persecutory delusions' (Freeman et al., 2002) places 'emotion' and 'reasoning' as side-by-side processes contributing to the development of persecutory beliefs that arise from attempts to make sense of anomalous experiences. Freeman et al. (2002) reference findings that support the hypothesis that "emotion has a direct contributory role to positive symptom development… it is the ubiquitous presence of emotional disturbance *prior* to full symptoms that is the key finding with regard to its potential influence on delusions". (p. 337). This model also highlights the role of sleep in the development of delusional beliefs within the context of the 'stress-vulnerability model' (Zubin & Spring, 1977), with authors suggesting that "arousal will be caused, and this is likely to be exacerbated by disturbances in sleep." (p. 333). Freeman's (2016) later work underscores sleep dysfunction as a key process in the maintenance of threat beliefs due to the impact on mood, emotion regulation and the influence of fatigue on individual's perceived ability to cope. Another cognitive model that references the role of sleep dysfunction in psychosis is the 'integrative cognitive approach to hallucinations and delusions' (Morrison, 2001), in which the author suggests "It is also likely that the physiological effects of sleep deprivation and drug use will be involved in the development and maintenance of psychosis" (p. 269). In a similar way to the cognitive model developed by Garety et al. (2001), this model emphasises the role of emotion in contributing to positive symptoms

indirectly through its influence on beliefs and interpretations of experiences rather than through a direct effect.

Models have also been developed with a specific emphasis on the role of emotion regulation. The ‘cognitive interpersonal model of psychosis’ (Gumley & Schwannauer, 2006) outlines the influence of early relational patterns and traumatic events in disrupting emotion regulation processes. Altogether the model suggests that these factors contribute to an individual's vulnerability to relapse following recovery from psychosis and that psychotic experiences develop as a maladaptive attempt cope with overwhelming emotional distress. The causal association between childhood trauma and psychotic experiences has been well established through research, and more recently emotion regulation has been found to partially mediate the influence of early trauma on symptom distress but not frequency (Lincoln et al., 2017). Gumley & Schwannauer (2007) propose that therapeutic interventions should focus on emotional recovery and adaptive regulation. For instance, in their ‘compassion focused model of recovery after psychosis’ (Gumley et al., 2010) Compassion Focused Therapy (CFT) is advocated as a beneficial intervention for addressing high levels of shame and stigma common for individuals with psychosis (Wood et al., 2017). Although primarily focusing on the importance of emotion regulation in the context of recovery, these models align with other newly developed models that emphasise the role of emotional distress in maintaining schizophrenia spectrum disorders (Hamm et al., 2015), reinforcing the growing recognition of emotion processes contributing to psychosis symptoms.

Many of the models above are syndrome models of psychosis, meaning that they view psychosis as a collection of interrelated symptoms (e.g. hallucinations, delusions and disorganised thinking). Modelling psychosis in this way has pragmatic value as it aligns with traditional diagnostic clustering of symptoms and supports with contextualising the interaction of interdependent processes. However, this view of psychosis also carries the risk of assuming that these symptoms arise from common underlying causes and mechanisms. In contrast, single-symptom approaches encourage dimensional assessment of experiences, such as the severity or frequency of hallucinations, over categorical groupings. This approach is supported by evidence showing that individual psychotic symptoms vary in heritability (Zavos et al., 2014), and research showing that the factor structure of psychotic symptoms is more complex than generally acknowledged (Peralta & Cuesta, 1999). Studying individual symptoms has distinct advantages. For example, research that focuses on specific symptoms can help clarify the detailed mechanisms involved in the onset and maintenance of psychosis (see Denecke et al., 2024). This symptom-focused approach allows for greater sensitivity to individual variability,

which is crucial for developing more personalised, targeted interventions with transdiagnostic relevance. Cognitive-behavioural interventions that target specific symptoms, such as delusions or hallucinations, have been found to produce stronger treatment effects than those seen in generic CBTp (Lincoln & Peters, 2019).

A conceptual distinction may also be drawn between traditional cognitive models and emotion regulation models, despite some overlapping features. Cognitive models primarily emphasise the role of cognitions, including biases in reasoning and maladaptive appraisals, in influencing changes in emotion, behaviour and resulting beliefs. These models typically focus on how cognition drives symptomology, suggesting that interventions targeting thought content and cognitions can improve clinical outcomes. In contrast, emotion regulation models focus more explicitly on how individuals attempt to modulate their emotional responses. While both frameworks acknowledge the interplay between cognition, emotion, and behaviour, emotion regulation models place greater emphasis on the dynamic strategies used to manage emotional experiences. Many of these strategies, such as cognitive reappraisal, behavioural avoidance, problem-solving or acceptance of experiences, may also be present in cognitive models and have roots in cognitive-behavioural therapy. However instead of emphasising the resulting reinterpretation of meaning, these strategies are conceptualised within emotion regulation models as active processes aimed at altering emotional impact. Rather than deny the role of cognitive processes in clinical symptomology, these models support with understanding a role for emotion independent of cognition in a field where there are longstanding debates about whether emotions are dependent on cognitive processes. For instance, Lazarus argued that cognitive appraisal is a necessary precondition for emotion, and that there would be no emotion without cognition (1982, 1984). In contrast, Leventhal and Scherer (1987) proposed that emotion and cognition often operate in an integrated reciprocal manner, with cognition functioning to help interpret and predict the environment while emotion rapidly evaluates its significance and impact on wellbeing. These conceptual nuances are important in developing our understanding of the causal mechanisms by which sleep loss influences psychotic experiences, as well as for identifying targets for effective clinical treatments.

The models outlined in this chapter provide an overview of our current understanding of the key risk factors associated with the onset and severity of psychotic experiences. This sets the stage for contextualising findings in the following chapters, as well as integrating them into existing theoretical and clinical models to consider their wider relevance.

Thesis aims and overview

This thesis portfolio aims to develop our understanding of the causal mechanisms that link sleep loss to the development of psychotic experiences, as well as explore the role of emotion regulation and related processes in bridging this relationship.

Chapter 2 presents a systematic review examining the impact of both sleep manipulation studies, and emotion-targeted interventions for alleviating psychotic experiences, on emotion regulation outcomes. Therefore, aiming to explore the relationship between sleep loss and emotion regulation, as well as between emotion regulation and psychotic symptoms, to elucidate the potential for this as a causal pathway for the development of psychosis.

Chapter 3, a bridging chapter, follows, connecting these findings and introducing key concepts to be explored in Chapter 4.

Chapter 4 presents the empirical research paper in which the causal mechanism linking sleep loss to the development of psychotic experiences is further explored. Negative affect, affect intolerance and dissociative experiences are proposed as mediators in this relationship. The experimental partial sleep restriction protocol, conducted with a non-clinical sample, aims to identify early increases in subclinical psychotic experiences following sleep loss, enhancing our understanding of how sleep difficulties impact the aetiology of psychosis.

Finally, Chapter 5 provides a synthesis of findings from the thesis portfolio, offers a critical appraisal of the research, and contextualises these results within our existing knowledge base. The theoretical and clinical implications are then discussed, followed by recommendations for future research and clinical practice. The chapter ends with reflections on the research process and final conclusions.

Chapter 2

Systematic Review

Emotion regulation: A bridge between sleep loss and psychotic experiences?

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Abstract

Background: Both sleep and emotion regulation difficulties have independently been associated with increased psychotic experiences. Given the influence of sleep on emotional functioning, it is plausible that emotion regulation is a currently unexplored mediating factor in the relationship between sleep and psychotic experiences. This review therefore aims to evaluate emotion regulation as a mechanism linking sleep loss and psychotic experiences.

Methods: A systematic search was carried out to identify i) studies manipulating sleep to test the effect on emotion regulation and ii) studies manipulating emotion regulation to test the effect on psychotic experiences. Identified studies were assessed for quality and combined using narrative synthesis.

Results: 15 papers fulfilled inclusion criteria. Sleep manipulation studies (n = 4) did not support an effect on emotion regulation. More studies were identified linking emotion regulation and psychosis (n = 11), in all cases these were intervention studies, predominantly of third wave therapies (e.g. Acceptance and Commitment Therapy). Few studies investigated the relationship between emotion regulation and specific psychotic experiences, and no identified studies directly tested the role of emotion regulation as a mechanism between sleep loss and psychotic experiences, limiting specificity of results.

Discussion: This review provides preliminary support for a relationship between emotion regulation difficulties and psychotic experiences. However, further detail is needed regarding causal mechanisms, including how individual domains of emotion regulation link to specific psychotic experiences. Clinical studies are also indicated to test the effect of sleep interventions on emotion regulation and psychotic symptoms in psychosis groups.

Keywords: sleep, insomnia, psychosis, emotion regulation, mechanism

Highlights:

- The review examines emotion regulation as a link between sleep loss and psychosis.
- A systematic review is undertaken of 15 studies investigating emotion regulation.
- Strong links were found between emotion regulation and psychotic experiences.
- The effect of sleep loss on emotion regulation requires more research.
- Additional research is needed to clarify mechanisms and reveal robust associations.

Introduction

Sleep loss has consistently been linked to psychotic experiences across clinical and non-clinical populations, with numerous studies indicating that sleep dysfunction can predict subsequent psychotic experiences (Reeve et al., 2015). Predictive clinical models have shown that sleep disturbances increase the likelihood of transition to psychosis in at-risk individuals (Ruhrmann et al., 2010), while retrospective studies investigating first-episode psychosis identify sleep disturbances as a common prodromal symptom (Tan et al., 2001; Yung & McGorry, 1996). Experimental sleep manipulation studies in non-clinical samples have demonstrated measurable increases in psychotic experiences following sleep deprivation (Hurdiel et al., 2015; Kahn-Greene et al., 2007; Petrovsky et al., 2014; Reeve et al., 2018), further supporting the causal role of sleep loss in psychosis development. While the mechanisms underlying this relationship remain unclear, negative affect is established as a partial mediator (Reeve et al., 2018). Another potential complementary mechanism is emotion regulation, which has independently been linked to both psychotic experiences and sleep loss. Gaining a deeper understanding of how sleep loss and emotion regulation interact in the onset of psychosis could enhance both clinical interventions and etiological models of this complex presentation.

Total sleep deprivation has been shown to disrupt resting-state functional connectivity in brain regions associated with the amygdala (Shao et al., 2014) and reduce the effectiveness of distraction and reappraisal during emotion regulation tasks (Stenson et al., 2021; Zhang et al., 2019). These impairments in emotion regulation functioning may influence an individual's ability or tendency to employ specific strategies, potentially leading to reliance on maladaptive approaches (e.g., ineffective or associated with negative outcomes) when adaptive strategies prove ineffective (Sheppes et al., 2014). A recent meta-analysis found that while sleep loss produced moderate to large shifts in both negative and positive mood states, its impact on adaptive and maladaptive emotion regulation was small or non-significant, respectively (Tomaso et al., 2021). However, these findings were based on studies involving youth samples, limiting their generalisability. Another review examining the relationship between sleep and emotion regulation highlighted that many studies focus on sleep-related changes to mood or emotion reactivity as indicators of emotion regulation effectiveness, rather than assessing regulatory efforts directly (Palmer & Alfano, 2017). By overlooking the role of emotion regulation strategy selection and implementation, existing research limits our ability to establish a clear mechanism linking sleep loss to psychopathology. These findings underscore the need for an updated review of recent experimental studies investigating self-reported emotion regulation

use, which could help clarify the causal relationship between sleep loss and emotion regulation.

Emotion regulation difficulties have been observed across the psychosis continuum, with changes occurring alongside symptom fluctuations (van Rossum et al., 2011). For instance, reduced use of reappraisal, which is generally considered an adaptive strategy, is associated with greater symptom severity and poorer clinical outcomes (Chapman et al., 2020; Westermann & Lincoln, 2011). Maladaptive emotion regulation strategy use has been found to predict subclinical paranoia (Westermann et al., 2013), while avoidance strategies are linked to increased vulnerability to hallucinations over time (Goldstone et al., 2012). In individuals with psychosis, the use of expressive suppression has been shown to predict momentary increases in beliefs related to mind reading and thought insertion, as well as visual and auditory hallucinations, and state paranoia (Kimhy et al., 2020; Nittel et al., 2018). These challenges also extend to the identification stage of regulation, as individuals with schizophrenia report difficulties in their awareness and description of emotional states (Lawlor et al., 2019), which may be a prerequisite for effectively implementing strategies at later stages of emotion regulation (Raugh & Strauss, 2022; Subic-Wrana et al., 2014). As a result, difficulties in emotion identification have been shown to moderate the impact of emotion regulation on psychotic experiences (Kimhy et al., 2020).

The relationship between emotion regulation and psychosis symptoms is important to consider in the context of recovery. Individuals with psychosis often report difficulties with goal-directed behaviour when experiencing unpleasant emotions and struggle to manage emotional distress while pursuing meaningful activities (Lawlor et al., 2019). They are also more vulnerable to experiencing heightened psychosocial stress, social withdrawal, and situational avoidance (Almuqrin et al., 2023), all of which are linked to increased paranoia and a lower quality of life (Fett et al., 2022). Research indicates that when individuals on the psychosis spectrum are guided in using adaptive regulation strategies, such as reappraisal, they can effectively reduce both subjective and objective measures of distress (Clamor et al., 2020; Laquidara et al., 2023). These findings highlight key intervention targets related to emotion regulation that may offer therapeutic benefits for individuals with psychosis. However, further research is needed to clarify the mechanisms of change and the role of emotion regulation in the maintenance of psychosis.

Preliminary evidence supports the mediating role of emotion regulation in bridging sleep loss to the development of psychotic experiences. One study examining the relationship between

insomnia and paranoid ideation, in both clinical and non-clinical participants using self-report measures, found that increased expressive suppression and reduced reappraisal use mediates this relationship (Grezellschak et al., 2017). This finding is further supported by an experimental partial sleep restriction study in non-clinical participants, which identified that increases in psychotic experiences were mediated by changes in negative affect and related processes (Reeve et al., 2018). Together, these studies suggest that emotion regulation may serve as a shared mechanism that bridges a causal pathway linking sleep loss to the development of psychotic experiences.

Identifying causal pathways in psychosis is essential for improving clinical interventions and outcomes. However, this remains a significant challenge due to the complexity of both psychosis and emotion regulation, as each involves multiple components and processes. By integrating sleep manipulation studies with psychosis intervention studies, that directly examine changes to emotion regulation, a causal pathway could emerge that links these concepts. Establishing this pathway would emphasise the value of preventative and targeted sleep interventions in improving psychosis outcomes (Freeman et al., 2015; Waite et al., 2023) and reinforce emotion regulation as a key mechanism in psychosis recovery (Garety et al., 2001; Gratz et al., 2015; Nardelli et al., 2023; Pishdadian et al., 2023).

Review aims

The current systematic review aims to synthesise findings from sleep manipulation studies, and emotion-targeted interventions that measure psychosis symptoms, with the objective of assessing evidence for emotion regulation as a mechanism bridging sleep loss and psychotic experiences. This aim was divided into the following two questions:

(RQ1) What effect does manipulating sleep have on emotion regulation in non-clinical populations?

(RQ2) What effect does modifying emotion regulation have on psychotic experiences?

Method

This review was prepared in accordance with PRISMA guidelines (Page et al., 2021, Appendix B) and registered on the PROSPERO International prospective register of systematic reviews prior to conducting the literature search (ID: CRD42024567253). The search was conducted in August 2024 (Appendix C) by searching title, abstract and keywords of studies using the terms outlined in Table 1.

The 'AMSTAR 2' checklist (Shea et al., 2017) was completed retrospectively and determined that this review is of 'moderate' quality (Appendix B).

With respect to protocol amendments, a decision was made during the abstract screening stage to exclude observational studies from RQ2. This was due to the large volume of such studies and their limited alignment with the review's aim of identifying causal mechanisms. In addition, during the full-text screening stage, the quality appraisal tools were revised. This change was necessary to accommodate the considerable number of studies lacking a comparator group and to enable more consistent quality assessments across study designs.

Inclusion Criteria

1. Reporting of a quantitative measure of emotion regulation that assessed multiple strategy domains.
2. Involving non-clinical participants exposed to a lack of sleep for RQ1.
3. Reporting of a measure of psychotic experiences (self- or clinician-report) for RQ2.
4. Involving modification of emotion regulation for RQ2.

Exclusion Criteria

1. Not available in English or as full texts.
2. Observational studies, systematic reviews or meta-analyses.
3. Reporting of a measure of emotion regulation that was exclusively qualitative, neural/physiological or measuring emotional reactivity as an indicator of emotion regulation effectiveness.
4. Included participants under the age of 13 or over 65, or with the presence of a neurodegenerative condition, stroke or brain injury.
5. Published prior to 1994 corresponding to the year of publication of the DSM-IV to ensure that populations with psychosis were comparable across studies.

Search terms related to emotion regulation were developed to include strategies in line with Gross' extended process model of emotion regulation (1998, 2001, 2002, 2015), and psychotic experiences terms focused on positive symptoms to enhance the specificity of retrieved studies. PubMed/MEDLINE, APA PsychInfo and Web of Science electronic databases were searched for reference lists, with grey literature included in the search. Citation lists were also checked for additional studies.

Table 1

Search terms used to search databases

Sleep loss		Psychotic experiences		Emotion regulation
Sleep* OR “sleep loss” OR “sleep depriv*” OR “sleep reduc*” OR “reduc* sleep” OR “sleep restrict*” OR “restrict* sleep” OR “sleep quality” OR “sleep deficit” OR “sleep fragment*” OR “fragment* sleep”	OR	Delus* OR hallucinat* OR psychosis OR psychotic OR paranoi* OR “disorganized speech” OR “disorganised speech” OR “disorganized thought” OR “disorganized thought” OR “disorganized thinking” OR “disorganized thinking” OR schizophrenia OR “psychotic disorder” OR “schizoaffective disorder”	AND	"Emotion *regulation" OR "affect *regulation" OR “emotion* reactivity” OR "experiential avoidance" OR "emotion* avoidance" OR "behavio* avoidance" OR “situation* avoid*” OR "expressive suppression" OR "thought suppression" OR "emotion* suppression" OR rumination OR distraction OR reapprais* OR “attentional deployment” OR “emotion regulation questionnaire” OR ERQ OR “cognitive emotion regulation questionnaire” OR CERQ OR “difficulties in emotion regulation scale” OR DERS

Screening, Data Extraction and Quality Appraisal

All results from the database searches were uploaded to Rayyan (Ouzzani et al., 2016) to support screening and de-duplication. A total of 5,818 duplicates were found, and an automation tool was used to resolve those with >95% similarity, leaving 847 duplicates to be manually addressed by the first author. Title and abstract screening was conducted by the first author, with any uncertainty regarding inclusion resolved through discussion between the authors. MJ and SR jointly conducted full-text screening for 58% of papers, while MJ screened the remaining papers independently, with any uncertainties resolved through discussion. A summary of studies excluded at the full-text stage, with reasons given, can be found in Appendix D.

Data were extracted by the first author from all studies that fulfilled the inclusion criteria. Data extracted included authors, year, country, study design, sample characteristics, type of exposure/intervention, control or comparison group, outcomes measured, results and conclusions.

Included studies were critically appraised for quality and risk of bias by the first author using the National Institute of Health’s quality checklists for 'Controlled Intervention Studies' and 'Before-After (Pre-Post) Studies with No Control Group' (National Institute of Health, 2021, Appendices E & F). These tools include items that support with quality assessment of the internal validity

and reliability of included studies. Each study was then given a quality rating that reflects the deduced quality and risk of bias (Appendix G).

Analysis

Due to the heterogeneity of study design, populations and outcomes, a meta-analysis was not appropriate. The findings were summarised and interpreted through a narrative synthesis, following the guidance of Popay et al. (2006). This method outlines a systematic process for identifying key themes, patterns, and relationships between study findings. Its flexible nature allowed for an exploration of not only *what* was found by the studies, but also *how* and *why* these results were observed, through examination of the mechanisms, processes and contextual factors.

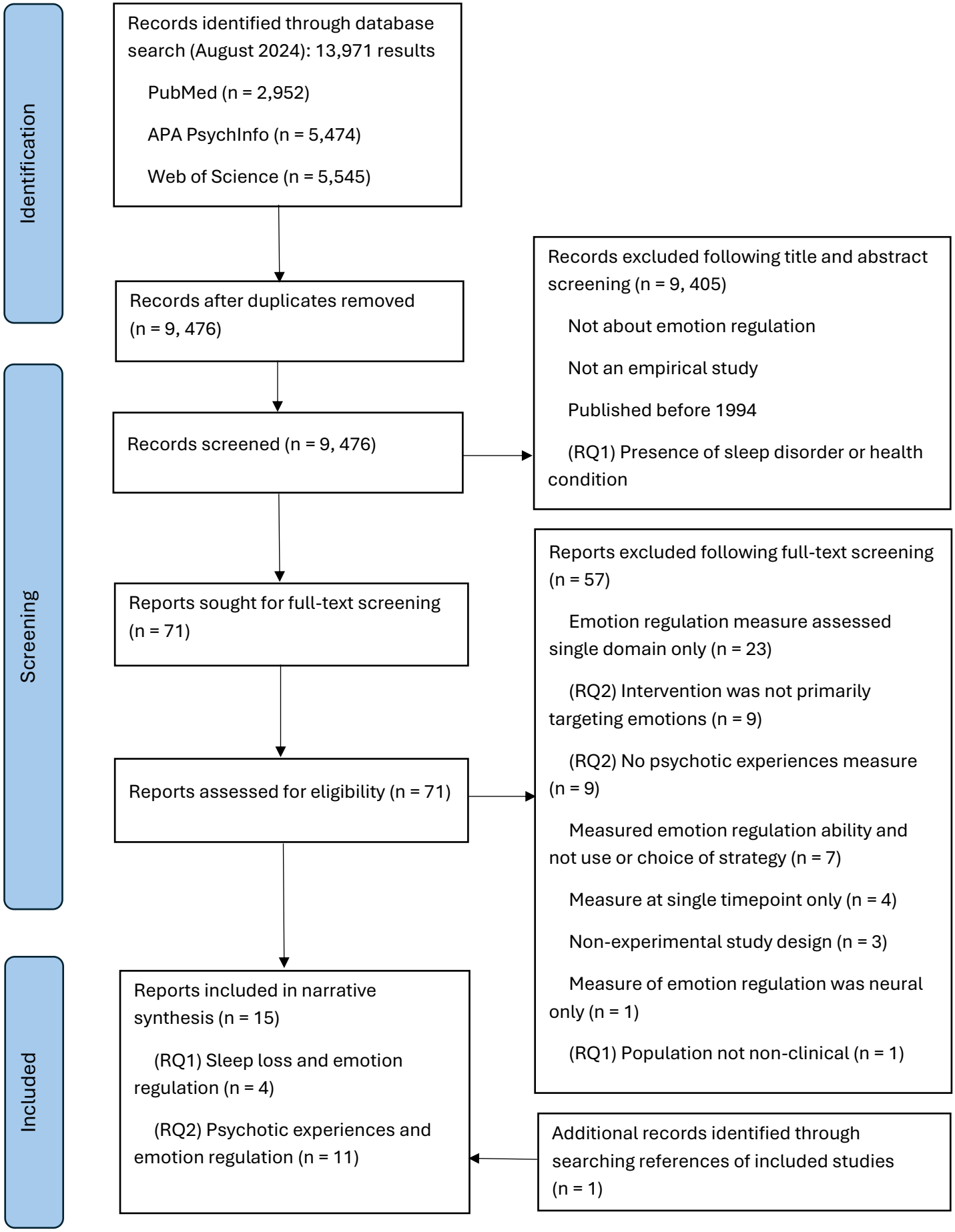
This approach is also designed to minimise bias and enhance the transparency of the synthesis process by providing clear guidelines for organising and interpreting complex evidence. Findings from the included studies are therefore summarised in a table containing an overview of study characteristics, outcomes and conclusions. The studies are then categorised into groups according to the research questions, and by similarity in outcomes measured, for synthesis in text to support with identifying themes in findings.

Results

The database searches yielded a preliminary 13,971 results once merged. After duplicate removal 9,476 results remained for title and abstract screening based on pre-determined eligibility criteria, which yielded 71 texts for full-text screening alongside one additional study identified through citation search. Fifteen studies were eligible for inclusion in the narrative synthesis, composed of 4 studies to address RQ1 and 11 studies to address RQ2. See Figure 1 for a PRISMA flow diagram summarising the selection process.

Figure 1

Flowchart of the identification, screening and selection of studies



Study characteristics

Descriptions of the included studies are given in Tables 2 and 3, including study details, sample size and characteristics, relevant measures used, a summary of findings and quality rating for each study.

All four studies investigating the experimental manipulation of sleep in non-clinical populations employed a Randomised Controlled Trial (RCT) design. Studies varied in their methods of sleep manipulation, including a full night of self-imposed sleep deprivation (n = 1), a single night of sleep continuity disruption with set waking intervals (n = 2), and sleep extension over seven nights (n = 1). All studies were conducted using young adult samples, with an average age ranging from 19 to 25 years.

Studies included that investigated psychotic experiences were all intervention studies, however, were mixed in design and approaches. Five studies used RCT methodology, comparing the treatment group to individuals that received routine care (n = 3) or an active treatment group (n = 2). The remaining six studies used a single-case ABA design (n = 1), single-case AB design (n = 1), were pilot studies or explored the effectiveness of a pre-existing intervention on psychotic experiences (n = 4). Intervention approaches included combined third-wave emotion regulation strategies training and practice (n = 3), Dialectical Behavioural Therapy (DBT) skills-based (n = 2), Acceptance and Commitment Therapy (ACT) interventions with mindfulness practices (n = 3), mindfulness based psychoeducation (n = 1), and adaptations of Cognitive Behavioural Therapy (CBT) with the addition of mindfulness (n = 1) or an ‘emotion-focus’ (n = 1). Interventions were primarily delivered in a group setting (n = 8).

Most studies were rated as 'good' (n = 7), or 'fair' (n = 7) in quality, and one was considered 'poor' quality (n = 1). The majority of studies were conducted in Europe (n = 8), with the remainder conducted in North America (n = 4) and Asia (n = 3).

Table 2

Overview of study characteristics and findings for studies investigating sleep loss and emotion regulation (n = 4)

Citation	Design/Intervention	N	Sample characteristics M age (SD), gender	Emotion regulation measure(s)	Comment on findings	Quality rating
Parsons and Young (2022)	Between-subjects 7-night baseline followed by 7-night 90min sleep extension vs sleep as usual	a) 38 intervention b) 34 control	Young adults reporting consistently sleeping <7 hours a night in the past 2 weeks. a) 20.61(1.81) years, 81.6% female b) 20.85(1.74) years, 85.3% female	Survey of 3-items from RPA (emotion-focus, self-focus, dampening) and 2-items from ERQ (cognitive reappraisal and suppression) reported via ESM at pseudorandom timepoints 6x daily.	No main effect of condition and no interaction effect of condition*week across all ER models.	Poor
Boon et al. (2023)	Within-subjects single-night sleep fragmentation (waking at 80min intervals) vs sleep as usual	69	Undergraduates. 20.86(2.88) years, 91.3% female	Survey assessing frequency of ER strategies used (cognitive reappraisal, distraction, acceptance, suppression, rumination, self-criticism) assessed at 4x 3h timepoints across day following sleep condition.	No significant difference in use of cognitive reappraisal, acceptance, self-criticism and suppression between sleep conditions, but higher use of rumination and distraction following sleep fragmentation. Increased use of self-criticism and rumination, and lower acceptance, associated with higher negative affect, with rumination mediating this relationship. Higher use of cognitive reappraisal and acceptance associated with higher scores in positive affect, and higher use of distraction and suppression associated with lower positive affect, with no significant effects found for mediation.	Fair
Campbell (2023)	Between-subjects single-night full sleep deprivation vs sleep as usual	a) 38 intervention b) 38 control	Undergraduates. a) 19.21(1.40) years, 60.5% male b) 19.08(1.12) years, 42.1% male	S-DERS and CERQ reappraisal subscale following sleep condition.	No significant differences in self-reported reappraisal or state ER difficulties across sleep conditions.	Fair
Reid et al. (2023)	Between-subjects single-night sleep continuity disruption (waking at 60min intervals) vs uninterrupted sleep in laboratory setting	a) 27 intervention b) 24 control	Good sleepers. a) 24.28(8.84) years, 74.1% female b) 23.08(3.34) years, 75.0% female	S-DERS at baseline and 4x 2h timepoints following sleep condition.	No significant group effect observed for next-day state ER difficulties.	Good

CERQ = Cognitive Emotion Regulation Questionnaire (Garnefski & Kraaij, 2006); ER = Emotion Regulation; ERQ = Emotion Regulation Questionnaire (Gross & John, 2003); ESM = Experience-Sampling Method; RPA = Responses to Positive Affect Questionnaire (Feldman et al., 2008); S-DERS = State Difficulties in Emotion Regulation Scale (Lavender et al., 2020).

