Exploring the Relationship Between Sleepiness, Psychosis and Mood:

A Quantitative Approach

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Thesis Portfolio Abstract

This thesis aimed to explore the relationship between hypersomnia, psychiatric symptoms, and wellbeing in the context of mental health disorders (depression and psychosis). A systematic review was conducted to synthesise existing research into hypersomnia in depression. This review examined 33 studies, all of them using quantitative design. The review confirmed that there are strong correlations between hypersomnia and depression, suggesting that hypersomnia has a negative impact on mood and increases the risk of future mood deterioration. The findings emphasised the need for more refined diagnostic criteria for hypersomnia, the use of objective measures for its assessment, and the integration of qualitative and mixed-methods research approaches to advance understanding in this area, in particular around the mechanisms underlying these associations.

An empirical study was then conducted in patients with psychosis to understand rates of hypersomnia in this group and differences between the patients with and without hypersomnia with respect to sleep, antipsychotic medication, clinical symptoms, and quality of life. Fifty-seven participants completed an online survey reporting on their sleep, clinical symptoms, and quality of life. The study found significant differences in gender and age, with hypersomnia patients exhibiting a higher likelihood of being younger and male, but no significant differences regarding diagnosis, medication type, or antipsychotic medication dose, clinical symptoms, quality of life, and physical activity between the study groups. One reason for this lack of significance may be the limited differences observed between groups, as even individuals who did not meet the criteria for hypersomnia reported extended sleep durations. Moreover, the study's insufficient power to detect anything other than large effects may have further restricted the capacity to identify more subtle but clinically relevant group differences. The study highlighted the need for longitudinal research and a focus on the direction of effect among

hypersomnia, clinical symptoms, and quality of life with the view of designing and offering tailored interventions targeting excessive sleepiness in people with psychosis.

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Finally, I dedicate this thesis to everyone who has struggled with psychosis. I am hoping this research will contribute to making a difference in their lives.

Chapter One

Introduction Chapter

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Introduction

Sleep disturbances are becoming acknowledged as transdiagnostic features that affect the trajectory, prognosis, and quality of life in patients with mental illness (Harvey et al., 2011; Wulff et al., 2010). While insomnia has garnered considerable research focus due to its strong associations with symptom intensification, relapse probability, and diminished treatment effectiveness (Baglioni et al., 2011; Freeman et al., 2020), hypersomnia is relatively under-researched—despite increasing evidence of its prevalence and substantial effects on worsening mood, increased risk of depression relapse and functional impairment (Kaplan & Harvey, 2009; Ohayon & Roth, 2003; Reeve et al., 2019).

Hypersomnia as a Sleep Symptom and Condition

The word "hypersomnia" can variously refer to subjective or objectively verified extended sleep duration (i.e., >9 hours at night, or 11 hours in a 24-hour period; Berkovski et al., 2016, Lammers et al., 2020) and/or excessive daytime sleepiness (EDS). However, assessing the prevalence of hypersomnia in various groups is challenging owing to the inherent ambiguity in its diagnosis and the symptom overlap with other sleep or medical conditions (Barateau et al., 2017). The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; American Psychiatric Association, 2013) includes a condition called "hypersomnolence disorder," which is identified as EDS—a strong desire to sleep, long sleep periods, or feeling very groggy after waking up—that cannot be fully explained by any medical or mental health issue.

The third edition of the International Classification of Sleep Disorders (ICSD-3), published by the American Academy of Sleep Medicine in 2014, uses the terms "hypersomnia" or "central disorders of hypersomnolence" (CDH) to specify rare (<0.05% prevalence) neurological sleep disorders such as narcolepsy, idiopathic hypersomnia (IH), and Kleine–

Levin syndrome (KLS). However, it also includes the far more common condition of hypersomnia linked to environmental factors (like insufficient sleep or substance use), medical conditions, or psychiatric disorders.

Hypersomnia in Mood Disorders

Hypersomnia is considered a secondary diagnostic criterion for mood disorders, specifically in conditions such as major depressive disorder (MDD), MDD with atypical characteristics, dysthymic disorder (DD), bipolar-related diagnoses (BD), and seasonal affective disorder (SAD), as noted by Kaplan and Harvey in 2009. For instance, the DSM-5 criteria require the presence of a sad mood and/or anhedonia, along with four out of nine other symptoms, including hypersomnia.

Depression and Hypersomnia

Depression is a complex mental condition with an estimated lifetime incidence of 10% in the general population (Kessing, 2007; Kessler et al., 2013) that exhibits a diverse range of symptoms and has a persistent nature.

Researchers identify both hypersomnia and insomnia as secondary symptoms of depression, along with changes in appetite or weight and in psychomotor activity. Traditionally, insomnia has been a greater focus of research and clinical attention, given the robust associations between it and low mood, risk, and poorer clinical outcomes. However, this has led to relative neglect of the role of hypersomnia.

Around 30% of individuals diagnosed with MDD exhibit symptoms of hypersomnia (Kaplan & Harvey, 2009). Additionally, a worsened prognosis for depression appears to be associated with hypersomnia. Prospective epidemiological studies have indicated that there is a 2.4 - 2.9-fold increased likelihood of subsequent depressive episodes in individuals with hypersomnia compared to individuals without hypersomnia or those with normal sleep

patterns. (Breslau et al., 1996; Ford & Cooper-Patrick, 2001). Further research suggests that hypersomnia seems to predict poor treatment outcomes in depression, often remaining as a residual symptom after therapeutic interventions, with residual symptom rates reported in as many as 40–50% of instances (Worthington et al., 1995; Iovieno et al., 2011). Moreover, hypersomnia is often considered one of the most distressing residual symptoms in individuals who do not achieve remission (Matza et al., 2003; Zimmerman et al., 2005), underscoring its considerable effect on quality of life and overall recovery.

Psychosis and Hypersomnia

Individuals diagnosed with schizophrenia and psychosis have consistently shown sleep disturbances since the first documentation of these disorders (Ferrarelli, 2020). In his case study descriptions, Kraepelin (1919) often refers to sleep problems and suggests that bed rest, supervision, sleep hygiene, and nutrition are the essential components of treatment. In addition, Bleuler (1950**)** said that these individuals had disrupted sleep due to their apprehension of potential harm while asleep. He also proposed that the traits and patterns of delusional thinking in people with schizophrenia were very similar to those observed in the sleep state. Furthermore, sleep difficulties are common in the early stages of the disorder before psychotic symptoms appear (Davies et al., 2017) and typically continue even after other symptoms have been addressed (Baandrup et al., 2013).

This patient population has reported various sleep disturbance issues, such as insomnia (Cohrs, 2008), disruption of circadian rhythm (Wulff et al., 2012), and nightmares (Sheaves et al., 2015). As with depression, there has been more of a focus on insomnia and sleep loss due to causal links with exacerbated psychotic symptoms, risk, and relapse (Reeve et al., 2018).

However, similar to depression, this group often presents with hypersomnia, a sleep presentation that has received relatively little attention. A cross-sectional study conducted by Sharma et al. (2016) found that 32% of a sample of 100 patients with schizophrenia who were taking medication had excessive sleepiness. Patients diagnosed with schizophrenia spectrum disorders (SSD) have been shown to have a higher incidence of EDS and abnormalities in their electroencephalograms (EEG), as demonstrated by Okruszek et al. in 2014. Moreover, research conducted by Reeve et al. (2019) revealed that 23% of people diagnosed with psychosis had symptoms of hypersomnia. In clinical practice, it is typical for symptoms of hypersomnia to be attributed to sedation induced by antipsychotic medication (Rehman et al., 2017; Faulkner et al., 2017). This sedation is often associated with the effects of these drugs on neurotransmitter systems, namely via the inhibition of histamine receptors (Miller, 2004).

Summary

In summary, hypersomnia is a frequently reported symptom in a range of mental disorders. Nevertheless, the assessment of its frequency is a challenge due to the inherent ambiguity and uncertainty associated with it (Barateau et al., 2017).

Both clinical and research contexts frequently observe the coexistence of hypersomnia disorders with mood symptoms, particularly depression. Nevertheless, the precise cause-and-effect link between these two situations remains uncertain. Hypersomnia symptoms are inherent to mood disorders like MDD, where they are acknowledged as a diagnostic criterion. Nonetheless, the underlying mechanisms and clinical implications of hypersomnia in mood disorders are much underexplored (Dauvilliers et al., 2013). This lack of research hinders comprehension of its function in prognosis, treatment efficacy, and overall effects on patients with MDD.

Remarkably, there is a notable absence of research elucidating the causes and sustaining factors of hypersomnia. However, research suggests that psychological factors may contribute to the onset and maintenance of hypersomnia. These psychological mechanisms include challenges with motivation and sustaining interest (Nofzinger et al., 1991; Billiard et al., 1994). Moreover, hypersomnia may also play a role in the onset and/or persistence of mood disorders, acting as a mechanism itself. Hypersomnia is often believed to be a result of accompanying mood disorders (Kaplan & Harvey, 2009). For example, depression-like symptoms may occur after long periods of sleep, such as fatigue, psychomotor slowness, and cognitive disruption. Additionally, a parallel lack of evidence is observed concerning the treatment of hypersomnia. (Kaplan & Harvey, 2009). Understanding the mental health impacts of hypersomnia, particularly in the context of depression, is crucial for comprehensively managing and treating individuals with psychosis.

Given the limited focus on hypersomnia compared to insomnia, this thesis aims to investigate the association between hypersomnia, psychiatric symptoms, and wellbeing within the framework of mental health difficulties, particularly depression and psychosis.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Publishing.

American Academy of Sleep Medicine. (2014). *International Classification of Sleep Disorders (*3rd ed.). American Academy of Sleep Medicine.

Baandrup, L., Jennum, P., Lublin, H., & Glenthoj, B. (2013). Treatment options for residual insomnia in schizophrenia. *Acta psychiatrica Scandinavica, 127*(1), 81–82. https://doi.org/10.1111/acps.12016

Baglioni, C., Battagliese, G., Feige, B., Spiegelhalder, K., Nissen, C., Voderholzer, U., ... & Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders, 135*(1-3), 10–19. https://doi.org/10.1016/j.jad.2011.01.011

Barateau, L., Lopez, R., Franchi, J. A., & Dauvilliers, Y. (2017). Hypersomnolence, Hypersomnia, and Mood Disorders. *Current psychiatry reports, 19*(2), 13. https://doi.org/10.1007/s11920-017-0763-0

Berkowski, J. A., & Shelgikar, A. V. (2016). Disorders of Excessive Daytime Sleepiness Including Narcolepsy and Idiopathic Hypersomnia. *Sleep medicine clinics, 11*(3), 365–378. https://doi.org/10.1016/j.jsmc.2016.04.005

Billiard, M., Dolenc, L., Aldaz, C., Ondze, B., & Besset, A. (1994). Hypersomnia associated with mood disorders: a new perspective. *Journal of psychosomatic research, 38 Suppl 1*, 41–47. https://doi.org/10.1016/0022-3999(94)90134-1

Bleuler, E. (1950). *Dementia Praecox or the Group of Schizophrenias*. International Universities Press.

Breslau, N., Roth, T., Rosenthal, L., & Andreski, P. (1996). Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biological psychiatry, 39*(6), 411–418. https://doi.org/10.1016/0006-3223(95)00188-3

Cohrs, S. (2008). Sleep disturbances in patients with schizophrenia. *CNS Drugs, 22*(11):939-62

Dauvilliers, Y., Lopez, R., Ohayon, M., & Bayard, S. (2013). Hypersomnia and depressive symptoms: methodological and clinical aspects. *BMC medicine, 11*, 78. https://doi.org/10.1186/1741-7015-11-78

Davies, G., Haddock, G., Yung, A. R., Mulligan, L. D., & Kyle, S. D. (2017). A systematic review of the nature and correlates of sleep disturbance in early psychosis. *Sleep medicine reviews*, 31, 25–38. https://doi.org/10.1016/j.smrv.2016.01.001

Faulkner, S., Bee, P. (2017). Experiences, perspectives and priorities of people with schizophrenia spectrum disorders regarding sleep disturbance and its treatment: a qualitative study. *BMC psychiatry, 17*(1), 158. https://doi.org/10.1186/s12888-017-1329-8

Ferrarelli, F. (2020). Sleep disturbances in schizophrenia and psychosis. *Schizophrenia research, 221*, 1–3. https://doi.org/10.1016/j.schres.2020.05.022

Ford, D. E., & Cooper-Patrick, L. (2001). Sleep disturbances and mood disorders: an epidemiologic perspective. *Depression and anxiety, 14*(1), 3–6. https://doi.org/10.1002/da.1041

Freeman, D., Sheaves, B., Waite, F., Harvey, A. G., & Harrison, P. J. (2020). Sleep disturbance and psychiatric disorders. *The Lancet Psychiatry, 7*(7), 628–637. https://doi.org/10.1016/S2215-0366(20)30136-X

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review, 31*(2), 225–235. https://doi.org/10.1016/j.cpr.2010.04.003

Iovieno, N., van Nieuwenhuizen, A., Clain, A., Baer, L., & Nierenberg, A. A. (2011). Residual symptoms after remission of major depressive disorder with fluoxetine and risk of relapse. *Depression and anxiety, 28*(2), 137–144. https://doi.org/10.1002/da.20768

Kaplan, K. A., & Harvey, A. G. (2009). Hypersomnia across mood disorders: A review and synthesis. *Sleep Medicine Reviews, 13*(4), 275–285. https://doi.org/10.1016/j.smrv.2008.08.001

Kessing, L. V. (2007). Epidemiology of subtypes of depression. *Acta psychiatrica Scandinavica. Supplementum, (433)*, 85–89. https://doi.org/10.1111/j.1600-0447.2007.00966.x

Kessler, R. C., & Bromet, E. J. (2013). The epidemiology of depression across cultures. *Annual review of public health, 34*, 119–138. https://doi.org/10.1146/annurev-publhealth-031912-114409

Kraepelin, E. (1919). *Dementia praecox and paraphrenia* (E. S. Barclay, Trans.). Livingston. (Original work published 1913 *in Textbook of Psychiatry,* 8th ed.)

Lammers, G. J., Bassetti, C. L. A., Dolenc-Groselj, L., Jennum, P. J., Kallweit, U., Khatami, R., Lecendreux, M., Manconi, M., Mayer, G., Partinen, M., Plazzi, G., Reading, P. J., Santamaria, J., Sonka, K., & Dauvilliers, Y. (2020). Diagnosis of central disorders of hypersomnolence: A reappraisal by European experts. *Sleep medicine reviews, 52*, 101306. https://doi.org/10.1016/j.smrv.2020.101306

Matza, L. S., Revicki, D. A., Davidson, J. R., & Stewart, J. W. (2003). Depression with atypical features in the National Comorbidity Survey: classification, description, and consequences. Archives of general psychiatry, 60(8), 817–826. https://doi.org/10.1001/archpsyc.60.8.817

Miller, D. D. (2004). Atypical antipsychotics: sleep, sedation, and efficacy. *Primary care companion to the Journal of clinical psychiatry, 6*(Suppl 2), 3–7.

Nofzinger, E.A, Thase, M.E., Reynolds, 3rd C.F., Himmelhoch, J.M., Mallinger, A., Houck, P. et al. (1991). Hypersomnia in bipolar depression: a comparison with narcolepsy using the multiple sleep latency test. *The American journal of psychiatry, 148*(9), 1177–1181. https://doi.org/10.1176/ajp.148.9.1177

Ohayon, M. M., & Roth, T. (2003). Place of chronic insomnia in the course of depressive and anxiety disorders. *Journal of Psychiatric Research, 37*(1), 9–15. https://doi.org/10.1016/S0022-3956(02)00052-3

Okruszek, L., Jernajczyk, W., Wierzbicka, A., Waliniowska, E., Jakubczyk, T., Jarema, M., & Wichniak, A. (2014). Daytime sleepiness and EEG abnormalities in patients treated with second generation antipsychotic agents. *Pharmacological Reports, 66*, 1077–1082.

Reeve, S., Nickless, A., Sheaves, B., Hodsoll, J., Roberts, A., & Freeman, D. (2019). Sleep loss as a trigger of severe mental illness: A population-based case-crossover study. *Sleep, 42*(10), zsz150. https://doi.org/10.1093/sleep/zsz150

Rehman, A., Waite, F., Sheaves, B., Biello, S., Freeman, D., & Gumley, A. (2017). Clinician perceptions of sleep problems, and their treatment, in patients with non-affective psychosis. *Psychosis, 9*(2), 129–139. https://doi.org/10.1080/17522439.2016.1206955

Sharma, P., Dikshit, R., Shah, N., Karia, S., & De Sousa, A. (2016). Excessive Daytime Sleepiness in Schizophrenia: A Naturalistic Clinical Study. Journal of clinical and diagnostic research: JCDR, 10(10), VC06–VC08. https://doi.org/10.7860/JCDR/2016/21272.8627

Wulff, K., Gatti, S., Wettstein, J. G., & Foster, R. G. (2010). Sleep and circadian rhythm *disruption in psychiatric and neurodegenerative disease. Nature Reviews Neuroscience, 11*(8), 589–599. https://doi.org/10.1038/nrn2868

Wulff, K., Dijk, D. J, Middleton, B., Foster, R. G., Joyce, E. M. (2012). Sleep and circadian rhythm disruption in schizophrenia. *Br J Psychiatry,200*(4):308-16. https://doi.org/10.1192/bjp.bp.111.096321

Zimmerman, M., McGlinchey, J. B., Posternak, M. A., Friedman, M., Boerescu, D., & Attiullah, N. (2005). Differences between minimally depressed patients who do and do not consider themselves to be in remission. *The Journal of clinical psychiatry, 66*(9), 1134–1138. https://doi.org/10.4088/jcp.v66n0908

Chapter Two

Systematic Review

Hypersomnia and Depression: A Narrative Synthesis

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Hypersomnia and Depression: A Narrative Synthesis

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Abstract

Background. Hypersomnia, while less extensively researched than insomnia, is a prevalent presentation in depression and is associated with more severe symptoms and poorer clinical outcomes. The causal link between hypersomnia and depression remains ambiguous and has been relatively underexplored in comparison to insomnia.

Methods. A systematic review of 33 papers was conducted by querying three major databases to evaluate current evidence on the associations, potential causal relationships, and proposed mechanisms connecting hypersomnia and depression in clinical patient groups. Studies were included according to established criteria, and results were synthesised narratively.

Results. The results suggest that hypersomnia in depression is associated with exacerbated depressive symptoms, elevated suicidality, and poor treatment outcomes, especially in cases of atypical and recurrent depression. Proposed mechanisms included modified neural connectivity, circadian disruption, and maladaptive sleep beliefs. Interventional studies indicate that treating hypersomnia may enhance depressive symptoms, suggesting that it may contribute to depression rather than be a simple consequence of it.

Conclusions. The review supports that hypersomnia is clinically relevant in depression, both as a marker of increased severity and potentially as a treatment target. The mechanisms between hypersomnia and depression remain under-researched but are worthy of further attention. Standardised terminology should be prioritised in future research, while qualitative and mixed-methods approaches may provide more profound insights and facilitate the development of targeted interventions, such as psychological approaches.

Introduction

Hypersomnia, a term composed of the Greek "hyper" ("excessive") and the Latin "somnus" ("sleep"), is recognised as a secondary diagnostic criterion for mood disorders. For instance, the diagnostic criteria for Major Depressive Disorder (MDD) rely on the presence of either a sad mood and/or anhedonia, along with at least four out of nine other symptoms, including hypersomnia or insomnia – as outlined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; American Psychiatric Association, 2013). Studies indicate that approximately 30% of individuals with depression experience hypersomnia, yet this condition often goes unnoticed (Kaplan & Harvey, 2009; Leibenluft et al., 1995).

Hypersomnia has been linked to poor clinical outcomes in depression, such as increased risk of treatment resistance, heightened suicide risk, and symptomatic recurrence (Plante et al., 2015; Zimmerman et al., 2005; Fitzgerald et al., 2011; Goldstein et al., 2008). Moreover, individuals with hypersomnia, according to a study by Ohayon et al. (2013), were 2-4 times more likely to report low quality of life compared to those without it. They also reported interference with socio-occupational activities and relationships. In bipolar disorder, hypersomnia has been identified as a potential predictor for a return to mania or hypomania (Kaplan et al., 2015), challenging the expectation that excessive sleepiness is strictly depressive in nature. Furthermore, hypersomnia may precede mood episodes, suggesting it could be both a symptom and a driver of mood disturbances (Gold & Sylvia, 2016; Grigolon et al., 2019). Further studies also highlighted that hypersomnia could cause cardiovascular disease (Lopes et al., 2013; Miller, 2011), metabolic syndrome (Andaku et al., 2015; Huang et al., 2016; Wasilewska et al., 2007), and automobile accidents (de Pinho et al., 2006; Risco et al., 2013; Tsai, 2010).

As indicated above, there is increasing interest in a potential causal role for hypersomnia in maintaining or exacerbating depression. This relationship may involve various potential pathways, including neurobiological, behavioural, and psychological factors.

Cholinergic systems influence rapid eye movement (REM) sleep, causing numerous anomalies in sleep architecture in MDD (Armitage, 2007). These systems are involved in controlling REM sleep, and people with MDD often have accelerated transitions into REM sleep and increased REM activity. These alterations, along with diminished deep slow-wave sleep (SWS) and impaired storage of sleep pressure, underscore specific patterns of sleep disturbance in depression. Although these alterations may explain the occurrence of hypersomnia in depression—as a compensatory response to inadequate restorative sleep—they do not explicitly clarify how hypersomnia may sustain or intensify depressive symptoms. Examining the potential role of hypersomnia in exacerbating depression, particularly via the facilitation of inactivity or the avoidance of emotionally demanding situations, is a significant and relatively understudied area.

A greater volume of research has investigated the mechanisms of hypersomnia in atypical depression. Gold and Chrousos (2002) suggested that some symptoms of atypical depression, including hypersomnia and heightened hunger, may be connected with diminished activity in the hypothalamic-pituitary-adrenal (HPA) axis. They propose that hypoarousal of the stress-regulating system and a deficit in corticotropin-releasing hormone (CRH) may contribute to these symptoms. Their work suggests a possible explanation for the development of hypersomnia; nevertheless, it is uncertain whether hypersomnia sustains low CRH activity and hence contributes to the persistence of depression. Additional research is required to investigate if hypersomnia directly affects the neurological pathways that underlie depression.

Psychological factors may also serve as plausible mechanisms that contribute to the relationship between hypersomnia and depression. These psychological processes include challenges pertaining to motivation and sustaining interest. (Nofzinger et al., 1991; Billiard et al., 1994). There is also a suggestion that hypersomnia might function as an avoidance for dealing with certain situations (Jacobson et al., 2001; Kaplan & Harvey, 2009). Hypersomnia is also associated with decreased socio-economic functioning among individuals with schizophrenia spectrum and bipolar disorders (Laskemoen et al., 2019). Gaining a better understanding of the relationship and mechanisms between hypersomnia and depression would enhance the ability to implement relevant interventions for this under-recognised and under-treated aspect of the clinical presentation.

Despite these findings, research on hypersomnia's role in depression has largely focused on its prevalence and association with broader outcomes, rather than its causal interaction with other low moods themselves or the underlying mechanisms for this. To address this gap, the current systematic review will explore how hypersomnia affects depressive symptoms and the potential mechanisms through which it exerts its influence.

Aims of the Review

Our aims for the systematic review were as follows:

To summarise existing research on the relationship between hypersomnia and low mood in depression, with a particular focus on potential causal links.

To identify relevant mechanisms that may elucidate the relationship between hypersomnia and low mood within the framework of depression

The inclusion criteria and scope of the review were deliberately broad, considering the ongoing debate about whether hypersomnia is a secondary symptom or an independent contributing factor.

Methods

This systematic review adhered to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses; Moher et al., 2009) guidelines for conducting systematic reviews. The review protocol was registered on PROSPERO (CRD42023416435).

Inclusion Criteria

In alignment with the aims of the review, no limitations were imposed on the study design to provide a thorough understanding of the associations and potential mechanisms between hypersomnia and depression. To qualify for inclusion in the current review, studies had to provide data on individuals (mean age: 18–65) diagnosed with depression. Depression had to be diagnosed using clinician-administered interviews, validated diagnostic instruments (e.g., DSM or ICD-10 criteria), or self-report measures, as indicated in the study. Additionally, the studies needed to incorporate either objective or subjective measures of hypersomnia and report on the relationship between depression and hypersomnia. To increase the scope of data collected, we used an inclusive methodology, integrating studies that recognised both depression and hypersomnia by diagnostic criteria or self-report instruments. This enabled us to synthesise a varied corpus of research pertinent to our aims. The review examined topics pertaining to causation without restricting inclusion based on research design or the capacity to infer causality. We included studies using various methodological designs (e.g., cross-sectional, longitudinal, and interventional) and assessed causal interpretations during synthesis.

Studies were excluded if they were non-peer-reviewed or non-primary research (e.g., case studies, systematic or narrative reviews, book chapters, conference papers, etc.), not available in English, or published prior to 1980. Studies involving Kleine-Levin syndrome, narcolepsy, obstructive sleep apnoea, or other physical health difficulties were excluded, as these organic

or neurological factors could confound the association between mood and hypersomnia or imply that mood changes and hypersomnia arise directly from these conditions.

Literature on idiopathic hypersomnia was also excluded from the review but was considered for relevant parallels in the discussion (e.g., whether treatments for idiopathic hypersomnia may be relevant to consider for hypersomnia alongside depression).

Search Strategy

We conducted a systematic search on three databases: PubMed (08/08/2024), PsycINFO (28/08/2024), and Web of Science (28/08/2024). The search terms were the phrases ("sleepiness" OR "hypersomnia" OR "somnolence" OR "oversleeping") AND ("depress*" OR "mood" OR "affective").

The search yielded a total of 19,358 academic publications. After removing duplicates, the titles and abstracts of 6238 papers were examined. Researcher IM examined all titles and abstracts. IM performed full-text screenings based on the inclusion and exclusion criteria, with 20% of the full-text screenings assessed by researcher SR. The study team discussed any disputes on inclusion and reached a consensus. Following the selection process, the review included 33 peer-reviewed papers (refer to Figure 1 for the PRISMA flowchart).

Quality and Bias

The Mixed Methods Appraisal Tool (MMAT; Hong et al., 2018) was used to undertake quality assessments of empirical papers. This tool was chosen for its capability to evaluate various study designs, including qualitative, quantitative, and mixed methods research. This review's extensive methodological inclusion criteria aimed to depict a comprehensive picture of the relationships and potential mechanisms connecting hypersomnia and depression, with the MMAT offering a reliable and adaptable framework for assessing study quality.

The initial evaluation of all studies relies on the following two screening questions:

1. Are there clear research questions?

2. Do the collected data allow addressing the research question?

The studies were evaluated using a quality assessment tool based on five criteria relevant to the study type (qualitative, randomised controlled trial, nonrandomised, quantitative descriptive, and mixed methods studies), yielding a score out of 5. These scores were used to provide a quality rating for each study, which informed the interpretation of the findings but were not applied as exclusion criteria.

After the first quality rating of all the studies, a second coder, KR, scored 10% of them. Any discrepancies were deliberated upon and settled through consensus (Table 1).

Data Extraction and Synthesis

The process of data extraction involved using an Excel document that contained key study characteristics, including the author, year of publication, design, sample size, sample characteristics, sleep/sleepiness measures, depression measures, and conclusions. We then used narrative synthesis to analyse the quantitative data obtained from the search. We used the narrative synthesis approach outlined in 'Guidance on the conduct of narrative synthesis in systematic reviews' (Popay et al., 2006).

The Popay et al. (2006) guidance on conducting narrative synthesis provides a systematic framework for synthesising data from many studies, making it especially beneficial for analysing intricate links, such as those between hypersomnia and depression. This methodology consists of three essential phases, each designed to ensure a clear and thorough synthesis of the evidence.

The first step, developing a preliminary synthesis, entails organising and summarising data to establish a fundamental comprehension of the research context. Research on hypersomnia and depression classifies studies based on the methodologies utilised to assess hypersomnia, such as self-reports or clinical diagnoses, and the tools used for evaluating depression, including the Beck Depression Inventory and the Patient Health Questionnaire-9, among others. Summarising these findings helped identify similarities, such as the correlation between hypersomnia and specific depressive symptoms, including low energy and feelings of worthlessness. Studies were categorised based on population variables, such as inpatient or community samples, to elucidate patterns pertinent to understanding the association between hypersomnia and depression.

The second step, examining links within the data, emphasised the identification and interpretation of patterns and differences between studies. Some studies may indicate that hypersomnia predicts the severity of depression, while others may emphasise that it is a result of depressive episodes. Discrepancies in data may be attributed to contextual variables like demographic variances, cultural interpretations of hypersomnia, or discrepancies in diagnostic criteria. This phase may also include investigating whether hypersomnia intensifies depressive symptoms via processes such as diminished daily functioning, guilt associated with excessive sleep, or social disengagement. Relationships were elucidated by incorporating theoretical models, such as frameworks that conceptualise hypersomnia as both a symptom of depression and a possible contributing factor to its chronicity.

The third step evaluated the robustness of the synthesis by rigorously assessing the quality of the included studies and identifying any biases. Research with rigorous designs, including the use of objective sleep metrics or longitudinal approaches, was prioritised in the evaluation.

The narrative synthesis progressively improved its understanding of hypersomnia and depression by incorporating new insights that strengthen its findings. Adhering to Popay et al.'s (2006) recommendations, the synthesis elucidated the complex relationship among hypersomnia and depression, offering a thorough and dependable overview of the data by exploring the characteristics of hypersomnia as both a symptom and a possible contributor to depression.

Results

Upon eliminating duplicates, we identified 6,238 unique records from the initial 19,358 search results. Of these 33 studies meet the inclusion criteria. The total number of studies evaluated for eligibility, as well as those eliminated or included at each level are outlined in a PRISMA flowchart (Fig. 1).

Figure 1

PRISMA flowchart

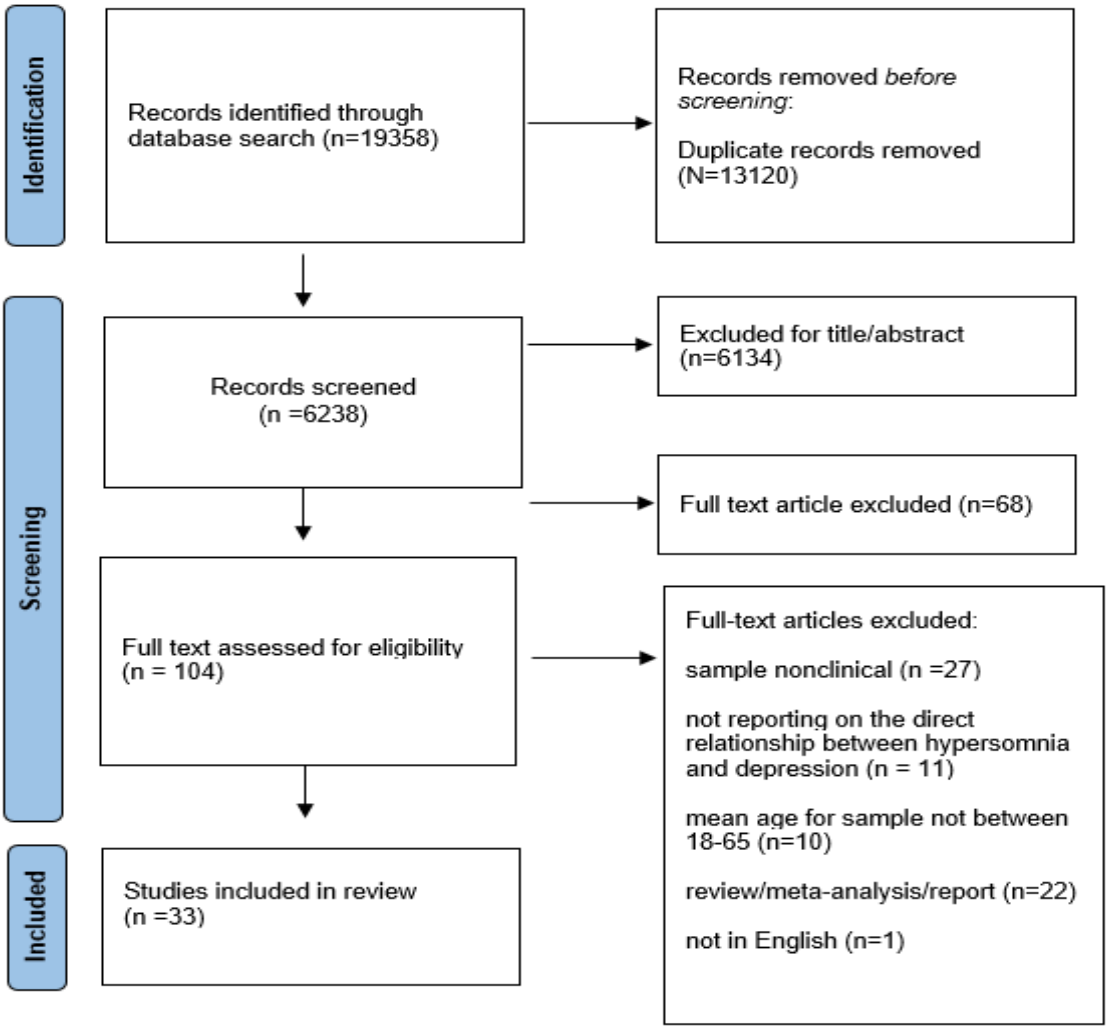

Table 1.

Summary of quality appraisals using the MMAT

Study	Quantitative criterion					Quality (%)
	6	7	8	9	10	
Bazin et al. (2024)	Y	Y	Y	CT	Y	80%
Blanchard et al. (2023)	Y	Y	Y	CT	Y	80%
Calati et al. (2010)	Y	Y	Y	CT	Y	80%
Cao et al. (2019)	Y	Y	Y	Y	Y	100%
Chellappa & Araujo (2006)	Y	Y	Y	CT	Y	80%
Chellappa & Araujo (2007)	Y	Y	Y	CT	Y	80%
Chen et al. (2021)	Y	Y	Y	CT	Y	80%
Cheung et al. (2024)	Y	Y	Y	CT	Y	80%
Claghorn et al. (1981)	Y	Y	Y	CT	Y	80%
Cook et al. (2021)	Y	Y	Y	CT	Y	80%
De Battista et al. (2003)	Y	Y	Y	Y	Y	100%
Dunlop et al. (2007)	Y	Y	Y	Y	Y	100%
Fava et al. (2007)	Y	Y	CT	Y	Y	80%
Garvey et al. (1984)	Y	Y	Y	CT	Y	80%
Hawkins et al. (1985)	Y	Y	Y	CT	Y	80%
Hein et al. (2019)	Y	Y	Y	Y	Y	100%
Jha et al. (2020)	Y	Y	Y	Y	CT	80%
Konuk et al. (2006)	Y	CT	Y	CT	Y	60%
Krystal et al. (2016)	Y	Y	Y	CT	Y	80%
Maruani et al. (2023) a	Y	Y	Y	CT	Y	80%
Maruani et al. (2023) b	Y	Y	Y	CT	Y	80%
Mume (2010)	Y	Y	Y	Y	Y	100%
Murru et al. (2019)	Y	Y	CT	CT	Y	60%
Patarroyo-Rodriguez et al. (2024)	Y	Y	Y	CT	Y	80%
Plante et al. (2012)	Y	Y	Y	CT	Y	80%
Plante et al. (2018)	Y	Y	Y	CT	Y	80%

Roberts et al. (2000)	Y	Y	Y	CT	Y	80%
Shen et al. (2011)	Y	Y	Y	CT	Y	80%
Soehner et al. (2014)	Y	Y	Y	CT	Y	80%
Thase et al. (2006)	Y	Y	Y	CT	Y	80%
YoEargon et al. (1997)	Y	Y	Y	CT	Y	80%
Zhang et al. (2021)	Y	Y	Y	Y	Y	80%
Zhao et al. (2021)	Y	Y	CT	CT	Y	60%

Description of Studies

All 33 peer-reviewed articles that met the inclusion criteria employed quantitative approaches. Tables 2, 3, and 4 show three types of studies that focused on the association between hypersomnia and depression: cross-sectional studies (19), longitudinal studies (4) and intervention/experimental studies (10), respectively. This review aimed to identify plausible mechanisms and investigate causal links between hypersomnia and depression. Although longitudinal and experimental studies provide more robust evidence for causal inference, cross-sectional studies were used to facilitate a more thorough synthesis of the existing evidence base. Due to the scarcity of rigorous studies directly addressing this link, a more inclusive strategy was used to identify emergent trends, testable hypotheses, and repeatable relationships that may guide future research. The method used allowed for the mapping of convergence and divergence across various research types, thereby enhancing the overall narrative synthesis. The combined number of participants across all studies was 14,404. Sample sizes fluctuated across the studies, ranging from as low as 21 in a cross-sectional study conducted by Plante et al. (2012) to as high as 3275 in a study conducted by Zhao et al. (2021). Among The studies recruited 31 participants from community samples, with two specifically targeting inpatients. Additionally, one study included participants from

both inpatient and community settings. All studies received a minimum quality rating of 60% in the MMAT, with 30 out of 33 studies having a quality rating of 80% or higher.

Criterion 6, concerning the appropriateness of the measurements used, was the most consistently met across studies. This is likely due to the widespread use of validated instruments for measuring hypersomnia and depression. However, a notable limitation was that most studies did not meet criterion 9 (MMAT 4.4), which assesses whether the risk of nonresponse bias is low. Nonresponse bias occurs when respondents differ systematically from nonrespondents on variables of interest, potentially skewing results. Only three studies explicitly addressed this issue: one study (Hein et al., 2019) minimised missing data by including only individuals who fully completed the questionnaires, while another study (Mume, 2010) included 67 of the 78 patients (85.9%) and excluded the remaining 11 due to incomplete data. A third study (Zhang et al., 2021) set a completion rate of greater than 85% (12 days of sleep records) as one of the data inclusion criteria. Despite these efforts, many studies did not report nonresponse rates, the characteristics of nonrespondents, or statistical adjustments for nonresponse, potentially limiting the generalisability of the findings.

Hypersomnia Assessment

The assessment of hypersomnia across various studies varied, with subjective measures being the most commonly employed. The Epworth Sleepiness Scale (ESS) was the predominant tool, used in 19 studies. Other subjective measures included the Pittsburgh Sleep Quality Index (PSQI; Calati et al., 2010; Cao et al., 2019; Cook et al., 2021), the Karolinska Sleepiness Scale (KSS; Jha et al., 2020), the Stanford Sleepiness Scale (SSS; Claghorn et al., 1981), the Hypersomnia Severity Index (HSI; Cook et al., 2021), and the Sleep Habits Questionnaire (SHQ; Chellappa & Araujo, 2007).

In contrast, seven studies incorporated objective measures of sleep, such as polysomnography (PSG), sleep electroencephalogram (EEG), wrist-worn actigraphy, and the Multiple Sleep Latency Test (MSLT). These tools were used in one longitudinal study (Zhang et al., 2021), four cross-sectional studies (e.g., Plante et al., 2012; Cook et al., 2021), and two intervention studies (Shen et al., 2011; Krystal et al., 2016).

The criteria for hypersomnia also differed across studies, suggesting diversity in its conceptualisation. For instance, Garvey et al. (1984) defined hypersomnia as sleeping more than one extra hour per day during the depressive episode compared to the pre-depression period, occurring at least four nights per week. In contrast, Zhao et al. (2021) adopted a broader definition, characterising hypersomnia as excessive sleepiness marked by either prolonged sleep duration or frequent daytime naps that occurred nearly every day over the past two weeks.

Mood Assessment

The criteria used for assessing mood across studies encompassed a range of diagnostic frameworks and tools. The most common measure was the Hamilton Rating Scale for Depression (HAM-D/HRDS/HDRS/HRSD), used in 12 studies (e.g., Plante et al., 2012; Zhao et al., 2021; Maruani et al., 2023b). Other widely used tools included the Beck Depression Inventory (BDI and BDI-II), utilised in seven studies (e.g., Calati et al., 2010; Krystal et al., 2016); the Montgomery-Åsberg Depression Rating Scale (MADRS) in six studies (e.g., Fava et al., 2007; Cook et al., 2021); and the Patient Health Questionnaire (PHQ-8 and PHQ-9) in three studies (e.g., Cao et al., 2019; Bazin et al., 2024).

In terms of diagnostic frameworks, the DSM (DSM-III, DSM-IV, DSM-IV-TR, and DSM-5) was the most frequently used, often in conjunction with structured interviews like the Structured Clinical Interview for DSM (SCID; Cook et al., 2021). Other diagnostic criteria

included the Research Diagnostic Criteria (RDC; Garvey et al., 1984; Hawkins et al., 1985) and the Mini-International Neuropsychiatric Interview (M.I.N.I.; Krystal et al., 2016; Chen et al., 2021). The World Health Organisation Composite International Diagnostic Interview (WHO-CIDI) was also employed in some studies (Soehner et al., 2014). While this diversity in diagnostic tools highlights the lack of a standardised approach to mood assessment, the frequent reliance on HAM-D, DSM-based criteria, and related structured interviews reflects their prominence in hypersomnia and depression research.

Cross-Sectional Studies

Most of the studies (16 out of 19) revealed a substantial correlation between hypersomnia and depression, highlighting hypersomnia as a salient characteristic of depressive illness. Bazin et al. (2024) discerned certain sleep patterns in patients with depression and hypersomnia, including extended total sleep duration and reduced REM latency. Likewise, Cheung et al. (2024) discovered that hypersomnolence in individuals with MDD was associated with heightened depression severity, elevated suicidal thoughts, and an increased propensity for atypical depression. Additional research (e.g., Chen et al., 2021; Mume, 2010) indicated that hypersomnia was associated with more severe depression symptoms and worse clinical outcomes, such as heightened functional impairment and chronicity of episodes (Murru et al., 2019). While other studies did not observe a clear correlation (Calati et al., 2010; Hawkins et al., 1985; YoEargon et al., 1997), the cumulative data indicates that hypersomnia is a notable indicator of depression severity.

Researchers have proposed several mechanisms to explain the relationship between hypersomnia and depression. Neurobiological research indicates that disturbances in brain circuits governing arousal and mood may underlie hypersomnia in depression. Plante et al. (2018) indicated decreased functional connectivity between the thalamus and striatum in

depressed patients exhibiting hypersomnia, suggesting modified neural network dynamics. Cognitive-behavioural frameworks highlight maladaptive sleep-related cognitions, including dysfunctional beliefs on the need for excessive sleep, which may sustain hypersomnia and intensify mood disorders (Cook et al., 2021). Circadian dysregulation is a significant component since research indicates that delayed or disturbed circadian rhythms contribute to hypersomnia, especially in atypical depression (Maruani et al., 2023a; Chen et al., 2021). This subtype, marked by hypersomnia in conjunction with increased hunger and mood reactivity, has been repeatedly recognised as a separate clinical phenotype (Garvey et al., 1984; Cheung et al., 2024).

In addition to symptom severity, hypersomnia is associated with heightened functional impairment and an elevated risk of suicide, particularly when it coexists with insomnia (Soehner et al., 2014). Further research reveals that hypersomnia predicts prolonged depressive episodes and an increased likelihood of recurrence, indicating it may function as a marker of illness chronicity (Murru et al., 2019). Familial aggregation studies suggest a possible genetic or hereditary factor in hypersomnia associated with depressive illnesses (Garvey et al., 1984). Nonetheless, information about treatment response is inconsistent, indicating that there must be further research to determine whether hypersomnia requires tailored therapy approaches.

Table 2.

Cross-sectional studies

Citation	Sample size	Sample characteristics	Sleep/sleepiness measures	Depression measures	Conclusions
Bazin et al (2024)	77	33 hypersomnia with depression 44 hypersomnia without depression Sleep centre	Video-PSG, sleep EEG, MSLT, ESS	DSM-IV criteria, CESD	Hypersomnolence in MDD is associated with longer total sleep time, more accurate sleep estimates, and reduced REM sleep latency. These findings suggest hypersomnia in depression has distinct sleep patterns, but further research is needed to identify mechanisms linking excessive sleep with mood disturbances in MDD.
Calati et al. (2010)	78	Female depressed outpatients	ESS, AIS, PSQI	M.I.N.I, HRSD	EDS in depression was not linked to sleep efficiency or the severity of depressive symptoms, suggesting that the relationship between hypersomnia and mood may involve other mechanisms yet to be identified.
Chellappa & Araujo (2006)	70	70 outpatients with depression	ESS	DSM-IV criteria, BDI	EDS was found in 57.1% of participants and was strongly linked to elevated depression levels, particularly suicidal thoughts. This suggests that EDS in depression may reflect more severe mood disturbances, with potential mechanisms yet to be fully understood.
Chellappa & Araujo (2007)	70	70 outpatients with MDD	SHQ, ICSD	DSM-IV criteria	Insomnia in depression was strongly linked to higher suicide risk, including active suicidal thoughts, intentions, and past attempts, highlighting a more significant connection between sleep disturbances and mood. While the mechanisms remain unclear, insomnia appears to have a stronger impact on suicidal ideation than excessive sleepiness.

Citation	Sample size	Sample characteristics	Sleep/sleepiness measures	Depression measures	Conclusions
Chen et al. (2021)	139	139 inpatients with unipolar depression	ESS	ICD-10, MINI, PHQ-9, BPRS	Hypersomnia in unipolar depression (UD) was found in 28.1% of inpatients and was associated with younger age and recurrent depression. This suggests hypersomnia may indicate a more severe form of UD, pointing to a potential distinct clinical subgroup.
Cheung et al. (2024)	252	252 outpatients with MDD	ESS, ISI, r-MEQ, GSQ	SIGH-ADS, HADS	Hypersomnolence in MDD patients is linked to higher depression severity, increased suicidal ideation, and a higher likelihood of atypical depression. It also increases the risk of depression non-remission, highlighting the need to address both sleep and mood symptoms in treatment for this more severe subgroup.
Claghorn et al. (1981)	102	51 depressed patients with hypersomnia 51 controls	SSS	Feighner criteria, BDI	Daytime sleepiness in patients was strongly linked to higher depression and anxiety levels, suggesting a potential connection between hypersomnia and mood disturbances.
Cook et al. (2021)	66	22 depression and hypersomnia 22 depression without hypersomnia 22 controls	DSM-5 criteria (hypersomnolence), DBAS-16, overnight PSG, ESS, PSQI, HSI, ISI	BDI-II, SCID	In individuals with MDD and comorbid hypersomnolence, elevated DBAS-16 scores suggest that maladaptive sleep-related cognitions may play a role in the link between hypersomnia and depression.
Garvey et al. (1984)	102	17 outpatients with depression and hypersomnia 85 outpatients with depression without hypersomnia	definition: sleeping an additional one or more hours per day during the index depression	RDC, SADS-L, HRSD, depressive subtype diagnosis, ratings of 26 individual symptoms of depression, longitudinal psychiatric history, family history.	Hypersomnia in individuals with MDD is linked to a distinct clinical profile, including increased appetite, weight gain, agitation, and a family history of depression. These associations suggest that hypersomnia may reflect a more severe or early-onset form of depression.
Hawkins et al. (1985)	28	14 depressed patients 14 controls	Sleep EEG	BDI, those who scored above 20 were administered an in-depth psychiatric interview (HRSD) or Zung Self-Rating Depression Scale), Research diagnostic criteria (depression), Depression Adjective Check List in conjunction with a mood adjective check list	Individuals with depression tend to sleep significantly longer than when they are well, with the depression group sleeping nearly twice as much as the control group. This suggests a link between hypersomnia and mood disturbances, potentially reflecting a disruption in sleep regulation mechanisms associated with depression.

Citation	Sample size	Sample characteristics	Sleep/sleepiness measures	Depression measures	Conclusions
Hein et al. (2019)	703	703 individuals with major depression (sleep laboratory)	ESS, ISI, PSG	DSM IV-TR, BDI	Atypical depression correlated with an increased incidence of EDS. Severe depression is a risk factor for EDS.
Maruani et al. (2023) a	125	77 patients with major depressive episode (MDE) and insomnia 48 patients with depression, insomnia and excessive sleepiness	ISI, PSQI, HSI, chronotype- H&O, GSS of the SPAQ-seasonality, actigraphy	MADRS, HADS	The depressed group with insomnia and EDS showed greater suicidal ideation, mood fluctuations, disrupted sleep and biological rhythms than the depressed group with insomnia without excessive sleepiness, suggesting a stronger link between hypersomnia and severe depression. These findings highlight the need for personalised treatments targeting both sleep disturbances and mood symptoms.
Mume (2010)	67	67 patients with depressive episode	ESS	ICD-10 criteria for depression, HDRS	44.8% of patients experienced EDS, with ESS scores strongly correlating with HDRS scores, indicating a significant link between hypersomnia and the severity of depression. This suggests that EDS may serve as an indicator of more severe depressive symptoms.
Murru et al. (2019)	2514	2091 patients with reduced sleep 423 patients with increased sleep	a statistical model predicting the likelihood of hypersomnia was performed	DSM-5 criteria, RDC	The hypersomnia group experienced longer durations of depressive states than the insomnia group, suggesting that hypersomnia is linked to a worse prognosis in acute depression. This highlights the potential impact of sleep disturbances on mood regulation and depressive outcomes.
Plante et al. (2012)	21	7 MDD patients with hypersomnia 7 MDD patients without hypersomnia 7 controls	PSG, sleep-diaries, wrist-worn actigraphy	SCID, HRSD, BDI II or IDS	The hypersomnia group exhibited decreased parieto-occipital slow wave activity (SWA), suggesting a link between excessive sleepiness and reduced brain activity in specific regions in MDD. This finding indicates that hypersomnia in MDD may reflect underlying changes in brain function, offering potential clinical insights into the relationship between sleep disturbances and mood regulation.