Table 3

Overview of study characteristics and findings for studies investigating psychotic experiences and emotion regulation (n = 11)

Citation	Design/Intervention	N	Sample characteristics M age (SD), gender	Emotion regulation measure(s)	Psychosis measure(s)	Comment on findings	Quality rating
Khoury et al. (2015)	Uncontrolled 8-session combined third-wave ER strategies group	12	Individuals with early psychosis. 29.08(8.13) years, 66.6% male	CERQ and FMI-short at baseline, post-treatment and 3-month follow-up.	BPRS at baseline, post-treatment and 3-month follow-up.	Some improvements in adaptive ER skills use over time that did not reach significance. No significant change in mindfulness score. Some improvement in total BPRS score post-intervention, not maintained at follow-up or reaching significance.	Fair
Lam et al. (2020)	RCT 8-session group mindfulness-based psychoeducation for ER vs TAU	a) 24 intervention b) 22 control	Individuals with schizophrenia spectrum diagnosis. a) Age 25-34 (4.2%), age 35-44 (8.3%), age 45-54 (41.7%), age 55+ (45.8%), 75.0% female b) Age 25-34 (13.6%), age 35-44 (13.6%), age 45-54 (45.5%), age 55+ (27.3%), 77.3% female	ERQ, SRRS and FFMQ-SF at baseline, post-treatment and 3-month follow-up.	PSYRATS at baseline, post-treatment and 3-month follow-up.	Significant reduction in rumination and increased total mindfulness across time. Interaction effect of reappraisal no longer significant after adjusting model for covariates. No significant differences found for rumination or expressive suppression over time. Significant effects of time on overall psychotic symptoms, hallucinations, physical characteristics, controllability and cognitive interpretation of hallucinations at follow-up. No significant effects of time on emotion characteristics of hallucinations and delusion subscales.	Good
Mehl et al. (2021)	RCT 6-month individual emotion-focused CBT for delusions vs WL control	a) 35 intervention b) 29 control	Individuals with delusions in the context of a psychotic disorder. a) 36.74(10.49) years, 48.6% female b) 36.66(10.69) years, 34.5% female	ERSQ and PTQ at baseline, post-intervention and 6-month follow-up.	PSYRATS delusions subscale, PDI-21, PANSS and PC at baseline, post-intervention and 6-month follow-up.	No effect found for ER mediators however scores improved across time. No significant differences between groups for improvements in PSYRATS delusions, PDI total, or PANSS scales across time aside from significant improvement in PANSS general psychopathology scale in treatment group.	Good

Ryan et al. (2021)	Uncontrolled 8-session group ER skills teaching	55	Individuals with schizophrenia spectrum diagnosis. 36.08(12.78) years, 60.0% male	DERS and SMQ at baseline, pre-intervention, post-intervention and one-month follow-up.	PSYRATS at baseline and one-month follow-up.	Significantly reduced difficulties with ER at post-intervention and follow-up compared to pre-intervention, and significant increase in mindfulness at both timepoints. Significant reductions in hallucination severity and distress, and delusional severity and distress, from pre-intervention to follow-up. Regression analyses showed that improvements in mindfulness, but not ER, were associated with reduced hallucination severity and distress, with no significant impact on delusions.	Good
Silva et al. (2021)	Single-case ABA 8-session ER skills rehearsal for paranoia	7	Individuals that met criteria for diagnosis of schizophrenia or schizoaffective disorder. 40.57(10.5) years, 3 male 3 female 1 non-binary	DERS at baseline, intervention and withdrawal phases every other day.	PC-state version at baseline, intervention and withdrawal phases every other day.	6 of 7 participants showed reductions in ER difficulties over time. 5 of 7 participants showed reductions in paranoia over time. Effects not always maintained at follow-up.	Fair
Khakbaz et al. (2022)	Single-case AB 15-session ACT	5	Individuals with schizophrenia spectrum diagnosis. 38.4(2.6) years, 3 male 2 female	DERS at 3x baseline timepoints, 5x intervention timepoints and 2x timepoints at 3-month follow-up.	PANSS at 3x baseline timepoints, 5x intervention timepoints and 2x timepoints at one-month follow-up.	4 of 5 participants showed reductions in ER difficulties over time. 4 of 5 participants showed reductions in symptoms over time. Effects were reduced at follow-up.	Fair
Simonson (2022)	Uncontrolled 12-session group DBT skills training	21	Outpatient transdiagnostic patients, subsample with elevated symptoms of psychosis, enrolled in DBT programme. 32.43 years, 76.2% female	DERS and DBT-WCCL at pre- and post-intervention.	SCL-90 merged psychoticism and paranoid ideation subscales at pre- and post-intervention.	Significant reduction in ER difficulties with medium effect, significant improvements in use of DBT skills with large effect, and significant decrease in use of dysfunctional coping strategies with medium effect, following treatment. Significant reduction in psychosis symptoms with large effect following treatment.	Fair

Von Hardenberg et al. (2022)	Uncontrolled 8-session ACT and mindfulness group	27	Inpatients with early psychosis. 23.33(4.83) years, 40.7% female	ERQ, ERI, ERSQ and SCS at baseline, 8-weeks post-intervention and 16-week follow-up.	PANSS, PC, PSYRATS and PDI-21 at baseline, 8-weeks post-intervention and 16-week follow-up.	Significant improvement in ER difficulties with small effect. No significant differences found for measures of emotion-specific regulation, or for subscales of cognitive reappraisal or expressive suppression. Significant improvement over time in general psychosis symptoms (total PANSS) and positive symptoms at all timepoints. Improvements across time for multiple scales measuring delusions, hallucinations, and negative symptoms.	Fair
Weintraub et al. (2023)	RCT 9-session remote group Mindfulness-Based Cognitive Therapy (MBCT) vs remote group CBT, both with parallel parent group.	a) 34 MBCT b) 32 CBT	Adolescents with mood or attenuated psychosis symptoms. a) 15.2(1.3) years, 73.5% female b) 15.1(1.5) years, 59.4% female	DERS at baseline, post-treatment and 3-month follow-up.	PQ-B at baseline, post-treatment and 3-month follow-up.	Both groups showed improvements in ER difficulties over time. CBT group associated with greater sustained improvements compared to MBCT at follow-up. Improvements in attenuated psychosis symptoms were comparable across groups.	Good
Wittkamp et al. (2023)	RCT 8-session group affect-regulation training (ART) vs self-help bibliotherapy (BT)	a) 68 ART b) 70 BT	Transdiagnostic 'at-risk- sample (distressing subclinical psychotic, depression and anxiety symptoms). a) 35.58(11.55), 61.8% female b) 37.25(12.59), 67.1% female	ERSQ measured bi-weekly during intervention, at 6- and 12-month follow-up, and ERSQ-ES by ESM with 5x timepoints over 6 days at pre- and post-intervention.	CAPE measured bi-weekly during intervention and at 6- and 12-month follow-up.	Greater improvements in emotion-specific ER for treatment condition compared to control group with a significant interaction effect of condition x time. No significant interaction effect for ERSQ at post-intervention. No significant interaction effects for psychotic-like experiences. Significant effect of time on CAPE positive symptom frequency and distress, and negative symptom frequency and distress, with both groups showing improvements.	Good
Zoromba et al. (2024)	RCT 6-session group ACT vs TAU	a) 33 ACT b) 32 TAU	Inpatients with primary psychoses. a) Age 20-30 (45.5%), age 30-40 (42.4%), age 40+ (12.1%), 63.3% male b) Age 20-30 (65.6%), age 30-40 (28.1%), age 40+ (6.3%), 62.5% male	DERS-16 and AAQ-II at pre- and post-intervention.	PANSS at pre- and post-intervention.	ACT group significantly improved both ER difficulties and psychological flexibility scores compared to TAU at post-intervention. ACT group showed significant reduction in psychosis symptoms compared to TAU at post-intervention.	Good

AAQ-II = Acceptance and Action Questionnaire version 2 (Bond et al., 2011); ACT = Acceptance and Commitment Therapy; BPRS = Brief Psychiatric Rating Scale (Ventura et al., 1993); CAPE = Community Assessment of Psychic Experiences (Stefanis et al., 2002); CBT = Cognitive Behavioural Therapy; CERQ = Cognitive Emotion Regulation Questionnaire (Garnefski & Kraaij, 2006); DBT-WCCL = DBT Ways of Coping Checklist (Neacsiu et al., 2010); DERS = Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004); DERS-16 = Difficulties in Emotion Regulation Scale – 16 item version (Bjureberg et al. 2016); ER = Emotion Regulation; ERI = Emotion Regulation Inventory (König, 2011); ERQ = Emotion Regulation Questionnaire (Gross & John, 2003); ERSQ = Emotion Regulation Skills Questionnaire (Berking & Znoj, 2008); ERSQ-ES = Emotion Regulation Skills Questionnaire - Emotion Specific (Ebert et al., 2013); ESM = Experience-Sampling Method; FFMQ-SF = Five Facet Mindfulness Questionnaire Short-Form (Baer et al., 2006); FMI-short = Freiburg Mindfulness Inventory short-version (Walach et al., 2006); PANSS = Positive and Negative Syndrome Scale (Kay et al., 1987); PC = Paranoia Checklist (Freeman et al., 2005); PDI-21 = Peters et al Delusions Inventory (Peters et al., 2004); PSYRATS = Psychotic Symptom Rating Scale (Haddock et al., 1999); PQ-B = Prodromal Questionnaire - Brief (Loewy et al., 2011); PTQ = Perseverative Thinking Questionnaire (Ehring et al., 2011); RPA = Responses to Positive Affect Questionnaire (Feldman et al., 2008); SCL-90 = Symptom Checklist-90 Revised (Derogatis, 1977); SCS = Self-Compassion Scale (Neff, 2003); SMQ = Southampton Mindfulness Questionnaire (Chadwick et al., 2008); SRRS = Short Ruminative Response Scale (Treynor et al., 2003); TAU = Treatment as Usual; WL = Waitlist.

(RQ1) Sleep loss and emotion regulation (n = 4 studies)

Difficulties in emotion regulation

Two studies investigated the impact of experimental sleep manipulation on state emotion regulation changes in non-clinical samples, focusing on momentary difficulties with acceptance, modulation, awareness, and clarity of emotions. Campbell (2023) exposed participants to a full night of sleep deprivation, while Reid et al. (2023) implemented sleep continuity disruption in a lab setting. Neither study detected significant differences in next-day state emotion regulation difficulties following sleep manipulation, despite the latter study observing a significant increase in state anxiety and decrease in positive affect.

However, the methodological limitations identified in the quality appraisal reduce confidence in these null findings. For instance, Campbell (2023) assessed state emotion regulation at a single timepoint and made comparisons at the group level post-intervention, which may not adequately capture the dynamic nature of within-person state changes. This study reported being appropriately powered to detect medium-size effects, however as the observed effect size was smaller than anticipated this raises concerns about the study's sensitivity to detect more subtle effects from sleep loss. Although Reid et al. (2023) effectively detected large effects for some measures, the absence of significant effects on emotion regulation are difficult to interpret with confidence. It is possible that this measure is more resistant to short-term changes, or alternatively that the study was underpowered to detect smaller, yet meaningful effects given that the a priori power analysis was based on detecting moderate-to-large effects. Additionally, both studies employed between-subjects designs, which may have introduced variability due to individual differences, such as habitual sleep quality and preferred emotion regulation approaches.

There are also challenges to the ecological validity of these findings. Reid et al. (2023) conducted their experiment in a controlled lab environment where participants were not exposed to the usual life stressors that may require modulation of their emotional response. In contrast, Campbell (2023) exposed participants to a full night of sleep deprivation, which is not typical for insomnia that usually consists of limited or interrupted hours of sleep.

Emotion regulation strategies

Three studies examined changes in emotion regulation strategy use following sleep manipulation. Campbell (2023) utilised a validated subscale to measure reappraisal, while the other two studies selected items from validated measures to create a survey assessing the

frequency of various adaptive and maladaptive strategies used. Following a full night of sleep deprivation, Campbell (2023) found no significant effects of time, condition, or interaction effects of sleep length on reappraisal. While the use of a validated measure adds strength to this finding, as the study was underpowered to detect smaller more subtle effects induced by a single night of exposure to sleep loss, this reduces confidence in the null results reported.

Parsons and Young (2022) investigated the effects 90-minute daily sleep extension over one week for individuals who reported consistently sleeping less than seven hours, assessing emotion regulation strategy use at six daily pseudorandom timepoints throughout the intervention week. They found no main effect of condition or interaction effects for changes to the modulation of positive affect using emotion-focus, self-focus or dampening strategies. Additionally, there were no significant effects found for changes to use expressive suppression or cognitive reappraisal, consistent with Campbell (2023). While the longer study period used by Parsons and Young (2022) may have increased their sensitivity to detect changes due to sleep manipulation, this study received a 'poor' quality rating due to the absence of true randomisation and use of listwise deletion rather than imputation methods for handling missing data, both of which raise concerns about potential bias in results. Furthermore, assessing emotion regulation using a single item for each strategy may have limited their ability to reliably detect changes.

Another study that assessed strategy use via survey was a within-subjects sleep fragmentation study by Boon et al. (2023). In line with the previous studies, they also found no significant change in the use of cognitive reappraisal between sleep conditions, as well as no significant change in acceptance, self-criticism or suppression. However, participants reported a significant increase in their use of rumination and distraction following sleep fragmentation. Boon et al. (2023) also investigated whether the use of these strategies predicted changes in affect. They showed that increased use of self-criticism and rumination, as well as reduced acceptance, were associated with higher negative affect. Additionally, the increased use of cognitive reappraisal and acceptance, along with the reduced use of distraction and suppression, all predicted increased positive affect. Furthermore, rumination was found to mediate the relationship between sleep fragmentation and negative affect, with no other significant mediation effects found. While these findings offer some evidence that fragmented sleep may influence the use of certain maladaptive strategies, confidence in the results is tempered by methodological limitations. As with Parsons and Young (2022), Boon et al. (2023) used a self-constructed survey rather than a validated emotion regulation measure, which was noted as a key weakness in the quality appraisal. This limits both internal validity and

comparability across studies. Nonetheless, Boon et al.'s use of a within-subjects design, and authors reporting that their final sample size meant that they were sufficiently powered to detect small-to-medium effects, adds strength to their conclusions.

Across these studies, findings for the influence of sleep manipulation on the use of emotion regulation strategies were mixed and should be interpreted with some caution. While each study contributes unique insights, common limitations such as inconsistent use of validated measures, and underpowered designs for detecting small effects, reduce the certainty of both positive and null results. The methods of sleep manipulation also varied across studies which further complicates comparability, and raises concerns about the generalisability of findings from full sleep deprivation or sleep extension to understanding the effects of insomnia.

(RQ2) Psychotic experiences and emotion regulation (n = 11 studies)

Difficulties in emotion regulation

Nine studies included a measure of context-specific emotion regulation difficulties or assessed participants' overall acceptance, modulation, awareness and clarity of emotions. Von Hardenberg et al. (2022) evaluated the effectiveness of an 8-session Acceptance and Commitment Therapy (ACT) and mindfulness group, finding no significant changes in emotion-specific regulation of negative or positive affective states, despite significant improvements in overall emotion regulation difficulties at follow-up. In contrast, Wittkamp et al. (2023) found that 8 sessions of affect regulation training led to significant interaction effects for momentary emotion-specific regulation, with greater improvements shown in the intervention group compared to the bibliotherapy group. It should be noted when interpreting these findings that von Hardenberg et al. (2022) received a quality rating of 'fair' and the authors noted that the modest sample sized used for their pilot study may have limited their sensitivity to detect a change through this mechanism. Meanwhile, Wittkamp et al. (2023) were rated as 'good' quality due to many methodological strengths and adequate sample size for detecting medium effects.

All nine studies assessed changes in participants' overall emotion regulation difficulties following intervention. Each study demonstrated significant improvements in participants' overall acceptance, modulation, awareness and clarity of emotions over time, with Zoromba et al. (2024) showing these improvements were significantly greater in the intervention group compared to treatment as usual. However, both Weintraub et al. (2023) and Wittkamp et al. (2023) did not observe significant interaction effects that would indicate greater outcomes for the treatment group relative to the comparator group. Additionally, Ryan et al. (2021) found no

statistical association between participants' improved emotion regulation difficulties and reduced psychotic experiences, while Mehl et al. (2021) found no evidence that emotion regulation difficulties mediated changes in psychosis symptoms. However, most of these studies were pilot studies that lacked a control condition and relied on small sample sizes, which limits their statistical power and reduces the precision of effect estimates. As such, significant findings may reflect overestimated effects and should be viewed as preliminary until replicated in larger, methodologically robust trials.

Regarding psychotic experiences, Mehl et al. (2021) reported marginally significant interaction effects for improvements in general psychopathology but no significant effects on positive and negative symptoms, delusions, or paranoia. Similarly, Wittkamp et al. (2023) found no significant interaction effects for psychotic-like experiences; however, both groups showed significant improvements over time in all scales of positive and negative symptom frequency and distress. Of the remaining seven studies, all reported significant improvements in measures of psychotic experiences following intervention. Five studies demonstrated reductions in measures of overall psychotic symptoms, while others examined specific experiences, demonstrating significant improvements in both positive and negative symptoms (n = 1), paranoia (n = 1), severity of hallucinations (n = 2), delusions (n = 2), and distress related to experiences of both hallucinations (n = 1) and delusions (n = 1).

These studies offer preliminary support for the potential benefit of emotion-targeted interventions for alleviating psychotic experiences. However, the variation in quality ratings should be held in mind when interpreting these findings, and it should be noted that improvements were not consistently sustained at follow-up or found to be superior to comparator groups. While tests examining associations between outcomes were limited, reported analyses did not demonstrate an association between measures of emotion regulation difficulties and psychotic experiences or indicate mediation by emotion regulation, and therefore larger trials are required before making causal or inferential conclusions.

Emotion regulation strategies

Six studies examined the impact of emotion-targeted interventions on regulation strategy use. Simonson (2022) found that following a 12-session DBT skills group, participants reported a significant reduction in their overall use of maladaptive coping strategies, alongside significant improvements in their frequency of skill use with large effect. In contrast, Khoury et al. (2015) found that a combined third-wave emotion regulation skills group did not significantly improve participants' overall adaptive emotion regulation skill use with medium effect. Both studies

were rated as 'fair' quality and relied on small sample sizes meaning their findings were prone to imprecision. Additionally, the sample used by Simonson (2022) were a sub-sample of transdiagnostic outpatients with elevated psychotic symptoms already enrolled in a DBT skills programme which may limit generalisability to clinical psychosis groups.

Some studies examined changes to the use of specific emotion regulation strategies. Among those investigating mindfulness (n = 3), Khoury et al. (2015) reported no significant change in scores following intervention, while both Lam et al. (2020) and Ryan et al. (2021) demonstrated significant improvements in self-reported mindfulness. Other studies explored adaptive emotion regulation strategies, including Zoromba et al. (2024) who found that a 6-session group ACT intervention significantly improved participants' psychological flexibility, a construct measured as one's ability to accept difficult emotions while staying committed to goals (Bond et al., 2011). Lam et al. (2020), Ryan et al. (2021), and Zoromba et al. (2024) were all rated as 'good' quality, reflecting sound methodology and sufficient power to detect moderate effects, which strengthens confidence in their findings.

Von Hardenberg et al. (2022) found no significant impact of an ACT group on changes in self-compassion or cognitive reappraisal. Similarly, while Lam et al. (2020) initially observed a significant interaction effect for reappraisal, this effect became non-significant after adjusting for covariates. Regarding maladaptive strategies, neither Lam et al. (2020) nor Von Hardenberg et al. (2022) observed significant changes in expressive suppression following intervention. Findings on the impact of interventions on ruminative thinking were mixed. Mehl et al. (2021) observed reductions over time, though no significant differences were found between the standard CBT and emotion-focused CBT groups. Similarly, Lam et al. (2020) reported a significant reduction in participants' self-reported rumination over time but found no significant interaction effects, indicating no additional benefit of the intervention over treatment as usual. Given that many of the included studies were small pilot trials, null findings cannot be assumed to indicate no effect as the limited statistical power may obscure true intervention effects.

With respect to psychotic experiences, Simonson (2022) demonstrated significant reductions in merged psychotic and paranoid symptom subscales following treatment with large effect. Von Hardenberg et al. (2022) demonstrated significant improvements across multiple scales of psychotic experiences at 16-week follow-up, including general, and positive symptoms, delusions, hallucinations, and both frequency and conviction in paranoid ideation, with no significant change to negative psychotic symptoms. Additionally, Zoromba et al. (2024) reported significant decreases in combined positive and negative symptoms, and subscales relating to

general psychopathology and negative symptoms, but no significant change to positive symptoms. In contrast, Khoury et al. (2015) observed some improvement in psychotic symptoms that did not reach significance. Mehl et al. (2021) reported a significant interaction of time and condition for general psychopathology, but no significant interactions were found for other scales, and similarly Lam et al. (2020) showed significant effects of time on overall psychotic symptoms, severity of hallucinations and related scales at follow-up, however no significant interaction effects. Finally, Ryan et al. (2021) demonstrated significant reductions in hallucination severity and distress, as well as in delusional severity and distress. Notably, improvements in hallucination severity and distress were associated with higher mindfulness scores, though no association was found for delusions scales.

These studies provide mixed evidence regarding the potential efficacy of emotion-targeted interventions for improving psychotic experiences through changes in emotion regulation strategy use. However the quality of these studies varied, and many were small pilot studies which would mean that conclusions about causal mechanisms would be premature. Furthermore, several studies lacked a control group (n = 3) or had either no follow-up (n = 2) or only short follow-up periods (n = 4), limiting conclusions about the sustained impact of these interventions. Variability in intervention design and sample characteristics further complicates the generalisability of findings, highlighting the need for larger, controlled trials with extended follow-up to determine the long-term efficacy of these approaches.

Quality and relevance appraisal

Included studies differed in their methodological design and statistical power, meaning that the strength and certainty of their conclusions vary considerably. For instance, limitations of studies included reliance on small sample sizes, high attrition rates, lack of control groups, and limited ecological validity which limits the generalisability of findings. The latter was especially true for the sleep studies that manipulated sleep through total sleep deprivation or sleep extension, which has limited relevance for understanding effects of sleep typical for insomnia on emotion regulation. Additionally, all four sleep manipulation studies were with youth samples, and overall, there was a paucity of studies that were eligible for inclusion, limiting the ability to draw conclusions about a relationship between sleep and emotion regulation.

The emotion-targeted intervention studies varied greatly by treatment modalities and sample characteristics, and many lacked detailed intervention descriptions, which hinders the identification of potential mechanisms of change. Many were pilot or feasibility studies with small sample sizes and no a priori power analyses, which limited their ability to detect small-to-

moderate effects or draw generalisable conclusions. Furthermore, the frequent absence of a comparison group and lack of long-term follow-up limits conclusions about sustained effects or attribution of outcome improvements to the interventions themselves. Finally, we were unable to identify a study that investigated emotion regulation in association with both sleep loss and psychotic experiences, which prevents firm conclusions about a causal mechanism linking these variables.

Discussion

This systematic review sought to synthesise empirical studies examining changes in emotion regulation, through the experimental manipulation of sleep in non-clinical populations (RQ1) and in relation to changes in psychotic symptoms in response to emotion-targeted interventions (RQ2). Overall, findings suggest that sleep loss does not significantly impact cognitive reappraisal use or state emotion regulation difficulties, though it may influence the use of rumination and distraction strategies. However, due to methodological limitations the absence of significant effects should not be taken as strong evidence against the influence of sleep on emotion regulation and these findings should be interpreted cautiously. The intervention studies targeting emotion regulation consistently demonstrated that improvements in emotion regulation difficulties coincided with reduced psychotic symptoms, and there was also preliminary evidence that increased mindfulness and acceptance towards experiences may be related to symptom improvement. However, as many studies were pilots with small samples and no comparator group, and tests of statistical association between outcomes were limited, causal or inferential conclusions about specific mechanisms remains unclear. The potential role of emotion regulation as a bridge between sleep loss and psychotic symptoms is yet to be established, as the studies in this review do not provide sufficient evidence to support robust conclusions.

The availability of studies for investigating a mechanism linking sleep loss with emotion regulation was limited and varied by sleep manipulation methods used. This variability should be considered when comparing findings. For example, sleep quality has been demonstrated to have superior predictive value over sleep quantity when assessing next-day mood and emotional functioning (Walker & van der Helm, 2009). The absence of significant effects in some studies may be attributed to most experiments manipulating sleep for only a single night, whereas sleep quality has been demonstrated to be more consistently associated with cognitive reappraisal over a week than within a 24-hour period (Mauss et al., 2013). Only Boon

et al. (2023) demonstrated significant differences in emotion regulation strategies assessed, potentially due to their within-subjects design, enhancing sensitivity to detect changes. However, the absence of a validated emotion regulation measure warrants cautious interpretation and comparison of findings. Their study showed that rumination and distraction use were both increased following sleep fragmentation, consistent with findings comparing strategy use in individuals with psychosis, whereby rumination and distraction use is elevated compared to controls (Ludwig et al., 2019), indicating a potential shared mechanism. Changes in strategies employed also differentially predicted changes to positive and negative affect, and the latter of these has been shown to mediate the relationship between sleep loss and psychotic experiences (Reeve et al., 2018). Altogether these findings provide preliminary indications that sleep loss may contribute to psychotic experiences via maladaptive emotion regulation strategy use rather than changes to state emotion regulation difficulties. However, given the limited scope and methodological constraints of the current evidence, further research is required with larger samples and validated measures to clarify these relationships.

All emotion-targeted interventions that investigated changes to overall acceptance, modulation, awareness and clarity of emotions demonstrated improvements over time, alongside alleviation of psychotic experiences (n = 9). This is consistent with previous research highlighting elevated emotion regulation difficulties in psychosis groups (Almuqrin et al., 2023; Fett et al., 2022; Lawlor et al., 2019). Zoromba et al. (2024) found that improvements were significantly greater in the intervention group compared to treatment as usual, whereas Weintraub et al. (2023) and Wittkamp et al. (2023) did not observe a significant difference between treatment and comparison groups, reflecting uncertainty in the presence of this relationship across studies. Furthermore, studies investigating relationships between outcomes did not find a significant association between changes in emotion regulation difficulties and psychotic experiences (Ryan et al., 2021), or mediation by emotion regulation (Mehl et al., 2021). There was some indication, however, that improvements in emotion regulation difficulties coincided more often with reductions in positive symptoms, such as hallucinations and delusions, than with negative symptoms. Regarding the potential role of emotion regulation difficulties in linking sleep loss with psychotic experiences, state difficulties appeared unaffected by sleep manipulation, meaning the overall findings do not support this perspective. However, since intervention studies assessed emotion regulation difficulties using trait-based measures, direct comparisons with state-level measures cannot be made, further complicating the ability to draw definitive conclusions.