Citation	Sample size	Sample characteristics	Sleep/sleepiness measures	Depression measures	Conclusions
Plante et al. (2018)	67	30 women with current depressive disorders 13 women with remitted depressive disorders 24 women controls	ESS	DSM-IV, DSM-5, RDoC, BDI-II, fmri scans	Decreased functional connectivity between the bilateral thalamus and left rostral striatum, particularly the caudate/putamen, was linked to EDS in individuals with depression. This suggests that disruptions in thalamostriatal connectivity may underlie the relationship between EDS and depression.
Soehner et al. (2014)	687	404 MDE with insomnia 44 MDE with hypersomnia 184 MDE with both insomnia and hypersomnia 55 MDE without sleep problems	QIDS-SR	WHO-CIDI, QIDS-SR	Co-occurring insomnia and hypersomnia symptoms in individuals with MDE were associated with more severe depression, higher rates of impulse control disorders, and suicide planning. This suggests that sleep disturbances, particularly when both insomnia and hypersomnia are present, may exacerbate depression severity and increase the risk of suicidal behaviour, with potential links to both mood regulation and substance use disorders.
YoEargon et al. (1997)	113	113 inpatients/outpatients with depression (69-insomnia, 20-hypersomnia,24-no sleep problems	sleep questions from the SADS	DSM-III criteria, HAM-D	Insomnia and hypersomnia are linked to suicidal behaviour in individuals with severe depression, suggesting that disrupted sleep patterns may contribute to mood instability and increase suicide risk.
Zhao et al. (2021)	3275	3275 patients with MDD 870 (no sleep problems), 2066 (insomnia only), 105 (hypersomnia only), 234 (insomnia + hypersomnia)	hypersomnia= if patients slept too much or had daytime napping nearly every day or more than half the days in the past two weeks	DSM-IV TR criteria, 64-symptom clinician-rating assessment questionnaire to assess the severity of symptoms of MDD	Individuals with MDD experiencing both insomnia and hypersomnia symptoms face greater functional impairment, increased suicide risk, and more severe depressive symptoms, highlighting the complex relationship between sleep disturbances and mood regulation.

AIS-the Athens Insomnia Scale; **BDI**-Beck Depression Inventory; **BPRS**-The Brief Psychiatric Rating Scale; **CESD**-Centre for Epidemiologic Studies Depression Scale; **DBAS-16-**Dysfunctional Beliefs and Attitudes About Sleep; **DSM III/ IV/V/DSM IV-TR**- The Diagnostic and Statistical Manual of Mental Disorders, third edition/ fourth edition/fifth edition/fourth edition, text revision; **EDS**-Excessive daytime sleepiness; **EEG**-electroencephalogram; **ESS**- Epworth Sleepiness Scale; **fmri**- Functional

magnetic resonance imaging; **GSQ**- General Sleep Questionnaire; **GSS**- Global Seasonality Score; **HADS**-Hospital Anxiety and Depression Scale; **HSI-**Hypersomnia Severity Index; **H&O**- Horne & Ostberg questionnaire; **HRSD/Ham-D**- Hamilton Rating Scale for Depression; **HSI**-Hypersomnia Severity Index; **ICSD**- International Classification of Sleep Disorders **IDS**- Inventory of Depressive Symptomology; **ISI-**Insomnia Severity Index; **MADRS -** Montgomery-Åsberg Depression Rating Scale; **MINI**-The Mini International Neuropsychiatric Interview; **MSLT**- Multiple sleep latency test; **PHQ-9-** Patient Health Questionnaire-9; **PSG-**Polysomnography; **PSQI-** The Pittsburgh Sleep Quality Index; **QIDS-SR**-Quick Inventory of Depressive Symptoms-Self Report; **RDC**-Research Diagnostic Criteria; **RDoC**-Research Domain Criteria; **R-MEQ-** The reduced Horne and ¨Ostberg Morningness and Eveningness Questionnaire; **SADS**- Schedule for Affective Disorders and Schizophrenia; **SADS-L** Schedule for Affective Disorders and Schizophrenia-Lifetime Version; **SCID-** The Structured Clinical Interview; **SHQ-** Sleep Habits Questionnaire; **SPAQ**- Seasonal Pattern Assessment Questionnaire; **SSS**- Stanford Sleepiness Scale; **WHO-CIDI-**World Health Organisation- Composite International Diagnostic Interview

Longitudinal Studies

Among the four longitudinal studies examined, three demonstrated unequivocal evidence that hypersomnia or excessive daytime sleepiness (EDS) might forecast future mood disorders, including depression and suicidality. Roberts et al. (2000) found that hypersomnia was associated with a heightened likelihood of subsequent depression, while symptoms like anhedonia and mood instability emerged as more significant predictors. Blanchard et al. (2023) extended the evidence by demonstrating that EDS and overnight sleep disruptions were predictive of depression among psychiatric inpatients.

The studies identified several potential mechanisms linking hypersomnia to depression. Roberts et al. (2000) highlighted mood instability as both a result and a possible catalyst of hypersomnia, creating a cyclical link that may impede recovery and elevate the risk of relapse. Blanchard et al. (2023) identified emotion regulation issues as a significant mediator between hypersomnia and mood abnormalities, indicating that impaired emotional processing may be fundamental to this association.

In addition to predictions and processes, the examined research uncovered significant discoveries. The frequent co-occurrence of hypersomnia with other sleep disorders indicates that patients require thorough sleep evaluations in cases of depression (Blanchard et al., 2023). Furthermore, the researchers showed that individuals experiencing heightened nocturnal sleep disruptions and daytime sleepiness had increased anxiety and an elevated risk of suicide. Similarly, Maruani et al. (2023b) recognised EDS as a predictor of suicidal thoughts in treatment-resistant depression, suggesting that hypnotic use may mitigate this risk. Zhang et al. (2021) highlighted the efficacy of wearable technology as a novel instrument for the remote monitoring of sleep and mood, potentially improving early

diagnosis and intervention strategies. These discoveries indicate that addressing hypersomnia and associated sleep disorders may be essential for enhancing mood outcomes and mitigating suicide risk in depression.

Table 3.

Longitudinal studies

Citation	Sample size	Sample characteristics	Sleep/sleepiness measures	Mood and other measures	Conclusions
Blanchard et al. (2023)	1500	1500 psychiatric inpatients	PSQI, ESS, DDNSI	PHQ-9	Individuals with greater nighttime sleep disturbances and daytime sleepiness showed increased suicide risk, anxiety, depression, and emotion regulation difficulties over time. This underscores a significant link between disrupted sleep patterns, including daytime sleepiness, and mood-related outcomes in inpatient psychiatry, highlighting the potential role of emotion regulation as a mechanism connecting daytime sleepiness and mood.
Maruani et al. (2023) b	261	261 patients with treatment resistant depression	PSQI, ESS, CSM	DSM-IV, MADRS	EDS is linked to suicidal thoughts in depressed patients, with a potential protective effect from hypnotic use. This suggests a connection between EDS and depression, particularly in the context of suicidal ideation, though the mechanisms remain unclear.
Roberts et al. (2000)	2370	Year 1994: depressed (206), not depressed (2164) Year 1995: Depressed (215) Not depressed (2155)	two items from the DSM-12D on trouble falling asleep or staying asleep (insomnia) and sleeping too much (hypersomnia)	DSM-IV criteria, DSM-12D	Hypersomnia is linked to an increased risk of future depression, but other symptoms, such as anhedonia and mood instability, appear to be more significant predictors. This highlights the need to explore the mechanisms connecting hypersomnia with mood disturbances.
Zhang et al. (2021)	368	368 participants with depression	Actigraphy (Fitbit wristband)	PHQ-8	Wearable devices offer a potential tool to monitor sleep patterns remotely, providing insights into how hypersomnia may reflect mood changes and aiding in depression assessment and progression tracking.

AUDADIS IV-Alcohol Use Disorder and Associated Disabilities Interview Schedule; **BDI**-Beck Depression Inventory; **CSM**- Composite Scale of Morningness; **DDNSI -** The Disturbing Dreams and Nightmares Severity Index; **DSM/DSM IV TR-**The Diagnostic and Statistical Manual of Mental Disorders, fourth edition/, text revision; **DSM - 12D**-12-item measure based on the DSM-IV diagnostic criteria; **HRSD**- Hamilton rating scale for depression; **ISI**-Insomnia Severity Index; **MADRS**- Montgomery- Asberg

Depression Rating Scale; **PHQ-8/9**-Patient Health Questionnaire-8/9; **PSG**- Polysomnography; **PSQI**- The Pittsburgh Sleep Quality Index; **SCID 5-PD**- Structured Clinical Interview for DSM-5 personality disorder; **SCID 5-RV-** Structured Clinical Interview for DSM-5 research version;

Intervention/Experimental Studies

Ten clinical studies examined the association between hypersomnia and depression, all using pharmacological interventions: modafinil (n=5), pimavanserin, brexpiprazole, vortioxetine, mirtazapine, and ketamine. Nine studies specifically investigated whether alleviating hypersomnia symptoms resulted in enhancements in depressed outcomes.

Modafinil, a wake-promoting agent, effectively alleviated excessive sleepiness and fatigue in people with MDD, especially in those with residual symptoms after antidepressant treatment (Fava et al., 2007; De Battista et al., 2003; Thase et al., 2006; Konuk et al., 2006). Certain investigations indicated slight improvements in mood and clinical results (Thase et al., 2006; Konuk et al., 2006). Nevertheless, not all outcomes were favourable—Dunlop et al. (2007) found no significant variation in overall sleepiness levels relative to placebo, and safety issues were identified, including exacerbations of suicidal ideation in many instances. Notwithstanding discrepancies, evidence indicates that modafinil may mitigate symptoms associated with hypersomnia, thus offering indirect advantages for mood.

Research indicates a bidirectional association between hypersomnia and depression, whereby excessive sleepiness intensifies depressive symptoms, and its alleviation may improve mood stability. The benefits of modafinil are likely due to its wake-promoting influence on dopamine and orexin pathways, which may alleviate fatigue and psychomotor retardation in MDD. Pimavanserin, Brexpiprazole, and Vortioxetine, which influence serotonin and dopamine systems, were associated with enhanced sleep efficiency, less daytime sleepiness, and concomitant decreases in depressive symptoms (Jha et al., 2020; Krystal et al., 2016; Cao et al., 2019).

Ketamine, a rapid-acting treatment for treatment-resistant depression, had favourable outcomes in individuals with hypersomnia, exhibiting elevated response rates and expediting

symptom alleviation relative to those without hypersomnia (Patarroyo-Rodriguez et al., 2024). This indicates that subgroups with hypersomnia may exhibit varied responses to treatment, underscoring the need for personalised strategies. Likewise, mirtazapine, an antidepressant, despite its sedative properties, decreased daytime sleepiness with time (Shen et al., 2011), highlighting that hypersomnia care differs according to the medication's mechanism.

Cao et al. (2019) showed that enhancements in sleep quality and reduced daytime sleepiness were significantly associated with decreased depression severity after Vortioxetine medication, indicating that enhanced sleep may facilitate mood recovery. Jha et al. (2020) and Krystal et al. (2016) corroborated this process, demonstrating that pharmaceutical interventions (pimavanserin and brexpiprazole, respectively) aimed at alleviating sleep disruptions also enhanced mood and daytime functioning. These data indicate a bidirectional link in which interrupted sleep leads to mood dysregulation and vice versa.

Numerous research studies (e.g., Fava et al., 2007; Thase et al., 2006) investigated Modafinil as an adjunctive treatment for SSRI partial responders, revealing that alleviating fatigue and hypersomnia enhanced energy and alertness, resulting in improved mood outcomes. This underscores fatigue and low energy as crucial elements connecting hypersomnia and depression.

In many studies (Konuk et al., 2006; Shen et al., 2011), pharmacological treatments designed to enhance daytime alertness correlated with simultaneous enhancements in mood. This reinforces the notion that excessive sleepiness is not just a sign of depression but a changeable factor influencing mood disruption.

The therapeutic efficacy of wake-promoting drugs, especially modafinil, was a prevalent topic. Despite some inconsistent outcomes (De Battista et al., 2003; Dunlop et al., 2007),

most trials indicated at least partial improvements in hypersomnia symptoms, particularly in instances when conventional antidepressants did not alleviate fatigue and sleepiness. Sedating antidepressants like mirtazapine (Shen et al., 2011) have been shown to alleviate daytime fatigue, indicating that judiciously selected sedatives may enhance sleep architecture, hence indirectly ameliorating hypersomnia and mood.

Findings suggest that addressing hypersomnia in MDD may improve treatment outcomes; nevertheless, results are inconsistent, and safety concerns persist. Additional research is required to identify which people experience the most benefit from wake-promoting interventions and to elucidate long-term effectiveness and associated risk factors.

Table 4.

Intervention/experimental studies

Citation	Sample size	Sample characteristics	Sleep/sleepiness measures	Mood and other measures	Conclusions
Cao et al. (2019)	146	92 patients with depression 54 controls	PSQI, ESS, ISI	DSM-V, MADRS	Improvements in depressive symptoms among individuals with MDD treated with Vortioxetine were closely linked to enhanced sleep quality, including daytime sleepiness. This suggests that better sleep may mediate reductions in depression severity, highlighting a potential mechanism connecting daytime sleepiness and depression.
De Battista et al. (2003)	136	136 patients with MDD	ESS	DSM-IV criteria, HAM-D	Modafinil may help manage residual fatigue and sleepiness in partial responders to antidepressants, suggesting its potential to target sleepiness as a mechanism influencing depression. No significant differences between Modafinil and placebo in overall HAM-D scores.
Dunlop et al. (2007)	72	72 patients with MDD	ESS	DSM IV criteria, MADRS, HDRS-31	While Modafinil showed no overall difference in ESS scores, it improved hypersomnia-related symptoms on the HDS, suggesting its potential to address hypersomnia as a mechanism contributing to depression in the short term.
Fava et al. (2007)	348	348 patients with MDD	ESS	DSM IV criteria, HAM-D	Modafinil augmentation rapidly improved wakefulness, fatigue, and depressive symptoms in SSRI partial responders, highlighting its role in targeting excessive sleepiness as a contributing mechanism in depression.
Jha et al. (2020)	207	207 patients with MDD	KSS, HDRS insomnia items	SCID-5-CV, MADRS, HDRS, unproductive days subscore	Pimavanserin improved sleep disturbances, including daytime sleepiness and overall functionality in MDD, underscoring the role of sleep regulation in addressing hypersomnia's contribution to depression.

Citation	Sample size	Sample characteristics	Sleep/sleepiness measures	Mood and other measures	Conclusions
Konuk et al. (2006)	25	25 patients with MDD (21 completed the 6-week augmentation trial)	ESS	HAM-D	Modafinil combined with SSRIs improved fatigue, sleepiness, and depression, highlighting its potential to address hypersomnia's role in depression.
Krystal et al. (2016)	44	44 patients with MDD	PSG recording, CSD-M), ISI, ESS	DSM-IV-TR criteria**,** MINI, ATRQ, MADRS	Brexpiprazole improved sleep physiology, daytime alertness, and sleepiness, shedding light on its role in addressing hypersomnia within depression.
Patarroyo-Rodriguez et al. (2024)	52	52 patients with treatment resistant depression	QIDS-SR	QIDS-SR	Individuals with hypersomnia and atypical depression showed faster, greater treatment responses, highlighting potential subtype-specific mechanisms linking hypersomnia and depression.
Shen et al. (2011)	74	42 patients with depression 32 controls	ESS, SSS, MSLT	DSM-IV criteria, HRDS-17, CES-D, BDI-II	Mirtazapine improved daytime sleepiness and fatigue in depression, suggesting sedating antidepressants may address hypersomnia-related mechanisms in depressive symptoms.
Thase et al. (2006)	245	245 patients with MDD (194 completed the study)	ESS	HAM-D, MADRS	Twelve weeks of Modafinil augmentation reduced sleepiness, fatigue, and depressive symptoms, supporting its role in addressing hypersomnia-related mechanisms in depression.

ATRQ- Massachusetts General Hospital Antidepressant Treatment Response Questionnaire; **CES-D-** Centre for Epidemiologic Studies Depression Scale; **CPFQ-**Massachusetts General Hospital—Cognitive and Physical Functioning Questionnaire; **CSD-M**- Consensus Sleep Diary for Morning; **DSM IV-TR**- The Diagnostic and Statistical Manual of Mental Disorders, fourth edition; **ESS**- Epworth Sleepiness Scale; **HRSD/Ham-D**- Hamilton Rating Scale for Depression; **ISI**-Insomnia Severity Index; **KSS**- Karolinska Sleepiness Scale; **MADRS**- Montgomery- Asberg Depression Rating Scale; **MINI**-The Mini International Neuropsychiatric Interview; **MSLT**- Multiple sleep latency test; **PSQI-** The Pittsburgh Sleep Quality Index**; QIDS-SR-** The Quick Inventory of Depressive Symptomatology-self-report; **SCID-CV**- Structured Clinical Interview for DSM-5, Clinician Version; **SSRIs-** Selective serotonin reuptake inhibitors

Discussion

This review sought to (1) outline current studies on the association between hypersomnia and depression, focussing on possible causal connections, and (2) identify pertinent mechanisms that may elucidate this relationship. The examined literature offers considerable evidence indicating a robust correlation between hypersomnia and depressed symptoms, as shown by cross-sectional, longitudinal, and interventional studies. Most cross-sectional studies (16 of 19) indicated that hypersomnia is strongly correlated with heightened depressive symptoms (Cheung et al., 2024; Chen et al., 2021; Mume, 2010), augmented functional impairment (Murru et al., 2019), and increased suicidality (Soehner et al., 2014). Moreover, hypersomnia is especially common in subtypes such as atypical depression, characterised by co-occurring symptoms such as mood reactivity and heightened hunger (Garvey et al., 1984; Cheung et al., 2024). Although some studies indicate no relationship between hypersomnia and depression (Calati et al., 2010), the aggregate findings robustly imply that hypersomnia is a clinically significant and relevant symptom in depressive disorders.

Longitudinal research provides further evidence, with many studies indicating that hypersomnia predicts future depressive symptoms and suicidality (Roberts et al., 2000; Blanchard et al., 2023; Maruani et al., 2023b). Challenges in emotion regulation, mood instability, and poor quality have been suggested as mechanisms that may underpin this relationship, reinforcing the notion of a cyclical dynamic in which hypersomnia both originates from and exacerbates depressive pathology (Roberts et al., 2000; Blanchard et al., 2023).

Experimental studies utilising pharmacological interventions, specifically wake-promoting agents such as modafinil, suggest that addressing hypersomnia may enhance mood, energy, and daytime functioning, particularly in individuals exhibiting residual depressive symptoms (Fava et al., 2007; Thase et al., 2006; Konuk et al., 2006). Likewise, recent pharmacological

treatments such as Pimavanserin, Brexpiprazole, and Vortioxetine have shown the capacity to concurrently mitigate somnolence and diminish the intensity of depressive symptoms (Jha et al., 2020; Krystal et al., 2016; Cao et al., 2019). Mirtazapine, a sedative antidepressant, was seen to enhance daytime performance over time (Shen et al., 2011), indicating that improved nocturnal sleep quality may provide subsequent advantages for hypersomnia and depressed symptoms.

Regarding the second aim, several neurobiological and psychological mechanisms have been suggested to elucidate the association between hypersomnia and depression. Functional neuroimaging research reveals modified thalamostriatal connectivity in depressed patients exhibiting hypersomnia, suggesting disturbances in arousal control networks (Plante et al., 2018). Circadian disruption and delayed sleep phase are prevalent, especially among people exhibiting unusual characteristics (Maruani et al., 2023a; Chen et al., 2021). Cognitive-behavioural theories highlight maladaptive attitudes about sleep and the utilisation of sleep as an avoidance mechanism, both of which may sustain hypersomnia and depression (Cook et al., 2021).

Additional clinical insights encompass the association between hypersomnia and the chronicity of depressive episodes (Murru et al., 2019), familial aggregation indicating heritability (Garvey et al., 1984), and the significant comorbidity of hypersomnia with other sleep disorders, including insomnia (Blanchard et al., 2023). The simultaneous presence of insomnia and hypersomnia dramatically elevates the risk of suicide (Soehner et al., 2014; Maruani et al., 2023a).

A significant drawback in the research is the variability in the definition and measurement of hypersomnia among studies. As anticipated, there was significant heterogeneity, complicating

direct comparisons. Increased uniformity in diagnostic criteria and measuring instruments would significantly improve the reliability and relevance of future results.

The lack of consensus on this issue complicates both the identification of the problem and the selection of suitable therapeutic strategies (Dauvilliers et al., 2013). Terminological inconsistencies—such as hypersomnolence (Cheung et al., 2024; Cook et al., 2021; Bazin et al., 2024), hypersomnia symptoms (Zhao et al., 2021; Soehner et al., 2014), EDS (Hein et al., 2019; Calati et al., 2010; Maruani et al., 2023a; Maruani et al., 2023b; Mume, 2010; Plante et al., 2018), daytime sleepiness (Blanchard et al., 2023; Claghorn, 1981; Jha et al., 2020; Cao et al., 2019), and excessive sleepiness (Fava et al., 2007)—illustrate the diversity of conceptualisations across studies. However, more precise operationalisation in particular studies could improve comparability and facilitate interpretation (Wescott et al., 2023). Furthermore, the variability of hypersomnia manifestations between researchers hinders the establishment of standardised treatment methods.

Furthermore, clinical studies examining pharmacological therapies have intriguing intervention opportunities, although they exhibit inconsistent results. Although modafinil is useful for the short-term alleviation of hypersomnia, its long-term effectiveness and overall influence on depressed symptoms are uncertain. Emerging evidence for medications such as ketamine and pimavanserin indicates the need for additional investigation of innovative treatment agents, especially those targeting the neurobiological causes of hypersomnia. Another notable gap lies in the lack of psychology intervention studies that investigate the reciprocal effects of treating hypersomnia and depression. There has been no study conducted to investigate if therapies that target mood could ease hypersomnia, or vice versa. This gap leaves a crucial space for investigation that has not been addressed. Due to this absence, a significant opportunity for future research to address the interconnected nature of these conditions has been brought to light.

The correlation between hypersomnia and depression is well documented; nevertheless, the underlying mechanisms are little comprehended. The inconsistency in the definition and evaluation of hypersomnia complicates the understanding of its function in depressive illnesses. The results highlight the need for a more cohesive treatment strategy that merges pharmaceutical and psychological therapies tailored to the person. Future research must emphasise the establishment of uniform criteria, conduct longitudinal studies to determine causation, and explore psychotherapy methods for addressing hypersomnia in depression.

Limitations

A limitation of this systematic review is its emphasis on a diagnostic methodology, with the majority of included papers depending on conventional categories such as MDD with hypersomnia. This may neglect the intricate manifestations of hypersomnia across diverse symptomatology. By characterising hypersomnia mostly as a characteristic of MDD rather than examining its function within wider transdiagnostic mechanisms, the review may inadequately depict its interaction with other phenomena, such as fatigue, low mood, or anhedonia. This focus on categorising diagnoses may limit a more nuanced understanding of hypersomnia as a symptom that encompasses several characteristics of psychopathology.

Another drawback of this analysis is its focus on clinical groups, namely those with severe or treatment-resistant depression. This emphasis yields significant insights into high-risk situations, but it may restrict the application of results to people with milder hypersomnia or those who do not seek professional care. Individuals with subthreshold or early-stage symptoms of hypersomnia may have distinct patterns that our research does not include. A comprehensive, transdiagnostic approach may effectively include the diversity of hypersomnia across many contexts, providing a more holistic understanding of its function in depression, including non-clinical or community environments.

The influence of antidepressants on sleep presents a further limitation. A significant number of participants in the analysed trials were undergoing pharmacological therapy; however, the impact of these drugs on hypersomnia was not universally considered. Antidepressants may profoundly modify sleep architecture, complicating distinctions between hypersomnia as an intrinsic characteristic of depression or, in some instances, a drug side effect. This mirrors actual clinical practice, where most individuals diagnosed with depression undergo pharmacological treatment; nonetheless, it complicates the ability to draw conclusive insights into the characteristics of hypersomnia in depression. Future research may investigate non-medicated cohorts or compare other types of antidepressants to further comprehension of their effects on sleep disruptions. Investigating the varying impacts of drugs on hypersomnia may reveal whether certain pharmacological treatments worsen or mitigate excessive sleepiness, thereby informing more personalised therapeutic approaches.

Collectively, these limitations underscore the intricacy of comprehending hypersomnia within the framework of depression. The review highlights significant correlations between the two; however, a more nuanced approach—one that transcends conventional diagnostic categories, includes non-clinical samples, and considers the impact of medication—may be essential to comprehensively understand hypersomnia and its ramifications for mental health.

Clinical Implications

The correlation between hypersomnia and depression suggests that there must be consistent monitoring, especially in people with atypical or recurrent depression. Adverse outcomes, including treatment resistance and a threefold heightened chance of non-remission in depression, are associated with hypersomnia (Cheung et al., 2024). Evidence indicates that hypersomnia may be associated with suicidality, which points to the importance of monitoring sleep disorders in the assessment of suicide risk (Soehner et al., 2014; Zhao et al.,

2021). Due to the inconsistency in definitions across research, standardising diagnostic criteria and integrating objective measurements, such as actigraphy, might enhance assessment precision and treatment planning. Recent studies on tailored therapy, including Cognitive Behavioural Therapy for Hypersomnia (CBT-H) and pharmaceutical agents such as Modafinil and Ketamine, indicate possible advantages; nevertheless, more rigorous trials are required to confirm their efficacy (Ong et al., 2020).

Identifying hypersomnia as a unique clinical characteristic within MDD may facilitate more targeted and effective therapies. Using integrated techniques to address mood and sleep disturbances—such as circadian-focused treatments, REM-targeting antidepressants, and organised routines—may improve patient results (Bazin et al., 2024). Collaboration among sleep experts, psychologists, and primary care doctors is crucial for comprehensive therapy. Furthermore, early intervention for people with hypersomnia, especially those with a family inclination towards depression, may alleviate the intensity and length of depressive episodes. Future investigations should examine the neurobiological causes of hypersomnia to enhance treatment approaches and optimise long-term results for this high-risk demographic.