In line with findings from the sleep manipulation studies, von Hardenberg et al. (2022) and Lam et al. (2020) both observed no significant change to the use of expressive suppression or cognitive reappraisal, despite reporting reduced psychotic experiences following intervention. This was unexpected given prior longitudinal research linking these strategies with symptom severity (Chapman et al., 2020; Kimhy et al., 2020; Nittel et al., 2018; Westermann & Lincoln, 2011), however suggests that based on these findings these strategies may have less direct influence than other regulatory processes in linking sleep difficulties and psychosis. Boon et al. (2023) showed that rumination use was increased following sleep manipulation. However, the effects of interventions on this strategy were mixed, with Mehl et al. (2021) and Lam et al. (2020) both reporting reductions in rumination over time alongside symptom alleviation, but these changes were not significantly greater than in control groups. As a result, the role of these strategies in linking sleep loss to psychotic experiences remains unclear.

Comparisons between related measures across sleep and intervention studies allows for cautious hypotheses about potential bridging mechanisms. For instance, Boon et al. (2023) found that self-criticism was unaffected by sleep manipulation, and Von Hardenberg et al. (2022) observed no significant change to self-compassion following intervention. Additionally, Boon et al. (2023) found that use of acceptance remained unchanged following sleep disruption, which contrasts with findings from Zoromba et al. (2024) who showed that psychological flexibility, involving acceptance of emotional states, was significantly improved along with psychosis symptoms following intervention. Boon et al. (2023) also demonstrated a significant increase in use of distraction following sleep fragmentation. While no intervention studies assessed this strategy directly, Lam et al. (2020) and Ryan et al. (2021) both reported that mindful approaches to experiences were employed more frequently by participants, alongside improvements in psychosis symptoms over time. Ryan et al. (2021) also reported a statistical association between higher mindfulness scores and reduced hallucination frequency and distress. These findings indicate that changes in use of mindfulness and acceptance strategies may link sleep loss with psychotic experiences. However, due to the variability in the measures used and the specific strategies investigated across studies, these potential bridging mechanisms cannot be identified with certainty. Consequently, these findings highlight the need for replication in larger, well-powered studies employing consistent and validated measures to better clarify these relationships.

Although improvements in overall psychotic symptoms were observed across studies, there were limited tests examining interactions between symptoms and emotion regulation measures, or reporting of scales for specific psychotic experiences, that would support with

identification of detailed mechanisms of change. For instance, Zoromba et al. (2024) noted no significant change in positive symptoms while negative symptoms improved, whereas von Hardenberg et al. (2023) noted the reverse. Given the similarities in intervention approaches and participant groups, this discrepancy may be attributed to differences in the emotion regulation mechanisms targeted. Zoromba et al. (2024) highlighted key intervention components aimed at alleviating negative symptoms, such as enhancing distress tolerance to support meaningful goal pursuit and social engagement. Ryan et al. (2021) was the only group intervention that incorporated individual formulations, which may have enhanced participants' awareness and application of skills (Redhead et al., 2015). Additionally, Mehl et al. (2021) noted therapist reports reflecting an emphasis on emotional stability over regulation skills during sessions and speculated that greater benefits might have been observed if regulation skills were prioritised. Similarly, Weintraub et al. (2023) reflected on parent's reporting a preference for the traditional CBT approach, which may have influenced their role in facilitating skills practice outside of sessions. Further research is needed to examine changes in specific psychotic experiences, and their relationship with emotion regulation measures, that provide detailed intervention descriptions. This would enhance our understanding of emotion regulation's role within a causal mechanism linking sleep loss to psychotic experiences.

Strengths and limitations

This systematic review provides the first synthesis of empirical studies of experimental sleep manipulation, and emotion-targeted interventions that assess changes in psychotic experiences, that examine emotion regulation effects. We conducted a comprehensive search across both scientific and grey literature, which enhances the validity of our findings. Most included studies were published in the last five years, which has allowed us to begin to address knowledge gaps relating to the connection between emotion regulation, sleep loss, and therapeutic outcomes. This has furthered our understanding of emotion regulation as a potential bridging mechanism between sleep disruption and psychosis.

The most common reason for exclusion was due to measures of emotion regulation only assessing a single domain. This distinction was made to improve comparability across studies as well as to provide a broader and more integrative view of emotion regulation that aligns with relevant models. However, this also limited the study pool and may have influenced our ability to explore more nuanced aspects of emotion regulation or specific strategies within our understanding of a causal mechanism. We also excluded studies that assessed changes to emotion regulation ability, rather than choice and use of strategies. As a result, surprisingly few

studies remained to address RQ1, which in combination with the heterogeneous assessment of changes to use of specific strategies across studies, limited the conclusions that could be drawn.

Psychological intervention studies were excluded if they were not primarily focused on emotion regulation. However, many studies provided only brief descriptions of the interventions, which may have led to the exclusion of studies where emotion regulation was an implicit component but not explicitly addressed by the authors. Additionally, many of the included studies lacked a comparison group and used small sample sizes, reducing the ability to draw inferential conclusions and the translational value of the findings. A couple of authors noted that these limitations were due to guidance for groups running during the COVID-19 pandemic. Finally, some studies evaluated outcomes with little to no long-term follow-up, potentially missing delayed changes in psychotic experiences that may emerge with consistent and sustained practice of emotion regulation skills.

Implications for future research and clinical practice

Future research should examine how specific emotion regulation strategies moderate or mediate the effects of sleep loss on psychotic experiences. A more standardised approach to measuring emotion regulation across studies, as well as investigations that examine sleep loss and psychotic experiences together, would provide deeper insight into the mechanisms linking insomnia and psychosis. This could be achieved through experimental sleep manipulation studies, or targeted sleep interventions, that assess changes in both emotion regulation and psychotic experiences. Furthermore, many of the included studies were pilot or feasibility studies which are not suitable for determining efficacy as they are underpowered to detect small effects (Leon et al., 2011). Replicating the studies included in this review with larger well-powered trials using control groups would strengthen the robustness of conclusions drawn. As most studies were published recently, this reflects growing scientific interest in this area that is likely to reveal valuable insights in the coming years.

As research investigating emotion regulation as a treatment mechanism develops, the cautious comparisons made in this review between intervention modalities and components could guide future investigations to focus on the potentially most efficacious drivers for alleviating psychotic experiences. Given that many interventions were delivered in group formats, the findings support the feasibility of group-based skills training, which could facilitate earlier intervention and greater accessibility compared to individual approaches if proven effective in larger trials. Additionally, it is important to distinguish between statistically significant and clinically

significant change when interpreting these findings. Studies that assess changes in goal ratings and participant feedback on intervention utility may offer a more comprehensive understanding of personally meaningful outcomes, which symptom-based measures alone may not fully capture.

Conclusion

This review provides preliminary support for the presence of relationships for emotion regulation with both sleep loss and severity of psychotic experiences, however the role of emotion regulation in potentially bridging sleep loss and psychotic experiences remains unclear. Due to variation in the quality and availability of studies, further research, particularly larger, well-powered trials that examine these outcomes together and investigate statistical associations or mediation effects, is required to strengthen conclusions and more clearly elucidate the specific mechanisms linking sleep loss to psychotic experiences.

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Conflict of interest

None to declare.

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Chapter 3

Bridging Chapter

Systematic review findings

The systematic review reported in the previous chapter concluded that, based on the currently available evidence, the role of emotion regulation in bridging sleep loss and psychotic experiences remains unclear. Additionally, findings revealed a gap in the literature due to the paucity of research investigating changes to emotion regulation difficulties and strategy use following experimental sleep manipulation, which incites challenges when investigating a causal relationship. Findings from the included sleep manipulation studies indicated that changes to emotion regulation choice and strategy use may be difficult to observe directly. This highlights the value of examining the potential moderating or mediating role for emotion regulation processes in linking sleep loss with psychotic experiences. Alternatively, investigating regulatory processes outside of Gross' extended process model of emotion regulation (1998, 2001, 2002, 2015), which informed the inclusion of studies in the systematic review, may reveal a more significant influence of sleep loss.

Studies that investigated changes to emotion regulation and psychotic symptoms following therapeutic interventions, where emotion regulation was targeted as the mechanism of change, revealed that improvements in both outcomes often co-occurred. This suggests a negative correlation between adaptive emotion regulation and psychotic symptom severity, highlighting the potential role of emotion regulation processes in the aetiology and maintenance of psychosis. However, few studies investigated a statistical association between emotion regulation and psychotic experiences or examined specific experiences (e.g. hallucinations) separately. Additionally, no study investigated changes in sleep or its role in this relationship. Further research is required to explore the association between sleep loss and psychotic experiences, as well as the potential mediating role of emotion regulation processes in this relationship. Experimental sleep restriction studies investigating these processes could provide valuable insights into the causal relationship between sleep loss and the onset of psychosis.

Alternative affective processes that may link sleep loss and psychotic experiences

'Affect' refers to the broad spectrum of emotional experiences, and 'affective processes' are the cognitive and physiological mechanisms involved in the interpretation, experience, regulation and expression of these experiences. These processes typically accompany affective experiences that occur along two dominant dimensions, otherwise known as 'positive' and

'negative' affect. Positive affect refers to emotional states that are pleasant or enjoyable and generally associated with wellbeing and positive experiences. While negative affect encompasses unpleasant emotional states associated with distress or discomfort (Díaz-García et al., 2020). Individuals may use emotion regulation strategies to modify the intensity, duration, or expression of positive and negative affective states to adapt to their environmental context or achieve their aims (Aldao & Plate, 2018).

Affect intolerance

Previous research investigating the relationship between sleep and emotion regulation has primarily focused on cognitive strategies, such as reappraisal and suppression (Vandekerckhove & Wang, 2017). Straus et al. (2024) propose that strategies like these, that are employed consciously or deliberately, are influenced by sleep in a less consistent way than 'implicit-automatic regulation' strategies, which involve unconscious learned responses. This suggests that investigating cognitive biases or automatic processes related to emotion regulation could provide deeper insights into how sleep disturbances affect emotional functioning, particularly in the context of linking sleep loss to psychotic experiences. Studies with undergraduates have shown a strong correlation between measures of difficulties in emotion regulation and affect intolerance (Stapinski et al., 2014). 'Affect intolerance' refers to the extent to which an individual holds negative beliefs about emotions and makes attempts to avoid or suppress them. Research has shown that individuals who perceive emotions as intolerable are more likely to rely on maladaptive emotion regulation strategies, such as suppression, avoidance, and rumination (Jeffries et al., 2015; Nowak et al., 2021). As a result, negative emotional states may be maintained or exacerbated over time and consequently contribute to the onset of psychopathology (Leyro et al., 2010).

Established cognitive models emphasise the relationship between subjective experience, associated appraisals, and the resulting affect (Beck, 1976), extending to perceptions of the emotional experiences themselves. For example, the 'cognitive model of panic disorder' (Clark, 1986, 1988) outlines that catastrophic misinterpretations of experiential sensations associated with feelings of anxiety are central to the escalation and maintenance of distress. Individual differences in beliefs about emotions being unacceptable or intolerable, and attempts made to control them, are often integral to clinical formulations explaining how psychological distress and maladaptive behaviours are maintained. This is due to the influence that an individual's perceived lack of control over their emotional experiences has in predicting negative psychological wellbeing and outcomes, as well as their likelihood to engage in maladaptive

avoidance strategies (De Castella et al., 2017). In the context of psychosis, individuals with clinical symptomology report believing that emotions are less controllable compared to the general population, and the extent to which these beliefs were held is associated with the frequency of maladaptive emotion regulation strategy use (Berglund et al., 2023). This highlights the potential contributing role of affect intolerance to the severity of psychosis, however an association between affect intolerance and sleep loss remains unclear.

Dissociative experiences

Affect intolerance has been shown through network analysis to be closely linked with dissociative experiences (Černis et al., 2022), with researchers hypothesising that an individual's perceived lack of ability to cope, or distressing appraisals of dissociative experiences, may increase the likelihood of dissociating (Černis et al., 2020). While the precise conceptualisation of dissociation is an ongoing debate among researchers and clinicians, the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) defines it as "a disruption and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control and behaviour" (American Psychiatric Association, 2013, p. 291). Dissociation is understood as a protective process of detachment in response to distressing events, which is adaptive in the short-term but becomes maladaptive when overutilized in the long-term (Lynn, 2005). Elevated dissociative symptoms are associated with emotional dysregulation in clinical samples, including difficulties with emotional awareness and acceptance (Nester et al., 2022). Furthermore, dissociative experiences have been shown to mediate the impact of sleep quality on psychological wellbeing in the general population (Cahill et al., 2025).

The early descriptions of schizophrenia were dissociative in nature, with Bleuler (1911) stating that "emotionally charged ideas or drives attain a certain degree of autonomy so that the personality falls into pieces. These fragments can then exist side by side and alternately dominate the main part of the personality, the conscious part of the patient." (p.143). This connection between dissociation and psychosis has been supported by a meta-analysis, which found a robust association between dissociation and both clinical and non-clinical positive psychotic symptoms, with a larger effect in non-clinical samples (Longden et al., 2020). Dissociation has also been shown to be highly connected to psychotic experiences, and indirectly connected to insomnia, through network analysis (Černis et al., 2021). Additionally, a systematic review of experimental sleep manipulation studies suggested a potential causal link between sleep loss and psychotic-like and dissociative experiences, emphasising the need for

further experimental studies to clarify these mechanisms and strengthen our understanding of causality (Barton et al., 2018). Altogether these findings suggest that dissociative experiences may play a mediating role in linking sleep loss to psychotic experiences.

Overview of empirical paper

The empirical paper presented in Chapter 4 investigates the impact of sleep loss on psychotic experiences following one night of partial sleep restriction in a non-clinical sample. Negative affect, affect intolerance and dissociative experiences are explored as potential mediators in this relationship.

The use of experimental sleep manipulation allows for exploration of a causal role of sleep disruption in the aetiology of psychotic experiences. If demonstrated, this would underscore the significance of sleep difficulties in the early identification and prevention of psychosis symptom development, as well as highlight the therapeutic value of interventions that target sleep or affective processes as a mechanism of change for alleviating psychotic experiences.

Chapter 4

Empirical Paper

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

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Abstract

Background and Hypotheses: Recent evidence suggests that sleep disturbances may precede and predict psychotic experiences, with affective processes mediating this relationship. We hypothesised that one night of sleep restriction in a non-clinical sample would increase self-reported psychotic experiences, with effects mediated by negative affect, affect intolerance and dissociative experiences.

Study Design: A within-subjects, cross-over experimental study was conducted with 58 undergraduate participants, comparing a single night of sleep loss (≤ 4 hours sleep) to a standard night of sleep (≥ 7 hours sleep).

Study Results: Small to large effects of sleep loss were found for increasing negative affect, affect intolerance, dissociative experiences, paranoia, hallucinations, cognitive disorganisation, anhedonia and distress related to experiences. Mediation analyses revealed that both dissociative experiences (46.2%), and combined mediators (89.1%), substantially mediated the effect of sleep loss on paranoia. Dissociative experiences also partially mediated the effect of sleep loss on hallucinations (38.5%). No significant mediation effects were found for the impact of sleep loss on cognitive disorganisation, anhedonia or distress related to experiences.

Conclusions: These findings support a causal role of sleep difficulties in the onset of psychosis, with mediation by negative affect, affect intolerance and dissociative experiences. The results emphasise the importance of further experimental research investigating the relationship between affective processes, particularly dissociative experiences and psychosis, as well as the role of sleep as a transdiagnostic intervention route in clinical populations.

Keywords: insomnia, psychosis, paranoia, hallucinations, aetiology, mechanisms

Introduction

Sleep disturbances are elevated in psychosis groups compared to the general population,[1] with between 36% to 44% meeting criteria for clinical insomnia.[2,3] While traditionally considered a secondary consequence of psychosis, recent reviews suggest that sleep difficulties precede and predict subsequent psychotic symptoms.[4] Furthermore, sleep disturbances contribute to the probability of transition from at risk mental state to first-episode psychosis.[5] Altogether this underscores the need for further research to deepen our understanding of how sleep contributes to the development and maintenance of psychosis by clarifying causal mechanisms.

Changes to negative affect is one established mechanism linking sleep disturbances to psychopathology, with chronic sleep deprivation being a significant predictor for the onset of affective disorders.[6] Reviews of experimental studies in healthy participants show that sleep loss has a greater effect on reducing positive affect than on increasing negative affect.[7] However, experimental studies have shown that negative affect significantly mediates the effect of sleep loss on psychotic experiences in non-clinical participants,[8] as well as its influence on paranoid thinking, as demonstrated through structural equation modelling.[9] These findings align with research linking changes in momentary affect to psychotic symptoms in the general population[10] and individuals at-risk for psychosis.[11]

Negative affect is associated with affect intolerance in non-clinical populations, characterised by beliefs about emotions being threatening alongside attempts to avoid or suppress them.[12] Individuals with psychosis report stronger beliefs in the uncontrollability of emotions, in association with the elevated use of maladaptive emotion regulation strategies (e.g. suppression) and more severe negative symptoms.[13] The use of experiential avoidance and expressive suppression is also linked to positive symptoms, including paranoia,[14,15,16] hallucinations,[17] and distress related to delusions and voice-hearing.[18,19] Similarly, individuals with sleep difficulties report challenges in accepting emotions and maintaining goal-directed behaviour when experiencing negative affect,[20] and those with insomnia are more inclined towards experiential avoidance and emotional dysregulation.[21] These shared characteristics suggest a potential mediating role for affect intolerance in linking sleep loss and psychosis.

Affect intolerance and dissociation self-reports are closely connected in the general population, with dissociation proposed to arise in response to threat-based appraisals of affect.[22,23] While definitions of dissociation vary amongst researchers,[24] dissociation is broadly understood as feeling ‘unreal’ or detached from one’s sense of self, experiences, or surroundings.[25] Network

analyses reveal strong connections between dissociation, negative affect, and psychotic experiences[26], and dissociation has been robustly linked to positive psychotic symptoms in both clinical and non-clinical samples.[27,28] Furthermore, both dissociation and experiential avoidance predict auditory hallucinations in clinical samples,[29] leading some researchers to propose that auditory hallucinations are dissociative in nature.[30,31] Experimental studies show that sleep loss increases dissociative experiences in non-clinical populations,[32,33,34,35] and self-report studies indicate that dissociation mediates the relationship between sleep quality and psychotic-like experiences.[36] However, to our knowledge this mechanism has not been explored experimentally to determine causation.

Currently, the mechanism linking sleep loss and psychotic experiences remains unclear. Sleep deprivation studies in non-clinical samples report mixed findings on negative affect and psychotic experiences.[8,37,38,39] It may be that sleep loss influences psychotic experiences differently, for instance the association with paranoia is more robustly demonstrated by findings.[4] Research also suggests that sleep deprivation differentially effects psychotic experiences in a time-dependent way.[40] These findings emphasise the need for experimental paradigms to establish a causal mechanism, with a focus on identifying mediating processes.

This study aims to examine the impact of sleep loss on subclinical psychotic experiences through experimental sleep manipulation in a non-clinical sample. To investigate causal mechanisms, it explores negative affect, affect intolerance, and dissociative experiences as mediators. The following hypotheses were tested:

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

Methods

Study design

A within-subjects cross-over experimental design included two counterbalanced conditions: a standard night of sleep (control condition, ≥ 7 hours sleep) and a night of partial sleep restriction (sleep loss condition, ≤ 4 hours sleep).

Participants

Participants were 58 undergraduate psychology students recruited via the Sona Systems participant recruitment portal (Appendix I).[41] Eligibility criteria were assessed during a remote screening interview.

Inclusion criteria

1. Good quality sleep was assessed using the Pittsburgh Sleep Index[42] with a score of ≤8 adjusted from the usual cutoff in line with recommendations for assessing student samples.[43]
2. Access to a smartphone or smartwatch during the study.

Exclusion criteria

1. A history of psychiatric disorder (assessed in interview).
2. Current mood disorder assessed by scoring above moderate range on the PHQ9,[44] or mood disorder vulnerability assessed by scoring above cut-off on the Mood Disorder Questionnaire.[45]
3. Presence of a medical condition that may be affected by a lack of sleep (e.g. epilepsy).
4. Regular consumption of medication that impacts sleep or mood.
5. Travel across time zones in the prior two weeks.
6. Requirement to drive, do late night shift work or operate heavy machinery while taking part.

95 participants expressed interest in taking part in the study, of which 77 were found eligible following screening. 19 participants from the 77 who consented to participate in the study were excluded from analysis due to at least one of the following reasons: did not complete or withdrew from study (n = 6), did not adhere to sleep length requirements for conditions following review of sleep diary and recordings (n = 8), did not complete questionnaires within timeframe required (n = 3), or missing a timepoint for questionnaire completion for the evenings following both sleep conditions (n = 6). This resulted in a final sample of 58 for analysis.

Ethical approval for this study was granted by the University of East Anglia FMH S-REC (ETH2324-0223; Appendix J) and this study was pre-registered on OSF (osf.io/dnju5). All participants gave written informed consent (Appendices K & L) and were granted course credits in return for participation. Aside from minor formatting edits, the protocol was amended prior to data analysis to reflect updated hypotheses and clarify model structure. Specifically, negative affect was added as a mediator variable, as it had been intended but was mistakenly omitted

from the original registration. Clarifications were also made to specify which measured variables served as proposed mediators versus outcomes in the parallel mediation model to ensure alignment with the intended analytic strategy. Additionally, exploratory hypotheses that were not pursued were removed from the protocol. These amendments were agreed upon before analyses began.

Measures

Demographics information was collected by interview following consent and before study commencement. During the study, participants completed assessments via Qualtrics[46] between the hours of 6pm-midnight on the evening before and after both sleep conditions, resulting in a total of four repeated-measures timepoints. All questionnaire timescales were adjusted to capture beliefs and experiences over the last 24 hours to allow for repeated administration.

Demographics

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

Sleep diary and sleep length recording

Following both nights of sleep, participants completed a sleep diary based on the Consensus Sleep Diary[47] which recorded subjective sleep and wake times, hours slept, night-time awakenings, daytime naps and consumption of substances (alcohol, caffeine and nicotine) that influence sleep and wakefulness. The duration of sleep for both conditions was objectively recorded using a smartwatch or smartphone app, which estimates sleep periods by monitoring motion, heart rate, and device activity. These methods have been shown to achieve an 85.9% accuracy rate in detecting sleep and wake states.[48] This data was used to check adherence to sleep conditions as a requirement for inclusion in analysis.

Proposed mediators

Depression Anxiety Stress Scale (DASS) short form:[49] a 21-item questionnaire that measures negative affect. There are three subscales: depression, anxiety and stress. Responses are given to each statement on a 4-point scale of agreement, with higher scores reflecting greater negative affect. This measure has demonstrated strong internal consistency (α = .81-.91 for subscales), as well as good convergent and discriminant validity in non-clinical samples. [49]

Affect Intolerance Scale (AIS) (Stapinski et al., 2014):[12] a 30-item questionnaire that measures appraisals of emotions as threatening and attempts to avoid or suppress unpleasant

emotions. Responses are given to each statement on a 6-point scale of agreement, with higher scores reflecting greater affect intolerance. This scale has demonstrated strong internal consistency (α = .94), as well as good test-retest reliability (*r* = .86), convergent and criterion validity in an undergraduate sample.[12] Permission to use this measure was obtained on 10th November 2023.

Felt Sense of Anomaly Questionnaire (FSAQ):[50] a 35-item questionnaire that measures dissociative experiences reflected as anomalous sensations, perceptions or cognitions that feel unusual or misaligned with one's typical sense of reality. This is captured by items that assess for an anomalous experience of the self, body, familiarity, emotion, and an altered sense of connection, agency and reality. Responses are given to each statement on a 5-point scale of agreement, with higher scores reflecting greater dissociative experiences. This measure has demonstrated excellent internal (α = .98) and test-retest reliability (ICC = .92), and good convergent validity in non-clinical samples, as well as adequate model fit in non-clinical and psychosis samples. [50]

Outcome variables

Specific Psychotic Experiences Questionnaire (SPEQ):[51] a questionnaire that measures various domains of psychotic experiences. There are six subscales: paranoia (15 items rated on a 6-point scale of agreement), hallucinations (9 items rated on a 6-point scale of agreement), cognitive disorganisation (11 items rated as a yes or no agreement), grandiosity (8 items rated on a 4-point scale of agreement), anhedonia (10 items rated on a 6-point scale of agreement, with the total score reverse scored) and observer-ratings (removed for this study due to not being suitable for self-report). Distress is also rated for each dimension on a 4-point scale, and the average of these ratings has been included as an outcome variable in addition to summed scores for each subscale. Higher scores in all scales reflect greater psychotic experiences. This measure has demonstrated good to excellent internal consistency (α = .77-.93) and test-retest reliability (*r* = .65-.74), [51] as well as sensitivity to changes in a non-clinical sample following partial sleep deprivation.[8]

Procedure

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

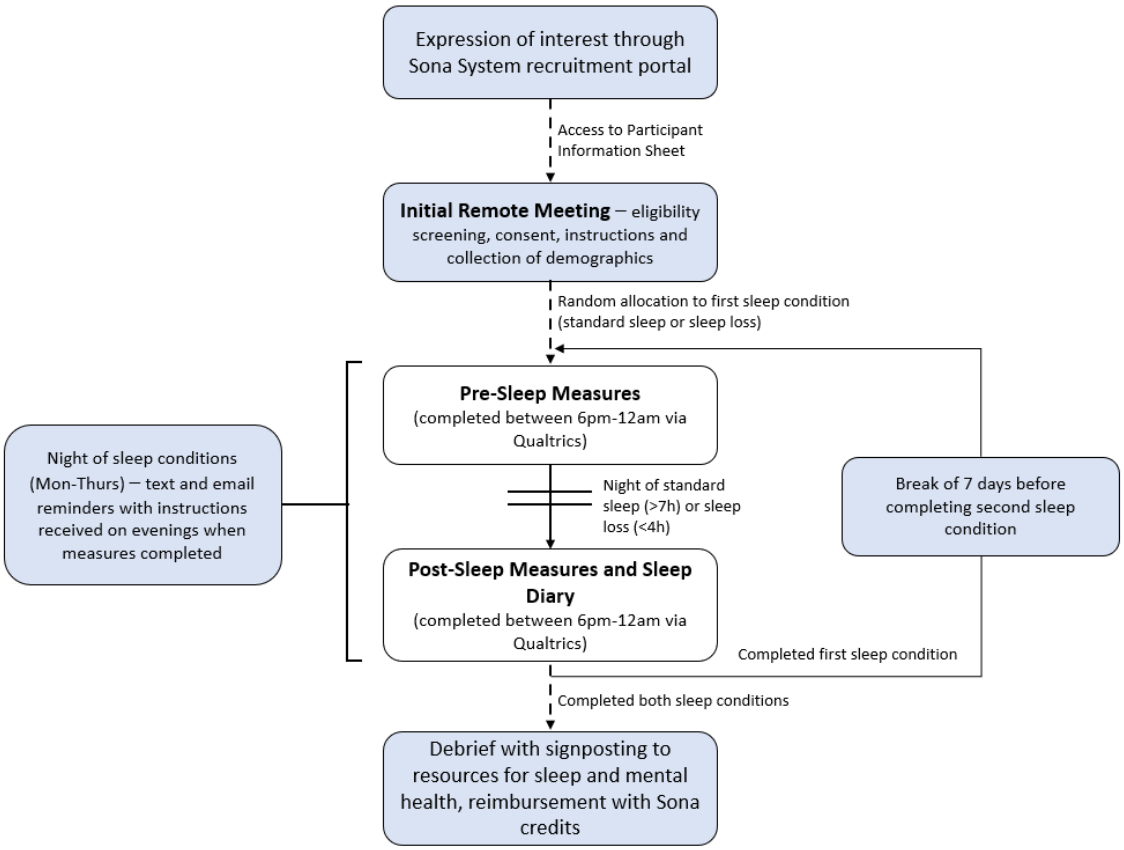
Participants were randomly assigned to their first sleep condition using a list generated via an online true random number service[52]. A list of numbers from 1 to 80, corresponding to the expected recruitment number plus an additional 20% for potential attrition, was randomized and then evenly split with the first 40 participant numbers allocated to complete the standard night of sleep in the first week and the remaining 40 to begin with the night of sleep loss. The allocation list was fixed prior to recruitment, and participants were assigned to conditions based on recruitment order. Recruitment order was determined by participant sign-ups to initial screening interviews on the SONA system which were independent of researcher involvement, ensuring allocation concealment and independence.