Research Implications

Future research must focus on creating consistent criteria and standardised methods for evaluating hypersomnia in depression. The inconsistency in measurements across research hinders data synthesis, pointing to the importance of objective instruments like polysomnography and actigraphy in conjunction with self-reports. Neuroimaging investigations may elucidate aberrant brain connection patterns, especially within mood and arousal networks, providing insights into the neurobiological causes of hypersomnia. Furthermore, investigations into reduced REM sleep latency in persons with both

hypersomnia and depression may guide innovative remedies, like REM-suppressing antidepressants or circadian-based therapies.

Psychological therapies are an inadequately examined domain, with CBT-H showing potential in preliminary investigations but necessitating more validation. Combining psychological and pharmaceutical therapies may provide a more tailored approach. Furthermore, research on genetics and epigenetics is a significant route for further exploration in the future. Although previous research has revealed that there is a family connection between hypersomnia and depression (Hamilton et al., 2020), more genetic studies that are more thorough might discover specific heritable variables that contribute to this correlation. An investigation of the relationship between genetic predispositions and environmental factors has the potential to enhance the development of individualised treatment plans. Finally, the incorporation of qualitative and mixed-method research would provide more in-depth insights into the experiences of those who suffer from hypersomnia and depression, which would, in turn, guide the creation of person-centred interventions.

Conclusion

This systematic review sought to identify and consolidate results from the current body of data about the processes that explain the correlation between hypersomnia and depression. The review uncovered clear connections between hypersomnia and depression, yet the mechanisms underlying this relationship remain elusive, meaning a lack of explanatory factors for this relationship in current literature. Improvement in methodological practices and more concerted attention to hypersomnia in clinical and research work will hopefully lead to clearer outcomes in future research. For now, it is important to not overlook hypersomnia as a marker of greater severity in depression.

References

Afonso, P., Figueira, M. L., & Paiva, T. (2014). Sleep-wake patterns in schizophrenia patients compared to healthy controls. *The world journal of biological psychiatry: the official journal of the World Federation of Societies of Biological Psychiatry, 15*(7), 517–524. https://doi.org/10.3109/15622975.2012.756987

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Publishing.

Andaku, D. K., D'Almeida, V., Carneiro, G., Hix, S., Tufik, S., & Togeiro, S. M. (2015). Sleepiness, inflammation and oxidative stress markers in middle-aged males with obstructive sleep apnea without metabolic syndrome: a cross-sectional study. *Respiratory research, 16*(1), 3. https://doi.org/10.1186/s12931-015-0166-x

Araghi, M. H., Jagielski, A., Neira, I., Brown, A., Higgs, S., Thomas, G. N., Taheri, S. (2013). The complex associations among sleep quality, anxiety-depression, and quality of life in patients with extreme obesity. *Sleep 36*, 1859–1865. https://doi.org/10.5665/sleep.3216

Armitage, R. (2007). Sleep and circadian rhythms in mood disorders. *Acta Psychiatrica Scandinavica, 115*(s433), 104–115. https://doi.org/10.1111/j.1600-0447.2007.00968.x

Barateau, L., Lopez, R., Franchi, J. A., & Dauvilliers, Y. (2017). Hypersomnolence, Hypersomnia, and Mood Disorders. *Current psychiatry reports, 19*(2), 13. https://doi.org/10.1007/s11920-017-0763-0

Bazin, B., Frija-Masson, J., Benzaquen, H., Maruani, J., Micoulaud-Franchi, J. A., Lopez, R., Philip, P., Bourgink, P., Lejoyeux, M., d'Ortho, M.-P., & Geoffroy, P. A. (2024). Major depressive disorder with hypersomnolence complaint: A comparison study with non-depressed

individuals examining objective biomarkers. *Journal of Affective Disorders, 352*, 422–428. https://doi.org/10.1016/j.jad.2024.02.030

Billiard, M., Dolenc, L., Aldaz, C., Ondze, B., & Besset, A. (1994). Hypersomnia associated with mood disorders: a new perspective. *Journal of psychosomatic research, 38*(Suppl 1), 41–47. https://doi.org/10.1016/0022-3999(94)90134-1

Blanchard, A. W., Rufino, K. A., Nadorff, M. R., & Patriquin, M. A. (2023). Nighttime sleep quality & daytime sleepiness across inpatient psychiatric treatment is associated with clinical outcomes. *Sleep Medicine, 110*, 235–242.

Calati, R., Gaspar-Barba, E., Cruz-Fuentes, C. S., Nenclares, A., Jimenez-Genchi, A., De Ronchi, D., & Serretti, A. (2010). Excessive daytime sleepiness in depressed women. *Psychiatry research, 179*(2), 171–175. https://doi.org/10.1016/j.psychres.2009.11.021

Cao, B., Park, C., Rosenblat, J. D., Chen, Y., Iacobucci, M., Subramaniapillai, M., Mansur, R. B., Zuckerman, H., Lee, Y., & McIntyre, R. S. (2019). Changes in sleep predict changes in depressive symptoms in depressed subjects receiving vortioxetine: An open-label clinical trial. *Journal of psychopharmacology, 33*(11), 1388–1394. https://doi.org/10.1177/0269881119874485

Chellappa, S. L., & Araújo, J. F. (2006). Excessive daytime sleepiness in patients with depressive disorder. *Revista brasileira de psiquiatria (Sao Paulo, Brazil: 1999), 28*(2), 126–129. https://doi.org/10.1590/s1516-44462006000200010

Chellappa, S. L., & Araújo, J. F. (2007). Sleep disorders and suicidal ideation in patients with depressive disorder. *Psychiatry research, 153*(2), 131–136. https://doi.org/10.1016/j.psychres.2006.05.007

Chen, Y., Zhang, L., Hu, S., Zhang, H., Sun, Q., Hong, M., Qian, Y., Lu, L., & Sun, H. (2021). Status and Correlates of Hypersomnia in Hospitalized Patients with Unipolar Depression - Beijing, Henan, and Shandong, China, August 2019-March 2021. *China CDC weekly, 3*(42), 879–882. https://doi.org/10.46234/ccdcw2021.217

Cheung, M. M. S., Lam, S. P., Chau, S. W. H., Chan, N. Y., Li, T. M. H., Wing, Y. K., & Chan, J. W. Y. (2024). Hypersomnolence is associated with non-remission of major depressive disorder. *Sleep Medicine, 119*, 35–43.

Claghorn, J. L., Mathew, R. J., Weinman, M. L., & Hruska, N. (1981). Daytime sleepiness in depression. *The Journal of clinical psychiatry, 42*(9), 342–343.

Cook, J. D., Rumble, M. E., Tran, K. M., & Plante, D. T. (2021). Potential Maladaptive Sleep-Related Cognitions in Depression with Comorbid Hypersomnolence: An Exploratory Investigation. *Behavioural sleep medicine, 19*(2), 232–242. https://doi.org/10.1080/15402002.2020.1732390

Dauvilliers, Y., Lopez, R., Ohayon, M., & Bayard, S. (2013). Hypersomnia and depressive symptoms: methodological and clinical aspects. *BMC medicine, 11*, 78. https://doi.org/10.1186/1741-7015-11-78

DeBattista, C., Doghramji, K., Menza, M. A., Rosenthal, M. H., Fieve, R. R., & Modafinil in Depression Study Group (2003). Adjunct modafinil for the short-term treatment of fatigue and sleepiness in patients with major depressive disorder: a preliminary double-blind, placebo-controlled study. *The Journal of clinical psychiatry, 64*(9), 1057–1064. https://doi.org/10.4088/jcp.v64n0911

de Pinho, R.S.N., da Silva-Junior, F.P., Bastos, J.P.C., Maia, W.S., de Mello, M.T., de

Bruin, V.M.S., de Bruin, P.F.C. (2006). Hypersomnolence and accidents in truck drivers: a cross-sectional study. *Chronobiology International, 23*(5), 963–971. https://doi.org/10.1080/07420520600920759

Dunlop, B. W., Crits-Christoph, P., Evans, D. L., Hirschowitz, J., Solvason, H. B., Rickels, K., Garlow, S. J., Gallop, R. J., & Ninan, P. T. (2007). Coadministration of modafinil and a selective serotonin reuptake inhibitor from the initiation of treatment of major depressive disorder with fatigue and sleepiness: A double-blind, placebo-controlled study. Journal of *Clinical Psychopharmacology, 27*(6), 614–619

Fava, M., Thase, M. E., DeBattista, C., Doghramji, K., Arora, S., & Hughes, R. J. (2007). Modafinil augmentation of selective serotonin reuptake inhibitor therapy in MDD partial responders with persistent fatigue and sleepiness. *Annals of Clinical Psychiatry, 19*(3), 153–159. https://doi.org/10.1080/10401230701464858

Fitzgerald, C. T., Messias, E., & Buysse, D. J. (2011). Teen sleep and suicidality: results from the youth risk behavior surveys of 2007 and 2009. *Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine, 7*(4), 351–356. https://doi.org/10.5664/JCSM.1188

Garvey, M. J., Mungas, D., & Tollefson, G. D. (1984). Hypersomnia in major depressive disorders. *Journal of affective disorders, 6*(3-4), 283–286. https://doi.org/10.1016/s0165-0327(84)80006-3

Gold, P. W., Chrousos, G. P. (2002). Organization of the stress system and its dysregulation in melancholic and atypical depression: high vs low CRH/NE states. *Molecular psychiatry, 7*(3), 254–275. https://doi.org/10.1038/sj.mp.4001032

Gold, A. K., Sylvia, L. G. (2016). The role of sleep in bipolar disorder. *Nature and science of sleep, 8*, 207–214. https://doi.org/10.2147/NSS.S85754

Goldstein, T. R., Bridge, J. A., & Brent, D. A. (2008). Sleep disturbance preceding completed suicide in adolescents. *Journal of consulting and clinical psychology, 76*(1), 84–91. https://doi.org/10.1037/0022-006X.76.1.84

Grigolon, R. B., Trevizol, A. P., Cerqueira, R. O., Lee, Y., Mansur, R. B., McIntyre, R. S., & Brietzke, E. (2019). Hypersomnia and Bipolar Disorder: A systematic review and meta-analysis of proportion. *Journal of affective disorders, 246*, 659–666. https://doi.org/10.1016/j.jad.2018.12.030

Hawkins, D. R., Taub, J. M., & Van de Castle, R. L. (1985). Extended sleep (hypersomnia) in young, depressed patients. *The American journal of psychiatry, 142*(8), 905–910. https://doi.org/10.1176/ajp.142.8.905

Hein, M., Lanquart, J. P., Loas, G., Hubain, P., & Linkowski, P. (2019). Prevalence and risk factors of excessive daytime sleepiness in major depression: A study with 703 individuals referred for polysomnography. *Journal of affective disorders, 243*, 23–32. https://doi.org/10.1016/j.jad.2018.09.016

Huang, J. F., Chen, L. D., Lin, Q. C., Chen, G. P., Yu, Y. H., Huang, J. C., & Zhao, J. M. (2016). The relationship between excessive daytime sleepiness and metabolic syndrome in severe obstructive sleep apnea syndrome. *The clinical respiratory journal, 10*(6), 714–721. https://doi.org/10.1111/crj.12276

Hong, Q. N., Gonzalez-Reyes, A., & Pluye, P. (2018). Improving the usefulness of a tool for appraising the quality of qualitative, quantitative and mixed methods studies, the

Mixed Methods Appraisal Tool (MMAT). *Journal of Evaluation in Clinical Practice, 24*(3), 459–467. https://doi.org/10.1111/jep.12884

Jacobson, N. S., Martell, C. R., & Dimidjian, S. (2001). Behavioral activation treatment for depression: Returning to contextual roots. *Clinical Psychology: Science and Practice, 8*(3), 255–270. https://doi.org/10.1093/clipsy.8.3.255

Jha, M. K., Fava, M., Freeman, M. P., Thase, M. E., Papakostas, G. I., Shelton, R. C., Trivedi, M. H., Dirks, B., Liu, K., & Stankovic, S. (2020). Effect of Adjunctive Pimavanserin on Sleep/Wakefulness in Patients with Major Depressive Disorder: Secondary Analysis From CLARITY. *The Journal of clinical psychiatry, 82*(1), 20m13425. https://doi.org/10.4088/JCP.20m13425

Kaplan, K. A., & Harvey, A. G. (2009). Hypersomnia across mood disorders: a review and synthesis. *Sleep medicine reviews, 13*(4), 275–285. https://doi.org/10.1016/j.smrv.2008.09.001

Kaplan, K. A., Gruber, J., Eidelman, P., Talbot, L. S., & Harvey, A. G. (2011). Hypersomnia in inter-episode bipolar disorder: does it have prognostic significance? *Journal of affective disorders, 132*(3), 438–444. https://doi.org/10.1016/j.jad.2011.03.013

Kaplan, K. A., McGlinchey, E. L., Soehner, A., Gershon, A., Talbot, L. S., Eidelman, P., Gruber, J., & Harvey, A. G. (2015). Hypersomnia subtypes, sleep and relapse in bipolar disorder. *Psychological medicine, 45*(8), 1751–1763. https://doi.org/10.1017/S0033291714002918

Konuk, N., Atasoy, N., Atik, L., & Akay, O. (2006). Open-label study of adjunct modafinil for the treatment of patients with fatigue, sleepiness, and major depression treated with selective serotonin reuptake inhibitors. *Advances in therapy, 23*(4), 646–654. https://doi.org/10.1007/BF02850053

Krystal, A. D., Mittoux, A., Meisels, P., & Baker, R. A. (2016). Effects of Adjunctive Brexpiprazole on Sleep Disturbances in Patients with Major Depressive Disorder: An Open-Label, Flexible-Dose, Exploratory Study. *The primary care companion for CNS disorders, 18*(5), 10.4088/PCC.15m01914. https://doi.org/10.4088/PCC.15m01914

Laskemoen, J. F., Simonsen, C., Büchmann, C., Barrett, E. A., Bjella, T., Lagerberg, T. V., Vedal, T. J., Andreassen, O. A., Melle, I., & Aas, M. (2019). Sleep disturbances in schizophrenia spectrum and bipolar disorders - a transdiagnostic perspective. *Comprehensive psychiatry*, *91*, 6–12. https://doi.org/10.1016/j.comppsych.2019.02.006

Leibenluft, E., Turner, E. H., Feldman-Naim, S., Schwartz, P. J.,Wehr, T. A., Rosenthal, N. E. (1995). Light therapy in patients with rapid cycling bipolar disorder: preliminary results. *Psychopharmacology Bulletin, 31*(4):705–710.

Lopes, J. M., Dantas, F. G., & Medeiros, J. L. (2013). Excessive daytime sleepiness in the elderly: association with cardiovascular risk, obesity and depression. *Revista brasileira de epidemiologia = Brazilian journal of epidemiology, 16*(4), 872–879. https://doi.org/10.1590/s1415-790x2013000400007

Maruani, J., Boiret, C., Leseur, J., Romier, A., Bazin, B., Stern, E., Lejoyeux, M., & Geoffroy, P. A. (2023a). Major depressive episode with insomnia and excessive daytime sleepiness: A more homogeneous and severe subtype of depression. *Psychiatry Research, 330*, 115603. https://doi.org/10.1016/j.psychres.2023.115603

Maruani, J., Molière, F., Godin, O., Yrondi, A., Bennabi, D., Richieri, R., El-Hage, W., Allauze, E., Anguill, L., Bouvard, A., Camus, V., Dorey, J. M., Etain, B., Fond, G., Genty, J. B., Haffen, E., Holtzmann, J., Horn, M., Kazour, F., Nguon, A. S., … Geoffroy, P. A. (2023b). Diurnal symptoms of sleepiness and dysfunction predict future suicidal ideation in a French cohort of

outpatients (FACE-DR) with treatment resistant depression: A 1-year prospective study about sleep markers. *Journal of affective disorders, 329*, 369–378. https://doi.org/10.1016/j.jad.2023.02.106

Miller, M. A. (2011). Association of inflammatory markers with cardiovascular risk and sleepiness. *Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine, 7*(5 Suppl), S31–S33. https://doi.org/10.5664/JCSM.1356

Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G., & PRISMA Group (2009). Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS medicine, 6*(7), e1000097. https://doi.org/10.1371/journal.pmed.1000097

Mume, C. O. (2010). Excessive daytime sleepiness among depressed patients. *The Libyan journal of medicine, 5*(1), 4626. https://doi.org/10.3402/ljm.v5i0.4626

Murru, A., Guiso, G., Barbuti, M., Anmella, G., Verdolini, N., Samalin, L., Azorin, J. M., Angst, J. J., Bowden, C. L., Mosolov, S., Young, A. H., Popovic, D., Valdes, M., Perugi, G., Vieta, E., Pacchiarotti, I., & BRIDGE-II-Mix Study Group (2019). The implications of hypersomnia in the context of major depression: Results from a large, international, observational study. European neuropsychopharmacology: the journal of the European College of *Neuropsychopharmacology, 29*(4), 471–481. https://doi.org/10.1016/j.euroneuro.2019.02.011

Nofzinger, E.A, Thase, M.E., Reynolds, 3rd C.F., Himmelhoch, J.M., Mallinger, A., Houck, P. et al. (1991). Hypersomnia in bipolar depression: a comparison with narcolepsy using the multiple sleep latency test. *The American journal of psychiatry, 148*(9), 1177–1181. https://doi.org/10.1176/ajp.148.9.1177

Ohayon, M. M. (2013). Narcolepsy is complicated by high medical and psychiatric comorbidities: a comparison with the general population. *Sleep medicine, 14*(6), 488–492. https://doi.org/10.1016/j.sleep.2013.03.002

Ong, J. C., Dawson, S. C., Mundt, J. M., & Moore, C. (2020). Developing a cognitive behavioural therapy for hypersomnia using telehealth: a feasibility study. *Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine, 16*(12), 2047–2062. https://doi.org/10.5664/jcsm.8750

Patarroyo-Rodriguez, L., Singh, B., Pazdernik, V., & Vande Voort, J. L. (2024). Hypersomnia as a predictor of response to intravenous ketamine/intranasal esketamine in treatment-resistant depression. *Journal of Affective Disorders, 349*, 272–276.

Plante, D. T., Landsness, E. C., Peterson, M. J., Goldstein, M. R., Wanger, T., Guokas, J. J., Tononi, G., & Benca, R. M. (2012). Altered slow wave activity in major depressive disorder with hypersomnia: a high-density EEG pilot study. *Psychiatry research, 201*(3), 240–244. https://doi.org/10.1016/j.pscychresns.2012.03.001

Plante, D. T. (2015). Hypersomnia in Mood Disorders: A Rapidly Changing Landscape. *Current sleep medicine reports, 1*(2), 122–130. https://doi.org/10.1007/s40675-015-0017-9

Plante, D. T., Birn, R. M., Walsh, E. C., Hoks, R. M., Cornejo, M. D., & Abercrombie, H. C. (2018). Reduced resting-state thalamostriatal functional connectivity is associated with excessive daytime sleepiness in persons with and without depressive disorders. *Journal of affective disorders, 227*, 517–520. https://doi.org/10.1016/j.jad.2017.11.054

Popay, J., Roberts, H., Sowden, A., Petticrew, M., Arai, L., Rodgers, M., Britten, N., Roen, K., & Duffy, S. (2006). Guidance on the conduct of narrative synthesis in systematic reviews: A

product from the ESRC Methods Programme. *Economic and Social Research Council (ESRC)*. Retrieved from https://www.lancaster.ac.uk/shm/research/nssr/research/dissemination/publications/NS_guidance_v1.pdf

Rehman, A., Waite, F., Sheaves, B., Biello, S., Freeman, D., & Gumley, A. (2017). Clinician perceptions of sleep problems, and their treatment, in patients with non-affective psychosis. *Psychosis, 9*(2), 129–139. https://doi.org/10.1080/17522439.2016.1206955

Risco, J., Ruiz, P., Mariños, A., Juarez, A., Ramos, M., Salmavides, F., Vega, J., Kruger, H., & Vizcarra, D. (2013). Excessive sleepiness prevalence in public transportation drivers of a developing country. *Traffic injury prevention, 14*(2), 145–149. https://doi.org/10.1080/15389588.2012.692493

Roberts, R. E., Shema, S. J., Kaplan, G. A., & Strawbridge, W. J. (2000). Sleep complaints and depression in an aging cohort: A prospective perspective. *The American journal of psychiatry, 157*(1), 81–88. https://doi.org/10.1176/ajp.157.1.81

Shen, J., Hossain, N., Streiner, D. L., Ravindran, A. V., Wang, X., Deb, P., Huang, X., Sun, F., & Shapiro, C. M. (2011). Excessive daytime sleepiness and fatigue in depressed patients and therapeutic response of a sedating antidepressant. *Journal of affective disorders, 134*(1-3), 421–426. https://doi.org/10.1016/j.jad.2011.04.047

Soehner, A. M., Kaplan, K. A., & Harvey, A. G. (2014). Prevalence and clinical correlates of co-occurring insomnia and hypersomnia symptoms in depression. *Journal of affective disorders, 167*, 93–97. https://doi.org/10.1016/j.jad.2014.05.060

Stern, C., Lizarondo, L., Carrier, J., Godfrey, C., Rieger, K., Salmond, S., Apóstolo, J., Kirkpatrick, P., & Loveday, H. (2020). Methodological guidance for the conduct of mixed methods systematic reviews. *JBI evidence synthesis, 18*(10), 2108–2118. https://doi.org/10.11124/JBISRIR-D-19-00169

Thase, M. E., Fava, M., DeBattista, C., Arora, S., & Hughes, R. J. (2006). Modafinil augmentation of SSRI therapy in patients with major depressive disorder and excessive sleepiness and fatigue: a 12-week, open-label, extension study. *CNS spectrums, 11*(2), 93–102. https://doi.org/10.1017/s1092852900010622

Tsai, S. C. (2010). Excessive sleepiness. *Clinics in chest medicine, 31*(2), 341–351. https://doi.org/10.1016/j.ccm.2010.02.007

Wasilewska, J., Jarocka-Cyrta, E., & Kaczmarski, M. (2007). Narcolepsy, metabolic syndrome and obstructive sleep apnea syndrome as the causes of hypersomnia in children. Report of three cases. *Advances in medical sciences, 52*(Suppl 1), 207–211.

Wescott, D. L., Franzen, P. L., Hasler, B. P., Miller, M. A., Soehner, A. M., Smagula, S. F., Wallace, M. L., Hall, M. H., & Roecklein, K. A. (2023). Elusive hypersomnolence in seasonal affective disorder: actigraphic and self-reported sleep in and out of depressive episodes. *Psychological medicine, 53*(4), 1313–1322. https://doi.org/10.1017/S003329172100283X

YoEargon, M. Y., Kara, H., & Solmaz, M. (1997). Sleep disturbances and suicidal behavior in patients with major depression. *The Journal of clinical psychiatry, 58*(6), 249–251. https://doi.org/10.4088/jcp.v58n0602

Zhang, Y., Folarin, A. A., Sun, S., Cummins, N., Bendayan, R., Ranjan, Y., Rashid, Z., Conde, P., Stewart, C., Laiou, P., Matcham, F., White, K. M., Lamers, F., Siddi, S., Simblett, S., Myin-

Germeys, I., Rintala, A., Wykes, T., Haro, J. M., Penninx, B. W., … RADAR-CNS Consortium (2021). Relationship Between Major Depression Symptom Severity and Sleep Collected Using a Wristband Wearable Device: Multicenter Longitudinal Observational Study. *JMIR mHealth and uHealth, 9*(4), e24604. https://doi.org/10.2196/24604

Zhao, J., Liu, H., Wu, Z., Wang, Y., Cao, T., Lyu, D., Huang, Q., Wu, Z., Zhu, Y., Wu, X., Chen, J., Wang, Y., Su, Y., Zhang, C., Peng, D., Li, Z., Rong, H., Liu, T., Xia, Y., Hong, W., … Fang, Y. (2021). Clinical features of the patients with major depressive disorder co-occurring insomnia and hypersomnia symptoms: a report of NSSD study. *Sleep medicine, 81*, 375–381. https://doi.org/10.1016/j.sleep.2021.03.005

Zimmerman, M., McGlinchey, J. B., Posternak, M. A., Friedman, M., Boerescu, D., & Attiullah, N. (2005). Differences between minimally depressed patients who do and do not consider themselves to be in remission. *The Journal of clinical psychiatry, 66*(9), 1134–1138. https://doi.org/10.4088/jcp.v66n0908

Chapter Three

Bridging Chapter

Word Count: 399

Bridging Chapter

The systematic review examined the relationship between hypersomnia and depression, synthesising the existing research on the topic. We analysed a total of 33 papers, which revealed the significant incidence of hypersomnia in people with depression and its robust correlation with this mood disorder. The results indicated that hypersomnia was associated with elevated depressive symptoms, increased functional impairment, and heightened suicidality. Nonetheless, despite these well-confirmed correlations, the exact processes governing this link remain inadequately understood. Multiple plausible causes have been suggested, including disturbances in circadian cycles, modifications in neurotransmitter systems, and maladaptive sleep-related cognitions. However, the scarcity of longitudinal designs and objective methods constrains our capacity to establish clear conclusions about causation. The review emphasised the significance of managing hypersomnia within the framework of depression, as unaddressed sleep abnormalities may hinder recovery and elevate the risk of chronic illness. Recent research indicates that hypersomnia in psychosis may possess considerable clinical significance, irrespective of its aetiology. Prolonged sleep duration and daytime sleepiness may intensify cognitive deficits, diminish motivation, and lead to social withdrawal—fundamental characteristics of psychotic disorders that already cause significant functional impairments. Moreover, sleep disruptions in psychosis are associated with heightened symptom intensity, elevated recurrence rates, and a worse overall prognosis (Reeve et al., 2021). The precise influence of hypersomnia on psychotic symptoms is ambiguous, given most research in this domain has concentrated on insomnia. Considering the intricate, reciprocal link between sleep disruptions and psychosis, comprehending the function of hypersomnia is crucial for formulating tailored therapies that enhance patient outcomes. This empirical research will investigate hypersomnia in people with psychosis, focusing on its incidence, clinical correlates, and effects on quality of life and physical

activity. By comparing individuals with psychosis and concomitant hypersomnia ('hypersomnia group') with those with psychosis without hypersomnia symptoms ('comparison group'), this study seeks to reveal previously unknown facts about an overlooked research field. In conclusion, the systematic review emphasised the robust correlation between hypersomnia and depression, while the empirical study broadened this inquiry to psychotic disorders, a domain that has yet to be explored. Comprehending the manifestation and ramifications of hypersomnia in psychosis may facilitate the development of more efficacious treatment methodologies and a more sophisticated approach to addressing sleep abnormalities in severe mental disorders. Considering the significant influence of sleep on cognitive, emotional, and functional results, more research in this domain is essential for enhancing the treatment and recovery pathways of patients with psychosis.