Participants completed the sleep manipulation between a Monday-Thursday evening of their choice, with both conditions being on the same day of the week to maintain consistency in daily schedules. Participants were encouraged to follow their usual routines as much as possible to reduce disruption and completed the study remotely at home. Participants were informed of the adverse events procedure during the remote screening interview and were provided written reminders in the email reminders for completing questionnaires and following each completion of remote questionnaires on Qualtrics. The procedure instructed participants to contact the researcher via email if they experienced any unexpected or harmful events during the study or wished to withdraw for any reason.

Following completion of the study participants were debriefed and signposted to resources for sleep and mental health (Appendix M). A diagram of the study procedure is shown in Figure 1.

Figure 1

Flowchart of the study procedure.



Statistical analysis

A priori power analysis performed using G*Power[53] to anticipate the sample size required for correlation analyses (one tailed, α=.05, ß=.8) estimated a minimum of N = 67 required to meet the desired power (Appendix N). Post-hoc power analysis using G*Power indicated that the final sample size of N = 58 retained satisfactory power of 0.75 (Appendix O), however the analysis may be underpowered to detect smaller effects resulting in potential Type II error inflation.

Data were cleaned, pre-analysis adherence to sleep conditions checked, and descriptive summaries of demographics, estimates of time slept and consumption of wakefulness substances (e.g. caffeine) were calculated using Microsoft Excel.[54] Where there were discrepancies between sleep length reported by sleep diary and objective recording the latter was chosen to reflect time slept. A threshold of <20% for imputation of missing data was chosen, and missing values replaced with the mean item score from the associated subscale. One SPEQ Anhedonia subscale for a single participant exceeded this threshold and therefore was not included in analysis. Total and subscale scores were calculated, and paired t-tests

were conducted to check for similarity in scores on subscales for data collected at baseline on the evenings before both sleep conditions. Two participants from each condition were missing baseline data which therefore was not included in this analysis. Paired t-tests were also conducted to check adherence to sleep diary estimates of sleep, as well as caffeine, alcohol and nicotine consumption across conditions.

To investigate whether scores in proposed mediating and outcome variables were correlated, a series of bivariate correlation analyses for both sleep conditions were performed using IBM Statistical Package for the Social Sciences (SPSS).[55] A Shapiro-Wilk test of normality was carried out on all variables (see Supplementary Material S.1) which informed the choice of Spearman's rho test of correlation as they did not meet criteria for parametric testing. Due to heterogeneity in scales, variables were standardised for comparison by conversion to z-scores for the purpose of correlation analyses. Variables chosen for the next stage of analysis were then prioritised based on the previously hypothesised plausible causal pathway and significant correlations.

Repeated measures mediation analyses were performed using the MEMORE macro tool for SPSS[55], which is based on the within-subject mediation approach described by Judd, Kenny and McClelland[56] and extended by Montoya and Hayes[57] into a path-analytic framework that supports the parallel mediation model employed in the present study. Total, direct and indirect effects of the sleep condition on outcome variables were calculated using percentile bootstrapping with 5,000 generated samples to produce standard errors and 95% confidence intervals. Percentile bootstrapping is currently recommended over other approaches that have been suggested as being too liberal resulting in increased Type I error.[58,59] Mediators were tested in a combined model for each psychotic experience outcome variable to examine the unique contribution of each mediator while accounting for shared variance among them. The causal assumptions required for a calculated indirect effect to be valid are plausibly held due to the within-subjects counterbalanced experimental design employed. Multiple comparison corrections were not applied as the comparisons were based on pre-defined hypotheses.

The proportion mediated for each mediator tested was calculated as the percentage of the indirect effect to the total effect. This effect size was chosen over other available metrics[60] to allow for comparison with previous studies[8] and to provide an easily interpretable and intuitive reflection of the relative contribution of indirect effects to the total effect that is accessible for a range of audiences. It should be noted that proportion metrics should be interpreted with caution as they can be unstable when the total effect is small or non-significant. Effect sizes for

candidate mediators were calculated using the pooled standard deviation across conditions as the denominator, which can be interpreted as equivalent to Cohen's *d*.[61] Statistical significance was set at $p < .05$.

Results

Adverse Events

No adverse events were reported as resulting from the sleep restriction. Of the six participants who consented but did not complete the study, two withdrew due to personal circumstances, and one due to feeling unwell, all in cases prior to study commencement. The remaining three participants were excluded due to non-response to emails or questionnaires within the required time window.

Demographics

The characteristics of the participants are presented in Table 1. The demographic characteristics of the total sample ($N = 58$) were similar across the counterbalanced orders, with $n = 29$ participants completing the sleep loss condition first and $n = 29$ participants completing the standard sleep condition in the first week. The mean age of participants was typical for an undergraduate sample ($M = 21.02$, $SD = 4.72$), aside from $n = 2$ participants over the age of 40. The sample were predominantly female (69.0%, $n = 40$), white (74.1%, $n = 43$) and British (82.8%, $n = 48$).

Table 1

Demographic characteristics of study participants

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

Comparison of baseline timepoints

No significant differences were found in any mediator or outcome variable between baseline assessments preceding each sleep condition (see Supplementary Material S.2).

Adherence to sleep conditions

The average sleep duration was 8 hours and 2 minutes for the control condition (M = 481.7 mins, SD = 57.3) and 3 hours and 45 minutes for the sleep loss condition (M = 225.3 mins, SD = 29.5), with a statistically significant reduction in time slept during partial sleep restriction ($p < .001$). No significant difference in the mean consumption of alcohol (.48 vs .49 units respectively, p = .910) or nicotine (0.71 vs 0.81 mg respectively, p = .910) over the last 24 hours was found across sleep conditions. An increase in caffeine consumption was recorded for the sleep loss condition (130.84 vs 88.99 mg in the control condition, p = .005).

Correlation analyses

The correlation matrices (see Supplementary Material S.3 and S.4) show the correlation coefficients between the variables assessed post-standard sleep and post-sleep loss. Both matrices reflect that total negative affect was significantly positively correlated with paranoia,

hallucinations, cognitive disorganisation, grandiosity, and distress for the sleep loss condition only, however, was not significantly correlated with scores for anhedonia across conditions.

Total affect intolerance score was significantly positively correlated with paranoia in the standard sleep condition only, and both conditions for hallucinations, cognitive disorganisation and distress, however, was not significantly correlated with scores for grandiosity or anhedonia across conditions. A similar pattern was found for dissociative experiences with paranoia, hallucinations, cognitive disorganisation, grandiosity and distress, but not anhedonia.

Effects of sleep loss on psychotic experiences and proposed mediators

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

Sleep loss was associated with a significant increase in negative affect, with a large effect, as well as significant increases in affect intolerance and dissociative experiences, both with small to medium effects.

Sleep loss was also significantly associated with increased paranoia, anhedonia, cognitive disorganisation, and distress related to experiences, all with small to medium effects. A significant increase in hallucinations was also observed, with a medium effect. No significant change in grandiosity was reported.

Table 2

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

	Standard Sleep Mean (*SD*)	Sleep Loss Mean (*SD*)	Effect (*SE*), *p*-value	95% *CI*	Effect Size
Psychotic Experiences					
Paranoia (SPEQ)	2.4 (3.7)	4.8 (6.8)	2.38 (.75), .002	.88 to 3.88	.45
Hallucinations (SPEQ)	0.7 (1.4)	2.2 (3.6)	1.48 (.39), <.001	.71 to 2.25	.59
Cognitive Disorganisation (SPEQ)	5.1 (3.8)	6.0 (3.5)	.97 (.27), <.001	.43 to 1.50	.27
Grandiosity (SPEQ)	2.4 (3.1)	2.2 (2.8)	-.17 (.33), .606	-.84 to .49	.06
Anhedonia (SPEQ)	14.6 (9.1)	17.0 (9.7)	2.21 (.99), .030	.22 to 4.2	.24
Distress Related to Experiences (SPEQ)	0.4 (1.0)	0.9 (1.5)	.45 (.14), .003	.16 to .73	.36
Proposed Mediating Variables					
Negative Affect (Total DASS)	5.2 (5.1)	11.3 (10.0)	6.09 (1.02), <.001	4.03 to 8.14	.81
Affect Intolerance (Total AIS)	83.1 (28.6)	96.4 (30.1)	13.33 (3.01), <.001	7.31 to 19.35	.45
Dissociative Experiences (Total FSAQ)	10.8 (14.5)	18.4 (20.1)	7.69 (1.65), <.001	4.38 to 11.00	.44

Mediation analyses

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

The relationship between sleep loss and paranoia was substantially mediated by dissociative experiences, meaning the relationship was attenuated when considering the indirect effect of dissociative experiences, which accounted for 46.2% of the association. There was also a significant combined indirect effect of negative affect, affect intolerance and dissociative experiences, accounting for 89.1% of the association between sleep loss and paranoia. No significant indirect effects were found for negative affect or affect intolerance independently.

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

Table 3

Mediation analyses for proposed mediators and psychotic experiences

Outcome	Total Effect, Estimate (*SE*), Bootstrap 95% *CI*, *P* value	Mediators Tested	Direct Effect, Estimate (*SE*), Bootstrap 95% *CI*, *p*-value	Indirect Effect, Bootstrap Estimate (SE), Bootstrap 95% *CI*	Proportion of Total Effect Mediated
SPEQ Paranoia Subscale	2.38 (.75), .88 to 3.88, .002*	Negative Affect *(Total DASS)*	.26 (.98), -1.72 to 2.24, .794	.55 (.94), -1.22 to 2.55	23.1%
		Affect Intolerance *(Total AIS)*		.47 (.44), -.18 to 1.64	19.7%
		Dissociative Experiences *(Total FSAQ)*		1.10 (.58), .04 to 2.39*	46.2%
		Combined *(DASS, AIS and FSAQ)*		2.12 (.95), .62 to 4.28*	89.1%
SPEQ Hallucinations Subscale	1.48 (.39), .71 to 2.25, <.001*	Negative Affect *(Total DASS)*	1.20 (.51), .18 to 2.23, .023*	-.34 (.63), -1.48 to .99	-23.0%
		Affect Intolerance *(Total AIS)*		.05 (.22), -.38 to .54	3.4%
		Dissociative Experiences *(Total FSAQ)*		.57 (.31), .02 to 1.24*	38.5%
		Combined *(DASS, AIS and FSAQ)*		.28 (.51), -.57 to 1.47	18.9%
SPEQ Cognitive Disorganisation Subscale	.97 (.27), .43 to 1.50, <.001*	Negative Affect *(Total DASS)*	.61 (.40), -.20 to 1.42, .138	.15 (.28), -.41 to .69	15.5%
		Affect Intolerance *(Total AIS)*		-.08 (.19), -.51 to .23	-8.2%
		Dissociative Experiences *(Total FSAQ)*		.29 (.21), -.06 to .78	29.9%
		Combined *(DASS, AIS and FSAQ)*		.36 (.27), -.17 to .89	37.1%
SPEQ Anhedonia Subscale	2.21 (.99), .22 to 4.20, .030*	Negative Affect *(Total DASS)*	.65 (1.30), -1.96 to 3.27, .618	1.70 (1.67), -1.27 to 5.14	76.9%
		Affect Intolerance *(Total AIS)*		-.65 (.52), -1.66 to .45	-29.4%
		Dissociative Experiences *(Total FSAQ)*		.51 (.84), -1.12 to 2.19	23.1%
		Combined *(DASS, AIS and FSAQ)*		1.56 (1.31), -.77 to 4.34	70.6%
SPEQ Distress Subscale	.45 (.14), .16 to .73, .003*	Negative Affect *(Total DASS)*	.43 (.20), .04 to .83, .031*	-.05 (.18), -.38 to .33	-11.1%
		Affect Intolerance *(Total AIS)*		-.07 (.07), -.23 to .05	-15.6%
		Dissociative Experiences *(Total FSAQ)*		.13 (.18), -.21 to .51	28.9%
		Combined *(DASS, AIS and FSAQ)*		.01 (.25), -.43 to .55	2.2%

Note. *$p < .05$

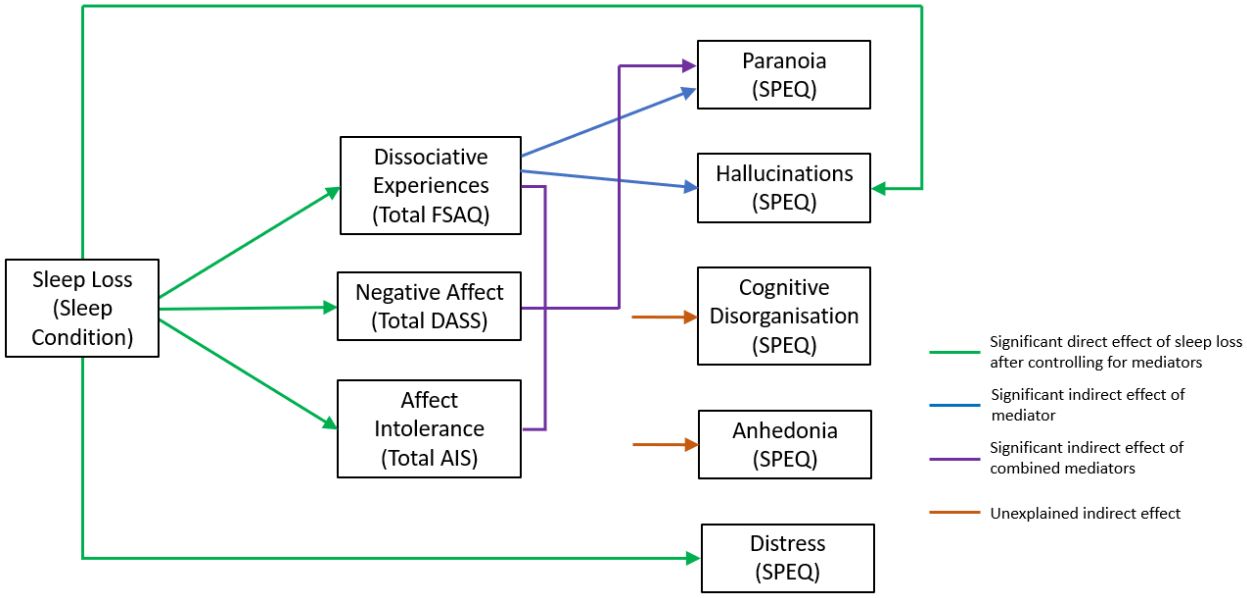
After accounting for the proposed mediators, the direct effects of sleep loss on both cognitive disorganisation and anhedonia were no longer significant. However, no significant mediation effects were observed for negative affect, affect intolerance, dissociative experiences, or combined mediators in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

mediation were found for the association between sleep loss and distress related to psychotic experiences.

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

Figure 2

Proposed causal pathway following mediation analyses



Discussion

The present study aimed to investigate whether a single night of sleep restriction increases subclinical psychotic experiences and to determine if this effect is mediated by heightened negative affect, affect intolerance, and dissociative experiences. In line with our first hypothesis, sleep loss resulted in a significant increase in paranoia, hallucinations, cognitive disorganisation, anhedonia, and distress related to these experiences, with small to medium effects. No significant change in grandiosity was found. Our second hypothesis was also confirmed, as negative affect, affect intolerance, and dissociative experiences were also all significantly increased in the sleep loss condition relative to standard sleep with small to large effects. For our third hypothesis, we successfully demonstrated that elevated dissociative experiences, and combined mediators, both substantially mediated increases in paranoia following sleep loss. Additionally, elevated dissociative experiences partially mediated the

association between sleep loss and increased hallucinations. The increases in outcome variables of cognitive disorganisation, anhedonia and distress related to psychotic experiences following sleep loss were not significantly mediated by the proposed variables. Overall, the results are consistent with a causal role for sleep deprivation in the development of subclinical psychotic experiences through mediation by affective processes.

Our study demonstrated that a single night of sleep loss was sufficient to cause a significant increase in paranoia, hallucinations, cognitive disorganisation, anhedonia, and distress associated with these experiences in a non-clinical sample. This finding supports a causal relationship between disrupted sleep and subclinical psychotic experiences and highlights the extent to which sleep loss increases vulnerability to these experiences. While prior prolonged sleep restriction and total sleep deprivation studies have shown mixed results, [8, 37,38,39] to our knowledge this is the first study to show that these changes can occur after just one night of partial sleep restriction. Our effect sizes were smaller than previous studies, likely due to the lesser loss of sleep since the effects of sleep loss are cumulative over time.[62] Additionally, our findings align with previous research indicating that sleep loss differentially influences individual psychotic experiences.[8]

One night of partial sleep restriction significantly increased next day negative affect in line with previous findings[8,63] and in contrast to others.[7] Regarding our other proposed mediating variables, results from prior studies are consistent with our findings in showing that sleep difficulties increase dissociative experiences.[32,33,34,35] To our knowledge the causal increase in affect intolerance following sleep loss is a novel finding. However, this is indirectly supported by a sleep deprivation study investigating changes to self-reported avoidance, an aspect of affect intolerance, which increased alongside negative affect.[64] These findings underscore the role of sleep loss in disrupting affective processes relevant to the development of psychopathology.

We found that increases in subclinical paranoia following sleep loss were substantially mediated by the combined effects of negative affect, affect intolerance and dissociative experiences. This aligns with prior research linking paranoia to both insomnia and the intolerance of distressing emotional states.[65,66] Additionally, our findings are consistent with evidence from clinical samples showing that maladaptive emotion regulation partially mediates the association between sleep difficulties and paranoid thinking.[67] Although we expected that negative affect would independently mediate this relationship,[8] the absence of this effect may be due to statistical underpowering. Similarly, no significant mediation effects were observed for cognitive disorganisation and anhedonia, despite the non-significant direct effects of sleep

loss after accounting for the proposed mediators, indicating a potential mediating contribution that did not reach significance. Furthermore, changes in distress related to psychotic experiences following sleep loss were not significantly influenced by the proposed mediators. This was unexpected given previous research highlighting the key role of emotion regulation processes in influencing distress in clinical samples,[68] suggesting that other mechanisms, such as thought control strategies[69] or reappraisal,[70] may better explain distress in response to psychotic experiences.

Dissociation was found to substantially mediate elevated paranoia, and partially mediate increased hallucinations, consistent with previous findings.[26,27,29,36] This may be explained by an attention bias towards affective stimuli as shown in non-clinical 'high dissociators',[71] leading to the attribution of greater attentive focus towards emotionally salient information with the potential to induce paranoid thinking.[72] Our finding that dissociation mediates hallucinatory experiences is also supported by previous studies that posit auditory hallucinations as dissociative in nature, such as dissociated components of the self or distressing past events.[30,31] The relationship between cognitive disinhibition and dissociation has been linked to hallucinations,[29,73] suggesting that cognitive processes may explain the remaining partial mediation of hallucinations after accounting for dissociation. Overall, these findings emphasise the key role of affective processes in the development of psychosis symptoms following sleep loss.

Strengths and limitations

The present study demonstrated several strengths in its methodology to enhance internal validity and minimise confounding variables. Sleep conditions were counterbalanced to reduce order and practice effects, and washout periods between conditions included to prevent carryover effects following sleep loss. Participants were encouraged to maintain their usual activities and participated remotely, enhancing the ecological validity and generalisability of findings to insomnia populations. The similarity in baseline scores before sleep manipulation suggests that the observed changes following sleep manipulation were unlikely due to unexplained variations between intervention weeks. Adherence to sleep conditions was verified by assessment of sleep length recordings, alongside assessment of the potential confounding effect of recently consumed wakefulness substances. A small increase in caffeine consumption during the sleep loss condition was found, which amounted to less than the content of a cup of black tea. Finally, our participants were predominantly young adults which

aligns with the typical age of onset for psychotic disorders,[74] providing greater insight into the aetiology of psychosis.

Several limitations should be considered when interpreting the results of this study. Due to time constraints during recruitment and difficulties with adherence to study instructions resulting in missing data, our final participant sample was smaller than originally planned, potentially introducing attrition bias. As a result, our analyses were underpowered, which increases the risk of Type II error. Conversely, we did not apply corrections for multiple comparisons since analyses were based on pre-specified hypotheses, which may have increased the likelihood of Type I error. Additionally, we relied on self-report measures to assess changes in affective processes, which are influenced by subjective awareness and interpretation of experiences, and studies investigating the correlation between self-reports and objective neural measures of emotion regulation have shown mixed results.[75,76] Finally, the timescale of these measures was adjusted to suit the 24-hour period between baseline and post-intervention assessments, which may have influenced the validity of the outcomes reported.

An important limitation concerns the original power calculation for this study, which was based on detecting a simple correlation of 0.3. Once the analysis approach was clarified, it became clear that this did not fully align with the study's primary aim, which was to detect minimally important effects of sleep restriction on subclinical psychotic experiences, later defined as group differences exceeding an effect size of 0.4 (Appendix P). Nevertheless, our post-hoc sensitivity analysis (Appendix P), together with the consistency of recruitment numbers and findings with the experimental study that we replicated[8], indicates that the study was in fact appropriately powered to detect moderate effects. A further limitation is the use of a one-tailed power calculation in the original calculation, which assumes a directional effect and therefore reduces the threshold for statistical significance. This approach would have been inappropriate as results in the opposite direction would have been considered theoretically meaningful if found.

Finally, an additional limitation of the current study was the use of a passive adverse events reporting approach, which relied on participants to self-identify and report any issues via email. This method may have led to underreporting, as participants could have forgotten, overlooked, or felt unmotivated to report events, which would have influenced opportunities for timely intervention or study adjustments if required.

Future directions and clinical implications

Replicating this study with larger and more diverse samples would support with establishing the mechanisms identified and improve the generalisability of these findings. Furthermore, using a sleep continuity disruption protocol rather than partial sleep restriction may produce more robust effects on reported affect[77] and provide valuable insights into whether these processes differ for individuals with insomnia characterised by frequent nighttime awakenings. Longitudinal experimental sleep manipulation could also reveal more pronounced changes in affective processes and psychotic experiences, while also facilitating mapping the temporal dynamics of changes in mediating and outcome variables. Given that our findings suggest changes in negative affect, affect intolerance and dissociative experiences following sleep loss contribute to psychotic symptoms, longitudinally measuring these outcomes as psychotic symptoms progress could offer valuable insights into their role in the aetiology of psychosis. If negative affect, affect intolerance and dissociation are shown to moderate changes in symptoms along the psychosis continuum, future studies could investigate targeted interventions to determine whether these variables act as mechanisms of change for improving psychotic symptoms. This would contribute to our growing appreciation for targeting sleep difficulties and enhancing adaptive affective processes as clinical mechanisms of change for individuals at risk of or experiencing symptoms of psychosis.[78,79]

Conclusion

Our findings support a model in which sleep difficulties act as a causal factor in the onset of psychotic experiences, offering insights into the mechanisms that drive the onset and maintenance of psychosis. This has significant implications for early intervention and prevention. Good quality sleep may serve as a protective factor against symptom development, and sleep difficulties should therefore be valued as a treatment target and early warning sign for clinicians working with individuals at higher risk for psychosis, such as those with genetic predispositions or prodromal symptoms. Additionally, negative affect, affect intolerance and dissociative experiences may serve as effective targets for symptom monitoring and intervention, and should be considered in clinical formulations that account for individual differences that influence the variability in presentations of psychosis.

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Conflict of interest

None to declare.

Supplementary Material

Table S.1

Shapiro-wilk test of normality output

	Post- Standard Sleep Shapiro Wilk test statistic, *p*-value	Post-Sleep Loss Shapiro Wilk test statistic, *p*-value
Affect Intolerance Scale (AIS) Total	.981, $p = .518$	.982, $p = .522$
Felt Sense of Anomaly Questionnaire (FSAQ) Total	.754, $p < .001$	.821, $p < .001$
Depression, Anxiety and Stress Scale (DASS) Total	.834, $p < .001$	.851, $p < .001$
Depression Subscale	.801, $p < .001$	.870, $p < .001$
Anxiety Subscale	.718, $p < .001$	.692, $p < .001$
Stress Subscale	.827, $p < .001$	.876, $p < .001$
Specific Psychotic Experiences Questionnaire (SPEQ)		
Paranoia Subscale	.673, $p < .001$	.722, $p < .001$
Hallucinations Subscale	.551, $p < .001$	.662, $p < .001$
Cognitive Disorganisation Subscale	.913, $p < .001$	.936, $p = .004$
Grandiosity Subscale	.768, $p < .001$	.773, $p < .001$
Anhedonia Subscale	.946, $p = .013$	.962, $p = .070$
Distress Related to Experiences Subscale	.485, $p < .001$	.636, $p < .001$

Note. N = 57 for standard sleep condition due to missing subscale.

Table S.2

Baseline scores of study variables and analysis of difference

	Pre-Standard Sleep Mean (*SD*)	Pre-Sleep Loss Mean (*SD*)	T-value (df), *p*-value
Affect Intolerance Scale (AIS) Total	96.3 (26.6)	97.0 (25.4)	0.29 (53), *p*= .772
Felt Sense of Anomaly Questionnaire (FSAQ) Total	16.4 (17.7)	15.2 (20.2)	0.50 (53), *p*= .618
Depression, Anxiety and Stress Scale (DASS) Total	7.4 (6.4)	7.5 (9.1)	0.11 (53), *p*= .915
Depression Subscale	1.9 (2.0)	2.0 (2.9)	0.18 (53), *p*= .861
Anxiety Subscale	2.1 (2.6)	2.1 (3.4)	0.12 (53), *p*= .902
Stress Subscale	3.5 (2.8)	3.5 (3.7)	0.00 (53), *p*= 1.00
Specific Psychotic Experiences Questionnaire (SPEQ)			
Paranoia Subscale	4.7 (6.3)	3.5 (5.3)	1.53 (53), *p*= .133
Hallucinations Subscale	1.9 (3.6)	1.1 (2.4)	1.82 (53), *p*= .074
Cognitive Disorganisation Subscale	5.1 (3.4)	4.8 (3.3)	1.02 (53), *p*= .312
Grandiosity Subscale	2.6 (2.2)	2.8 (2.7)	0.78 (53), *p*= .438
Anhedonia Subscale	14.6 (8.5)	14.5 (8.4)	0.20 (53), *p*= .841
Distress (related to experiences) Subscale	0.6 (1.2)	0.5 (1.1)	0.76 (53), *p*= .451

Note. N = 54 due to missing data sets, two-tailed paired t-test

Table S.3

Correlation matrix for study variables in post-standard sleep condition

	M (*SD*)	1	2	3	4	5	6	7	8	9	10	11	12
1. AIS_Total	83.1 (28.6)	-											
2. FSAQ_Total	10.8 (14.5)	.509***	-										
3. DASS_Dep	1.7 (1.9)	.370**	.675***	-									
4. DASS_Anx	1.1 (1.7)	.212	.587***	.569***	-								
5. DASS_Str	2.3 (2.5)	.285*	.348**	.486***	.464***	-							
6. DASS_Total	5.2 (5.1)	.319*	.606***	.805***	.772***	.827***	-						
7. SPEQ_Par	2.4 (3.7)	.405**	.546***	.473***	.415**	.365**	.452***	-					
8. SPEQ_Hal	0.7 (1.4)	.363**	.617***	.479***	.431***	.385**	.526***	.440***	-				
9. SPEQ_CD	5.1 (3.8)	.454***	.588***	.417**	.292*	.232	.334*	.423***	.356**	-			
10. SPEQ_Gra	2.4 (3.1)	.039	.287*	.320*	.409**	.318*	.412**	.408**	.261*	.111	-		
11. SPEQ_Anh	14.6 (9.1)	-.081	.137	-.049	.028	-.066	-.045	.070	.187	.020	-.079	-	
12. SPEQ_Dis	0.4 (1.0)	.341**	.282*	.248	.196	.290*	.250	.314*	.190	.512***	.067	.022	-

Note. *p < .05 (two-tailed), **p < .01 (two-tailed), ***p < .001 (two-tailed). AIS_Total: Affect Intolerance Scale Total, FSAQ_Total: Felt Sense of Anomaly Questionnaire Total, DASS_Dep: DASS Depression Subscale, DASS_Anx: DASS Anxiety Subscale, DASS_Str: DASS Stress Subscale, DASS_Total: Depression Anxiety Stress Scale Total, SPEQ_Par: SPEQ Paranoia Subscale, SPEQ_Hal: SPEQ Hallucinations Subscale, SPEQ_CD: SPEQ Cognitive Disorganisation Subscale, SPEQ_Gra: SPEQ Grandiosity Subscale, SPEQ_Anh: SPEQ Anhedonia Subscale, SPEQ_Dis: SPEQ Distress Subscale.