Chapter Four

Empirical Paper

Hypersomnia in psychosis: assessment of relationship with clinical features, symptoms, and wellbeing

This paper has been developed for submission to PLoS ONE Journal.

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Hypersomnia in psychosis: assessment of relationship with clinical features, symptoms, and wellbeing

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Abstract

Background. Psychosis is often accompanied by excessive sleepiness and disruptions in sleep patterns. Excessive sleepiness in psychosis may be attributed to the sedative properties of antipsychotic medications; however, other factors such as depression and low activity might also play a role.

Methods. This research conducted a cross-sectional analysis to compare individuals with psychosis who screened positive for hypersomnia (hypersomnia group, n=24) to those who did not (comparison group, n=33) with respect to clinical features, psychiatric symptoms, and functional variables.

Results. There were significant differences in gender and age across the groups, with psychosis patients experiencing hypersomnia more likely to be younger and male. We found no significant differences in diagnosis, medication type, or antipsychotic medication dose. The results indicated greater severity in psychiatric symptoms (paranoia, hallucinations, depression, and anxiety) and reduced quality of life and physical activity in the hypersomnia group; however, these differences did not reach statistical significance relative to the comparison group.

Discussion. We found that hypersomnia symptoms were not related to differences in medication type or dosage, contrary to expectations. Hypersomnia symptoms also appeared to be related to a more severe clinical presentation. However, significant differences were limited, potentially due to (below-threshold) hypersomnia symptoms being common in the comparison group and reduced power due to lower-than-expected sample size. Future studies should consider using objective methods, alternative thresholds for hypersomnia, or investigating daytime sleepiness and extended sleep separately to improve the clarity of their findings.

Introduction

Hypersomnia, i.e., difficulties with sleepiness and oversleeping, is a common sleep difficulty among individuals with psychosis, with 23% of patients and 48% of clinicians reporting issues (Reeve et al., 2019, 2021; Barrett et al., 2020). Patients and clinicians often link the symptoms associated with excessive sleepiness to drowsiness caused by antipsychotic medication (Faulkner et al., 2017; Reeve et al., 2021). This phenomenon stems from the known impact of antipsychotics on the histaminergic system (Miller et al., 2004) and subsequent sedation. Research indicates that people with psychosis often have longer sleep lengths than those without clinical disorders (Martin et al., 2001; Chouinard et al., 2004; Poulin et al., 2010; Afonso et al., 2014). Nonetheless, hypersomnia in psychosis may not be exclusively attributable to medication. Kluge et al. (2012) found that individuals with schizophrenia experiencing psychosis while on medication reported more sleepiness than non-clinical controls. This result indicates that sleepiness in psychosis may arise via prolonged sleep duration, disturbed sleep architecture, sedative side effects, or inherent neurobiological pathways associated with the condition.

Nevertheless, within psychosis, many studies have focused on insomnia and other sleep-related issues, including nightmares (Waite et al., 2020). In part, this is because these concerns are more immediately linked to sleep loss and subsequent clinically significant outcomes like relapse and distress and because evidence-based therapies available for the general population can be adapted for individuals with psychosis (Waite et al., 2016). Therefore, despite its common presentation, hypersomnia receives relatively less attention, similar to depression (Kaplan & Harvey, 2009).

Nevertheless, hypersomnia symptoms may affect clinical outcomes. If excessive sleepiness causes social isolation, it may sustain paranoia by preventing the chance to gather evidence that contradicts their beliefs about threats (Freeman, 2016). It may further aggravate cognitive

impairments, including diminished attention and memory, which are prevalent in psychosis and may obstruct everyday functioning (Wykes et al., 2011; Fatouros-Bergman et al., 2014). Moreover, excessive sleepiness may diminish motivation and energy levels, possibly exacerbating adverse symptoms of psychosis, including apathy and social withdrawal (Bowie & Harvey, 2006; Fervaha et al., 2014). Beyond psychiatric outcomes, excessive sleepiness may also have a negative effect on physical health, leading to sedentary behaviour. People with psychosis on average spend around 3 hrs more each day sedentary than the general population (Stubbs et al., 2016). Sedentary activity has been associated with an elevated risk of cardiac or metabolic health issues, resulting in greater death rates among those who sleep for extended durations (Cappuccio et al., 2010). This phenomenon can be linked to the disparity in life expectancy for those with psychosis, with a premature mortality rate 10 to 20 years higher than that of the general population (Walker et al., 2015). The prevalence of cardiovascular disease among individuals with psychosis primarily accounts for this discrepancy (Lawrence et al., 2013). Excessive sleepiness may also hinder patients' ability to engage in treatment by stopping them from attending or actively participating in visits with mental health teams (Reeve et al., 2021).

Additional factors, including depression and inactivity that have not been thoroughly investigated in existing research, may serve as significant targets for mitigating sleepiness, as they lead to a loop of inactivity and fatigue, hence worsening excessive sleepiness in psychosis. People experiencing psychosis often report depression, characterised by hypersomnia as a prominent symptom (Vorontsova et al., 2013). A qualitative study indicates that excessive sleep might sometimes function as a conscious coping method or 'escape' strategy (Faulkner & Bee, 2017; Waite et al., 2016). Hypersomnia is believed to perpetuate itself via a detrimental cycle, whereby extended sleep induces more sleep inertia (Trotti, 2017), resulting in ongoing sleepiness (Kaplan & Harvey, 2009). To mitigate this, people may

engage in excessive sleep, thereby sustaining the cycle. Moreover, inactivity may result in physical deconditioning, diminishing overall energy levels and complicating daytime wakefulness (Vancampfort et al., 2017). Challenging maladaptive beliefs about energy and sleep (e.g., the notion that more sleep is always advantageous) and promoting incremental modifications to wake-up practices may facilitate the disruption of this cycle and enhance daytime functioning (Sheaves et al., 2018). Promoting incremental modifications to wake-up timings and exercise levels may mitigate excessive sleepiness and improve overall functioning (Reeve et al., 2021). Also, presenting sleepiness only because of antipsychotic medication has unintentionally constrained research into its wider implications for mental health and well-being in this patient population, along with possible treatment strategies (Reeve et al., 2021).

Research Questions

This study aims to clarify the role of hypersomnia in psychosis by comparing patients with psychosis and comorbid hypersomnia (‘hypersomnia group’) against patients with psychosis who do not report hypersomnia symptoms (‘comparison group’) with respect to the below questions:

Are there differences in demographic factors (age, gender, employment status) or clinical features (diagnosis, medication status, or medication dosage) between the hypersomnia and comparison groups?

Are other sleep-related difficulties more pronounced in the hypersomnia group than in the comparison group?

Does the hypersomnia group report more severe psychiatric symptoms (paranoia, hallucinations, anxiety, depression), lower quality of life, or reduced physical activity compared to the comparison group?

Methods

Design

The study was an online survey using a cross-sectional, between-subjects design. A comparison was made between two groups: one consisting of individuals who reported symptoms meeting the threshold for hypersomnia and another group of individuals with psychosis who did not report signs meeting the threshold for hypersomnia. We assessed the groups using an identical set of measures.

Procedure

The recruitment for the study occurred via two routes. The research predominantly recruited participants using poster advertising in suitable patient-orientated settings (e.g., service waiting rooms) and by requesting care teams to distribute advertising cards to eligible individuals. Participants were instructed to access the online information sheet (Appendix D) and permission form (hosted on JISC Online Surveys) by following a link provided on both the poster and cards (Appendix E). Participants granted informed consent (Appendix F) using an online platform before proceeding to answer the survey questions. Additionally, we provided a "consent to contact" option to aid participants in completing the online survey. Care teams enquired if patients were willing to be contacted by researchers. The care team shared the patient's contact information with the researchers if the patient gave permission for contact. The researchers then reached out to the patient to help them access the online survey and address any questions about the study.

The survey link was also disseminated via various social media platforms (Twitter, Reddit, and Facebook peer support groups) to enlist participants who met the specific criteria (between the ages of 18 and 65, diagnosed with non-affective psychosis) across the United Kingdom. Prospective participants clicked the link to access the same information sheet and

permission form as patients within the NHS Trust. From there, they were able to continue with the survey if they were willing to participate.

Participants

A total of 60 individuals completed the survey, including participants from the adult community mental health teams (n=14) and early intervention in psychosis services (EIP) within an NHS Trust across East Anglia (n=14), as well as those who responded to advertisements on social media (n=22). Furthermore, 22 participants engaged with social media advertisements, while an additional 10 did not specify a team affiliation—these are assumed to either originate from social media or be affiliated with clinical teams but omit this information.

We performed a sensitivity analysis using G*Power 3.1 (Faul et al., 2009) prior to recruitment to assess the feasibility of identifying a significant group difference. The research, with a significance level of $\alpha = 0.05$ (two-tailed) and a sample size of 80 individuals (40 per group), would possess 80% power to identify a medium-to-large effect size (Cohen's $d = 0.63$). Nonetheless, owing to recruiting constraints, only 57 individuals were included in the final analysis. This reduced the statistical power of the investigation, indicating it was only adequately powered to identify large effects ($d = 0.76$). Consequently, lesser but potentially meaningful effects may have been undetected.

Inclusion Criteria

The inclusion criteria were that participants:

- Were aged between 18 and 65
- Were fluent in English (i.e., able to complete the online survey)

- They self-reported a diagnosis of either first-episode psychosis or schizophrenia spectrum disorder.

Exclusion Criteria

The exclusion criteria included:

- Individuals with a primary diagnosis of affective disorder (e.g., depression or bipolar disorder)
- Individuals with a primary substance use disorder
- Those with significant neurological difficulties
- Individuals diagnosed with moderate to severe learning disabilities to an extent that would make participation in an online survey inappropriate

Before participating, we asked those who accessed the survey to confirm they did not meet any of the above criteria. Sixty prospective participants successfully completed the web-based survey; however, three individuals were excluded from the analysis as they expressed uncertainty regarding their respective diagnoses. Additionally, the study excluded one participant due to their indication of residency in the United States. Therefore, the analysis included 57 participants.

Measures

The online survey used the following measures: demographics, sleep-related measures, psychiatric symptoms measures, mood-related measures, and quality of life measures.

Demographic and Clinical Data

We asked participants to provide information on their age, gender, ethnicity, level of completed education, and occupation. Additionally, we enquired about their diagnosis and what NHS clinical team the participant was under (if any, for those recruited online).

Lastly, we asked them to disclose any prescribed antipsychotic medication, along with its dosage. We converted the medications to their specified daily dosage equivalents for dopaminergic and histaminergic drugs, following the methodology from Reeve et al. (2021). We conducted the calculations to provide a standardised method for comparing the doses of various antipsychotic medications across the study groups. Information on demographics and medical background questions can be found in Appendix A.

Sleep-Related Questions

Participants were asked about their nightly sleep duration and whether they slept over 11 hours in a 24-hour period or over 9 hours a night over the last month. Participants were also asked about their experiences over the last month regarding excessive daytime sleepiness, specifically whether they felt excessively sleepy to the extent that it was challenging to stay awake or engage in activities. Additionally, participants were queried about the frequency of experiencing excessive sleepiness during a typical week and whether they reported it at least 3 days/week, which meant they met the threshold for hypersomnia. Sleepiness questions can be found in Appendix B.

The Insomnia Severity Index (ISI) by Morin et al., 2011, is a self-report questionnaire consisting of seven items. It is used to assess the severity of insomnia symptoms experienced by an individual during the last month. The Likert scale used in this study assigns a rating from 0 to 4 to each item, with 0 indicating no difficulty and 4 indicating a very severe issue. The total score, which ranges from 0 to 28, is derived from these ratings. The questionnaire has strong internal consistency, as shown by a Cronbach's α coefficient of 0.91 (Morin et al., 2011).

The Epworth Sleepiness Scale (ESS) by Johns, 1992, is an eight-item self-report questionnaire designed to evaluate symptoms of excessive daytime sleepiness. Participants

are requested to assess, using a 4-point scale ranging from 0 to 3, their typical likelihood of dozing off or falling asleep during eight distinct activities. The ESS score, calculated as the sum of the scores for the eight items, ranges from 0 to 24. The internal consistency of responses to the eight questions was assessed using Cronbach's alpha. In ten separate studies, the alpha values varied from 0.73 to 0.90, with a mean of 0.82 (Johns, 1992; Hagell & Broman, 2007). The test-retest reliability of ESS scores, measured over a period of a few weeks to a few months, has been assessed using the intraclass correlation coefficient. This value has varied between 0.81 and 0.93 in five separate investigations, namely Gibson et al., 2006; Izci et al., 2008; Cho et al., 2011; and van der Heide et al., 2015.

The Hypersomnia Severity Index (HSI) by Kaplan et al., 2019, is a concise tool designed to measure the degree of severity and functional impairment caused by excessive sleepiness. It has been validated for use in patients with psychiatric disorders. The questionnaire consists of nine items, and the total score may range from 0 to 36. If the score is 10 or more, it may indicate the presence of hypersomnia over the preceding month. The HSI demonstrated a satisfactory level of internal consistency, with a coefficient of 0.82. The convergent validity with the ESS was quite satisfactory, with a correlation coefficient of 0.44 (Kaplan et al. 2019).

The Integrated Tiredness Index (ITI) by Reeve et al., in prep., is an 8-item survey that measures the effects of tiredness experienced in the previous week. It consists of a sleepiness subscale and a fatigue subscale. The questionnaire uses a Likert scale that ranges from "0 (not at all)" to "4 (extreme)." Individual subscale scores may be used independently for a two-factor analysis or aggregated into a total sum score (ITI-t) to measure overall tiredness.

The Fatigue Severity Scale (FSS) by Krupp et al., 1989 is a questionnaire consisting of nine items that assesses the level of exhaustion experienced as a symptom in various chronic

illnesses and conditions. The scale assesses the impact of fatigue on daily functioning by investigating its association with motivation, physical activity, employment, family, and social life. It also asks respondents to rate how easily they tire and how much it affects them. Responses are rated on a 7-point scale, with 1 indicating "strongly disagree" and 7 indicating "strongly agree." Thus, the minimum possible score is nine, and the maximum is 63. In the first psychometric analysis conducted by Krupp et al. (1989), the FSS demonstrated an internal consistency of 0.88.

Psychiatric Symptom Measures

The Depression, Anxiety, and Stress Scale—21 Items (DASS-21) by Lovibond & Lovibond, 1995, is a questionnaire that provides three assessments of depression, anxiety, and stress by using a set of 21 questions that individuals answer about their own emotional state. We only used the depression and anxiety subscales for this current study. Participants evaluate each of the seven things on a scale from 0 ("did not apply at all") to 3 ("applied very much"). Higher scores indicate increased levels of anxiety and depression. The three measures have shown exceptional internal consistency, with all Cronbach coefficients over 0.90.

The Specific Psychotic Experiences Questionnaire (SPEQ) by Ronald et al., 2014, is a set of questionnaires designed to assess six features of psychotic experiences: paranoia, hallucinations, cognitive disorganisation, grandiosity, anhedonia, and negative symptoms. This current research used only the hallucinations subscale. This subscale has nine items, each assessed on a uniform scale, leading to a total score of 45. All six subscales of the SPEQ showed exceptional internal consistency and stability over a period of nine months, with correlation coefficients ranging from .65 to .74 (all $P < .001$). Principal component analysis, as shown by Ronald et al. (2014), was used to establish the construct validity of the SPEQ by confirming its division into six distinct subscales.

The revised Green et al. Paranoid Thoughts Scale (R-GPTS) by Freeman et al., 2019, is a tool used to quantify paranoia and comprises two 16-item measures that evaluate thoughts of reference (Part A) and concepts of persecution (Part B). The overall score for each subscale is calculated by summing the items. A score of 18 or above on the R-GPTS Persecution scale suggests the existence of strong paranoid ideation and a probable persecutory delusion, as per Freeman et al. (2019). The R-GPTS has excellent reliability throughout the whole range of severity, offering an accurate evaluation of paranoia suitable for both non-clinical and clinical environments.

Quality of Life Measures

Recovering Quality of Life (ReQoL) by Keetharuth et al., 2018, is a concise assessment tool consisting of 10 items. It was developed to assess the quality of life of people with different mental health difficulties, with a specific emphasis on the rehabilitation process. All items are rated on a five-point Likert scale, from "none of the time" to "most or all of the time." The total score is determined by adding together the scores of all items, except the physical health item. A higher score indicates a superior quality of life. The intra-class correlation (ICC) for the ReQoL-10 measure was 0.85 ($P < 0.01$) for both the general population sample ($n = 488$) and the patient sample ($n = 279$), indicating a strong agreement in general mental health ratings across both groups. Additionally, the internal consistency, as measured by Cronbach's α, was found to be 0.96, indicating a high level of reliability (Keetharuth et al., 2018).

The SIMPAQ (Simple Physical Activity Questionnaire) by Rosenbaum, 2020, is a short questionnaire consisting of five items. It was developed by a diverse global team of experts as a therapy instrument for assessing physical activity and sedentary behaviour in individuals with mental illnesses. The 5-item tool involves interviewing people about several elements of their physical activity in the last seven days. This comprises time spent in bed during the

night, time spent inactive (including sleeping), time spent walking, time spent exercising, and time involved in incidental activity. The total hours entered in the five SIMPAQ boxes should total about 24 hours. For this current study, an adapted version of the SIMPAQ was used. We did not ask participants about the time they spent on incidental activities. The SIMPAQ has strong test-retest reliability, with a Spearman rho coefficient of roughly 0.70. The criterion validity of moderate-to-vigorous physical activity is comparable to that of research conducted with samples from the general population (Rosenbaum, 2020). Appendix C contains complete versions of each outcome measure.

Analysis

Participants were classified into the hypersomnia group if they met the following criteria based on the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; American Psychiatric Association, 2013) and the International Classification of Sleep Disorders, Third Edition (ICSD-3; American Academy of Sleep Medicine, 2014); they reported excessive sleepiness at least three days per week, and either (a) slept more than nine hours per night over the past month or (b) averaged 11 or more hours of total sleep in a 24-hour period over the past month.

The analyses were conducted using the statistical software Jamovi (2023). A between-subjects t-test, or in some instances, a Mann-Whitney U test (for non-parametric data) was employed for continuous variables, while Chi-square analysis was applied for categorical data to compare the study measures between the two groups. The testing adopted a two-tailed approach with a predetermined significance level of $p \leq 0.05$.

Patient and Public Participation (PPI)

Patient and public participation (PPI) involves conducting research collaboratively with patients and/or the general public (INVOLVE, 2018). The engagement of patients and the

public can contribute to making research more patient-centred and relevant to a broader population (Hahn et al., 2017). Consequently, contact was established with the psychosis lived experience group in the NHS Trust to seek advice regarding the selection of measures and the study process. We decided not to use full sleep diaries or activity monitoring after consulting with PPI. We also modified the language on the information sheets to enhance accessibility and minimise the use of technical jargon.

Ethical Approval

The study received ethical approval from the London Bloomsbury Research Ethics Committee (REC) and reference number 23/LO/0085 (Appendix G). The University of East Anglia acted as the study sponsor.

Results

Question (Q) 1: Are there differences in demographic (age, gender, employment) or clinical features (diagnosis, medication status, medication dosage) between the hypersomnia and comparison groups?

Table 1 summarises the demographic and clinical findings. Statistically significant differences in gender and age were observed between groups ($p<0.05$) with a greater percentage of male participants who were also younger in the hypersomnia group (n=11, 45.8%) versus the comparison group (n=7, 21.2%). There were no differences in terms of ethnicity, level of education completed, occupation, or diagnosis between the hypersomnia and comparison groups. There were no significant distinctions in medication types or dosages between the two study groups. The hypersomnia group was prone to being administered fewer antipsychotics and other medications (n=10, 43.5%) than the comparison group (n=16, 48.5%) and at slightly higher CPZ dosages (mean difference =35), but these differences did not reach significance.

Q2: Are sleep-related difficulties more severe in the hypersomnia group compared to the comparison group?

In the study, 37 participants, accounting for 64.9% of the sample, reported experiencing excessive sleepiness during the day to the extent that it was challenging to stay awake or engage in activities. Additionally, 33 participants, comprising 57.8% of the sample, reported sleeping for over 9 hours per night. Twenty-four participants, constituting 42.1% of the sample, reported sleeping for over 11 hours in a 24-hour period (i.e., including naps).

The findings from sleep measures are presented in Table 2. The hypersomnia group exhibited significantly higher scores on the sleepiness and hypersomnia scales ($p<0.001$). Additionally, they indicated increased fatigue and tiredness relative to the comparison group. The degree of

insomnia did not, however, differ significantly between the groups. There were no significant differences in nightly sleep duration between the two groups. However, significant differences were observed between groups regarding the frequency of sleepiness, with the hypersomnia group reporting approximately 5.25 days a week when excessive sleepiness is dominant ($p<0.001$), compared to 1.82 days per week in the comparison group.

Table 1. *Demographic and clinical differences between the hypersomnia group and comparison group*

	Hypersomnia (n=24) (57.9%)	Comparison group (n=33) (42.1%)	Difference statistic [a]	p-value	Effect size (d)	95% Confidence Interval	
Gender-n (%)			3.90	0.04			
Male	11 (45.8)	7 (21.2)				**Lower**	**Upper**
Female	13 (54.2)	26 (78.8)					
Age-average years (SD)	33.1 (8.62)	40.6 (10.6)	2.85	0.006	0.76	0.21	1.30
Occupational status-n (%)							
Unemployed	15 (25.0)	15 (45.5)	7.30	0.19			
Employed	16 (66.6)	13 (39.4)					
Homemaker/Student/Retired	8 (8.3)	5 (15.2)					
Ethnicity-n (%)			0.83	0.66			
Any White Background	22 (91.7)	30 (90.9)					
BAME	2 (8.3)	3 (9.1)					
Level of education completed n (%)			1.95	0.37			
Primary education	0	2 (6.1%)					
Secondary education	9 (37.5%)	9 (27.3%)					
Tertiary education	15 (62.5%)	22 (66.7%)					
Diagnosis-n%			3.83	0.14			
Schizophrenia	10 (41.7)	6 (18.2)					
SZA/SPD/DD [b]	4 (16.6)	7 (21.2)					
PNOS (including FEP) [c]	10 (41.6)	20 (60.6)					
Medications prescribed n (%)			0.57	0.74			
Antipsychotics only	6 (26.1)	6 (18.2)					
Other medication [d]	3 (13.0)	3 (9.1)					
Both antipsychotics +other medication	10 (43.5)	16 (48.5)					
Antipsychotic dosage–mean (SD) [e]							
Defined Daily Dose (DDD)	1.15 (0.77)	1.29 (0.7)	0.56	0.57	0.18	-0.47	0.84

Chlorpromazine equivalent (CPZ)	459 (428)	424 (302)	-0.28	0.77	-0.09	-0.76	0.57
Sleep							
Hours of sleep/night	8.67 (1.62)	7.58 (2.35)	285	0.07	0.28	-2.00	7.78
Sleepiness frequency (days/week)	5.25 (1.36)	1.82 (2.71)	141	< .001	0.64	-5.00	-3.00

[a] Chi-square statistic for gender, occupational status, ethnicity, diagnosis, and medications prescribed, t-statistic for age and antipsychotic dose, Mann Witney test for non-parametric data (sleep)
[b] Schizoaffective/schizotypal/delusional disorder
[c] Psychotic disorders not otherwise specified (including first episode psychosis)
[d] Includes antidepressants, anxiolytics, mood stabilisers, hypnotics
[e] Statistics only include participants prescribed antipsychotic medication i.e. n =16 in hypersomnia group, n =22 in comparison group

Table 2. *Sleep-related differences between hypersomnia and comparison groups*

	Hypersomnia	Comparison group	Difference statistic[a]	p-value	Effect size (d)	95% Confidence Interval	
	Mean (SD)	Mean (SD)				Lower	Upper
Sleepiness (ESS)	11.5 (3.66)	7.15 (5.77)	221	0.005	0.44	-7.00	- 2.00
Hypersomnia (HSI)	21.3 (4.41)	14.33 (6.89)	-4.31	< .001	-1.15	-10.13	- 3.70
Insomnia (ISI)	13.9 (4.29)	13.48 (6.03)	378	0.77	0.04	- 4.00	3.00
Fatigue (FSS)	44.0 (8.97)	36.09 (15.41)	271	0.04	0.31	- 17.00	- 2.44
Tiredness (ITI)	20.9 (4.53)	15.06 (7.50)	212	0.003	0.46	- 9.00	- 2.00

[a] Mann-Witney for ESS, ISI, FSS, ITI as Levene's test was significant ($p < .05$), suggesting a violation of the assumption of equal variances, and t statistic for HSI

Q3: Does the group experiencing hypersomnia report heightened psychiatric symptoms as well as a lower quality of life and reduced physical activity in comparison to the comparison group?