Table S.4

Correlation matrix for study variables in post- sleep loss condition

	M (*SD*)	1	2	3	4	5	6	7	8	9	10	11	12
1. AIS_Total	96.4 (30.1)	-											
2. FSAQ_Total	18.4 (20.1)	.407**	-										
3. DASS_Dep	3.7 (3.4)	.582***	.481***	-									
4. DASS_Anx	2.4 (3.6)	.364**	.648***	.581***	-								
5. DASS_Str	5.2 (4.6)	.503***	.503***	.570***	.516***	-							
6. DASS_Total	11.3 (10.0)	.587***	.644***	.826***	.782***	.864***	-						
7. SPEQ_Par	4.8 (6.8)	.201	.633***	.449***	.489***	.491***	.576***	-					
8. SPEQ_Hal	2.2 (3.6)	.353**	.631***	.301*	.443***	.387**	.467***	.403**	-				
9. SPEQ_CD	6.0 (3.5)	.557***	.652***	.562***	.480***	.455***	.568***	.296*	.330*	-			
10. SPEQ_Gra	2.2 (2.8)	.084	.375**	.299*	.329*	.369**	.409**	.475***	.231	.122	-		
11. SPEQ_Anh	17.0 (9.7)	.122	.117	.067	.154	.113	.090	.033	.118	.135	-.139	-	
12. SPEQ_Dis	0.9 (1.5)	.314*	.539***	.445***	.551***	.328*	.482***	.468***	.298*	.555***	.053	.248	-

Note. *p < .05 (two-tailed), **p < .01 (two-tailed), ***p < .001 (two-tailed). AIS_Total: Affect Intolerance Scale Total, FSAQ_Total: Felt Sense of Anomaly Questionnaire Total, DASS_Dep: DASS Depression Subscale, DASS_Anx: DASS Anxiety Subscale, DASS_Str: DASS Stress Subscale, DASS_Total: Depression Anxiety Stress Scale Total, SPEQ_Par: SPEQ Paranoia Subscale, SPEQ_Hal: SPEQ Hallucinations Subscale, SPEQ_CD: SPEQ Cognitive Disorganisation Subscale, SPEQ_Gra: SPEQ Grandiosity Subscale, SPEQ_Anh: SPEQ Anhedonia Subscale, SPEQ_Dis: SPEQ Distress Subscale.

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Chapter 5

Discussion and Critical Evaluation Chapter

This thesis aimed to develop our understanding of the causal mechanisms that link sleep loss to the development of psychotic experiences, as well as to explore the role of emotion regulation and related processes in bridging this relationship. This chapter will provide a synthesis of findings from the systematic review and empirical study and position these results within the context of existing knowledge, followed by a critical appraisal of the research highlighting strengths and limitations. The theoretical and clinical implications are then discussed, followed by recommendations for future research and clinical practice. The chapter ends with reflections on the research process and final conclusions.

Overview of findings

Systematic review

The systematic review aimed to synthesise findings from experimental sleep manipulation studies in non-clinical samples, and emotion-targeted interventions for alleviating psychosis symptoms, with the objective of assessing evidence for emotion regulation as a mechanism linking sleep loss to the development of psychotic experiences. Fifteen studies, composed of four sleep manipulation studies and eleven intervention studies, met eligibility criteria and were included in the narrative synthesis for analysis and quality appraisal.

The findings revealed a paucity of studies investigating the impact of sleep loss on emotion regulation difficulties and strategy use. In addition, due to variability in sleep manipulation methods used and the absence of validated measures, conclusions about this relationship were tentatively drawn. There was no evidence to suggest that sleep loss affected changes in momentary state emotion regulation difficulties. This suggests that short-term sleep disruption does not lead to alterations in this aspect of emotion regulation that could contribute to a causal mechanism for the development of psychosis. However, research in transdiagnostic samples suggests that trait measures of emotion regulation more reliably predict symptom severity than situational use (Adolph et al., 2024), highlighting the need for future sleep manipulation studies to investigate changes in trait emotion regulation measures longitudinally. Following sleep disruption, one study observed significant increases in the use of rumination and distraction following exposure to sleep fragmentation. This finding is consistent with clinical psychosis samples, which also report an elevated use of these strategies relative to the general population (Ludwig et al., 2019), indicating a potential shared mechanism.

Studies identified mindfulness and acceptance approaches as adaptive emotion regulation strategies linked with improvements in psychotic experiences, and one study found an association between improvements in self-reported mindfulness and reductions in hallucination severity. This aligns with findings from meta-analyses, which suggest that mindfulness is a promising mechanism for change in individuals with psychosis (Ellett et al., 2023). While no sleep studies directly examined changes in mindfulness, framing mindfulness as focused rather than distracted attention allows for cautious comparisons with findings of increased distraction following sleep disruption, potentially indicating a linking mechanism. Additionally, across studies trait emotion regulation difficulties were consistently demonstrated to improve alongside psychotic experiences, particularly positive symptom subscales, suggesting a relationship between these outcomes. However, further research with larger samples is needed to confirm a robust association between these variables as many of the studies included in the review were pilot or feasibility trials and therefore robust causal or inferential conclusions are premature.

One sleep manipulation study revealed various examples of changes to emotion regulation strategy use differentially predicting changes to positive and negative affect, as well as a mediating role of rumination in the association between sleep disruption and elevated negative affect. This indicates that changes in emotion regulation following sleep loss may be challenging to measure directly, but that valuable insights into the relationship between sleep difficulties and psychosis may be revealed through the investigation of mediation by emotion regulation processes. The review highlighted the limited number of sleep manipulation studies investigating changes to emotion regulation choice and use, as well as research examining direct associations between emotion regulation and psychotic experiences, or these variables in relation to sleep difficulties. This underscores the need for additional experimental studies to address this gap in the literature to clarify causal mechanisms.

Empirical paper

The empirical research paper aimed to investigate the causal influence of sleep loss on the development of psychotic experiences using an experimental sleep manipulation protocol in a non-clinical sample. To further examine linking mechanisms, negative affect, affect intolerance and dissociative experiences were explored as potential mediating variables.

The results of the study revealed that one night of partial sleep deprivation (≤ 4 hours sleep) significantly increased self-reported negative affect, affect intolerance, dissociative experiences, paranoia, hallucinations, cognitive disorganisation, anhedonia and distress

related to experiences. This is consistent with observations of elevated sleep difficulties in psychosis samples (Cederlöf et al., 2022), as well as studies indicating that sleep difficulties predict subsequent psychotic experiences (Reeve et al., 2015). To our knowledge this is the first study that has demonstrated significant increases in psychotic experiences after only one night of sleep restriction, and the first to causally link sleep loss with elevated affect intolerance. These findings highlight the significant role sleep plays in influencing individuals' vulnerability to the development of psychopathology.

Mediation analyses revealed that the combined effect of negative affect, affect intolerance and dissociative experiences, substantially mediated the association between sleep loss and increased paranoia (89.1%). Additionally, dissociative experiences independently significantly mediated the relationship between sleep loss and paranoia (46.2%) and partially mediated the association between sleep loss and increased hallucinations (38.5%). No other significant indirect effects of proposed mediators on psychotic experience outcomes were found. This is consistent with previous findings using self-report measures in non-clinical samples, which demonstrated the mediating role of dissociation in linking sleep loss to psychotic-like experiences (Creatura et al., 2022). However, using experimental sleep manipulation allows for more confident conclusions about the causal contribution of sleep difficulties to the onset of psychosis through this mechanism.

Overall synthesis of studies and findings

Taken together, these findings suggest a potential causal influence of sleep loss, and mediating role of regulatory affective processes, in psychosis symptom development. While these results offer valuable insights for theoretical and clinical understandings of psychosis onset and maintenance, further research is needed to confirm and clarify these relationships.

Studies included in the systematic review indicated the potential for trait emotion regulation difficulties (individuals' overall acceptance, modulation, awareness and clarity of emotions), as well as elevated distraction and decreased use of mindfulness and acceptance strategies, in linking the effects of sleep disruption with the severity of psychotic experiences. The empirical study confirmed the causal role of sleep loss in the development of subclinical psychotic experiences, and established processes including dissociative coping, heightened negative affect and affect intolerance as mediating variables. Overall, these findings tentatively suggest that following sleep disruption, an individual's tendency to cope through disengagement from their current emotional experiences and the eliciting stimuli is enhanced, and this may consequently increase the severity of psychotic experiences such as paranoia and

hallucinations. This conclusion aligns with previous findings demonstrating that individuals show a preference for using engagement strategies (e.g. reappraisal) when faced with low-intensity negative situations, and disengagement strategies (e.g. distraction) when emotions are more distressing, which consequently disrupts emotional processing of events (Sheppes et al., 2011, 2014). If this pattern persists, this could exacerbate and maintain emotional distress, thereby increasing vulnerability to developing psychotic experiences. Although this hypothesis should be explored through further research before drawing firm conclusions.

Critical appraisal of thesis portfolio strengths and limitations

The systematic review and empirical study both offer novel contributions to the existing evidence base, supporting a deeper understanding of regulatory affective processes that mechanistically link sleep loss with psychotic experiences. The review synthesised research that was predominantly published in the last five years, which allowed us to compare and contrast recent findings to address knowledge gaps. Additionally, the use of narrative synthesis for analysis of findings supported with the contextualisation and exploration of complexity when hypothesising potential bridging mechanisms. Regarding the empirical study, the experimental investigation of within-person changes to psychotic experiences following partial sleep restriction allowed for direct testing of causality with confounding variables controlled for, enhancing our ability to establish cause-and-effect relationships. Additionally, conducting the study with an undergraduate sample enabled the investigation of psychotic experiences in an age group most vulnerable to psychosis onset (Solmi et al., 2022), providing valuable insights into a demographic most relevant for understanding the aetiology of psychosis. Participants also completed the study remotely to minimise disruptions to daily life which supported with maintaining ecological validity, enhancing the generalisability of findings to real world settings.

Several limitations were encountered during the systematic review process. Despite the large number of studies identified through database searches, only a few were eligible for inclusion. This disparity is partly due to the chosen search terms, such as ‘distraction,’ which are relevant to multiple research topics. To address this, terms like ‘attention’ and ‘affect’ were intentionally excluded from the search. However, these decisions posed challenges in achieving specificity in the results. The complexity of emotion regulation as a multicomponent concept also introduced subjectivity when considering which strategies *are* and *aren’t* emotion regulation. To compensate for this challenge, we excluded studies that only measured one domain of emotion regulation in line with Gross’ extended process model of emotion regulation (1998, 2001, 2002, 2015), however this may have limited our ability to evaluate a mechanistic role of individual

strategies or explore emotion regulation approaches outside of this model. Additionally, the included studies showed significant variation in their use of validated measures, sleep manipulation protocols, and the level of detail provided for intervention descriptions. Furthermore, many studies relied on small sample sizes without comparator groups, and did not assess changes in individual psychotic experiences or examine statistical associations with emotion regulation measures. These limitations hindered the ability to draw robust conclusions about potential bridging mechanisms.

The use of an undergraduate sample for the empirical study was appropriate for testing our hypotheses. However, since this age group is commonly examined in published sleep manipulation research, our findings contribute only limited additional value to the broader evidence base. The 24-hour period between baseline and post-sleep timepoints meant that we altered the timeframe of instructions for self-report measures, which may have influenced their validity and therefore the reliability of findings. Additionally, the use of self-report measures for our study increases the risk of demand characteristics from participants. Due to the nature of the study design we were unable to blind participants to their sleep condition, and therefore their reporting may have been influenced by their expectations of how sleep loss would impact their experiences. Finally, we examined the effects of partial sleep restriction rather than alternative sleep manipulation methods such as sleep fragmentation. Insomnia can be characterised by difficulty falling asleep or early morning awakening which would limit total sleep time, or alternatively by difficulties maintaining the continuity of sleep (NHS, 2024). This means that we cannot necessarily generalise our findings to individuals with insomnia characterised by fragmented sleep.

Theoretical implications

The combined findings from the systematic review and empirical study highlight the potential mediating role of regulatory affective processes in linking insomnia to psychosis symptom development. This contributes to our current appreciation of the relationship between affective mechanisms and symptom severity, as well as the influential role of sleep difficulties in the aetiology of psychosis.

The development of theoretical models in clinical psychology is a critical foundation for understanding complex phenomena such as symptom-related distress. This process typically begins with the formulation of a testable hypothesis in the target population, grounded in existing psychological theory, clinical observation, or gaps identified through a comprehensive review of the empirical literature. This allows for the identification of key constructs and

potential causal relationships that can be empirically tested, for example by using experimental designs with mediation analyses to test mechanistic pathways as in the empirical project in this portfolio. The empirical findings can then be used to refine theoretical models, for instance by adjusting the relationships between constructs or through the addition of newly identified mediators, to highlight potential treatment targets. These are then used to develop targeted treatment manuals and protocols that are piloted for feasibility and efficacy in small scale studies to refine the protocol, before being scaled to large scale randomised controlled trials (RCTs) with a mechanistic analysis to test whether changes in the target mechanism mediate clinical outcomes. If shown to be clinically effective, the research findings may then be translated for clinical application through their inclusion in clinician training programs and clinical guidelines. The outcomes of intervention studies and real-world application then feed back into refining theoretical models, following a reciprocal and iterative pathway to the development of effective clinical interventions. This process should be held in mind when contextualising the findings from this portfolio into existing theoretical models and when discussing potential clinical implications.

Findings across the studies in this portfolio indicate that sleep loss is linked to the development of psychotic experiences through the heightened use of disengagement coping (e.g. distraction, dissociation) in response to emotional distress. These strategies occur at the ‘attentional deployment’ stage of Gross’ extended process model of emotion regulation (1998, 2001, 2002, 2015), which involves shifting attention away from emotionally distressing stimuli as a means of regulating affect. This suggests that individuals experiencing sleep disruption may rely more on these strategies, potentially exacerbating psychotic symptoms over time by preventing adaptive emotional processing and reinforcing maladaptive coping patterns.

The 'stress-vulnerability model' (Zubin & Spring, 1977), which provides a framework for understanding the factors that contribute to psychosis, includes stress-inducing factors that may encompass poor sleep quality. This relationship between sleep disruption and stress is supported by previous research, which has demonstrated that sleep deprivation increases plasma cortisol levels the following day, indicating a disrupted stress response (Leproult et al., 1997). In addition, studies show that sleep and stress are closely intertwined, with each influencing the other in a bidirectional manner (see review by Hirotsu et al., 2015). Our findings support this model, emphasising the significance of sleep difficulties in increasing vulnerability to developing psychosis.

Our findings also complement the 'cognitive model of positive psychotic symptoms' proposed by Garety et al. (2001), which highlights how emotional changes interact with cognitive processes, particularly in response to anomalous experiences like paranoid beliefs or hallucinations. Our findings extend this model by suggesting that sleep difficulties, and the resulting changes to regulatory affective processes, may play an influential role in shaping emotional responses that feed back into the processing of psychotic experiences. Our results indicate that disruptions in sleep could exacerbate emotional dysregulation or dissociative coping, increasing vulnerability to the misinterpretation of unusual sensory experiences and further intensifying psychotic symptoms. These insights underscore the importance of considering sleep and affective processes as central mechanisms in the aetiology and maintenance of psychosis.

Finally, our findings indirectly relate to the 'cognitive-interpersonal model of psychosis' (Gumley & Schwannauer, 2006), which emphasises the impact of early relational patterns and traumatic experiences on emotion regulation that influence vulnerability to relapse. Poor sleep quality and stress have been linked to childhood trauma through multilevel modelling (O'Connor et al., 2023), and impaired sleep has been shown to partially mediate the effect of interpersonal trauma on psychotic experiences (Herms et al., 2024). Additionally, childhood trauma has also been shown to correlate with dissociative experiences in individuals with schizophrenia (Sar et al., 2009). This suggests that exposure to early life stress, and an associated tendency towards dissociation, may contribute to the severity of psychotic experiences following sleep loss, in line with our findings from the empirical study. However, further research is needed to confirm this extended mechanism. Furthermore, the 'cognitive-interpersonal model of psychosis' proposes that psychotic experiences may develop as a way of coping with overwhelming emotional distress, mirroring our findings, which highlight the role of emotion regulation processes in relation to negative affect in symptom development. Our findings extend this view through consideration of the impact of sleep loss on maladaptive emotion regulation that consequently exacerbates psychotic symptoms.

Interpreting the findings from this portfolio through the frameworks of cognitive-behavioural and emotion regulation models of emotional distress highlights important distinctions. Cognitive-behavioural models emphasise how reasoning biases, maladaptive appraisals, and avoidance behaviours contribute to maintaining distress. In contrast, emotion regulation models focus on the selection and flexibility of strategies individuals use to modify emotional responses and the resulting distress. While these models share certain features, such as their recognition of the influence of cognitive appraisals and regulatory strategies on emotional outcomes, the present

findings point more directly to mechanisms central to emotion regulation theory. As the findings indicate the potential mediating role of disengagement from emotional experiences, they support a view in which disruptions in strategy selection and emotional responses may be critical in linking sleep loss to the onset of psychotic experiences, processes that may not be fully addressed within traditional cognitive-behavioural frameworks. This perspective is consistent with the empirical work underpinning this project, which found affective processes to be reliable mediators of changes in psychotic symptoms following sleep loss, whereas many of the cognitive processes and beliefs investigated were not (Reeve et al., 2018). Considering that selected factors from cognitive models explain approximately 40% to 67% of the variance in self-reported persecutory delusions in non-clinical and at-risk populations (Freeman et al., 2005; Freeman & Loe, 2023; Gin et al., 2021), and acknowledging conceptual overlap between models, emotion regulation processes may help clarify the remaining unexplained variation. Future research should aim to further delineate where these models converge and diverge in explaining emotional distress in psychosis and their respective roles in the causal pathways linking sleep loss to the development of psychotic experiences.

Clinical implications

The findings from this thesis portfolio provide preliminary support for early intervention and prevention approaches for psychosis based on the mechanisms studied, situated within the broader context of theory development and translational research focused on identifying mechanisms underlying psychosis risk. These findings contribute to the early stages of model refinement, as experimental research in non-clinical populations with the inclusion of mediation tests has been recognised as a valuable step in the development of psychological treatments for psychosis (Clark, 2004; Freeman, 2024). However, before these findings can inform clinical practice, several critical steps in validation and adaptation are required. These include replicating the results in clinical populations with established or emerging psychotic symptoms, examining whether the same mechanisms are active and clinically significant, and adapting methodologies to accommodate the complexities of clinical presentations. Furthermore, individuals with lived experience should be involved in building a robust evidence base of treatment credibility on a foundation of early proof-of-concept testing and demonstration of a large clinical effect before planning a detailed implementation strategy (Freeman, 2024). Therefore, the following clinical implications are proposed with caution and should only be considered for implementation as the broader body of research continues to establish and validate these mechanistic pathways. Further work is essential to refine these findings within clinical populations before they can inform intervention development.

The results of the empirical study emphasise the significance of sleep difficulties as a potential early indicator of psychosis. When considered alongside the growing evidence base, these findings may support earlier recognition and intervention for individuals before symptoms progress. This is particularly relevant for individuals who are frequently exposed to disrupted sleep, such as shift workers or new parents, or those at increased risk for insomnia. This includes individuals who are experiencing depressive other psychiatric symptoms, comorbid medical conditions, or those facing chronic stress due to environmental or social factors including recent separation, grief, unemployment or financial difficulties (Dollander, 2002).

Given the relationships observed between the severity of psychotic experiences, sleep difficulties, and emotion regulation in this portfolio, considering these factors when formulating psychosis symptom presentations has potential to facilitate a deeper understanding of individual differences. This could then inform the provision of targeted interventions, such as CBT for insomnia which has been demonstrated to be highly effective for improving self-reported sleep for individuals with persistent delusions or hallucinations (Freeman et al., 2015). Additionally, addressing sleep problems in individuals at-risk for developing psychosis, or those with prodromal symptoms, may reduce the likelihood of transition to psychosis. Recent research has shown that this approach is both feasible and effective for reducing insomnia symptoms, in addition to psychotic experiences, at 3- and 9-month follow-up (Waite et al., 2023).

Previous research has increasingly recognised difficulties in emotion regulation as a key mechanism underlying the development and maintenance of psychopathology (Sheppes et al., 2015) highlighting the value of targeting emotion regulation as a mechanism of change (Gratz & Tull, 2010). The findings from the systematic review suggest that enhancing individuals' emotional acceptance and awareness, as well as their ability to access and use adaptive emotion regulation strategies, is associated with reductions in the severity of psychotic experiences. Many of the studies included in the systematic review were pilot studies with small sample sizes, however, if these findings were replicated in larger scale RCTs, the presence of this relationship would underscore the importance of incorporating emotion regulation skills training for individuals with psychosis, helping to inform and shape future intervention approaches. Ensuring that these skills were included in treatment frameworks, including CBT manuals where they are often not explicitly incorporated (Lincoln et al., 2022), has the potential to enhance therapeutic outcomes and support more effective coping. Furthermore, the empirical paper demonstrated that dissociative experiences and beliefs about the uncontrollability of emotions (a component of affect intolerance) mediate the impact of sleep

loss on psychotic experiences. This finding highlights these variables as promising treatment targets that warrant further empirical investigation, offering clinicians working with individuals with psychosis potential avenues for intervention to mitigate the effects of sleep disruption (Varese et al., 2020).

The recommendations made here regarding early intervention and prevention of psychosis hold potential relevance for public health and policy, provided they are verified through robust future research. One of the primary recommendations following a recent independent investigation into the current challenges faced by the NHS was a shift towards prevention in healthcare (Darzi, 2024). This will be a key area of focus in upcoming public health policy and guidance, such as the next NHS 10-year plan (Department of Health and Social Care, 2024), that will then inform service design and commissioning. This will offer an opportunity moving forwards to integrate preventative sleep interventions into the core treatment offer from community mental health services. Additionally, incorporating sleep difficulties into routine screening and monitoring for psychosis risk in primary care settings, and ensuring that individuals at risk of or experiencing sleep difficulties are identified and monitored for emerging psychosis symptoms, has the potential to enhance early identification and improve clinical outcomes.

Sleep difficulties are not currently included in the most recent clinical guidance for psychosis prevention and management (National Institute for Health and Care Excellence, 2014). The findings of this portfolio, along with emerging evidence highlighting the role of sleep disruption in the aetiology of psychosis, suggest that sleep-related factors may be included in future reviews of professional guidance. Given the debilitating impact of schizophrenia spectrum disorders on the individuals affected, their support systems, and public health costs (Aceituno et al., 2019), once empirically established the mechanistic targets highlighted in this portfolio have the potential to yield economic benefits for healthcare services, while also enhancing early intervention and treatment outcomes for individuals at risk of or experiencing psychosis, ultimately enhancing their quality of life.

Research implications and future directions

Collectively, the findings from this portfolio emphasise the need for further research to clarify the mechanisms linking sleep loss with psychotic experiences. The systematic review highlighted a clear need for more sleep manipulation studies that explore changes in emotion regulation difficulties and strategy use. Such research could offer valuable insights into the impact of sleep difficulties transdiagnostically. Additionally, future studies should prioritise manipulating sleep through sleep restriction or sleep fragmentation, rather than total sleep

deprivation, as these methods are more reflective of the sleep disturbances typical in insomnia. Replicating the empirical study using sleep fragmentation, or over an extended period, could provide further insights into how sleep disruption influences psychotic experiences. Longitudinal research would also be beneficial in mapping the temporal characteristics of changes to psychotic experiences and regulatory affective processes. This would deepen our understanding of these mechanisms and assist with identifying additional targets for early intervention and prevention.

Studies investigating the relationship between emotion regulation and psychotic experiences should aim to explore direct associations between these measures and examine how specific emotion regulation strategies relate to individual psychotic experiences, such as hallucinations and paranoia. Research in this area, particularly with larger samples and the inclusion of control groups, would greatly enhance our understanding of the mechanisms that contribute to alleviating psychosis symptoms, with potential benefits for clinical practice. Furthermore, it would be valuable to assess how emotion regulation difficulties and strategy use influence the severity, frequency, and duration of symptoms, as well as to determine whether certain strategies are more effective in managing specific psychotic experiences.

Future research on emotion regulation, and its relationship with sleep disruption and psychotic experiences, may benefit from moving beyond the categorical limitations of adaptive and maladaptive strategy use frequency. Recently, experts in the field have emphasised the importance of 'emotion regulation flexibility', which is conceptualised as an individual's sensitivity to context, available repertoire of strategies, and responsiveness to feedback (Aldao et al., 2015; Bonanno & Burton, 2013). Understanding how individuals with psychosis differ in their emotion regulation flexibility may reveal valuable treatment targets for enhancing the strategic matching and effectiveness of their emotion regulation approaches. Furthermore, exploring how emotion regulation flexibility is influenced by sleep disruption would deepen our understanding of mechanisms linking insomnia to psychosis.

Author reflections

Conducting this research has been a fascinating experience and enhanced my appreciation for the importance of emotion processes in driving psychopathology. In my experience, clinically formulating and targeting emotion regulation tends to be overlooked in favour of cognitive and behavioural change, which may be due to the difficulty with conceptualising emotion regulation processes. Through conducting this thesis portfolio, I have deepened my ability to map appropriate language and frameworks onto emotional experiences which will have ongoing

benefits for my own clinical practice. This general preference for cognitive and behavioural change may also reflect Western cultural ideals, which generally place greater value on individual agency and change in favour of engaging with and expressing difficult emotional experiences (Ramzan & Amjad, 2017). I hope that the research I've conducted will contribute to a greater appreciation of the factors influencing an individual's emotional resilience, and the significance of emotional processes, for individuals with psychosis. Investigating the potential mechanistic pathways in the aetiology of psychosis has been an incredibly fascinating process. Similarly, I hope that the work detailed in this portfolio will play a role in advancing early intervention and prevention approaches, ultimately leading to more effective support for individuals at risk of or currently experiencing psychosis.

The research process presented several challenges, principally the sheer scale and attention to detail required to conduct research at this level. However, I am already aware of the confidence and skills this experience has equipped me with, which will undoubtedly benefit my future research endeavours. The empirical study was anxiety-inducing at times, particularly since all measures were completed remotely, leaving me completely unaware about the outcome of the results until it came to analysis. Conducting an experimental study that I knew would induce a level of psychological discomfort and functional impairment was unfamiliar as a clinician, whose primary focus is usually to alleviate distress. It was surprising; however, how willing participants were to undergo a night of partial sleep restriction, which may reflect the typical student lifestyle of participants that involves frequent late nights studying or socialising. As a result, some individuals who expressed interest were disappointed when they did not meet the inclusion criteria, and it was challenging to manage this while also navigating a conversation that included signposting them to resources for sleep or mental health that might benefit them. The use of supervision was invaluable for reorientating myself to the bigger picture of the research, and for finding balance in my roles as both a clinician and a researcher.

Final conclusions

The overall findings from this portfolio offer valuable contributions to our theoretical understanding of the aetiology of psychosis. The empirical project confirmed that significant increases in psychotic experiences can be observed after just a single night of sleep loss in a non-clinical sample, evidencing the significant role of sleep disturbances in influencing individuals' vulnerability to these experiences. The results from this portfolio also support the existence of a close relationship between psychotic experiences and regulatory affective processes, in particular dissociation, emphasizing the value of emotion regulation as a key

treatment target for alleviating symptoms. When considered alongside the growing evidence base, these findings may contribute to the development of effective early intervention and prevention strategies, as well as targeted interventions that improve clinical outcomes and quality of life for individuals with psychosis.

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Appendices

List of Appendices

Appendix A. Clinical Psychology Review: Author Guidelines

Writing and formatting

Title page

You are required to include the following details in the title page information:

- Article title. Article titles should be concise and informative. Please avoid abbreviations and formulae, where possible, unless they are established and widely understood, e.g., DNA).
- Author names. Provide the given name(s) and family name(s) of each author. The order of authors should match the order in the submission system. Carefully check that all names are accurately spelled. If needed, you can add your name between parentheses in your own script after the English transliteration.
- Affiliations. Add affiliation addresses, referring to where the work was carried out, below the author names. Indicate affiliations using a lower-case superscript letter immediately after the author's name and in front of the corresponding address. Ensure that you provide the full postal address of each affiliation, including the country name and, if available, the email address of each author.
- Corresponding author. Clearly indicate who will handle correspondence for your article at all stages of the refereeing and publication process and also post-publication. This responsibility includes answering any future queries about your results, data, methodology and materials. It is important that the email address and contact details of your corresponding author are kept up to date during the submission and publication process.
- Present/permanent address. If an author has moved since the work described in your article was carried out, or the author was visiting during that time, a "present address" (or "permanent address") can be indicated by a footnote to the author's name. The address where the author carried out the work must be retained as their main affiliation address. Use superscript Arabic numerals for such footnotes.

Abstract

You are required to provide a concise and factual abstract which does not exceed 250 words. The abstract should briefly state the purpose of your research, principal results and major conclusions. Some guidelines:

- Abstracts must be able to stand alone as abstracts are often presented separately from the article.
- Avoid references. If any are essential to include, ensure that you cite the author(s) and year(s).
- Avoid non-standard or uncommon abbreviations. If any are essential to include, ensure they are defined within your abstract at first mention.

Keywords

You are required to provide 1 to 7 keywords for indexing purposes. Keywords should be written in English. Please try to avoid keywords consisting of multiple words (using "and" or "of").

We recommend that you only use abbreviations in keywords if they are firmly established in the field.

Highlights

You are required to provide article highlights at submission.

Highlights are a short collection of bullet points that should capture the novel results of your research as well as any new methods used during your study. Highlights will help increase the discoverability of your article via search engines. Some guidelines:

- Submit highlights as a separate editable file in the online submission system with the word "highlights" included in the file name.
- Highlights should consist of 3 to 5 bullet points, each a maximum of 85 characters, including spaces.

We encourage you to view example article highlights and read about the benefits of their inclusion.

Tables

Tables must be submitted as editable text, not as images. Some guidelines:

- Place tables next to the relevant text or on a separate page(s) at the end of your article.
- Cite all tables in the manuscript text.
- Number tables consecutively according to their appearance in the text.
- Please provide captions along with the tables.
- Place any table notes below the table body.
- Avoid vertical rules and shading within table cells.

We recommend that you use tables sparingly, ensuring that any data presented in tables is not duplicating results described elsewhere in the article.

Figure captions

All images must have a caption. A caption should consist of a brief title (not displayed on the figure itself) and a description of the image. We advise you to keep the amount of text in any image to a minimum, though any symbols and abbreviations used should be explained.

Color artwork

If you submit usable color figures with your accepted article, we will ensure that they appear in color online.

Please ensure that color images are accessible to all, including those with impaired color vision. Learn more about color and web accessibility.

For articles appearing in print, you will be sent information on costs to reproduce color in the printed version, after your accepted article has been sent to production. At this stage, please indicate if your preference is to have color only in the online version of your article or also in the printed version.

Article structure

Article sections

Divide your manuscript into clearly defined sections covering all essential elements using headings.

Glossary

Please provide definitions of field-specific terms used in your article, in a separate list.

Footnotes

We advise you to use footnotes sparingly. If you include footnotes in your article, ensure that they are numbered consecutively.

You may use system features that automatically build footnotes into text. Alternatively, you can indicate the position of footnotes within the text and present them in a separate section at the end of your article.

Acknowledgements

Include any individuals who provided you with help during your research, such as help with language, writing or proof reading, in the acknowledgements section. Acknowledgements should be placed in a separate section which appears directly before the reference list. Do not include acknowledgements on your title page, as a footnote to your title, or anywhere else in your article other than in the separate acknowledgements section.

Funding sources

Authors must disclose any funding sources who provided financial support for the conduct of the research and/or preparation of the article. The role of sponsors, if any, should be declared in relation to the study design, collection, analysis and interpretation of data, writing of the report and decision to submit the article for publication. If funding sources had no such involvement this should be stated in your submission.

List funding sources in this standard way to facilitate compliance to funder's requirements:

Funding: This work was supported by the National Institutes of Health [grant numbers xxxx, yyyy]; the Bill & Melinda Gates Foundation, Seattle, WA [grant number zzzz]; and the United States Institutes of Peace [grant number aaaa].

It is not necessary to include detailed descriptions on the program or type of grants, scholarships and awards. When funding is from a block grant or other resources available to a university, college, or other research institution, submit the name of the institute or organization that provided the funding.

If no funding has been provided for the research, it is recommended to include the following sentence:

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Appendices

We ask you to use the following format for appendices:

- Identify individual appendices within your article using the format: A, B, etc.
- Give separate numbering to formulae and equations within appendices using formats such as Eq. (A.1), Eq. (A.2), etc. and in subsequent appendices, Eq. (B.1), Eq. (B. 2) etc. In a similar way, give separate numbering to tables and figures using formats such as Table A.1; Fig. A.1, etc.

Journal specific information

Manuscripts should be prepared according to the guidelines set forth in the most recent publication manual of the American Psychological Association. Of note, section headings should not be numbered.

Manuscripts should ordinarily not exceed 50 pages, including references and tabular material. Exceptions may be made with prior approval of the Editor in Chief. Manuscript length can often be managed through the judicious use of appendices. In general the References section should be limited to citations actually discussed in the text. References to articles solely included in meta-analyses should be included in an appendix, which will appear in the on line version of the paper but not in the print copy. Similarly, extensive Tables describing study characteristics, containing material published elsewhere, or presenting formulas and other technical material should also be included in an appendix. Authors can direct readers to the appendices in appropriate places in the text.

It is authors' responsibility to ensure their reviews are comprehensive and as up to date as possible (at least to 3 months within date of submission) so the data are still current at the time of publication. Authors are referred to the PRISMA Guidelines (http://www.prisma-statement.org/) for guidance in conducting reviews and preparing manuscripts. Adherence to the Guidelines is not required, but is recommended to enhance quality of submissions and impact of published papers on the field.

References

References within text

Any references cited within your article should also be present in your reference list and vice versa. Some guidelines:

- References cited in your abstract must be given in full.
- We recommend that you do not include unpublished results and personal communications in your reference list, though you may mention them in the text of your article.
- Any unpublished results and personal communications included in your reference list must follow the standard reference style of the journal. In substitution of the publication date add "unpublished results" or "personal communication."
- References cited as "in press" imply that the item has been accepted for publication.

Linking to cited sources will increase the discoverability of your research.

Before submission, check that all data provided in your reference list are correct, including any references which have been copied. Providing correct reference data allows us to link to abstracting and indexing services such as Scopus, Crossref and PubMed. Any incorrect surnames, journal or book titles, publication years or pagination within your references may prevent link creation.

We encourage the use of Digital Object Identifiers (DOIs) as reference links as they provide a permanent link to the electronic article referenced.

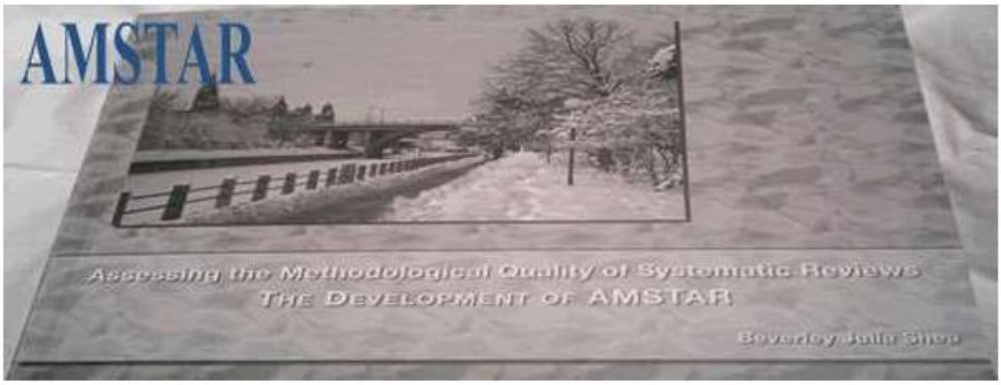
Reference style

Citations in the text should follow the referencing style used by the American Psychological Association. You are referred to the *Publication Manual of the American Psychological Association,* Seventh Edition (2020) ISBN 978-1-4338-3215-4.

The reference list should be arranged alphabetically and then chronologically. More than one reference from the same author(s) in the same year must be identified by the letters 'a', 'b', 'c', etc., placed after the year of publication.

Appendix B. AMSTAR 2 and PRISMA 2020 Checklists

AMSTAR 2 Checklist



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AMSTAR 2 Results

Printer Friendly Version

Article Name:
Emotion regulation: A bridge between sleep loss and psychotic experiences?

You are currently logged on as Guest. You need to be logged on as a member to submit your score.
Log On

Emotion regulation: A bridge between sleep loss and psychotic experiences? is a Moderate quality review

1. Did the research questions and inclusion criteria for the review include the components of PICO? Yes

2. Did the report of the review contain an explicit statement that the review methods were established prior to the conduct of the review and did the report justify any significant deviations from the protocol? Partial Yes

3. Did the review authors explain their selection of the study designs for inclusion in the review? No

4. Did the review authors use a comprehensive literature search strategy? Partial Yes

5. Did the review authors perform study selection in duplicate?	Yes
6. Did the review authors perform data extraction in duplicate?	No
7. Did the review authors provide a list of excluded studies and justify the exclusions?	Yes
8. Did the review authors describe the included studies in adequate detail?	Yes
9. Did the review authors use a satisfactory technique for assessing the risk of bias (RoB) in individual studies that were included in the review?	
RCT	Partial Yes
NRSI	Partial Yes
10. Did the review authors report on the sources of funding for the studies included in the review?	Yes
11. If meta-analysis was performed did the review authors use appropriate methods for statistical combination of results?	
RCT	0
NRSI	0
12. If meta-analysis was performed, did the review authors assess the potential impact of RoB in	0

individual studies on the results of the meta-analysis or other evidence synthesis?	
13. Did the review authors account for RoB in individual studies when interpreting/ discussing the results of the review?	Yes
14. Did the review authors provide a satisfactory explanation for, and discussion of, any heterogeneity observed in the results of the review?	Yes
15. If they performed quantitative synthesis did the review authors carry out an adequate investigation of publication bias (small study bias) and discuss its likely impact on the results of the review?	0
16. Did the review authors report any potential sources of conflict of interest, including any funding they received for conducting the review?	Yes

To cite this tool: Shea BJ, Reeves BC, Wells G, Thuku M, Hamel C, Moran J, Moher D, Tugwell P, Welch V, Kristjansson E, Henry DA. AMSTAR 2: a critical appraisal tool for systematic reviews that include randomised or non-randomised studies of healthcare interventions, or both. BMJ. 2017 Sep 21;358:j4008.

<< Back

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Section and Topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	✓
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	✓
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	✓
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	✓
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	✓
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	✓
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	✓
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	✓
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	✓
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	X
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	✓
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	✓
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	X
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	✓
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	X
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	✓
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	✓
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	X
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	X
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	X
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	X

Section and Topic	Item #	Checklist item	Location where item is reported
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	✓
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	✓
Study characteristics	17	Cite each included study and present its characteristics.	✓
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	✓
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	X
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	✓
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	X
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	X
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	X
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	X
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	X
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	✓
	23b	Discuss any limitations of the evidence included in the review.	✓
	23c	Discuss any limitations of the review processes used.	✓
	23d	Discuss implications of the results for practice, policy, and future research.	✓
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	✓
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	✓
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	✓
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	✓
Competing interests	26	Declare any competing interests of review authors.	✓
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	X

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n71. This work is licensed under CC BY 4.0. To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/

Appendix C. Systematic Review Search Strategy

Search dates: Initial search to be conducted in August 2024. Likely to run an updated search with the same search terms/databases in July 2025 for studies published since the initial search.

Databases: PubMed/MEDLINE, PsychINFO and Web of Science.

Restrictions: Papers must be empirical studies, must be available in English and must have been published since 1994.

Search terms:

> Sleep* OR “sleep loss” OR “sleep depriv*” OR “sleep reduc*” OR “reduc* sleep” OR “sleep restrict*” OR “restrict* sleep” OR “sleep quality” OR “sleep deficit” OR “sleep fragment*” OR “fragment* sleep”
>
> OR
>
> Delus* OR hallucinat* OR psychosis OR psychotic OR paranoi* OR “disorganized speech” OR “disorganised speech” OR “disorganized thought” OR “disorganized thought” OR “disorganized thinking” OR “disorganized thinking” OR schizophrenia OR “psychotic disorder” OR “schizoaffective disorder”
>
> AND
>
> "Emotion *regulation" OR "affect *regulation" OR “emotion* reactivity” OR "experiential avoidance" OR "emotion* avoidance" OR "behavio* avoidance" OR “situation* avoid*” OR "expressive suppression" OR "thought suppression" OR "emotion* suppression" OR rumination OR distraction OR reapprais* OR “attentional deployment” OR “emotion regulation questionnaire” OR ERQ OR “cognitive emotion regulation questionnaire” OR CERQ OR “difficulties in emotion regulation scale” OR DERS

MEDLINE Search Input

((Sleep* OR "sleep loss" OR "sleep depriv*" OR "sleep reduc*" OR "reduc* sleep" OR "sleep restrict*" OR "restrict* sleep" OR "sleep quality" OR "sleep deficit" OR "sleep fragment*" OR "fragment* sleep") OR (Delus* OR hallucinat* OR psychosis OR psychotic OR paranoi* OR "disorganized speech" OR "disorganised speech" OR "disorganized thought" OR "disorganized thought" OR "disorganized thinking" OR "disorganized thinking" OR schizophrenia OR "psychotic disorder" OR "schizoaffective disorder")) AND ("Emotion *regulation" OR "affect *regulation" OR "emotion* reactivity" OR "experiential avoidance" OR "emotion* avoidance" OR "behavio* avoidance" OR "situation* avoid*" OR "expressive suppression" OR "thought suppression" OR "emotion* suppression" OR rumination OR distraction OR reapprais* OR "attentional deployment" OR "emotion regulation questionnaire" OR ERQ OR "cognitive emotion regulation questionnaire" OR CERQ OR "difficulties in emotion regulation scale" OR DERS)

PsychInfo Search Input

((Sleep* OR "sleep loss" OR "sleep depriv*" OR "sleep reduc*" OR "reduc* sleep" OR "sleep restrict*" OR "restrict* sleep" OR "sleep quality" OR "sleep deficit" OR "sleep fragment*" OR "fragment* sleep")

OR

(Delus* OR hallucinat* OR psychosis OR psychotic OR paranoi* OR "disorganized speech" OR "disorganised speech" OR "disorganized thought" OR "disorganised thought" OR "disorganized thinking" OR "disorganised thinking" OR schizophrenia OR "psychotic disorder" OR "schizoaffective disorder"))

AND

("Emotion* regulation" OR "affect* regulation" OR "emotion* reactivity" OR "experiential avoidance" OR "emotion* avoidance" OR "behavio* avoidance" OR "situation* avoid*" OR "expressive suppression" OR "thought suppression" OR "emotion* suppression" OR rumination OR distraction OR reapprais* OR "attentional deployment" OR "emotion regulation questionnaire" OR ERQ OR "cognitive emotion regulation questionnaire" OR CERQ OR "difficulties in emotion regulation scale" OR DERS)

Web of Science Search Input

TS=(("Sleep*" OR "sleep loss" OR "sleep depriv*" OR "sleep reduc*" OR "reduc* sleep" OR "sleep restrict*" OR "restrict* sleep" OR "sleep quality" OR "sleep deficit" OR "sleep fragment*" OR "fragment* sleep")

OR

(Delus* OR hallucinat* OR psychosis OR psychotic OR paranoi* OR "disorganized speech" OR "disorganised speech" OR "disorganized thought" OR "disorganised thought" OR "disorganized thinking" OR "disorganised thinking" OR schizophrenia OR "psychotic disorder" OR "schizoaffective disorder"))

AND

("Emotion* regulation" OR "affect* regulation" OR "emotion* reactivity" OR "experiential avoidance" OR "emotion* avoidance" OR "behavio* avoidance" OR "situation* avoid*" OR "expressive suppression" OR "thought suppression" OR "emotion* suppression" OR rumination OR distraction OR reapprais* OR "attentional deployment" OR "emotion regulation questionnaire" OR ERQ OR "cognitive emotion regulation questionnaire" OR CERQ OR "difficulties in emotion regulation scale" OR DERS))

Appendix D. Studies Excluded Following Full-Text Screening

Study Citation	Reason for Exclusion
Bahlinger et al., 2022	Measure at single timepoint only
Baker et al., 2011	Measured emotion regulation ability and not use or choice of strategy
Baum et al., 2014	Measured emotion regulation ability and not use or choice of strategy
Becker et al., 2020	(RQ1) Population not non-clinical
Benasi et al., 2024	Emotion regulation measure assessed single domain only
Burhan & Karadere, 2021	Emotion regulation measure assessed single domain only
Burke et al., 2020	Emotion regulation measure assessed single domain only
Campbell et al., 2020	Abstract of full text already included
Campbell et al., 2022	Emotion regulation measure assessed single domain only
Capuano et al., 2016	(RQ2) No psychosis measure
Chien et al., 2019	Emotion regulation measure assessed single domain only
Cruz-Sanabria et al., 2022	Emotion regulation measure assessed single domain only
Dhaka & Naveen, 2018	Measured emotion regulation ability and not use or choice of strategy
Dingle et al., 2017	(RQ2) No psychosis measure
Eack et al., 2009	(RQ2) Intervention was not primarily targeting emotions
Eack et al., 2016	(RQ2) Intervention was not primarily targeting emotions
Freeman et al., 2015	(RQ2) Intervention was not primarily targeting emotions
Gaudiano et al., 2013	Emotion regulation measure assessed single domain only
Gaudiano et al., 2015	Emotion regulation measure assessed single domain only
Heggdal et al., 2016	Emotion regulation measure assessed single domain only
Hepworth et al., 2011	Emotion regulation measure assessed single domain only
Hsu & Ouyang, 2021	(RQ2) Intervention was not primarily targeting emotions
Hutton et al., 2014	(RQ2) Intervention was not primarily targeting emotions
Igra et al., 2023	Wrong study design
Jansen & Morris, 2017	Emotion regulation measure assessed single domain only
Jolley et al., 2020	Emotion regulation measure assessed single domain only
Kalak et al., 2012	Wrong study design
Khakbaz et al., 2023	(RQ2) Intervention was not primarily targeting emotions
Langlois et al., 2020	Emotion regulation measure assessed single domain only
Lawlor et al., 2022	(RQ2) No psychosis measure
Li et al., 2023	Measure at single timepoint only
Lincoln et al., 2015	Measure at single timepoint only
Lombardo et al., 2016	Measured emotion regulation ability and not use or choice of strategy
Lowery et al., 2014	Emotion regulation measure assessed single domain only
Mak et al., 2021	(RQ2) No psychosis measure
Manning, 2019	(RQ2) Intervention was not primarily targeting emotions
Martins et al., 2018	Emotion regulation measure assessed single domain only
Nguyen, 2021	Measure at single timepoint only
Paetzold et al., 2022	(RQ2) No psychosis measure
Reddy et al., 2017	Measured emotion regulation ability and not use or choice of strategy
Ricarte et al., 2014	(RQ2) Intervention was not primarily targeting emotions
Salvi et al., 2016	(RQ2) No psychosis measure
Scarinci et al., 2021	(RQ2) No psychosis measure
Selvi et al., 2015	Emotion regulation measure assessed single domain only

Sharma et al., 2021	Emotion regulation measure assessed single domain only
Shi et al., 2023	Emotion regulation measure assessed single domain only
Spidel et al., 2018	Emotion regulation measure assessed single domain only
Spidel et al., 2019	Emotion regulation measure assessed single domain only
Spinelli et al., 2020	(RQ2) No psychosis measure
Stenson et al., 2021	Measured emotion regulation ability and not use or choice of strategy
Tagalidou et al., 2019	Emotion regulation measure assessed single domain only
Tamm, 2019	Measured emotion regulation ability and not use or choice of strategy
Taraku et al., 2023	Measure of emotion regulation neural only
Ten Brink et al., 2020	Wrong study design
Trousselard et al., 2016	Emotion regulation measure assessed single domain only
Vezmar et al., 2024	(RQ2) No psychosis measure
Weintraub et al., 2023	(RQ2) Intervention was not primarily targeting emotions
Xue et al., 2024	Emotion regulation measure assessed single domain only

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1 Bahlinger, K., Lincoln, T. M., & Clamor, A. (2022). Recovery after stress—autonomic and subjective arousal in individuals with psychosis compared to healthy controls. *Schizophrenia Bulletin*, *48*(6), 1373-1383.

2 Baker, L. D., Lymn, J., Wingfield, M., Wickens, R., Baldwin, D. D., & Garner, M. (2011). Associations between disturbed sleep, emotion regulation, attention to threat and cognitive control in healthy volunteers. *Journal of Psychopharmacology, 25*(8), A56-A56.

3 Baum, K. T., Desai, A., Field, J., Miller, L. E., Rausch, J., & Beebe, D. W. (2014). Sleep restriction worsens mood and emotion regulation in adolescents. *Journal of Child Psychology and Psychiatry*, *55*(2), 180-190.

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6 Burhan, H. Ş., & Karadere, E. (2021). Effectiveness of acceptance and commitment therapy for patients with psychosis being monitored at a community mental health center: A six-month follow-up study. *Alpha Psychiatry*, *22*(4), 206-211.

7 Burke, A. S., Shapero, B. G., Pelletier-Baldelli, A., Deng, W. Y., Nyer, M. B., Leathem, L., Namey, L., Landa, C., Cather, C., & Holt, D. J. (2020). Rationale, methods, feasibility, and preliminary outcomes of a transdiagnostic prevention program for at-risk college students. *Frontiers in Psychiatry*, *10*, 1030.

8 Campbell, R. L., Lindsay, E., Vance, A., Nguyen, A., Feldner, M., & Leen-Feldner, E. (2020). 0220 Sleep Deprivation Increases Self-Reported But Not Behavioral Avoidance. *Sleep*, *43*, A85-A86.

9 Campbell, R. L., Feldner, M. T., & Leen-Feldner, E. W. (2022). An experimental test of the effects of acute sleep deprivation on affect and avoidance. *Journal of Behavior Therapy and Experimental Psychiatry*, *77*, 101770.

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Appendix E. NHLBI National Institute of Health's (2021) Quality Assessment Tool for Controlled Intervention Studies with Guidance

Criteria	Yes	No	Other (CD, NR, NA)*
1. Was the study described as randomized, a randomized trial, a randomized clinical trial, or an RCT?			
2. Was the method of randomization adequate (i.e., use of randomly generated assignment)?			
3. Was the treatment allocation concealed (so that assignments could not be predicted)?			
4. Were study participants and providers blinded to treatment group assignment?			
5. Were the people assessing the outcomes blinded to the participants' group assignments?			
6. Were the groups similar at baseline on important characteristics that could affect outcomes (e.g., demographics, risk factors, co-morbid conditions)?			
7. Was the overall drop-out rate from the study at endpoint 20% or lower of the number allocated to treatment?			
8. Was the differential drop-out rate (between treatment groups) at endpoint 15 percentage points or lower?			
9. Was there high adherence to the intervention protocols for each treatment group?			
10. Were other interventions avoided or similar in the groups (e.g., similar background treatments)?			
11. Were outcomes assessed using valid and reliable measures, implemented consistently across all study participants?			
12. Did the authors report that the sample size was sufficiently large to be able to detect a difference in the main outcome between groups with at least 80% power?			
13. Were outcomes reported or subgroups analyzed prespecified (i.e., identified before analyses were conducted)?			
14. Were all randomized participants analyzed in the group to which they were originally assigned, i.e., did they use an intention-to-treat analysis?			

Quality Rating (Good, Fair, or Poor)

Rater #1 initials:

Rater #2 initials:

Additional Comments (If POOR, please state why):

*CD, cannot determine; NA, not applicable; NR, not reported

Guidance for Assessing the Quality of Controlled Intervention Studies

The guidance document below is organized by question number from the tool for quality assessment of controlled intervention studies.

Question 1. Described as randomized

Was the study described as randomized? A study does not satisfy quality criteria as randomized simply because the authors call it randomized; however, it is a first step in determining if a study is randomized

Questions 2 and 3. Treatment allocation–two interrelated pieces

Adequate randomization: Randomization is adequate if it occurred according to the play of chance (e.g., computer generated sequence in more recent studies, or random number table in older studies).
Inadequate randomization: Randomization is inadequate if there is a preset plan (e.g., alternation where every other subject is assigned to treatment arm or another method of allocation is used, such as time or day of hospital admission or clinic visit, ZIP Code, phone number, etc.). In fact, this is not randomization at all–it is another method of assignment to groups. If assignment is not by the play of chance, then the answer to this question is no.
There may be some tricky scenarios that will need to be read carefully and considered for the role of chance in assignment. For example, randomization may occur at the site level, where all individuals at a particular site are assigned to receive treatment or no treatment. This scenario is used for group-randomized trials, which can be truly randomized, but often are "quasi-experimental" studies with comparison groups rather than true control groups. (Few, if any, group-randomized trials are anticipated for this evidence review.)

Allocation concealment: This means that one does not know in advance, or cannot guess accurately, to what group the next person eligible for randomization will be assigned. Methods include sequentially numbered opaque sealed envelopes, numbered or coded containers, central randomization by a coordinating center, computer-generated randomization that is not revealed ahead of time, etc.

Questions 4 and 5. Blinding

Blinding means that one does not know to which group–intervention or control–the participant is assigned. It is also sometimes called "masking." The reviewer assessed whether each of the following was blinded to knowledge of treatment assignment: (1) the person assessing the primary outcome(s) for the study (e.g., taking the measurements such as blood pressure, examining health records for events such as myocardial infarction, reviewing and interpreting test results such as x ray or cardiac catheterization findings); (2) the person receiving the intervention (e.g., the patient or other study participant); and (3) the person providing the intervention (e.g., the physician, nurse, pharmacist, dietitian, or behavioral interventionist).

Generally placebo-controlled medication studies are blinded to patient, provider, and outcome assessors; behavioral, lifestyle, and surgical studies are examples of studies that are frequently blinded only to the outcome assessors because blinding of the persons providing and receiving the interventions is difficult in these situations. Sometimes the individual providing the intervention is the same person performing the outcome assessment. This was noted when it occurred.