The measures focused on psychiatric symptoms are displayed in Table 3. We detected no significant differences between the study groups on any psychiatric symptoms or general functioning measures. Examination of confidence intervals and effect sizes, where effect sizes are higher than 0.3, suggest that hallucinations, paranoia, and depression are more severe among individuals reporting hypersomnia. However, this finding needs to be interpreted with caution due to its lack of significance.

Anxiety and quality of life appeared to be more severely affected in the hypersomnia group. Additionally, individuals in the hypersomnia group exhibited higher levels of time spent in bed and walking, while those in the comparison group had higher levels of sedentary time and exercise. Notably, we observed medium effect sizes (greater than 0.5) for both bedtime and sedentary time. However, we advise caution in interpreting these findings, as the differences between groups did not reach statistical significance.

Table 3. *Symptoms differences between groups*

Symptom	Hypersomnia	Comparison group	Difference statistic[a]	p-value	Effect size (d)	95% Confidence Interval	
	Mean (SD)	Mean (SD)				Lower	Upper
Psychotic experiences (SPEQ, R-GPTS)							
Hallucinations	20.58 (12.27)	15.27 (13.43)	-1.52	0.13	-0.41	-12.27	1.65
Reference	15.75 (10.50)	12.24 (9.10)	-1.34	0.18	-0.36	-8.72	1.71
Persecution	18.08 (12.77)	14.03 (10.95)	-1.28	0.20	-0.34	-10.37	2.26
Negative affect (DASS)							
Depression	13.70 (4.40)	11.30 (5.67)	-1.73	0.08	-0.46	-5.19	0.37
Anxiety	9.16 (4.54)	8.57 (5.07)	-0.45	0.65	-0.12	-3.20	2.02
Quality of life (ReQoL)	17.75 (6.34)	19.12 (6.77)	0.77	0.44	0.20	-2.17	4.91
Physical activity (SIMPAQ) [b]							
Time in bed	10.19 (1.80)	8.57 (2.41)	-1.95	0.06	-0.73	-3.30	0.07
Sedentary time	4.68 (4.40)	7.32 (5.20)	1.44	0.16	0.53	-1.10	6.38
Time spent in walking	1.59 (2.26)	0.80 (0.66)	96.0	0.80	0.05	-1.00	0.57
Time spent in exercise	0.40 (0.81)	0.50 (0.98)	0.31	0.75	0.11	-0.55	0.76

[a] Mann-Witney for "Time spent walking" as Levene's test was significant ($p < .05$), suggesting a violation of the assumption of equal variances, and t statistic for the rest of domains

[b] Statistics only include participants who completed SIMPAQ, each domain referred to hours/day per activity

Discussion

This cross-sectional study aimed to further our understanding of the frequency, impact, and experience of excessive sleepiness in people with psychotic disorders. The study revealed statistically significant differences in both gender and age between the groups, with a higher representation of younger male participants in the hypersomnia group. there were no indicated differences in medication dose or type between the groups, despite this being the main assumed source of hypersomnia symptoms (via sedative effects). There were also no significant differences in clinical symptoms (depression, paranoia, hallucinations), although the differences that were observed all indicated greater severity in the hypersomnia group. Furthermore, there were no significant differences found between the study groups regarding quality of life and physical activity, despite the hypersomnia group indicating higher severity in quality of life and spending more time in bed. One potential explanation is that the comparison group also reported high levels of hypersomnia symptoms, which could have mitigated the observable differences between the groups.

The hypersomnia group had a greater percentage of male participants and was, on average, younger than the comparator cohort. This corresponds with other studies indicating that hypersomnia symptoms are more prevalent among younger males (Pallesen et al., 2007); however, additional research has shown inconclusive findings regarding gender differences and indicated that women reported significantly higher levels of hypersomnia than men (Liu et al., 2000). No significant differences were seen in ethnicity, educational attainment, work position, or clinical diagnosis between the two groups, indicating that hypersomnia symptoms in psychosis are not inherently affected by sociodemographic variables.

The high incidence of co-occurring insomnia symptoms was an interesting discovery in this study, with 51% of participants (29 out of 57) meeting the clinical threshold for insomnia (ISI > 15). This underscores the intricacy of sleep disruptions in psychosis, as it is possible for

both excessive daytime sleepiness and difficulty initiating sleep to coexist. Although high ISI scores indicate inadequate sleep quality, it is crucial to emphasise that the ISI is designed to evaluate the effects of insomnia-related symptoms on one's ability to function. ISI items were presented on a distinct page from hypersomnia and other sleep-related queries in this study, which helped reduce confusion and ensured greater measurement clarity. This design is consistent with previous research that utilised the ISI in conjunction with other sleep measures, and there is no indication of misinterpretation **(**Cheung et al., 2024). The overlap between insomnia and hypersomnia is in line with prior research in psychosis and depression, where multiple forms of sleep disturbance frequently co-occur together (Freeman et al., 2020). These results underscore the significance of evaluating and treating sleep difficulties in this population by taking into account the combined impact of hypersomnia and insomnia on daily functioning.

Regarding medication use, the hypersomnia group appeared to receive fewer antipsychotic and other medications, but at slightly higher CPZ-equivalent doses, with the average difference being approximately 1.4 mg of olanzapine. Previous research indicates that second-generation antipsychotics, especially olanzapine and clozapine, are significantly linked to excessive sleepiness and hypersomnia (Cohrs, 2008; Krystal et al., 2008). The lack of an indicated difference in medication use across the groups indicates that hypersomnia in psychosis may be linked to illness-related factors rather than just pharmacological effects. This information corresponds with research indicating that antipsychotic treatment is a recognised contributor to excessive sleepiness in individuals with psychosis. Reeve et al. (2021) highlighted that excessive sleepiness is not only due to drug effects but is also associated with general activity levels and the existence of sleep problems.

The hypersomnia cohort had markedly elevated scores on sleepiness and hypersomnia metrics ($p<0.001$) and reported increased fatigue and tiredness relative to the comparison

group. These results corroborate current research that demonstrates a robust association between hypersomnia and excessive sleepiness, diminished alertness, and heightened fatigue in patients with psychotic illnesses (Reeve et al., 2019; Reeve et al., 2021). Notably, despite these subjective complaints, there were no significant differences in total nightly sleep duration or insomnia severity between the groups, indicating that hypersomnia may not be inherently associated with an increased amount of sleep but rather with the quality of wakefulness. A significant finding indicated that the hypersomnia group had excessive sleepiness on roughly 5.25 days per week, in contrast to 1.82 days in the comparison group ($p<0.001$). This conclusion corroborates other studies indicating that people with psychotic illnesses often suffer from hypersomnia symptoms (Laskemoen et al., 2019; Waite et al., 2020).

No statistically significant differences were detected between the groups for clinical symptoms or overall functioning. Nonetheless, effect sizes indicated patterns of heightened hallucinations, paranoia, and depression in the hypersomnia group, aligning with prior research that associates excessive sleepiness with exacerbated psychiatric symptoms in psychotic diseases (Reeve et al., 2021). Although these results did not achieve statistical significance, they correspond with studies indicating a bidirectional association between sleep disorders and mental health, whereby hypersomnia may aggravate mood symptoms and vice versa (Wulff et al., 2012; Formica et al., 2023). This association is well reported in affective disorders, where hypersomnia is often associated with increased severity of depression and suicidal thoughts (Tsuno et al., 2005). Regarding quality of life, people in the hypersomnia group reported elevated anxiety levels and diminished general well-being. The results corroborate prior studies demonstrating that excessive sleepiness correlates with poorer quality of life, depression, and anxiety in psychotic illnesses (Reeve et al., 2019). The hypersomnia group allocated more time in bed and walking, whereas the comparison group

participated in more sedentary behaviour and organised exercise. Medium effect sizes (>0.5) for time in bed and sedentary duration indicate substantial differences; however, they did not achieve statistical significance. The findings align with previous research suggesting that hypersomnia correlates with altered activity patterns, whereby patients tend to participate in more passive movements while facing difficulties with organised physical exercise (Reeve et al., 2021). Considering that physical inactivity is a major risk factor for metabolic problems in adults with mental illnesses (Vancampfort et al., 2015), treating sleep difficulties may be key to encouraging healthy exercise habits in this demographic.

Study Strengths

Firstly, the research established a specific threshold for hypersomnia, based on DSM-5 and ICSD-3 diagnostic criteria, to accurately identify people with hypersomnia in a consistent and standardised manner. This improved the reliability and validity of the results, enabling more precise comparisons and analyses.

Secondly, we engaged PPI contributors throughout the study's design phase. Although this engagement was limited, integrating insights from people with lived experience enhanced the study's focus and assured increased relevance to the target demographic. Despite the small degree of participation, first contributions from experienced individuals may enhance the relevance and sensitivity of study design (Lindenmeyer et al., 2007; Thompson et al., 2009). The method may empower the engaged public and help individuals find meaning in challenging events (Patterson et al., 2014; Simpson et al., 2014).

Study Limitations

We conducted a pre-study power analysis, which revealed that a sample size of 80 participants was required to obtain sufficient statistical power (80% power at $\alpha = 0.05$) to detect the expected effect size. Nevertheless, the study was unable to detect the anticipated

magnitude of effects since only 60 participants were enrolled, which was a result of recruitment limitations. However, we were only able to recruit 60 participants, meaning the study was underpowered to detect the specified effect size. Additionally, the online study design prevented targeted recruitment to balance group sizes. While several potential reasons could explain the non-significant results observed in this study, it is important to note that a likely reason is the limited statistical power due to the smaller-than-planned sample size. This consequently restricted our ability to detect smaller but potentially meaningful effects. As a result, our sample only had sufficient power to detect large effects, which may explain the lack of significant findings. Due to limitations in sample size and sensitivity, the study incorporated confidence intervals and effect sizes as additional markers to assess meaningful changes between the study groups.

The lack of statistical significance in certain analyses may have been influenced by sample size, methodological considerations, or a lack of heterogeneity regarding sleepiness between groups. A methodological concern pertains to the absence of Bonferroni corrections or similar adjustments for multiple comparisons in this research. Although such adjustments are often used to mitigate the likelihood of Type I errors in multiple testing scenarios, their implementation was considered superfluous in this instance, given none of the statistical tests produced significant findings before modification. Nonetheless, it is crucial to emphasise that the results remained nonsignificant even in the absence of these adjustments. This openness guarantees clarity in interpreting the data and emphasises the strength of the indicated absence of significance. Subsequent research using bigger sample sizes or other approaches may further investigate the possibility of significant connections between excessive sleepiness and clinical symptoms.

Another limitation of our study is the potential lack of generalisability due to the predominantly white sample. Data on these groups is necessary to avoid gaps in health

outcomes, access to healthcare, and treatment effectiveness that may otherwise remain unnoticed and unattended. Minority groups may have distinct health obstacles, cultural influences, and societal issues affecting health that need tailored interventions. Additionally, these groups may experience less-than-optimal health outcomes as a result of the creation and assessment of culturally appropriate and efficient therapies for hypersomnia.

The study relied solely on self-reported measures for assessing sleepiness. The absence of objective measures, such as actigraphy or polysomnography, introduces a potential limitation. Utilising objective measures, such as the Multiple Sleep Latency Test (MSLT) and continuous polysomnography (PSG) recordings, would provide a more comprehensive and accurate evaluation of sleepiness, reducing potential biases, such as memory recall bias or social desirability bias. Furthermore, while subjective measurements may reflect changes in people's views and experiences of sleepiness and sleep quality, objective measures could also be valuable for long-term monitoring of sleep patterns.

Furthermore, the study's cross-sectional design limits our ability to establish causal relationships. Longitudinal studies would provide a more dynamic understanding of the relationship between excessive sleepiness, psychopathological symptoms, and quality of life over time.

Clinical Implications

Excessive sleepiness is an area that has been understudied. The findings indicate the importance of creating precise definitions and methods to measure excessive sleepiness in psychosis. Moreover, involving service users in this process can help understand how they perceive oversleeping and its impact on them. Therefore, evaluating the patient's awareness of excessive sleepiness, comprehension of treatment choices, and outlook on living with it might provide a chance to provide necessary information (Ong et al., 2022).

Prior research has shown evidence supporting the use of behavioural methods to decrease both perceived and measured sleepiness and enhance functional performance (Helmus et al., 1997); Mullington & Broughton, 1993; Rogers et al., 2001. A typical behavioural approach for managing excessive sleepiness is to schedule afternoon naps in advance to enhance alertness and cognitive performance. Research also investigated the effects of implementing a regular nighttime sleep routine with or without planned naps (Rogers et al., 2001). Manuals have emphasised the need to provide consistent light exposure every morning to effectively synchronise the circadian rhythm (Wirz-Justice et al., 2013). Providing light in the morning is beneficial for addressing challenges with waking up, especially for those with circadian rhythm delay, hypersomnia, or sedation in the morning due to medication. Additionally, behavioural experiments can help patients understand that light may enhance levels of alertness and vitality, which can be an effective method for napping in the afternoon (Sheaves et al., 2018).

The combined burden of both insomnia and hypersomnia in this sample may have substantial clinical consequences. Persistent insomnia may contribute to heightened stress, increased sensitivity to mood disorders, and worsening of psychotic symptoms, while hypersomnia and excessive sleepiness can lead to poor cognitive performance, diminished motivation, and difficulty with everyday tasks. The interaction between these sleep disorders might potentially affect overall quality of life, functional results, and even treatment response (Reeve et al., 2019). Given these possible repercussions, a more comprehensive knowledge of sleep disorders in psychosis is required, particularly how these symptoms interact, whether they reflect discrete subtypes of sleep dysfunction, and how best to tailor therapies.

Furthermore, the prevalence of increased sedentary behaviour in the sample indicates a need for specific treatments to enhance daily functioning, with a clear emphasis on addressing clinical symptoms and daily functioning related to sedentary conduct. Implementing new

strategies to decrease sedentary behaviours, along with treatments focused on knowledge and skills, might provide significant advantages for daily functioning and health outcomes (Strassnig et al., 2012).

Research implications

Future studies need to include more longitudinal study designs. Most of the current research is cross-sectional, restricting the capacity to determine causation among hypersomnia, mental symptoms, and quality of life. Longitudinal studies would enable researchers to monitor changes in sleep patterns and psychiatric symptoms over time, elucidating whether hypersomnia exacerbates mental health issues or whether it is a byproduct of preexisting psychiatric disorders. Moreover, such research might examine whether targeted therapies for hypersomnia, like behavioural sleep interventions, medication modifications, or cognitive-behavioural therapy for insomnia (CBT-I), result in enhancements in psychiatric symptoms and general well-being.

Furthermore, more research should investigate the efficacy of psychological therapies in alleviating hypersomnia symptoms. Investigating cognitive behavioural therapy for hypersomnia (CBT-H) with other psychotherapy methods, including mindfulness-based treatments and behavioural activation, may provide significant insights into non-pharmacological treatment techniques. Considering the significant correlation among hypersomnia, depression, and diminished quality of life, psychological therapies may provide a viable strategy for enhancing daytime functioning, motivation, and overall mental health outcomes.

Further research also needs to investigate the biological factors that contribute to hypersomnia in patients with psychotic illnesses. Examining neurobiological elements, including modifications in neurotransmitter systems (e.g., dopamine, serotonin, and orexin)

and circadian rhythm disturbances, may elucidate the pathophysiology of hypersomnia and guide the formulation of more efficacious treatment approaches. Comprehending these processes may aid in distinguishing hypersomnia as a separate symptom domain instead of an overlapping characteristic of other mental disorders.

Future research must prioritise the recruitment of larger and more varied samples to improve the generalisability of the results. Contemporary research often depends on very small, homogeneous cohorts, which may inadequately represent the heterogeneity of hypersomnia symptoms among diverse populations. Enhancing the variety of research participants, specifically regarding ethnicity, socioeconomic status, and clinical history, will facilitate the identification of possible cultural and environmental factors influencing hypersomnia and its related symptoms.

Moreover, although self-reported measures of sleepiness provide significant subjective insights and are prevalent in sleep research, the inclusion of objective sleep assessments such as polysomnography, actigraphy, or the MSLT would enhance the reliability and validity of the findings. Integrating these methodologies would provide a more thorough assessment of sleep patterns and their underlying processes, thereby improving comprehension of hypersomnia in further studies.

Addressing these gaps will enable future research to enhance the knowledge of hypersomnia in psychotic illnesses, hence enhancing diagnosis, treatment, and overall patient outcomes.

Conclusion

These findings focused on the differences in excessive sleepiness, psychiatric symptoms, and quality of life in individuals with psychosis. Although we observed some significant differences between groups, further investigation is necessary due to the nuanced nature of these variables and their directions of effect. This study contributes to the ongoing discourse in the field, providing valuable insights for clinicians and researchers working towards a more comprehensive understanding of hypersomnia in the context of psychosis. There is growing evidence suggesting that sleep disturbance plays a role in causing psychosis (Reeve et al., 2015; Freeman et al., 2017; Reeve et al., 2018). Enhancing sleep may potentially help with psychosis, offering a promising new therapy focus. Regardless of any connection to psychotic experiences, evaluating and addressing hypersomnia may be crucial for treating psychotic episodes, mood, and overall wellbeing in people with psychosis.

By directing future research efforts and influencing clinical approaches, these findings add to the ongoing discussion about the relationship between hypersomnia and mental health in the early stages of psychosis. For instance, further research could help develop a cognitive-behavioural programme to address hypersomnia in people with psychosis, which may lead to symptom reduction and improved quality of life.

References

Afonso, P., Figueira, M. L., & Paiva, T. (2014). Sleep-wake patterns in schizophrenia patients compared to healthy controls. *The world journal of biological psychiatry: the official journal of the World Federation of Societies of Biological Psychiatry, 15*(7), 517–524. https://doi.org/10.3109/15622975.2012.756987

American Psychiatric Association. (2013). Cautionary statement for forensic use of DSM-5. In *Diagnostic and statistical manual of mental disorders* (5th ed., p. 280). American Psychiatric Publishing.

American Academy of Sleep Medicine. (2014). *International Classification of Sleep Disorders* (3rd ed.). American Academy of Sleep Medicine

Barrett, E. A., Aminoff, S. R., Simonsen, C., & Romm, K. L. (2020). Opening the curtains for better sleep in psychotic disorders - considerations for improving sleep treatment. *Comprehensive psychiatry, 103*, 152207. https://doi.org/10.1016/j.comppsych.2020.152207

Bowie, C. R., & Harvey, P. D. (2006). Cognitive deficits and functional outcome in schizophrenia. *Neuropsychiatric Disease and Treatment, 2*(4), 531–536

Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. *Sleep, 33*(5), 585–592. https://doi.org/10.1093/sleep/33.5.585

Cheung, M. M. S., Lam, S. P., Chau, S. W. H., Chan, N. Y., Li, T. M. H., Wing, Y. K., & Chan, J. W. Y. (2024). Hypersomnolence is associated with non-remission of major depressive disorder. *Sleep Medicine, 119*, 35–43.

Cho, Y. W., Lee, J. H., Son, H. K., Lee, S. H., Shin, C., & Johns, M. W. (2011). The reliability and validity of the Korean version of the Epworth sleepiness scale. *Sleep & breathing = Schlaf & Atmung, 15*(3), 377–384. https://doi.org/10.1007/s11325-010-0343-6

Chouinard, S., Poulin, J., Stip, E., & Godbout, R. (2004). Sleep in untreated patients with schizophrenia: a meta-analysis. *Schizophrenia bulletin*, *30*(4), 957–967. https://doi.org/10.1093/oxfordjournals.schbul.a007145.

Cohrs, S. (2008). Sleep disturbances in patients with schizophrenia. *CNS Drugs, 22*(11), 939–962.

Dauvilliers, Y., Lopez, R., Ohayon, M., & Bayard, S. (2013). Hypersomnia and depressive symptoms: methodological and clinical aspects. *BMC medicine, 11*, 78. https://doi.org/10.1186/1741-7015-11-78

Fatouros-Bergman, H., Cervenka, S., Flyckt, L., Edman, G., & Farde, L. (2014). Meta-analysis of cognitive performance in drug-naïve patients with schizophrenia. *Schizophrenia Research, 158*(1-3), 156–162.

Faul, F., Erdfelder, E., Buchner, A., & Lang, A. G. (2009). Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. *Behaviour research methods, 41*(4), 1149–1160. https://doi.org/10.3758/BRM.41.4.1149

Faulkner, S., Bee, P. (2017). Experiences, perspectives and priorities of people with schizophrenia spectrum disorders regarding sleep disturbance and its treatment: a qualitative study. *BMC psychiatry, 17*(1), 158. https://doi.org/10.1186/s12888-017-1329-8

Fervaha, G., Foussias, G., Agid, O., & Remington, G. (2014). Motivational and neurocognitive deficits are central to negative symptoms of schizophrenia. *Schizophrenia Research, 159*(2-3), 501–508.

Formica, M. J. C., Fuller-Tyszkiewicz, M., Hickie, I., Olive, L., Wood, S. J., Purcell, R., Yung, A. R., Phillips, L. J., Nelson, B., Pantelis, C., McGorry, P. D., & Hartmann, J. A. (2023). The relationship between subjective sleep disturbance and attenuated

psychotic symptoms after accounting for anxiety and depressive symptoms. *Schizophrenia research*, *258*, 84–93. https://doi.org/10.1016/j.schres.2023.07.019

Freeman, D. (2016). Persecutory delusions: a cognitive perspective on understanding and treatment. *The lancet. Psychiatry, 3*(7), 685–692. https://doi.org/10.1016/S2215-0366(16)00066-3

Freeman, D., Sheaves, B., Goodwin, G. M., Yu, L. M., Nickless, A., Harrison, P. J., Emsley, R., Luik, A. I., Foster, R. G., Wadekar, V., Hinds, C., Gumley, A., Jones, R., Lightman, S., Jones, S., Bentall, R., Kinderman, P., Rowse, G., Brugha, T., Blagrove, M., … Espie, C. A. (2017). The effects of improving sleep on mental health (OASIS): a randomised controlled trial with mediation analysis. *The lancet. Psychiatry, 4*(10), 749–758. https://doi.org/10.1016/S2215-0366(17)30328-0

Freeman, D., Loe, B., Kingdon, D., Startup, H., Molodynski, A., Rosebrock, L., . . . Bird, J.C. (2019). The revised Green et al., Paranoid Thoughts Scale (R-GPTS): Psychometric properties, severity ranges, and clinical cut-offs. *Psychological Medicine, 1*-10. https://doi.org/10.1017/S0033291719003155

Freeman, D., Sheaves, B., Waite, F., Harvey, A. G., & Harrison, P. J. (2020). Sleep disturbance and psychiatric disorders. *The lancet. Psychiatry, 7*(7), 628–637. https://doi.org/10.1016/S2215-0366(20)30136-X

Gibson, E. S., Powles, A. C., Thabane, L., O'Brien, S., Molnar, D. S., Trajanovic, N., Ogilvie, R., Shapiro, C., Yan, M., & Chilcott-Tanser, L. (2006). "Sleepiness" is serious in adolescence: two surveys of 3235 Canadian students. *BMC public health, 6*, 116. https://doi.org/10.1186/1471-2458-6-116

Hagell, P., & Broman, J. E. (2007). Measurement properties and hierarchical item structure of the Epworth Sleepiness Scale in Parkinson's disease. *Journal of sleep research, 16*(1), 102–109. https://doi.org/10.1111/j.1365-2869.2007.00570.x

Hahn, D. L., Hoffmann, A. E., Felzien, M., LeMaster, J. W., Xu, J., & Fagnan, L. J. (2017). Tokenism in patient engagement. *Family practice, 34*(3), 290–295. https://doi.org/10.1093/fampra/cmw097

Helmus, T., Rosenthal, L., Bishop, C., Roehrs, T., Syron, M. L., & Roth, T. (1997). The alerting effects of short and long naps in narcoleptic, sleep deprived, and alert individuals. *Sleep, 20*(4), 251–257. https://doi.org/10.1093/sleep/20.4.251

INVOLVE. (2018). *National Standards for Public Involvement*. Available at: https://drive.google.com/file/d/1MRzOnq_dXTU96vWfwpe5-wkYBa8mUu3o/view

Izci, B., Ardic, S., Firat, H., Sahin, A., Altinors, M., & Karacan, I. (2008). Reliability and validity studies of the Turkish version of the Epworth Sleepiness Scale. *Sleep & breathing = Schlaf & Atmung, 12*(2), 161–168. https://doi.org/10.1007/s11325-007-0145-7

Jamovi project. (2023). *Jamovi* (Version 2.3) [Computer Software]. Retrieved from https://www.jamovi.org

Johns, M. W. (1992). Reliability and factor analysis of the Epworth Sleepiness Scale. *Sleep*, *15*(4), 376-381. https://doi.org/10.1093/sleep/15.4.376

Kaplan, K. A., Harvey, A. G. (2009). Hypersomnia across mood disorders: a review and synthesis. *Sleep medicine reviews, 13*(4), 275–285. https://doi.org/10.1016/j.smrv.2008.09.001

Kaplan, K. A., Plante, D. T., Cook, J. D., & Harvey, A. G. (2019). Development and validation of the Hypersomnia Severity Index (HSI): A measure to assess

hypersomnia severity and impairment in psychiatric disorders. *Psychiatry research, 281*, 112547. https://doi.org/10.1016/j.psychres.2019.112547

Keetharuth, A. D., Brazier, J., Connell, J., Bjorner, J. B., Carlton, J., Taylor Buck, E., Ricketts, T., McKendrick, K., Browne, J., Croudace, T., & Barkham, M. (2018). Recovering Quality of Life (ReQoL): a new generic self-reported outcome measure for use with people experiencing mental health difficulties. *The British journal of psychiatry: The journal of mental science, 212*(1), 42–49. https://doi.org/10.1192/bjp.2017.10

Kluge, M., Himmerich, H., Wehmeier, P. M., Rummel-Kluge, C., Dalal, M., Hinze-Selch, D., Kraus, T., Dittmann, R. W., Pollmächer, T., & Schuld, A. (2012). Sleep propensity at daytime as assessed by Multiple Sleep Latency Tests (MSLT) in patients with schizophrenia increases with clozapine and olanzapine. *Schizophrenia research, 135*(1-3), 123–127. https://doi.org/10.1016/j.schres.2011.12.017

Krystal, A. D, Goforth, H. W., Roth, T. (2008). Effects of antipsychotic medications on sleep in schizophrenia. *Int Clin Psychopharmacol, May;23*(3):150-60. https://doi.org/10.1097/YIC.0b013e3282f39703

Krupp, L. B., LaRocca, N. G., Muir-Nash, J., & Steinberg, A. D. (1989). The fatigue severity scale. Application to patients with multiple sclerosis and systemic lupus erythematosus. *Archives of Neurology*, *46*, 1121–1123.