Question 6. Similarity of groups at baseline

This question relates to whether the intervention and control groups have similar baseline characteristics on average especially those characteristics that may affect the intervention or outcomes. The point of randomized trials is to create groups that are as similar as possible except for the intervention(s) being studied in order to compare the effects of the interventions between groups. When reviewers abstracted baseline characteristics, they noted when there was a significant difference between groups. Baseline characteristics for intervention groups are usually presented in a table in the article (often Table 1).

Groups can differ at baseline without raising red flags if: (1) the differences would not be expected to have any bearing on the interventions and outcomes; or (2) the differences are not statistically significant. When concerned about baseline difference in groups, reviewers recorded them in the comments section and considered them in their overall determination of the study quality.

Questions 7 and 8. Dropout

"Dropouts" in a clinical trial are individuals for whom there are no end point measurements, often because they dropped out of the study and were lost to followup.

Generally, an acceptable overall dropout rate is considered 20 percent or less of participants who were randomized or allocated into each group. An acceptable differential dropout rate is an absolute difference between groups of 15 percentage points at most (calculated by subtracting the dropout rate of one group minus the dropout rate of the other group). However, these are general rates. Lower overall dropout rates are expected in shorter studies, whereas higher overall dropout rates may be acceptable for studies of longer duration. For example, a 6-month study of weight loss interventions should be expected to have nearly 100 percent followup (almost no dropouts–nearly everybody gets their weight measured regardless of whether or not they actually received the intervention), whereas a 10-year study testing the effects of intensive blood pressure lowering on heart attacks may be acceptable if there is a 20-25 percent dropout rate, especially if the dropout rate between groups was similar. The panels for the NHLBI systematic reviews may set different levels of dropout caps.

Conversely, differential dropout rates are not flexible; there should be a 15 percent cap. If there is a differential dropout rate of 15 percent or higher between arms, then there is a serious potential for bias. This constitutes a fatal flaw, resulting in a poor quality rating for the study.

Question 9. Adherence

Did participants in each treatment group adhere to the protocols for assigned interventions? For example, if Group 1 was assigned to 10 mg/day of Drug A, did most of them take 10 mg/day of Drug A? Another example is a study evaluating the difference between a 30-pound weight loss and a 10-pound weight loss on specific clinical outcomes (e.g., heart attacks), but the 30-pound weight loss group did not achieve its intended weight loss target (e.g., the group only lost 14 pounds on average). A third example is whether a large percentage of participants assigned to one group "crossed over" and got the intervention provided to the other group. A final example is when one group that was assigned to receive a particular drug at a particular dose had a large percentage of participants who did not end up taking the drug or the dose as designed in the protocol.

Question 10. Avoid other interventions

Changes that occur in the study outcomes being assessed should be attributable to the interventions being compared in the study. If study participants receive interventions that are not part of the study protocol and could affect the outcomes being assessed, and they receive these interventions differentially, then there is cause for concern because these interventions could bias results. The following scenario is another example of how bias can occur. In a study comparing two different dietary interventions on serum cholesterol, one group had a significantly higher percentage of participants taking statin drugs than the other group. In this situation, it would be impossible to know if a difference in outcome was due to the dietary intervention or the drugs.

Question 11. Outcome measures assessment

What tools or methods were used to measure the outcomes in the study? Were the tools and methods accurate and reliable–for example, have they been validated, or are they objective? This is important as it indicates the confidence you can have in the reported outcomes. Perhaps even more important is ascertaining that outcomes were assessed in the same manner within and between groups. One example of differing methods is self-report of dietary salt intake versus urine testing for sodium content (a more reliable and valid assessment method). Another example is using BP measurements taken by practitioners who use their usual methods versus using BP measurements done by individuals trained in

a standard approach. Such an approach may include using the same instrument each time and taking an individual's BP multiple times. In each of these cases, the answer to this assessment question would be "no" for the former scenario and "yes" for the latter. In addition, a study in which an intervention group was seen more frequently than the control group, enabling more opportunities to report clinical events, would not be considered reliable and valid.

Question 12. Power calculation

Generally, a study's methods section will address the sample size needed to detect differences in primary outcomes. The current standard is at least 80 percent power to detect a clinically relevant difference in an outcome using a two-sided alpha of 0.05. Often, however, older studies will not report on power.

Question 13. Prespecified outcomes

Investigators should prespecify outcomes reported in a study for hypothesis testing–which is the reason for conducting an RCT. Without prespecified outcomes, the study may be reporting ad hoc analyses, simply looking for differences supporting desired findings. Investigators also should prespecify subgroups being examined. Most RCTs conduct numerous post hoc analyses as a way of exploring findings and generating additional hypotheses. The intent of this question is to give more weight to reports that are not simply exploratory in nature.

Question 14. Intention-to-treat analysis

Intention-to-treat (ITT) means everybody who was randomized is analyzed according to the original group to which they are assigned. This is an extremely important concept because conducting an ITT analysis preserves the whole reason for doing a randomized trial; that is, to compare groups that differ only in the intervention being tested. When the ITT philosophy is not followed, groups being compared may no longer be the same. In this situation, the study would likely be rated poor. However, if an investigator used another type of analysis that could be viewed as valid, this would be explained in the "other" box on the quality assessment form. Some researchers use a completers analysis (an analysis of only the participants who completed the intervention and the study), which introduces significant potential for bias. Characteristics of participants who do not complete the study are unlikely to be the same as those who do. The likely impact of participants withdrawing from a study treatment must be considered carefully. ITT analysis provides a more conservative (potentially less biased) estimate of effectiveness.

General Guidance for Determining the Overall Quality Rating of Controlled Intervention Studies

The questions on the assessment tool were designed to help reviewers focus on the key concepts for evaluating a study's internal validity. They are not intended to create a list that is simply tallied up to arrive at a summary judgment of quality.

Internal validity is the extent to which the results (effects) reported in a study can truly be attributed to the intervention being evaluated and not to flaws in the design or conduct of the study–in other words, the ability for the study to make causal conclusions about the effects of the intervention being tested. Such flaws can increase the risk of bias. Critical appraisal involves considering the risk of potential for allocation bias, measurement bias, or confounding (the mixture of exposures that one cannot tease out from each other). Examples of confounding include co-interventions, differences at baseline in patient characteristics, and other issues addressed in the questions above. High risk of bias translates to a rating of poor quality. Low risk of bias translates to a rating of good quality.

Fatal flaws: If a study has a "fatal flaw," then risk of bias is significant, and the study is of poor quality. Examples of fatal flaws in RCTs include high dropout rates, high differential dropout rates, no ITT analysis or other unsuitable statistical analysis (e.g., completers-only analysis).

Generally, when evaluating a study, one will not see a "fatal flaw;" however, one will find some risk of bias. During training, reviewers were instructed to look for the potential for bias in studies by focusing on the concepts underlying the questions in the tool. For any box checked "no," reviewers were told to ask: "What is the potential risk of bias that may be introduced by this flaw?" That is, does this factor cause one to doubt the results that were reported in the study?

Appendix F. NHLBI National Institute of Health's (2021) Quality Assessment Tool for Before-After (Pre-Post) Studies with No Control Group with Guidance

Criteria	Yes	No	Other (CD, NR, NA)*
1. Was the study question or objective clearly stated?			
2. Were eligibility/selection criteria for the study population prespecified and clearly described?			
3. Were the participants in the study representative of those who would be eligible for the test/service/intervention in the general or clinical population of interest?			
4. Were all eligible participants that met the prespecified entry criteria enrolled?			
5. Was the sample size sufficiently large to provide confidence in the findings?			
6. Was the test/service/intervention clearly described and delivered consistently across the study population?			
7. Were the outcome measures prespecified, clearly defined, valid, reliable, and assessed consistently across all study participants?			
8. Were the people assessing the outcomes blinded to the participants' exposures/interventions?			
9. Was the loss to follow-up after baseline 20% or less? Were those lost to follow-up accounted for in the analysis?			
10. Did the statistical methods examine changes in outcome measures from before to after the intervention? Were statistical tests done that provided p values for the pre-to-post changes?			
11. Were outcome measures of interest taken multiple times before the intervention and multiple times after the intervention (i.e., did they use an interrupted time-series design)?			
12. If the intervention was conducted at a group level (e.g., a whole hospital, a community, etc.) did the statistical analysis take into account the use of individual-level data to determine effects at the group level?			

Quality Rating (Good, Fair, or Poor) (see guidance)

Rater #1 Initials:

Rater #2 Initials:

Additional Comments (If POOR, please state why):

*CD, cannot determine; NA, not applicable; NR, not reported

Guidance for Assessing the Quality of Before-After (Pre-Post) Studies With No Control Group

The guidance document below is organized by question number from the tool for quality assessment of controlled intervention studies.

Question 1. Study question

Did the authors describe their goal in conducting this research? Is it easy to understand what they were looking to find? This issue is important for any scientific paper of any type. Higher quality scientific research explicitly defines a research question.

Question 2. Eligibility criteria and study population

Did the authors describe the eligibility criteria applied to the individuals from whom the study participants were selected or recruited? In other words, if the investigators were to conduct this study again, would they know whom to recruit, from where, and from what time period?

Here is a sample description of a study population: men over age 40 with type 2 diabetes, who began seeking medical care at Phoenix Good Samaritan Hospital, between January 1, 2005 and December 31, 2007. The population is clearly described as: (1) who (men over age 40 with type 2 diabetes); (2) where (Phoenix Good Samaritan Hospital); and (3) when (between January 1, 2005 and December 31, 2007). Another sample description is women who were in the nursing profession, who were ages 34 to 59 in 1995, had no known CHD, stroke, cancer, hypercholesterolemia, or diabetes, and were recruited from the 11 most populous States, with contact information obtained from State nursing boards.

To assess this question, reviewers examined prior papers on study methods (listed in reference list) when necessary.

Question 3. Study participants representative of clinical populations of interest

The participants in the study should be generally representative of the population in which the intervention will be broadly applied. Studies on small demographic subgroups may raise concerns about how the intervention will affect broader populations of interest. For example, interventions that focus on very young or very old individuals may affect middle-aged adults differently. Similarly, researchers may not be able to extrapolate study results from patients with severe chronic diseases to healthy populations.

Question 4. All eligible participants enrolled

To further explore this question, reviewers may need to ask: Did the investigators develop the I/E criteria prior to recruiting or selecting study participants? Were the same underlying I/E criteria used for all research participants? Were all subjects who met the I/E criteria enrolled in the study?

Question 5. Sample size

Did the authors present their reasons for selecting or recruiting the number of individuals included or analyzed? Did they note or discuss the statistical power of the study? This question addresses whether there was a sufficient sample size to detect an association, if one did exist.

An article's methods section may provide information on the sample size needed to detect a hypothesized difference in outcomes and a discussion on statistical power (such as, the study had 85 percent power to detect a 20 percent increase in the rate of an outcome of interest, with a 2-sided alpha of 0.05). Sometimes estimates of variance and/or estimates of effect size are given, instead of sample size calculations. In any case, if the reviewers determined that the power was sufficient to detect the effects of interest, then they would answer "yes" to Question 5.

Question 6. Intervention clearly described

Another pertinent question regarding interventions is: Was the intervention clearly defined in detail in the study? Did the authors indicate that the intervention was consistently applied to the subjects? Did the research participants have a high level of adherence to the requirements of the intervention? For example, if the investigators assigned a group to 10 mg/day of Drug A, did most participants in this group take the specific dosage of Drug A? Or did a large percentage of participants end up not taking the specific dose of Drug A indicated in the study protocol?

Reviewers ascertained that changes in study outcomes could be attributed to study interventions. If participants received interventions that were not part of the study protocol and could affect the outcomes being assessed, the results could be biased.

Question 7. Outcome measures clearly described, valid, and reliable

Were the outcomes defined in detail? Were the tools or methods for measuring outcomes accurate and reliable–for example, have they been validated or are they objective? This question is important because the answer influences confidence in the validity of study results.

An example of an outcome measure that is objective, accurate, and reliable is death–the outcome measured with more accuracy than any other. But even with a measure as objective as death, differences can exist in the accuracy and reliability of how investigators assessed death. For example, did they base it on an autopsy report, death certificate, death registry, or report from a family member? Another example of a valid study is one whose objective is to determine if dietary fat intake affects blood cholesterol level (cholesterol level being the outcome) and in which the cholesterol level is measured from fasting blood samples that are all sent to the same laboratory. These examples would get a "yes."

An example of a "no" would be self-report by subjects that they had a heart attack, or self-report of how much they weight (if body weight is the outcome of interest).

Question 8. Blinding of outcome assessors

Blinding or masking means that the outcome assessors did not know whether the participants received the intervention or were exposed to the factor under study. To answer the question above, the reviewers examined articles for evidence that the person(s) assessing the outcome(s) was masked to the participants' intervention or exposure status. An outcome assessor, for example, may examine medical records to determine the outcomes that occurred in the exposed and comparison groups. Sometimes the person applying the intervention or measuring the exposure is the same person conducting the outcome assessment. In this case, the outcome assessor would not likely be blinded to the intervention or exposure status. A reviewer would note such a finding in the comments section of the assessment tool.

In assessing this criterion, the reviewers determined whether it was likely that the person(s) conducting the outcome assessment knew the exposure status of the study participants. If not, then blinding was adequate. An example of adequate blinding of the outcome assessors is to create a separate committee whose members were not involved in the care of the patient and had no information about the study participants' exposure status. Using a study protocol, committee members would review copies of participants' medical records, which would be stripped of any potential exposure information or personally identifiable information, for prespecified outcomes.

Question 9. Followup rate

Higher overall followup rates are always desirable to lower followup rates, although higher rates are expected in shorter studies, and lower overall followup rates are often seen in longer studies. Usually an acceptable overall followup rate is considered 80 percent or more of participants whose interventions or exposures were measured at baseline. However, this is a general guideline.

In accounting for those lost to followup, in the analysis, investigators may have imputed values of the outcome for those lost to followup or used other methods. For example, they may carry forward the baseline value or the last observed value of the outcome measure and use these as imputed values for the final outcome measure for research participants lost to followup.

Question 10. Statistical analysis

Were formal statistical tests used to assess the significance of the changes in the outcome measures between the before and after time periods? The reported study results should present values for statistical tests, such as p values, to document the statistical significance (or lack thereof) for the changes in the outcome measures found in the study.

Question 11. Multiple outcome measures

Were the outcome measures for each person measured more than once during the course of the before and after study periods? Multiple measurements with the same result increase confidence that the outcomes were accurately measured.

Question 12. Group-level interventions and individual-level outcome efforts

Group-level interventions are usually not relevant for clinical interventions such as bariatric surgery, in which the interventions are applied at the individual patient level. In those cases, the questions were coded as "NA" in the assessment tool.

General Guidance for Determining the Overall Quality Rating of Before-After Studies

The questions in the quality assessment tool were designed to help reviewers focus on the key concepts for evaluating the internal validity of a study. They are not intended to create a list from which to add up items to judge a study's quality.

Internal validity is the extent to which the outcome results reported in the study can truly be attributed to the intervention or exposure being evaluated, and not to biases, measurement errors, or other confounding factors that may result from flaws in the design or conduct of the study. In other words, what is the ability of the study to draw associative conclusions about the effects of the interventions or exposures on outcomes?

Critical appraisal of a study involves considering the risk of potential for selection bias, information bias, measurement bias, or confounding (the mixture of exposures that one cannot tease out from each other). Examples of confounding include co-interventions, differences at baseline in patient characteristics, and other issues throughout the questions above. High risk of bias translates to a rating of poor quality; low risk of bias translates to a rating of good quality. Again, the greater the risk of bias, the lower the quality rating of the study.

In addition, the more attention in the study design to issues that can help determine if there is a causal relationship between the exposure and outcome, the higher quality the study. These issues include exposures occurring prior to outcomes, evaluation of a dose-response gradient, accuracy of measurement of both exposure and outcome, and sufficient timeframe to see an effect.

Generally, when reviewers evaluate a study, they will not see a "fatal flaw," but instead will find some risk of bias. By focusing on the concepts underlying the questions in the quality assessment tool, reviewers should ask themselves about the potential for bias in the study they are critically appraising. For any box checked "no" reviewers should ask, "What is the potential risk of bias resulting from this flaw in study design or execution?" That is, does this factor lead to doubt about the results reported in the study or doubt about the ability of the study to accurately assess an association between the intervention or exposure and the outcome?

The best approach is to think about the questions in the assessment tool and how each one reveals something about the potential for bias in a study. Specific rules are not useful, as each study has specific nuances. In addition, being familiar with the key concepts will help reviewers be more comfortable with critical appraisal. Examples of studies rated good, fair, and poor are useful, but each study must be assessed on its own.

Appendix G. Quality Appraisal Tables

Quality Assessment of Controlled Intervention Studies

Criteria	Parsons and Young (2022)	Boon et al. (2023)	Cambell (2023)	Reid et al. (2023)	Lam et al. (2020)	Mehl et al. (2021)	Weintraub et al. (2023)	Wittkamp et al. (2023)	Zoromba et al. (2024)
1. Was the study described as randomized, a randomized trial, a randomized clinical trial, or an RCT?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
2. Was the method of randomization adequate (i.e., use of randomly generated assignment)?	No	CD	CD	Yes	Yes	Yes	Yes	Yes	Yes
3. Was the treatment allocation concealed (so that assignments could not be predicted)?	No	CD	CD	Yes	Yes	CD	CD	CD	Yes
4. Were study participants and providers blinded to treatment group assignment?	NA	NA	NA	NA	NA	NA	NA	NA	NA
5. Were the people assessing the outcomes blinded to the participants' group assignments?	NR	NR	No	NR	Yes	NR	NR	NR	Yes
6. Were the groups similar at baseline on important characteristics that could affect outcomes (e.g., demographics, risk factors, co-morbid conditions)?	CD	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
7. Was the overall drop-out rate from the study at endpoint 20% or lower of the number allocated to treatment?	No	Yes	No	Yes	Yes	No	Yes	No	Yes
8. Was the differential drop-out rate (between treatment groups) at endpoint 15 percentage points or lower?	CD	Yes	CD	Yes	Yes	Yes	Yes	Yes	Yes
9. Was there high adherence to the intervention protocols for each treatment group?	Yes	Yes	Yes	Yes	Yes	CD	Yes	Yes	Yes
10. Were other interventions avoided or similar in the groups (e.g., similar background treatments)?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

11. Were outcomes assessed using valid and reliable measures, implemented consistently across all study participants?	No	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
12. Did the authors report that the sample size was sufficiently large to be able to detect a difference in the main outcome between groups with at least 80% power?	No	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes
13. Were outcomes reported or subgroups analyzed prespecified (i.e., identified before analyses were conducted)?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
14. Were all randomized participants analyzed in the group to which they were originally assigned, i.e., did they use an intention-to-treat analysis?	No	No	No	Yes	Yes	Yes	Yes	Yes	CD
Quality rating (Good, Fair, or Poor) with rationale	Poor – absence of true randomisation and concealment from participants, poor attrition rate with no use of ITT analysis and adaptation of validated measures	Fair – low attrition and crossover design strengthen internal validity, however adaptation of validated measures and absence of ITT analysis introduce some risk of bias	Fair – internal validity supported by high adherence, good sample size and choice of analysis however poor attrition, reporting gaps and no ITT analysis	Good – methodological strengths in randomisation and concealment, strong adherence with low attrition due to lab setting, and appropriate statistical analysis	Good – small but methodologically sound study with limited attrition, appropriate power and ITT analysis	Good – difficulties with attrition however strengths in use of ITT analysis, good sample size and 12-month follow-up	Good – some reporting gaps, active treatment group with low attrition, high treatment fidelity and good use of ITT analysis	Good – difficulties with attrition however strengths in sample size, treatment adherence, active treatment comparator group and analysis (with ITT)	Good – lack of follow-up, however strengths in randomisation and concealment, appropriate attrition and use of an independent researcher for analysis

Note. CD, cannot determine; ITT, intention-to-treat; NA, not applicable; NR, not reported

Quality Assessment of Before-After (Pre-Post) Studies With No Control Group

Criteria	Khoury et al. (2015)	Ryan et al. (2021)	Silva et al. (2021)	Khakbaz et al. (2022)	Simonson (2022)	von Hardenberg et al. (2022)
1. Was the study question or objective clearly stated?	Yes	Yes	Yes	Yes	Yes	Yes
2. Were eligibility/selection criteria for the study population prespecified and clearly described?	Yes	Yes	Yes	Yes	Yes	Yes
3. Were the participants in the study representative of those who would be eligible for the test/service/intervention in the general or clinical population of interest?	Yes	Yes	CD	CD	No	Yes
4. Were all eligible participants that met the prespecified entry criteria enrolled?	No	No	No	No	Yes	No
5. Was the sample size sufficiently large to provide confidence in the findings?	No	Yes	No	No	No	No
6. Was the test/service/intervention clearly described and delivered consistently across the study population?	Yes	Yes	Yes	Yes	No	Yes
7. Were the outcome measures prespecified, clearly defined, valid, reliable, and assessed consistently across all study participants?	Yes	Yes	Yes	Yes	Yes	Yes
8. Were the people assessing the outcomes blinded to the participants' exposures/interventions?	No	No	No	No	No	No
9. Was the loss to follow-up after baseline 20% or less? Were those lost to	No	Yes	No	Yes	Yes	No

follow-up accounted for in the analysis?						
10. Did the statistical methods examine changes in outcome measures from before to after the intervention? Were statistical tests done that provided p values for the pre-to-post changes?	Yes	Yes	Yes	Yes	Yes	Yes
11. Were outcome measures of interest taken multiple times before the intervention and multiple times after the intervention (i.e., did they use an interrupted time-series design)?	Yes	Yes	Yes	Yes	No	Yes
12. If the intervention was conducted at a group level (e.g., a whole hospital, a community, etc.) did the statistical analysis take into account the use of individual-level data to determine effects at the group level?	No	Yes	NA	NA	No	No
Quality rating (Good, Fair, or Poor) with rationale	Fair – small sample size with high attrition in clinical population of interest, good treatment description and appropriate measures with follow-up	Good – incomplete enrolment however good sample size for a pilot study, detailed treatment description, choice of outcomes and use of multilevel analysis	Fair – small sample with high early attrition, however good choice of measures and analysis with follow-up	Fair – small pilot sample with incomplete enrolment, however good choice of measures and analysis with follow-up	Fair – good attrition and choice of emotion regulation measures with transparency in reporting, however limited treatment description and generalisability of sample with no follow-up	Fair –good treatment description, choice of measures and follow-up, appropriate non-parametric tests with imputation of missing data however analysis does not adjust for intra group correlation

Note. CD, cannot determine; ITT, intention-to-treat; NA, not applicable; NR, not reported

Appendix H. Schizophrenia Bulletin: Author Guidelines

Manuscript Preparation

Title Page

This page should consist of (i) the complete title of the manuscript, (ii) a running title not to exceed 50 characters including spaces, (iii) the full name of each author and the authors' institutional affiliations, (iv) name, complete address, telephone, fax, and e-mail address of the corresponding author, and (v) separate word counts of the abstract and text body. Please note that there can only be one corresponding author, per journal style

Manuscript Length

Manuscripts should be concisely worded and must not exceed 6,000 words for regular articles and major reviews, 2,500 words for invited special features, or 1,500 words for editorials and first-person accounts. The word count is inclusive of the text body (excluding abstract, figure legends, acknowledgements, references) and must appear together with the abstract word count on the title page of the manuscript. Word counts are checked upon submission and at revision and manuscripts exceeding the prescribed limits will be returned.

Abstract

For both major reviews and regular articles, provide a structured abstract of no more than 250 words comprised of the following sections with a blank line between each section: Background and Hypothesis, Study Design, Study Results, Conclusions. Unstructured abstracts are acceptable for invited special features. Abstracts should not contain unexplained abbreviations. Up to six key words that do not appear as part of the title should be provided at the end of the abstract.

Main Text

Unsolicited original manuscripts reporting novel experimental findings should be comprised of these sections, in this order: Abstract, Introduction (maximum of 650 words for Introduction), Methods, Results, Discussion (maximum of 1500 words for Discussion section), Acknowledgments, References, and Figure Legends.

The Introduction (maximum of 650 words for Introduction) should situate the study in the context of the literature and explain the goals of the study and the hypotheses tested.

The Methods section should provide sufficient detail to enable replication of the study. Citing earlier work is appropriate (even necessary) but using the citation in lieu of a full and clear description of methodology is not. Supplementary materials can be used for detailed presentation of unusually complicated study protocols and methods.

The Results section should present the results of the statistical analyses of all major study hypotheses clearly and succinctly. Each statistical test should be presented in detail with precise reporting of p-values to the appropriate number of decimal places considering the measures involved. Reporting of confidence intervals should accompany the primary results. Statistical tests should be accompanied by an estimate of effect size.

The validity of statistical testing methods and model assumptions should be carefully examined. All missing data should be reported. Statistical strategies for handling incomplete data including mixed models and multiple imputation methods should be described by clearly defined model specification and assumptions. Sensitivity analysis are strongly recommended. Multiple testing correction is required

for simultaneous multiple comparisons. When publicly available data is used (e.g., in meta-analysis), reproducible statistical analysis reports should be included to ensure high reproducibility.

Supplementary materials can be used for more detailed presentation of descriptive data, individual subject data where appropriate, and additional statistical analyses that are not critical for understanding the main findings of the study.

The Discussion section (maximum of 1500 words for Discussion section) should highlight the main findings of the study and concisely relate the findings to the published literature in the area. Both the significance and limitations of the work should be addressed. Extensive speculation is discouraged.

Number pages consecutively beginning with the title page. Spelling should conform to that used in Merriam-Webster's Collegiate Dictionary, eleventh edition. Clinical laboratory data may be expressed in conventional rather than Système International (SI) units.

Acknowledgments

These should be as brief as possible but include the names of sources of logistical support.

References

Authors are encouraged to be circumspect in compiling the reference section of their manuscripts.

Please note: references to other articles appearing in the same issue of the journal must be cited fully in the reference list.

Each reference should be cited in consecutive numerical order using superscript arabic numerals, and reference style should follow the recommendations in the *American Medical Association Manual of Style*, 11th edition.

Journal names should be abbreviated in accordance with *Index Medicus*.

Manuscripts in which the references do not follow this format will be returned for retyping. References to meeting abstracts, material not yet accepted for publication, or personal communications are not acceptable as listed references and instead should be listed parenthetically in the text. It is the authors' responsibility for obtaining the necessary permissions from colleagues to include their work as a personal communication.

Note : In the online version of *Schizophrenia Bulletin* there are automatic links from the reference section of each article to cited articles in Medline. This is a useful feature for readers, but is only possible if the references are accurate. It is the responsibility of the author to ensure the accuracy of the references in the submitted article. Downloading references directly from Medline is highly recommended.

Figures and Tables

Full length manuscripts including regular and invited theme articles should contain no more than a combined total of 5 tables and figures. Theme introductions and special features are limited to 2 tables or figures (total). Figures and tables must be referred to using arabic numbers in order of their appearance in the text (e.g., Figure 1, Figure 2, Table 1, Table 2, etc.).

Tables should be created with the table function of a word processing program; spreadsheets are not acceptable. Include only essential data, and format the table in a manner in which it should appear in the text. Each table must fit on a single manuscript page and have a short title that is self-explanatory without reference to the text. Footnotes can be used to explain any symbols or abbreviations appearing in the table. Do not duplicate data in tables and figures.

Please be aware that the figure requirements for initial online submission (peer review) and for reproduction in the journal are different. Initially, it is preferred to embed your figures within the word processing file or upload them separately as low-resolution images (.jpg, .tif, or .gif files). However, upon submission of a revised manuscript, you will be required to supply high-resolution .tif files for reproduction in the journal (1200 d.p.i. for line drawings and 300 d.p.i. for color and half-tone artwork). It is advisable to create high-resolution images first as these can be easily converted into low-resolution images for online submission. Figure legends should be typed separately from the figures in the main text document. Additional information on preparing your figures for publication can be found at OUP's Preparing and submitting your manuscript page.