Laskemoen, J. F., Simonsen, C., Büchmann, C., Barrett, E. A., Bjella, T., Lagerberg, T. V., Vedal, T. J., Andreassen, O. A., Melle, I., & Aas, M. (2019). Sleep disturbances in schizophrenia spectrum and bipolar disorders - a transdiagnostic perspective. *Comprehensive psychiatry, 91*, 6–12. https://doi.org/10.1016/j.comppsych.2019.02.006

Lawrence, D., Hancock, K. J., & Kisely, S. (2013). The Gap in Life Expectancy from Preventable Physical Illness in Psychiatric Patients in Western Australia: Retrospective Analysis of Population Based Registers. *BMJ, 346*, 1. https://doi.org/10.1136/bmj.f2539

Lindenmeyer, A., Hearnshaw, H., Sturt, J., Ormerod, R., & Aitchison, G. (2007). Assessment of the benefits of user involvement in health research from the Warwick Diabetes Care Research User Group: a qualitative case study. *Health expectations: an international journal of public participation in health care and health policy, 10*(3), 268–277. https://doi.org/10.1111/j.1369-7625.2007.00451.x

Liu, X., Uchiyama, M., Kim, K., Okawa, M., Shibui, K., Kudo, Y., Doi, Y., Minowa, M., & Ogihara, R. (2000). Sleep loss and daytime sleepiness in the general adult population of Japan. *Psychiatry research, 93*(1), 1–11. https://doi.org/10.1016/s0165-1781(99)00119-5

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales (*2nd ed.). Psychology Foundation of Australia.

Martin, J., Jeste, D. V., Caliguiri, M. P., Patterson, T., Heaton, R., & Ancoli-Israel, S. (2001). Actigraphic estimates of circadian rhythms and sleep/wake in older schizophrenia patients. *Schizophrenia research, 47*(1), 77–86. https://doi.org/10.1016/s0920-9964(00)00029-3

Miller, D. D. (2004). Atypical antipsychotics: sleep, sedation, and efficacy. Primary care companion to the *Journal of clinical psychiatry, 6*(Suppl 2), 3–7.

Monti, J. M., & Monti, D. (2005). Sleep disturbance in schizophrenia. International *Review of Psychiatry, 17*(4), 247-253. https://doi.org/10.1080/09540260500104516

Morin, C. M., Belleville, G., Bélanger, L., & Ivers, H. (2011). The Insomnia Severity Index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep, 34*(5), 601–608. https://doi.org/10.1093/sleep/34.5.601

Mullington, J., Broughton, R. (1993). Scheduled naps in the management of daytime sleepiness in narcolepsy-cataplexy. *Sleep*, 16(5), 444–456. https://doi.org/10.1093/sleep/16.5.444

Ong, J.C., Schuiling, M. D. (2022). CBT-I for patients with hypersomnia disorders. In S. Nowakowski, S. N., Garland, M. A. Grandner, J. Leisha, L. J. Cuddihy (Eds). *Adapting Cognitive Behavioural Therapy for Insomnia*. Academic Press. https://doi.org/10.1016/B978-0-12-822872-2.00018-9

Pallesen, S., Nordhus, I. H., Omvik, S., Sivertsen, B., Tell, G. S, Bjorvatn, B. (2007). Prevalence and risk factors of subjective sleepiness in the general adult population. Sleep. May;30(5):619-24. https://doi.org/10.1093/sleep/30.5.619

Patterson, S., Trite, J., Weaver, T. (2014). Activity and views of service users involved in mental health research: UK survey. *British Journal of Psychiatry, 205*, 68-75

Pérez-Carbonell, L., & Leschziner, G. (2018). Clinical update on central hypersomnias. *Journal of thoracic disease, 10*(Suppl 1), S112–S123. https://doi.org/10.21037/jtd.2017.10.161

Poulin, J., Chouinard, S., Pampoulova, T., Lecomte, Y., Stip, E., & Godbout, R. (2010). Sleep habits in middle-aged, non-hospitalized men and women with schizophrenia: a comparison with healthy controls. *Psychiatry research, 179*(3), 274–278. https://doi.org/10.1016/j.psychres.2009.08.009

Reeve, S., Sheaves, B., & Freeman, D. (2015). The role of sleep dysfunction in the occurrence of delusions and hallucinations: A systematic review. *Clinical psychology review, 42*, 96–115. https://doi.org/10.1016/j.cpr.2015.09.001

Reeve, S., Emsley, R., Sheaves, B., & Freeman, D. (2018). Disrupting Sleep: The Effects of Sleep Loss on Psychotic Experiences Tested in an Experimental Study with Mediation Analysis. *Schizophrenia bulletin, 44*(3), 662–671. https://doi.org/10.1093/schbul/sbx103

Reeve, S., Sheaves, B., & Freeman, D. (2019). Sleep Disorders in Early Psychosis: Incidence, Severity, and Association with Clinical Symptoms. *Schizophrenia bulletin*, *45*(2), 287–295. https://doi.org/10.1093/schbul/sby129

Reeve, S., Sheaves B., Freeman, D. (2021). Excessive sleepiness in patients with psychosis: An initial investigation. *PLoS ONE 16*(1): e0245301. https://doi.org/10.1371/journal.pone.0245301

Rogers, A. E., Aldrich, M. S., & Lin, X. (2001). A comparison of three different sleep schedules for reducing daytime sleepiness in narcolepsy. *Sleep*, *24*(4), 385–391. https://doi.org/10.1093/sleep/24.4.385.

Ronald, A., Sieradzka, D., Cardno, A. G., Haworth, C. M. A., McGuire, P., & Freeman, D. (2014). Characterization of psychotic experiences in adolescence using the Specific Psychotic Experiences Questionnaire: Findings from a study of 5000 16-year-old twins. *Schizophrenia Bulletin, 40*(4), 868–877. https://doi.org/10.1093/schbul/sbt106

Rosenbaum, S., Morell, R., Abdel-Baki, A., Ahmadpanah, M., Anilkumar, T. V., Baie, L., Bauman, A., Bender, S., Boyan Han, J., Brand, S., Bratland-Sanda, S., Bueno-Antequera, J., Camaz Deslandes, A., Carneiro, L., Carraro, A., Castañeda, C. P., Castro Monteiro, F., Chapman, J., Chau, J. Y., Chen, L. J., … Ward, P. B. (2020). Assessing physical activity in people with mental illness: 23-country reliability and validity of the simple physical activity questionnaire (SIMPAQ). *BMC psychiatry, 20*(1), 108. https://doi.org/10.1186/s12888-020-2473-0

Sheaves, B., Isham, L., Bradley, J., Espie, C., Barrera, A., Waite, F., Harvey, A. G., Attard, C., & Freeman, D. (2018). Adapted CBT to Stabilize Sleep on Psychiatric Wards: a Transdiagnostic Treatment Approach. *Behavioural and cognitive psychotherapy, 46*(6), 661–675. https://doi.org/10.1017/S1352465817000789

Simpson, A., Jones, J., Barlow, S., Cox, L. (2014). Adding SUGAR service user and carer collaboration in mental health nursing research. *Journal of Psychosocial Nursing and Mental Health Services, 52*, 22-30

Strassnig, M., Caceda, R., Newcomer, J., Harvey, P. D. (2012). Cognitive deficits, obesity and disability in schizophrenia. *Translational Neuroscience*, *3*(4), 345–354. https://doi.org/10.2478/s13380-012-0046-5

Stubbs, B., Williams, J., Gaughran, F., & Craig, T. (2016). How sedentary are people with psychosis? A systematic review and meta-analysis. *Schizophrenia research, 171*(1-3), 103–109. https://doi.org/10.1016/j.schres.2016.01.034

Thompson, J., Barber, R., Ward, P. R., Boote, J. D., Cooper, C. L., Armitage, C. J. (2009). Health researchers' attitudes towards public involvement in health research. *Health Expectations, 12*, 209-220.

Trotti, L. M. (2017). Waking up is the hardest thing I do all day: Sleep inertia and sleep drunkenness. Sleep medicine reviews, 35, 76–84. https://doi.org/10.1016/j.smrv.2016.08.005

Tsuno, N., Besset, A., & Ritchie, K. (2005). Sleep and depression. *Journal of Clinical Psychiatry, 66*(10), 1254–1269. https://doi.org/10.4088/JCP.v66n1008

Vancampfort, D., Firth, J., Schuch, F. B., Rosenbaum, S., Mugisha, J., Hallgren, M., Probst, M., Ward, P. B., Gaughran, F., De Hert, M., Carvalho, A. F., & Stubbs, B. (2017). Sedentary behavior and physical activity levels in people with schizophrenia, bipolar

disorder and major depressive disorder: a global systematic review and meta-analysis. *World psychiatry: official journal of the World Psychiatric Association (WPA), 16*(3), 308–315. https://doi.org/10.1002/wps.20458

van der Heide, A., van Schie, M. K., Lammers, G. J., Dauvilliers, Y., Arnulf, I., Mayer, G., Bassetti, C. L., Ding, C. L., Lehert, P., & van Dijk, J. G. (2015). Comparing Treatment Effect Measurements in Narcolepsy: The Sustained Attention to Response Task, Epworth Sleepiness Scale and Maintenance of Wakefulness Test. *Sleep, 38*(7), 1051–1058. https://doi.org/10.5665/sleep.4810

Vorontsova, N., Garety, P., & Freeman, D. (2013). Cognitive factors maintaining persecutory delusions in psychosis: The contribution of depression. *Journal of Abnormal Psychology, 122*(4), 1121–1131. https://doi.org/10.1037/a0034952

Waite, F., Myers, E., Harvey, A. G., Espie, C. A., Startup, H., Sheaves, B., & Freeman, D. (2016). Treating Sleep Problems in Patients with Schizophrenia. *Behavioural and cognitive psychotherapy*, *44*(3), 273–287. https://doi.org/10.1017/S1352465815000430

Waite, F., Sheaves, B., Isham, L., Reeve, S., & Freeman, D. (2020). Sleep and schizophrenia: From epiphenomenon to treatable causal target. *Schizophrenia research, 221*, 44–56. https://doi.org/10.1016/j.schres.2019.11.014

Walker, E. R., McGee, R. E., & Druss, B. G. (2015). Mortality in mental disorders and global disease burden implications: a systematic review and meta-analysis. *JAMA psychiatry, 72*(4), 334–341. https://doi.org/10.1001/jamapsychiatry.2014.2502

Wirz-Justice, A., Benedetti, F., & Terman, M. (2013). *Chronotherapeutics for affective disorders: A clinician's manual for light and wake therapy (*2nd rev. ed.). Karger

Wulff, K., Dijk, D. J, Middleton, B., Foster, R. G., Joyce, E. M. (2012). Sleep and circadian rhythm disruption in schizophrenia. *Br J Psychiatry,200*(4):308-16. https://doi.org/10.1192/bjp.bp.111.096321

Wykes, T., Huddy, V., Cellard, C., McGurk, S. R., & Czobor, P. (2011). A meta-analysis of cognitive remediation for schizophrenia: Methodology and effect sizes. *American Journal of Psychiatry, 168*(5), 472–485.

Chapter Five

Discussion and Critical Evaluation

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Discussion and critical evaluation chapter

This section examines the outcomes of both the systematic review and the empirical study, highlighting their distinct and collective impacts on the domain of clinical psychology. In addition to examining the strengths and limitations of the research, this chapter also investigates broader clinical and research applications. We also include the researcher's reflections.

Summary of Findings

The systematic review sought to examine the incidence of hypersomnia in people with depression and its substantial association with mood changes. The results indicated persistent correlations between hypersomnia and depression in the current literature, with evidence indicating that addressing depression may also mitigate hypersomnia symptoms. Nevertheless, our understanding of the precise processes governing this association remains inadequate. The research highlights the need for thorough assessment and management of hypersomnia in depressed individuals and promotes the use of objective and standardised metrics to improve the precision and dependability of future studies in this domain.

The primary objective of the empirical project was to enhance our comprehension of the frequency, consequences, and subjective encounters associated with excessive sleepiness among individuals diagnosed with psychotic illnesses. The findings of the research indicated significant differences in gender and age across the various groups, with a higher proportion of male participants in the hypersomnia group. There were no statistically significant differences seen among the groups with regards to diagnosis, type of medication, or dosage of antipsychotic medication. There were no statistically significant differences in the severity of psychiatric symptoms among the groups. However, it is worth noting that the group experiencing hypersomnia symptoms had heightened levels of hallucinations, paranoia, and

depression. There were no significant differences in quality of life or physical activity across the groups. However, the results indicated that individuals in the hypersomnia group exhibited longer periods of bed rest and engaged in more sedentary behaviours.

Overall, the results highlight the significance of hypersomnia as an under-recognised but influential concern in mental health. Hypersomnia is correlated with poorer mood and heightened sedentary behaviour across several diagnostic categories, despite no notable variations in drug type or illness severity. This underscores a distinct gap in our understanding of the processes behind hypersomnia in clinical populations and accentuates the need for more focused assessment and intervention strategies. The results demonstrate the particular importance of hypersomnia in the context of depression co-occurring with psychosis, a presentation that is frequently characterised by more severe functional impairments and complex clinical requirements. This is particularly important because depression is now acknowledged as a prevalent and prognostically significant aspect of psychotic disorders, which affects both the course of recovery and the persistence of symptoms. The interaction between depressive symptoms and psychotic processes has the potential to exacerbate sleep disturbances, indicating that hypersomnia may be a symptom of a more complex or treatment-resistant subtype of illness. Additional research using standardised measures and longitudinal designs is essential for formulating effective, individualised therapies that may tackle both sleep disturbances and their wider functional and emotional consequences.

Critical Appraisal

The systematic review focused on the relationship between hypersomnia and depression. Interestingly, the literature highlights a bidirectional relationship. As an example, people with depression are prone to developing hypersomnia (Hein et al., 2019). Furthermore, the

presence of hypersomnia might intensify physical sensations (appetite, agitation, and headaches), hence exacerbating the individual's low mood (Garvey et al., 1984).

A surprising finding also emerged regarding the connection between hypersomnia and an elevated risk of suicidal thoughts and behaviours (YoEargon et al., 1997; Zhao et al., 2021; Maruani et al., 2023b, etc.). This offers important perspectives on the development of specific therapies designed to mitigate the likelihood of suicide among people experiencing hypersomnia, thereby enhancing overall mental health outcomes.

The empirical project aimed to examine excessive sleepiness in the context of psychosis by comparing two groups (one experiencing hypersomnia symptoms and the other not). Remarkably, both groups had heightened levels of excessive sleepiness. Furthermore, there were no statistically significant differences detected across the groups in terms of medication use or the severity of clinical symptoms. The result suggests that there may be other variables, apart from medication and symptom management, that might potentially influence the occurrence of hypersomnia in people diagnosed with psychosis. Moreover, the study highlights the need to adopt a holistic methodology for comprehending and addressing hypersomnia in individuals with psychosis, encompassing the examination of behavioural, environmental, and neurobiological elements.

Engaging in research within the realm of psychosis has been a significant departure from my previous experiences working directly in clinical settings. Motivated by the limited opportunities for both research and clinical interventions for this population, I embarked on this project with a determination to contribute to filling this gap within the literature.

Throughout the course of this endeavour, I encountered numerous challenges, particularly when I began participant recruitment. This aspect was one of the most formidable hurdles, surpassing even the complexities I had previously navigated within mental health settings.

There were moments when I doubted my ability to recruit enough participants for the study, yet these instances of uncertainty ultimately served as catalysts for resilience and determination.

Moreover, venturing into a systematic review as a novice presented its set of challenges. However, despite initial discomfort, this experience has been invaluable in fostering my understanding, knowledge, and skill set within this domain.

Overall, working on this portfolio has broadened my methodological skills and underscored the significant influence of quantitative approaches in shaping my understanding of this complex area. Additionally, undertaking this portfolio has strengthened my enthusiasm for investigating the complexities of psychosis, particularly within the context of early intervention services, and has deepened my curiosity for the relationship between sleep, psychosis and their collective impact on the mood and quality of life of people with psychosis.

In summary, this journey has been marked by both challenges and growth, propelling me to expand my horizons and deepen my commitment to contributing meaningfully to the field of psychosis.

The researcher's and supervisor’s positions and expertise were crucial factors to consider throughout the research process. The researcher had experience dealing with acute mental health and clinically with psychosis, had expertise in establishing relationships with this group, and was skilled at offering support for successful communication, addressing the social communication challenges often faced by this group. For instance, the researcher's skills facilitated the creation of a safe environment for discussing experiences and potentially distressing information when interacting with patients, their families, and carers or clinicians at the clozapine clinic. The supervisor's extensive experience in both psychosis research and

clinical work in early intervention services for psychosis was extremely beneficial for the current study. Their expertise in two areas allowed them to provide a wide range of information, as they were knowledgeable in research literature and had practical experience dealing with people with psychosis. Their knowledge of relevant research helped make well-informed decisions and suggestions, ensuring the project was founded on evidence-based methods. The supervisor's extensive ties in the clinical community were a valuable tool that helped foster cooperation and create partnerships. They expedited the research process and promoted collaborations by communicating with clinicians from community mental health teams and early intervention in psychosis services, organising meetings, and presenting our research proposal to staff. Moreover, their interaction with people who had firsthand experience added an important aspect to the endeavour, guaranteeing that the viewpoints and voices of those with lived psychotic experiences were included in the research, which emphasised the need to focus on the perspectives of those directly affected by psychosis.

Additionally, regular supervision meetings were crucial for overseeing different facets of the research process and addressing any issues that arose. The meetings served as a formal platform for discussing progress, resolving challenges, and guaranteeing compliance with portfolio deadlines.

The supervisor's assistance and encouragement during these sessions were essential in overcoming difficulties and sustaining momentum throughout the journey. They provided valuable knowledge and perspectives that assisted in identifying and solving problems, improving methods, and making well-informed choices at crucial moments. Furthermore, the frequency of these sessions promoted responsibility and uniformity, guaranteeing that advancements were constantly supervised and any deviations from the research plan were swiftly dealt with. This proactive strategy helps prevent possible obstacles and keeps progress moving steadily towards finishing the project. The monthly supervision sessions facilitated a

collaborative dynamic that was crucial in guiding the research project to its successful completion.

Systematic Review Critical Review

The strengths of the systematic review included the use of a study rating tool, which enhanced the rigour and objectivity of the review process. Additionally, the review encompassed a wide range of study designs, including cross-sectional, longitudinal, and intervention studies, thereby offering an in-depth look at the existing literature on the topic. This approach allowed for a more nuanced view of the relationship between hypersomnia and depression, facilitating more robust conclusions and implications for clinical practice and future research.

The systematic review elucidated the correlations between hypersomnia and depression while prompting critical examination of many fundamental ideas and obstacles in this research domain. A critical concern is the inconsistency in how hypersomnia is defined and measured across various research studies. The combination of hypersomnia with similar categories, such as excessive daytime sleepiness, generates ambiguity since these disorders may originate from different causes despite their common presentations. Certain studies assess hypersomnia through subjective self-reports of excessive sleep duration, whilst others emphasise excessive daytime sleepiness, which may not consistently align with extended sleep duration. This diversity complicates study comparisons and undermines the validity of general findings about its function in mental health issues. Moreover, conventional psychiatric diagnosis systems often categorise hypersomnia as a major depressive disorder with unusual characteristics. This approach may oversimplify the presentation by categorising it only as a symptom of depression rather than seeing its potential as an autonomous or transdiagnostic phenomenon. Hypersomnia often coexists with fatigue,

anhedonia, and cognitive impairment, complicating the isolation of its distinct effects on psychological discomfort. Furthermore, the connection between hypersomnia and insomnia, often considered diametrically opposed, is more complex than it seems. Both symptoms may coexist or alternate within the same person, indicating a common imbalance in mood and arousal systems. The intersection of these sleep disorders undermines the validity of existing diagnostic paradigms, which often depend on subjective self-reports and lack verifiable biomarkers. This epistemic constraint hampers causal explanations since several roles and processes underpinning hypersomnia remain implicit or confined to self-reported symptoms. Considering these obstacles, there is a need for more sophisticated diagnostic criteria and multimodal assessment strategies that integrate both subjective experiences and objective sleep measures to improve the precision and therapeutic applicability of hypersomnia research.

The legitimacy of diagnostic frameworks used to detect hypersomnia in depression requires more examination. A multitude of research mostly depends on subjective self-report measures, such as the Epworth Sleepiness Scale (ESS) or Pittsburgh Sleep Quality Index (PSQI), which are susceptible to memory bias and variations in individual perception. These approaches may inadequately screen for hypersomnia or differentiate it from other sleep disorders. Objective measures such as polysomnography or actigraphy are infrequently used, hindering the capacity to validate results using physiological data.

The epistemological identification of causal links between hypersomnia and depression presents significant obstacles. Numerous processes of relevance, such as disturbances in mood control or arousal pathways, are implicit and not readily apparent. This dependence on subjective evaluations or indirect inferences limits the capacity to differentiate causation from correlation. Longitudinal and interventional studies, while providing some insights, often neglect the bidirectional or dynamic aspects of this interaction, so confusing conclusions.

This review emphasised the significance of comprehending hypersomnia in depression while also stressing the need for conceptual clarity, methodological rigour, and a more integrated approach to examining the relationship between sleep and mood disorders. Mitigating these constraints may facilitate the development of more accurate and effective treatment strategies.

Empirical Project Critical Review

The empirical project defined a threshold for hypersomnia using diagnostic criteria from the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association, 2013) and the International Classification of Sleep Disorders (3rd ed.; ICSD-3; American Academy of Sleep Medicine, 2014). The rationale for this methodological choice was to improve the transparency of the findings, allowing more accurate comparisons and analyses.

The study notes the inclusion of experts by experience, often referred to as patient and public involvement (PPI). The engagement and ownership of participants may be enhanced by engaging people who have personal experience with psychosis, therefore building a collaborative connection between researchers and participants. This methodology has the potential to provide study outcomes that are more significant and effective since people with direct experience contribute vital ideas and perspectives to the research endeavour.

Nevertheless, the research does have numerous shortcomings. The absence of statistical significance in some results might be due to a lower-than-expected sample size or to unexpected intergroup homogeneity in terms of sleepiness. Further investigation using larger sample sizes and alternative methodologies has the potential to provide a more definitive understanding of the associations between hypersomnia and clinical symptoms or features in psychosis.

A further limitation is the exclusive dependence on self-reported measures for evaluating hypersomnia, hence introducing possible biases. Utilising objective indicators like actigraphy or polysomnography would provide a more thorough and precise assessment of sleepiness, hence mitigating the biases linked to self-report measures. Furthermore, the study's cross-sectional methodology and the unequal allocation of individuals among groups pose limitations on the establishment of causal correlations. A more comprehensive evaluation of the association between hypersomnia, clinical symptoms, and quality of life throughout time might be achieved by the implementation of longitudinal studies.

Overall Clinical Implications

The therapeutic consequences of comprehending the interaction of hypersomnia, clinical symptoms, and wellbeing within the framework of depression and psychosis are of considerable importance. For instance, treating hypersomnia by using sleep interventions or taking pharmaceuticals can potentially enhance mood symptoms (Jha et al., 2020). Roberts et al. (2000) closely link hypersomnia to an increased risk of developing depression in the future. This underscores the importance of vigilance among clinicians when identifying hypersomnia in individuals with depression.

Moreover, hypersomnia has been associated with suicidal thoughts (Chellappa & Araujo, 2006) and attempts (YoEargon et al., 1997). Therefore, it is crucial for clinicians to conduct a thorough evaluation of suicide risk in people diagnosed with hypersomnia and mood disorders and thereafter employ suitable therapies to effectively reduce this risk.