Wherever possible figures should be submitted in their desired final size, to fit the width of a single (88 mm) or at most a double (180 mm) column width. All letters and numerals appearing in a particular figure should be of the same size and in proportion to the overall dimensions of the drawing. Letter labels used in figures should be in upper case in both the figure and the legend. The journal reserves the right to reduce the size of illustrative material.

Each figure should have a separate legend that clearly identifies all symbols and abbreviations used. The legend should be concise and self-explanatory and should contain enough information to be understood without reference to the text.

Note : All tables and figures reproduced from a previously published manuscript must cite the original source (in the figure legend or table footnote) and be accompanied by a letter of permission from the publisher of record or the copyright owner.

Figure accessibility and alt text

Incorporating alt text (alternative text) when submitting your paper helps to foster inclusivity and accessibility. Good alt text ensures that individuals with visual impairments or those using screen readers can comprehend the content and context of your figures. The aim of alt text is to provide concise and informative descriptions of your figure so that all readers have access to the same level of information and understanding, and that all can engage with and benefit from the visual elements integral to scholarly content. Including alt text demonstrates a commitment to accessibility and enhances the overall impact and reach of your work.

Alt text is applicable to all images, figures, illustrations, and photographs.

Alt text is only accessible via e-reader and so it won't appear as part of the typeset article.

Detailed guidance on how to draft and submit alt text.

Supplementary Material

Supporting material that is not essential for inclusion in the full text of the manuscript, but would nevertheless benefit the reader, can be made available by the publisher as supplementary material, linked to the online manuscript. The material should not be essential to understanding the conclusions of the paper, but should contain data that is additional or complementary and directly relevant to the article content. Such information might include more detailed methods, extended data sets/data analysis, or additional figures (including color). All text and figures must be provided *in separate files from the manuscript files labeled as supplementary material* in suitable electronic formats. Instructions for the preparation of supplementary material can be found on the supplementary material webpage.

All material to be considered as supplementary material must be submitted at the same time as the main manuscript for peer review. It cannot be altered or replaced after the paper has been accepted for

publication. Please indicate clearly the material intended as supplementary material upon submission. Also ensure that the supplementary material is referred to in the main manuscript where necessary.

Appendix I. SONA Recruitment Advertisement

Study Name: Experimental study examining the relationship between sleep loss and unusual experiences in undergraduates.

Study Type: Quantitative.

RPS Credits: 12 RPS Credits.

Duration: 30mins initial meeting and 2x 24-hour periods for experimental conditions (each including 2x 30mins to complete questionnaires remotely and either normal or sleep loss condition).

Description: Once you have expressed an interest in participating then you will be invited to attend an initial meeting over MS Teams to discuss the study with the lead researcher Maddie. During this meeting you will also be asked to complete screening questionnaires relating to sleep and mood to confirm that you are eligible to take part, which should take altogether around 30 minutes. Following this if you would like to participate in the study then you will be asked to provide consent to do so by signing an electronic form.

You and Maddie will then agree upon a date (between Monday-Thursday) that suits you to complete the first night of sleep. You will be randomly allocated either a night of standard sleep (a minimum of 7 hours of sleep) or sleep loss (around 4 hours of sleep). If you are allocated the sleep loss condition, you will be asked to stay up later and wake up at the same time as usual. The time that you will need to stay up until will be discussed with Maddie at the initial meeting, and tips for staying up late safely will be provided electronically. You will be asked to complete a set of questionnaires in the evening before sleep, and then asked to stay awake the following day and complete the same set of questionnaires the following evening between 6-12pm remotely using Qualtrics software, completing these questionnaires should take around 30 minutes. You will also be asked to upload a screenshot of your recorded sleep length for this night from your phone or smartwatch onto Qualtrics, as well as your completed sleep diary (including hours slept, nicotine and caffeine use).

On the same day of the following week (e.g. if the first night of sleep was a Monday, then the next night of sleep would also be a Monday) you will complete your second night of sleep (either the standard sleep or sleep loss night, whichever you have not already completed). You will then be asked to complete evening questionnaires before the sleep condition, stay awake during the day and complete the same set of questionnaires, and upload your sleep length screenshot and diary that evening in the same way as before.

After completing both nights of sleep, you will receive a debrief sheet that includes information about the study and resources relating to sleep and mental health. Once the sleep length screenshots, sleep diaries and completed questionnaire responses have been received via Qualtrics then SONA credits for your participation will be released, and you will be invited to enter a prize draw to win 1 of 5 £20 Amazon vouchers (optional).

Eligibility Requirements: Participants must have a method of objectively recording the length of time slept via a smartwatch, an app on a smartphone, etc. to participate in this study. You will also need a laptop or computer to complete the required tasks the morning after the sleep condition is completed. You must be between the ages of 18-65. You must also have 'good quality sleep' which will be assessed at the initial meeting. You must not have a history of psychiatric or current mood disorder. You must not have a medical condition that may be affected by a lack of sleep, such as epilepsy, or be taking regular medication that impacts your sleep or mood. You must also not be travelling across time zones 2 weeks prior to taking part and not be required to drive, operate heavy machinery or do shift work following the sleep loss condition. These requirements will be screened for and checked during the initial meeting.

If you do not meet these inclusion and exclusion criteria and so are not eligible to participate, but are interested in the study and would like to receive updates about its progress, then please email the primary researcher at Madeleine.Johnson@uea.ac.uk to express your interest in this.

Website: [n/a]

Researchers: Maddie Johnson (Trainee Clinical Psychologist), Dr Sarah Reeve (Research Supervisor and Lecturer in Clinical Psychology) and Dr Jo Bower (Research Supervisor and Lecturer in Psychology).

Appendix J. The University of East Anglia's Faculty of Medicine and Health Sciences Research Ethics Subcommittee Approval

University of East Anglia
Norwich Research Park
Norwich. NR4 7TJ

Email: ethicsmonitor@uea.ac.uk
Web: www.uea.ac.uk

Study title: Experimental study examining the relationship between sleep loss and unusual experiences in undergraduates.

Application ID: ETH2324-0223

Dear Madeleine,

Your application was considered on 10th January 2024 by the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee).

The decision is: **approved**.

You are therefore able to start your project subject to any other necessary approvals being given.

If your study involves NHS staff and facilities, you will require Health Research Authority (HRA) governance approval before you can start this project (even though you did not require NHS-REC ethics approval). Please consult the HRA webpage about the application required, which is submitted through the IRAS system.

This approval will expire on **1st March 2025**.

Please note that your project is granted ethics approval only for the length of time identified above. Any extension to a project must obtain ethics approval by the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee) before continuing.

It is a requirement of this ethics approval that you should report any adverse events which occur during your project to the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee) as soon as possible. An adverse event is one which was not anticipated in the research design, and which could potentially cause risk or harm to the participants or the researcher, or which reveals potential risks in the treatment under evaluation. For research involving animals, it may be the unintended death of an animal after trapping or carrying out a procedure.

Any amendments to your submitted project in terms of design, sample, data collection, focus etc. should be notified to the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee) in advance to ensure ethical compliance. If the amendments are substantial a new application may be required.

Approval by the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee) should not be taken as evidence that your study is compliant with the UK General Data Protection Regulation (UK GDPR) and the Data Protection Act 2018. If you need guidance on how to make your study UK GDPR compliant, please contact the UEA Data Protection Officer (dataprotection@uea.ac.uk).

Please can you send your report once your project is completed to the FMH S-REC (fmh.ethics@uea.ac.uk).

I would like to wish you every success with your project.

On behalf of the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee)

Yours sincerely,

Dr Paul Linsley

Appendix K. Participant Information Sheet

Experimental study examining the relationship between sleep loss and unusual experiences in undergraduates.

Version 1.0, December 2023

Researchers: Madeleine Johnson (Trainee Clinical Psychologist, Primary Researcher), Dr Sarah Reeve (Primary Research Supervisor) and Dr Jo Bower (Secondary Research Supervisor).

We would like to invite you to take part in our experimental study investigating the relationship between sleep deprivation and unusual experiences. Participation in this study is voluntary so to help you to reach an informed decision we will explain more about the purpose of the study and what it involves. Thank you for taking the time to read this information and consider taking part.

Please read the following information carefully while considering whether you would like to take part in our study. If you have any questions before taking part, then please feel free to get in contact with a member of the research team using the details below.

What is the purpose of this research?

We are interested in whether reducing sleep leads to an increase in unusual experiences when compared to a night of usual sleep in a non-clinical population. There is an abundance of research that has shown associations between individuals who struggle with disrupted sleep and also have unusual experiences, however there is a lack of experimental evidence demonstrating that sleep loss is a direct cause of unusual experiences, or many efforts to explain the mechanism of this effect.

This study will allow us to better understand why some individuals are more likely to have unusual experiences and the process by which this may occur. With this knowledge we may reveal new early signs shown by individuals that unusual experiences are developing and identify specific targets for helping to manage these experiences and reduce distress for these people.

This research is being carried out as part of a Doctorate thesis in Clinical Psychology at the University of East Anglia (UEA).

Who is being invited to take part?

We are interested in recruiting individuals who:

- Are 18-65 years old.
- Have good quality sleep.
- Do not have a history of psychiatric disorder or current mood disorder.
- Do not have a medical condition that may be affected by a lack of sleep, such as epilepsy, or are taking regular medication that impacts their sleep or mood.
- Have access to a smartphone or Fitbit while taking part.
- Will not be travelling across time zones 2 weeks prior to taking part.
- Are not required to do shift work or operate heavy machinery while taking part.

However, these criteria will be assessed in an initial meeting prior to participation in the study.

What would taking part involve?

Once agreeing to take part, we will be in contact to arrange an initial meeting with the primary researcher so that we can complete some questionnaires, assess your eligibility for the study and provide an opportunity for you to ask further questions.

Within a month of the initial meeting (the specific dates will be decided to fit with what is convenient for your schedule), you will be asked to take part in both experimental conditions, the order of which will be randomised. The first experimental condition would require you to sleep as you normally would, while the second would require you to restrict your sleep to a total of 4 hours over the night. For both conditions a set of questionnaires that takes around 30 minutes to complete will be carried out remotely the evening before sleep and the evening afterwards. This means that most of the study will be completed remotely or at home, and we encourage participants to continue with their daily activities as usual to minimise disruption.

We also ask participants to record their sleep over the night for each condition using a diary and electronic device so that we can confirm that the 2 nights of sleep are appropriately different in length. Once both conditions are completed along with the questionnaires, and the sleep diaries and sleep length screenshots have been uploaded to Qualtrics, then participants will be debriefed about the study. They will then be compensated with 12 SONA participation credits for their involvement and included in a prize draw to win one of five £20 Amazon vouchers if they would like to be as an additional thank you for your time.

Do I have to take part?

No, your participation is entirely voluntary. If after reading this information sheet you express an interest in taking part, then you will be asked to provide consent at the end of the initial meeting.

Can I stop taking part if I change my mind?

Yes, if for any reason you would like to stop participation in the study then you can withdraw at any time. In this case you would not be required to provide a reason as to why, and there would be no consequences for this decision. If by that point you had already completed measures, then your data would not be used and be destroyed.

What are the possible benefits of taking part?

The direct benefits to you as an individual for taking part would include the acquisition of SONA credits and optional entry into the prize draw for vouchers, as well as signposting to services and resources that may help to improve your sleep at the end of the study. We also hope that your participation would help us to increase our understanding of the relationship between sleep loss and unusual experiences. This would provide a worthy contribution to academic literature and may guide clinical services and improve care.

What are the possible disadvantages and risks of taking part?

Although we have made adjustments to improve the comfort of participants where possible, we are aware that reduced sleep can cause disruption and impairs functioning (including cognitive and motor performance). 'Sleep Restriction Tips' will be provided to support with practicing sleep restriction safely, and we remind individuals that they can withdraw from the study at any time. We recommend that if any adverse events occur following sleep restriction, then the participant should withdraw from the study immediately, seek restorative sleep and contact the primary researcher to let them know when possible. The debrief from the study includes signposting to services that offer information and support for improving sleep, as well as general mental health, and we encourage individuals to seek support if any distress continues after restorative sleep.

Another disadvantage of participation is the time taken to complete measures throughout the study. We have selected shorter versions of measures where possible to reduce this burden, and have adjusted the amount of participation credits earned to try to appropriately compensate for this time, however we are mindful that the time taken to participate still differs from other available studies.

What will happen to the information I provide?

All study data will be stored securely and will only be accessible by the research team during the course of the study. This study requires the collection of some personal data such as phone numbers to send text reminders on the day of experimental condition and questionnaires completion, and email addresses if the individual opts-in to participate in a prize draw for vouchers or to receive study updates. This information will not be used for any other purpose except those mentioned, will be stored separately from the main study data so that your responses are not associated with identifiable information, and will be permanently deleted once the study concludes in March 2025.

Following the conclusion of the study, anonymous data will be archived in the UEA psychology department repository and may be used for future research and publication purposes. However, by this stage all data will be completely anonymous and impossible to link back to individual participants, ensuring confidentiality.

What will happen to the results?

The data collected over the course of the study will be analysed and the findings written up and submitted as part of a Doctoral thesis in Clinical Psychology at UEA. These findings may then be submitted for publication in a scientific journal or presented at research conferences or used in

future research, however by this point the data will be combined and presented in a way that no individual data or details could possibly be identified.

Who is organising, funding and reviewing this study?

This study is organised and funded by the Doctoral Programme in Clinical Psychology at the UEA. It has been reviewed and approved by the UEA Faculty of Medicine and Health Sciences Research Ethics Committee [ETH2324-0223].

Can I get in touch?

If you have any questions, concerns, or would like to know more about the study then please feel free to contact the team using the details below:

Madeleine Johnson (Trainee Clinical Psychologist, Primary Researcher)

Doctoral Programme in Clinical Psychology, Department of Clinical Psychology, Norwich Medical School, University of East Anglia, Norwich, NR4 7TJ. Email: Madeleine.Johnson@uea.ac.uk

Dr Sarah Reeve (Research Supervisor and Lecturer in Clinical Psychology)

Doctoral Programme in Clinical Psychology, Department of Clinical Psychology, Norwich Medical School, University of East Anglia, Norwich, NR4 7TJ. Email: Sarah.Reeve@uea.ac.uk

Or if you would like to contact a member of course staff independent to the study:

Professor Sian Coker (Deputy Programme Director for UEA Clinical Psychology Doctorate programme) Doctoral Programme in Clinical Psychology, Department of Clinical Psychology, Norwich Medical School, University of East Anglia, Norwich, NR4 7TJ. Email: S.Coker@uea.ac.uk

Thank you for your time and consideration.

Appendix L. Consent Form

University of East Anglia

Title of Project: Experimental study examining the relationship between sleep loss and unusual experiences in undergraduates.

Name of Researchers: Madeleine Johnson (Trainee Clinical Psychologist), Dr Sarah Reeve (Lecturer in Clinical Psychology) and Dr Jo Bower (Lecturer in Psychology)

Please respond Yes or No below

1. I confirm that I have read the Participant Information sheet dated December 2023 (version 1.0) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without consequence. ☐

3. I have a smartphone/watch I can use to record my sleep length and I am willing to upload a screenshot to Qualtrics of both sleep nights for the study. ☐

4. I agree to provide my number so that I can be texted as a reminder to complete the morning measures. I understand that my number will not be used for any other purpose, and will be deleted once I finish the study. ☐

5. I am aware that this study is not recommended for people who have physical or mental health conditions which may be exacerbated by sleep loss. ☐

6. If I have 4 hours of sleep, I agree to follow the 'Sleep Restriction Tips' provided so that I am aware of how to practice sleep restriction safely. ☐

7. If I have 4 hours of sleep, I agree not to participate in any risky or dangerous activities until I have had recovery sleep (e.g. driving, operating heavy machinery). ☐

8. I understand what will happen to the information that I provide and who will be able to access this information. ☐

9. I agree that the anonymised data collected for this study can be used for the purposes of future research studies conducted within UEA. ☐

10. I consent to participate in this study. ☐

Appendix M. Debrief Sheet

Experimental study examining the relationship between sleep loss and unusual experiences in undergraduates.

Researchers: Madeleine Johnson (Trainee Clinical Psychologist, Primary Researcher), Dr Sarah Reeve (Primary Research Supervisor) and Dr Jo Bower (Secondary Research Supervisor).

Thank you for giving your time and energy to support this research, it is greatly appreciated.

This is a study investigating the mechanism by which sleep loss influences unusual experiences (such as hearing things that are not there or feeling unreal). We hope that your participation will help us to increase our understanding of the relationship between sleep loss and unusual experiences, this would inform a worthy contribution to the academic literature and may guide clinical services and improve care. You completed the measures included in this study for research and not diagnostic purposes, but if you are concerned about your responses then please look at the resources below or contact your GP or local mental health service.

We encourage you to prioritise using the following nights to practice restorative sleep, perhaps by allocating time today for a nap or going to sleep earlier this evening, using the resources below for advice on how to improve your sleep if needed. If you have experienced any distress from taking part in this study, then we would encourage you to reach out to a trusted friend or family member for support. If distress experienced is significant or continues after restorative nights of sleep, then we would advise you to contact your GP for advice and guidance.

We would like to remind you that all data collected for the purposes of this study will be stored securely with access granted to only the researchers listed above. If you have opted-in to enter the prize draw for vouchers, or to receive study updates, then your email address will be stored securely until the end of the study. Prize winners will then be notified, and final study updates sent when the study concludes in March 2025, at which point all personal data will be permanently deleted.

Again, we **thoroughly** appreciate your time and effort given to this study.

If you would like to be entered into the prize draw to potentially win one of five £20 vouchers, or would like to receive study updates, then please email the primary researcher to let them know at Madeleine.Johnson@uea.ac.uk.

This debrief sheet is downloadable through Qualtrics if you would like to store a copy to refer to at a later time.

The Sleep Charity
https://thesleepcharity.org.uk/ - for information, support and courses on sleep including tips and techniques to help you to improve your sleep. Information is also available on sleep deprivation, sleep disorders and signs for when to seek additional support.
The National Sleep Helpline: **03303 530 541** – speak to trained sleep advisors confidentially (open Mon/Tues/Thurs 7-9pm, Mon/Wed 9-11am)

Sleep Foundation, Best Sleep Apps
https://www.sleepfoundation.org/best-sleep-apps - recommended apps for improving sleep, who they are recommended for, and information related to subscriptions.

MIND – Mental Health Charity
https://www.mind.org.uk/ - for information, support and resources relating to mental health.
https://www.mind.org.uk/information-support/types-of-mental-health-problems/sleep-problems/about-sleep-and-mental-health/ - for information about the relationship between sleep and mental health and how they influence each other.
Infoline: **0300 123 3393** – for information and signposting (open 9am-6pm Mon-Fri)

UEA Student Services, Wellbeing
https://www.uea.ac.uk/uea-life/student-support/wellbeing - advice, guidance, and signposting for students.

The Wellbeing Service – Norfolk and Waveney Mental Health Service
https://www.wellbeingnands.co.uk/norfolk/ - local mental health support including workshops, self-help, peer support and talking therapies available online or through self-referral.

Should you have additional questions or concerns, or would like to discuss the study further, then please contact a member of the research team below:

Madeleine Johnson (Trainee Clinical Psychologist, Primary Researcher)

Doctoral Programme in Clinical Psychology, Department of Clinical Psychology, Norwich Medical School, University of East Anglia, Norwich, NR4 7TJ. Email: Madeleine.Johnson@uea.ac.uk

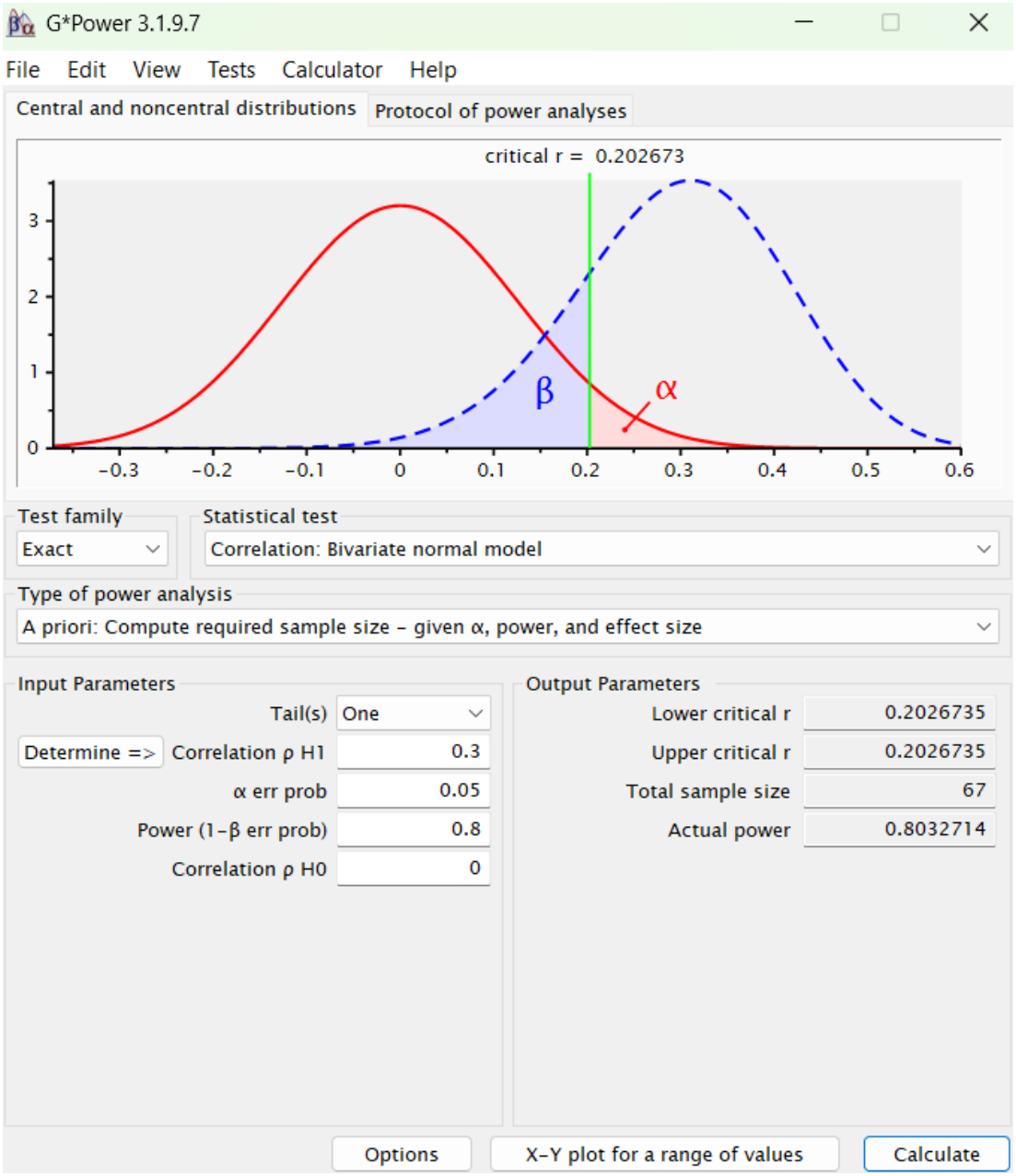
Dr Sarah Reeve (Research Supervisor and Lecturer in Clinical Psychology)

Doctoral Programme in Clinical Psychology, Department of Clinical Psychology, Norwich Medical School, University of East Anglia, Norwich, NR4 7TJ. Email: Sarah.Reeve@uea.ac.uk

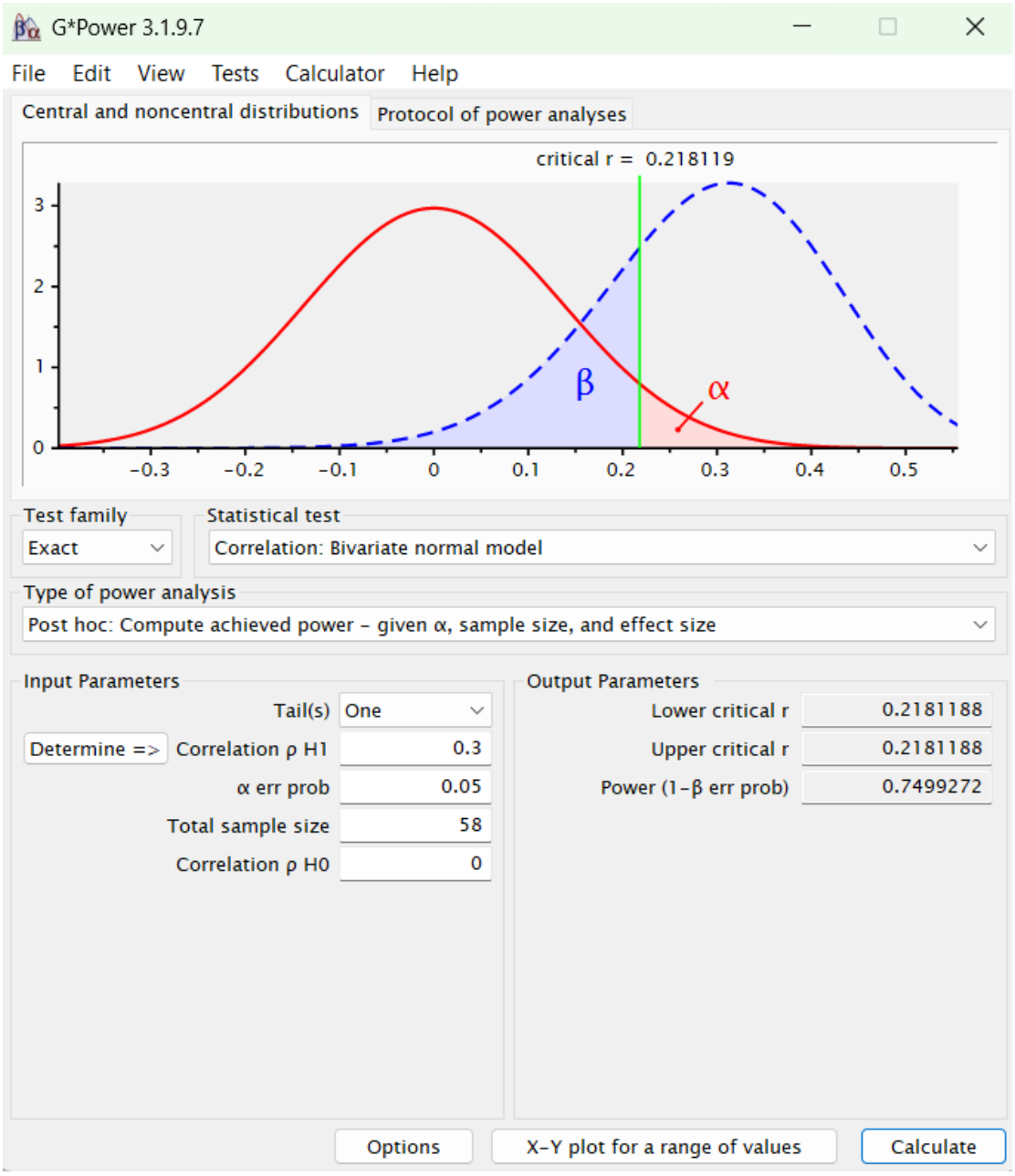
Or if you would like to contact a member of course staff independent to the study:

Professor Sian Coker (Deputy Programme Director for UEA Clinical Psychology Doctorate programme) Doctoral Programme in Clinical Psychology, Department of Clinical Psychology, Norwich Medical School, University of East Anglia, Norwich, NR4 7TJ. Email: S.Coker@uea.ac.uk

Appendix N. G*Power Screenshot for Priori Sample Size Calculation



Appendix O. G*Power Screenshot for Post Hoc Power Analysis Calculation



Appendix P. G*Power Screenshot for Post Hoc Repeated Measures Sensitivity Calculation