Overall, it is essential to acknowledge the correlation between hypersomnia and depression to achieve a precise diagnosis, facilitate the selection of appropriate treatments, and enhance clinical outcomes. To optimise patient treatment and improve overall functionality and

quality of life, it is recommended that clinicians employ a comprehensive approach that addresses both conditions.

It is also important for healthcare staff to regularly evaluate hypersomnia in individuals with psychosis. From the perspective of clinicians, sleep issues are widespread in individuals with psychotic illnesses and have detrimental effects, including symptom severity, mood, and daily activities (Rehman et al., 2017). This holds significance since clinicians assert that enhancing sleep yields several advantages, such as ameliorating psychotic symptoms (96%), augmenting energy levels (91%), promoting physical activity (90%), ameliorating emotional symptoms (88%), and enhancing physical well-being (86%), according to Barrett et al., 2020). However, there is a shortage of organised evaluations and the implementation of suggested therapeutic approaches. The observed contradiction may be partially attributed to the clinicians' perceived obstacles to sleep therapy, such as their acknowledged lack of understanding about sleep evaluation and treatment, as well as their opinions that sleep treatment is too challenging (Rehman et al., 2017; Barrett et al., 2020).

Therefore, creating treatment pathways is a crucial objective in clinical practice. There is a need for increased recognition of the significance of sleep among both staff and individuals with psychosis. Training courses would facilitate the ability of staff members to recognise, evaluate, and address sleep-related issues (Rehman et al., 2017).

Also, integrating sleep disruptions into the overall treatment strategy for people with psychosis is crucial. This may require a comprehensive strategy encompassing several fields of study, such as medication, psychotherapy, and lifestyle treatments that are customised to target individual symptoms and concerns related to sleep. There is an urgent need for the implementation of sleep and circadian therapies within regular practice settings, often by clinicians who possess little or no expertise in the field of sleep and circadian research, as

well as evidence-based psychological treatments (EBPTs). Moreover, there are regular practice environments that lack the necessary resources to provide training and monitoring. For instance, multicomponent transdiagnostic therapies, like TranS-C, may provide therapists with a straightforward method that may be beneficial for patients with several sleep and circadian challenges, including hypersomnia (Harvey, 2022).

Effective management of depressive symptoms is of utmost importance in patients with psychosis since there is a significant correlation between hypersomnia and alterations in mood. Interventions that focus on mood management, such as cognitive restructuring and training in emotion regulation skills, may be especially advantageous (Waite et al., 2016; Kimhy et al., 2020; Lawlor et al., 2022).

Moreover, consistent surveillance of sleep patterns and depressive symptoms is crucial to monitoring changes over time and adapting treatment approaches appropriately. Enhancing treatment adherence and results and avoiding relapse may be achieved by offering continuous support and psychoeducation to patients and their families (Iuso et al., 2023) on the possible consequences of hypersomnia on mood and general functioning.

Future Directions for Systematic Review and Empirical Paper

For future systematic reviews, expanding the search criteria to include "mood disorder" may enhance the identification of relevant studies exploring the relationship between hypersomnia and mood disorders, particularly focusing on conditions such as bipolar disorder and seasonal affective disorder.

In the future, potential paths for the empirical paper might involve increasing the sample size to improve statistical power and broaden the generalisability of results. Also, using objective measures (actigraphy, polysomnography) to assess sleepiness would offer a more comprehensive assessment.

Moreover, investigating the temporal correlation between sleepiness and clinical symptoms over an extended period would enable the examination of causal pathways and the discovery of possible predictors or mediators of results.

More importantly, exploring the association between sleepiness and clinical symptoms like mood, paranoia, and hallucinations by using correlation or exploratory regression might clarify mechanisms linking sleepiness and mental symptoms.

Conclusion

The objective of this thesis portfolio was to examine hypersomnia in two specific contexts: depression and psychosis. The portfolio included a systematic review to investigate the correlation between hypersomnia and depression, followed by a quantitative empirical study that explored hypersomnia in individuals diagnosed with psychosis.

The findings of the systematic review and the empirical paper, although examined independently, together enhance our understanding of the intricate relationship between hypersomnia, depression, and psychosis, as well as their shared implications for clinical treatment and future research.

The comprehensive study identified a definitive association between hypersomnia and depression, emphasising their bidirectional relationship and the possibility that treating depression may mitigate hypersomnia symptoms. The empirical investigation further corroborates that people diagnosed with psychosis and experiencing hypersomnia also report increased levels of depression and other mental symptoms. Both findings indicate the interconnectedness between mood difficulties and sleep disorders, especially with depression and psychosis.

Both results suggest that there are thorough evaluations and therapies addressing hypersomnia. The systematic review indicates that identifying and addressing hypersomnia in

individuals with depression can improve mood regulation and overall functioning, whereas the empirical project emphasises the role of psychological therapies to address hypersomnia symptoms in people with psychosis. The systematic review aimed to provide comprehensive knowledge of hypersomnia in depression, while the empirical paper offered particular demographic insights, emphasising gender and age disparities in hypersomnia frequency among patients with psychosis. This variation adds a level of specificity to the systematic review's results, indicating that certain demographic characteristics may affect the incidence and severity of hypersomnia, necessitating more individualised treatment strategies.

The systematic review revealed a possible association between hypersomnia and an elevated risk of suicidal ideation and attempts. This element is especially pertinent to the empirical project's results, whereby increased depression and psychotic symptoms in the hypersomnia group indicate that there must be vigilant monitoring of patients with these co-occurring illnesses for suicide risk. The aggregated findings indicate that tackling hypersomnia may be essential in suicide prevention initiatives.

The methodological challenges encountered in both studies—specifically, the dependence on subjective self-report measures in the systematic review and the constraints of the empirical study's cross-sectional design—underscore the necessity for future research to use more objective sleep assessments, larger sample sizes, and longitudinal designs. The two studies together indicate that improving measurement precision and broadening the research scope may provide a more comprehensive understanding of the processes connecting hypersomnia to mood and psychotic illnesses.

Both studies advocate for a comprehensive strategy in tackling hypersomnia, especially in those with mood and psychotic disorders. The systematic review supports the use of objective and standardised metrics for assessing hypersomnia, while empirical research highlights the

need to account for environmental, behavioural, and neurobiological elements in the management of sleep problems associated with psychosis. These results indicate that a comprehensive treatment strategy—incorporating sleep treatments, psychological therapy, and meticulous medication management—may be most successful in meeting the intricate requirements of patients with hypersomnia, depression, and psychosis.

The systematic review and empirical project together offer complementary analyses of the influence of hypersomnia on depression and psychosis. The review presents a comprehensive theoretical framework, and the empirical investigation supplies more detailed clinical data. Both underscore the need for integrated, patient-centred interventions for controlling hypersomnia, focusing on the intricate connections among sleep, mood, and psychotic symptoms. These results indicate the need for more research and therapeutic initiatives to enhance the evaluation, management, and comprehension of hypersomnia in these groups.

The combined findings from the systematic review and empirical study highlight the necessity for continued investigation into hypersomnia among individuals affected by depression and psychosis, stressing its impact on clinical symptoms and overall well-being. Through ongoing research, it is possible to advance the development of more effective therapies, increase the provision of services, and ultimately improve the quality of life for people impacted by hypersomnia.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Publishing.

American Academy of Sleep Medicine. (2014). *International classification of sleep disorders* (3rd ed.). American Academy of Sleep Medicine.

Barrett, E. A., Aminoff, S. R., Simonsen, C., & Romm, K. L. (2020). Opening the curtains for better sleep in psychotic disorders - considerations for improving sleep treatment. *Comprehensive psychiatry*, 103, 152207. https://doi.org/10.1016/j.comppsych.2020.152207

Chellappa, S. L., & Araújo, J. F. (2006). Excessive daytime sleepiness in patients with depressive disorder. *Revista brasileira de psiquiatria (Sao Paulo, Brazil: 1999), 28*(2), 126–129. https://doi.org/10.1590/s1516-44462006000200010

Garvey, M. J., Mungas, D., & Tollefson, G. D. (1984). Hypersomnia in major depressive disorders. *Journal of affective disorders*, *6*(3-4), 283–286. https://doi.org/10.1016/s0165-0327(84)80006-3

Harvey, A. G. (2022). Treating sleep and circadian problems to promote mental health: perspectives on comorbidity, implementation science and behaviour change. *Sleep, 45*(4), zsac026. https://doi.org/10.1093/sleep/zsac026

Hein, M., Lanquart, J. P., Loas, G., Hubain, P., & Linkowski, P. (2019). Prevalence and risk factors of excessive daytime sleepiness in major depression: A study with 703 individuals referred for polysomnography. *Journal of affective disorders, 243*, 23–32. https://doi.org/10.1016/j.jad.2018.09.016

Iuso, S., Severo, M., Trotta, N., Ventriglio, A., Fiore, P., Bellomo, A., & Petito, A. (2023). Improvements in Treatment Adherence after Family Psychoeducation in Patients

Affected by Psychosis: Preliminary Findings. *Journal of personalized medicine, 13*(10), 1437. https://doi.org/10.3390/jpm13101437

Jha, M. K., Fava, M., Freeman, M. P., Thase, M. E., Papakostas, G. I., Shelton, R. C., Trivedi, M. H., Dirks, B., Liu, K., & Stankovic, S. (2020). Effect of Adjunctive Pimavanserin on Sleep/Wakefulness in Patients with Major Depressive Disorder: Secondary Analysis From CLARITY. *The Journal of clinical psychiatry, 82*(1), 20m13425. https://doi.org/10.4088/JCP.20m13425

Kimhy, D., Lister, A., Liu, Y., Vakhrusheva, J., Delespaul, P., Malaspina, D., Ospina, L. H., Mittal, V. A., Gross, J. J., & Wang, Y. (2020). The impact of emotion awareness and regulation on psychotic symptoms during daily functioning. *NPJ schizophrenia, 6*(1), 7. https://doi.org/10.1038/s41537-020-0096-6

Lawlor, C., Vitoratou, S., Duffy, J., Cooper, B., De Souza, T., Le Boutillier, C., Carter, B., Hepworth, C., & Jolley, S. (2022). Managing emotions in psychosis: Evaluation of a brief DBT-informed skills group for individuals with psychosis in routine community services. *The British journal of clinical psychology, 61*(3), 735–756. https://doi.org/10.1111/bjc.12359

Maruani, J., Molière, F., Godin, O., Yrondi, A., Bennabi, D., Richieri, R., El-Hage, W., Allauze, E., Anguill, L., Bouvard, A., Camus, V., Dorey, J. M., Etain, B., Fond, G., Genty, J. B., Haffen, E., Holtzmann, J., Horn, M., Kazour, F., Nguon, A. S., … Geoffroy, P. A. (2023b). Diurnal symptoms of sleepiness and dysfunction predict future suicidal ideation in a French cohort of outpatients (FACE-DR) with treatment resistant depression: A 1-year prospective study about sleep markers. *Journal of affective disorders, 329*, 369–378. https://doi.org/10.1016/j.jad.2023.02.106

Rehman, A., Waite, F., Sheaves, B., Biello, S., Freeman, D., & Gumley, A. (2017). Clinician perceptions of sleep problems, and their treatment, in patients with non-affective psychosis. *Psychosis, 9*(2), 129–139. https://doi.org/10.1080/17522439.2016.1206955

Vancampfort, D., Stubbs, B., Mitchell, A. J., De Hert, M., Wampers, M., Ward, P. B., Rosenbaum, S., & Correll, C. U. (2015). Risk of metabolic syndrome and its components in people with schizophrenia and related psychotic disorders, bipolar disorder and major depressive disorder: a systematic review and meta-analysis. *World psychiatry: official journal of the World Psychiatric Association (WPA), 14*(3), 339–347. https://doi.org/10.1002/wps.20252

Waite, F., Myers, E., Harvey, A. G., Espie, C. A., Startup, H., Sheaves, B., & Freeman, D. (2016). Treating Sleep Problems in Patients with Schizophrenia. *Behavioural and cognitive psychotherapy, 44*(3), 273–287. https://doi.org/10.1017/S1352465815000430

YoEargon, M. Y., Kara, H., & Solmaz, M. (1997). Sleep disturbances and suicidal behavior in patients with major depression. *The Journal of clinical psychiatry, 58*(6), 249–251. https://doi.org/10.4088/jcp.v58n0602

Zhao, J., Liu, H., Wu, Z., Wang, Y., Cao, T., Lyu, D., Huang, Q., Wu, Z., Zhu, Y., Wu, X., Chen, J., Wang, Y., Su, Y., Zhang, C., Peng, D., Li, Z., Rong, H., Liu, T., Xia, Y., Hong, W., … Fang, Y. (2021). Clinical features of the patients with major depressive disorder co-occurring insomnia and hypersomnia symptoms: a report of NSSD study. *Sleep medicine, 81*, 375–381. https://doi.org/10.1016/j.sleep.2021.03.005

Portfolio Reference List

Afonso, P., Figueira, M. L., & Paiva, T. (2014). Sleep-wake patterns in schizophrenia patients compared to healthy controls. *The world journal of biological psychiatry: the official journal of the World Federation of Societies of Biological Psychiatry, 15*(7), 517–524. https://doi.org/10.3109/15622975.2012.756987

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). American Psychiatric Publishing.

American Psychiatric Association. (2013). Cautionary statement for forensic use of DSM-5. In *Diagnostic and statistical manual of mental disorders* (5th ed., p. 280). American Psychiatric Publishing.

American Academy of Sleep Medicine. (2014). *International Classification of Sleep Disorders* (3rd ed.). American Academy of Sleep Medicine.

Andaku, D. K., D'Almeida, V., Carneiro, G., Hix, S., Tufik, S., & Togeiro, S. M. (2015). Sleepiness, inflammation and oxidative stress markers in middle-aged males with obstructive sleep apnea without metabolic syndrome: a cross-sectional study. *Respiratory research, 16*(1), 3. https://doi.org/10.1186/s12931-015-0166-x

Araghi, M. H., Jagielski, A., Neira, I., Brown, A., Higgs, S., Thomas, G. N., Taheri, S. (2013). The complex associations among sleep quality, anxiety-depression, and quality of life in patients with extreme obesity. *Sleep 36*, 1859–1865. https://doi.org/10.5665/sleep.3216

Armitage, R. (2007). Sleep and circadian rhythms in mood disorders. *Acta Psychiatrica Scandinavica, 115*(s433), 104–115. https://doi.org/10.1111/j.1600-0447.2007.00968.x

Baandrup, L., Jennum, P., Lublin, H., & Glenthoj, B. (2013). Treatment options for residual insomnia in schizophrenia. *Acta psychiatrica Scandinavica, 127*(1), 81–82. https://doi.org/10.1111/acps.12016

Baglioni, C., Battagliese, G., Feige, B., Spiegelhalder, K., Nissen, C., Voderholzer, U., ... & Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders, 135*(1-3), 10–19. https://doi.org/10.1016/j.jad.2011.01.011

Barateau, L., Lopez, R., Franchi, J. A., & Dauvilliers, Y. (2017). Hypersomnolence, Hypersomnia, and Mood Disorders. *Current psychiatry reports, 19*(2), 13. https://doi.org/10.1007/s11920-017-0763-0

Barrett, E. A., Aminoff, S. R., Simonsen, C., & Romm, K. L. (2020). Opening the curtains for better sleep in psychotic disorders - considerations for improving sleep treatment. *Comprehensive psychiatry, 103*, 152207. https://doi.org/10.1016/j.comppsych.2020.152207

Bowie, C. R., & Harvey, P. D. (2006). Cognitive deficits and functional outcome in schizophrenia. *Neuropsychiatric Disease and Treatment, 2*(4), 531–536.

Bazin, B., Frija-Masson, J., Benzaquen, H., Maruani, J., Micoulaud-Franchi, J. A., Lopez, R., Philip, P., Bourgink, P., Lejoyeux, M., d'Ortho, M.-P., & Geoffroy, P. A. (2024). Major depressive disorder with hypersomnolence complaint: A comparison study with non-depressed individuals examining objective biomarkers. *Journal of Affective Disorders, 352*, 422–428. https://doi.org/10.1016/j.jad.2024.02.030

Berkowski, J. A., & Shelgikar, A. V. (2016). Disorders of Excessive Daytime Sleepiness Including Narcolepsy and Idiopathic Hypersomnia. *Sleep medicine clinics, 11*(3), 365–378. https://doi.org/10.1016/j.jsmc.2016.04.005

Billiard, M., Dolenc, L., Aldaz, C., Ondze, B., & Besset, A. (1994). Hypersomnia associated with mood disorders: a new perspective. *Journal of psychosomatic research, 38*(Suppl 1), 41–47. https://doi.org/10.1016/0022-3999(94)90134-1

Blanchard, A. W., Rufino, K. A., Nadorff, M. R., & Patriquin, M. A. (2023). Nighttime sleep quality & daytime sleepiness across inpatient psychiatric treatment is associated with clinical outcomes. *Sleep Medicine, 110*, 235–242.

Bleuler, E. (1950). *Dementia Praecox or the Group of Schizophrenias*. International Universities Press.

Breslau, N., Roth, T., Rosenthal, L., & Andreski, P. (1996). Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biological psychiatry, 39*(6), 411–418. https://doi.org/10.1016/0006-3223(95)00188-3

Calati, R., Gaspar-Barba, E., Cruz-Fuentes, C. S., Nenclares, A., Jimenez-Genchi, A., De Ronchi, D., & Serretti, A. (2010). Excessive daytime sleepiness in depressed women. *Psychiatry research, 179*(2), 171–175. https://doi.org/10.1016/j.psychres.2009.11.021

Cao, B., Park, C., Rosenblat, J. D., Chen, Y., Iacobucci, M., Subramaniapillai, M., Mansur, R. B., Zuckerman, H., Lee, Y., & McIntyre, R. S. (2019). Changes in sleep predict changes in depressive symptoms in depressed subjects receiving vortioxetine: An open-label clinical trial. *Journal of psychopharmacology, 33*(11), 1388–1394 https://doi.org/10.1177/0269881119874485

Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. *Sleep, 33*(5), 585–592. https://doi.org/10.1093/sleep/33.5.585

Chellappa, S. L., & Araújo, J. F. (2006). Excessive daytime sleepiness in patients with depressive disorder. *Revista brasileira de psiquiatria (Sao Paulo, Brazil: 1999), 28*(2), 126–129. https://doi.org/10.1590/s1516-44462006000200010

Chellappa, S. L., & Araújo, J. F. (2007). Sleep disorders and suicidal ideation in patients with depressive disorder. *Psychiatry research, 153*(2), 131–136. https://doi.org/10.1016/j.psychres.2006.05.007

Chen, Y., Zhang, L., Hu, S., Zhang, H., Sun, Q., Hong, M., Qian, Y., Lu, L., & Sun, H. (2021). Status and Correlates of Hypersomnia in Hospitalized Patients with Unipolar Depression - Beijing, Henan, and Shandong, China, August 2019-March 2021. *China CDC weekly, 3*(42), 879–882. https://doi.org/10.46234/ccdcw2021.217

Cheung, M. M. S., Lam, S. P., Chau, S. W. H., Chan, N. Y., Li, T. M. H., Wing, Y. K., & Chan, J. W. Y. (2024). Hypersomnolence is associated with non-remission of major depressive disorder. *Sleep Medicine, 119*, 35–43.

Cho, Y. W., Lee, J. H., Son, H. K., Lee, S. H., Shin, C., & Johns, M. W. (2011). The reliability and validity of the Korean version of the Epworth sleepiness scale. *Sleep & breathing = Schlaf & Atmung, 15*(3), 377–384. https://doi.org/10.1007/s11325-010-0343-6

Chouinard, S., Poulin, J., Stip, E., & Godbout, R. (2004). Sleep in untreated patients with schizophrenia: a meta-analysis. *Schizophrenia bulletin, 30*(4), 957–967. https://doi.org/10.1093/oxfordjournals.schbul.a007145

Claghorn, J. L., Mathew, R. J., Weinman, M. L., & Hruska, N. (1981). Daytime sleepiness in depression. *The Journal of clinical psychiatry, 42*(9), 342–343.

Cohrs, S. (2008). Sleep disturbances in patients with schizophrenia. *CNS Drugs, 22*(11):939-62.

Cook, J. D., Rumble, M. E., Tran, K. M., & Plante, D. T. (2021). Potential Maladaptive Sleep-Related Cognitions in Depression with Comorbid Hypersomnolence: An Exploratory Investigation. *Behavioural sleep medicine, 19*(2), 232–242. https://doi.org/10.1080/15402002.2020.1732390

Dauvilliers, Y., Lopez, R., Ohayon, M., & Bayard, S. (2013). Hypersomnia and depressive symptoms: methodological and clinical aspects. *BMC medicine, 11*, 78. https://doi.org/10.1186/1741-7015-11-78

Davies, G., Haddock, G., Yung, A. R., Mulligan, L. D., & Kyle, S. D. (2017). A systematic review of the nature and correlates of sleep disturbance in early psychosis. *Sleep medicine reviews*, 31, 25–38. https://doi.org/10.1016/j.smrv.2016.01.001

DeBattista, C., Doghramji, K., Menza, M. A., Rosenthal, M. H., Fieve, R. R., & Modafinil in Depression Study Group (2003). Adjunct modafinil for the short-term treatment of fatigue and sleepiness in patients with major depressive disorder: a preliminary double-blind, placebo-controlled study. *The Journal of clinical psychiatry, 64*(9), 1057–1064. https://doi.org/10.4088/jcp.v64n0911

de Pinho, R.S.N., da Silva-Junior, F.P., Bastos, J.P.C., Maia, W.S., de Mello, M.T., de Bruin, V.M.S., de Bruin, P.F.C. (2006). Hypersomnolence and accidents in truck drivers: a cross-sectional study. *Chronobiology International, 23*(5), 963–971. https://doi.org/10.1080/07420520600920759

Dunlop, B. W., Crits-Christoph, P., Evans, D. L., Hirschowitz, J., Solvason, H. B., Rickels, K., Garlow, S. J., Gallop, R. J., & Ninan, P. T. (2007). Coadministration of modafinil and a selective serotonin reuptake inhibitor from the initiation of treatment of major depressive

disorder with fatigue and sleepiness: A double-blind, placebo-controlled study. Journal of *Clinical Psychopharmacology, 27*(6), 614–619.

Fatouros-Bergman, H., Cervenka, S., Flyckt, L., Edman, G., & Farde, L. (2014). Meta-analysis of cognitive performance in drug-naïve patients with schizophrenia. *Schizophrenia Research, 158*(1-3), 156–162.

Faul, F., Erdfelder, E., Buchner, A., & Lang, A. G. (2009). Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. *Behaviour research methods, 41*(4), 1149–1160. https://doi.org/10.3758/BRM.41.4.1149

Faulkner, S., Bee, P. (2017). Experiences, perspectives and priorities of people with schizophrenia spectrum disorders regarding sleep disturbance and its treatment: a qualitative study. *BMC psychiatry, 17*(1), 158. https://doi.org/10.1186/s12888-017-1329-8

Fava, M., Thase, M. E., DeBattista, C., Doghramji, K., Arora, S., & Hughes, R. J. (2007). Modafinil augmentation of selective serotonin reuptake inhibitor therapy in MDD partial responders with persistent fatigue and sleepiness. *Annals of Clinical Psychiatry, 19*(3), 153–159. https://doi.org/10.1080/10401230701464858

Ferrarelli, F. (2020). Sleep disturbances in schizophrenia and psychosis. *Schizophrenia research, 221*, 1–3. https://doi.org/10.1016/j.schres.2020.05.022

Fervaha, G., Foussias, G., Agid, O., & Remington, G. (2014). Motivational and neurocognitive deficits are central to negative symptoms of schizophrenia. *Schizophrenia Research, 159*(2-3), 501–508.

Fitzgerald, C. T., Messias, E., & Buysse, D. J. (2011). Teen sleep and suicidality: results from the youth risk behavior surveys of 2007 and 2009. *Journal of clinical sleep medicine: JCSM:*

official publication of the American Academy of Sleep Medicine, 7(4), 351–356. https://doi.org/10.5664/JCSM.1188

Ford, D. E., & Cooper-Patrick, L. (2001). Sleep disturbances and mood disorders: an epidemiologic perspective. *Depression and anxiety, 14*(1), 3–6. https://doi.org/10.1002/da.1041

Formica, M. J. C., Fuller-Tyszkiewicz, M., Hickie, I., Olive, L., Wood, S. J., Purcell, R., Yung, A. R., Phillips, L. J., Nelson, B., Pantelis, C., McGorry, P. D., & Hartmann, J. A. (2023). The relationship between subjective sleep disturbance and attenuated psychotic symptoms after accounting for anxiety and depressive symptoms. *Schizophrenia research, 258*, 84–93. https://doi.org/10.1016/j.schres.2023.07.019

Freeman, D. (2016). Persecutory delusions: a cognitive perspective on understanding and treatment. *The lancet. Psychiatry, 3*(7), 685–692. https://doi.org/10.1016/S2215-0366(16)00066-3

Freeman, D., Sheaves, B., Goodwin, G. M., Yu, L. M., Nickless, A., Harrison, P. J., Emsley, R., Luik, A. I., Foster, R. G., Wadekar, V., Hinds, C., Gumley, A., Jones, R., Lightman, S., Jones, S., Bentall, R., Kinderman, P., Rowse, G., Brugha, T., Blagrove, M., … Espie, C. A. (2017). The effects of improving sleep on mental health (OASIS): a randomised controlled trial with mediation analysis. *The lancet. Psychiatry, 4*(10), 749–758. https://doi.org/10.1016/S2215-0366(17)30328-0

Freeman, D., Loe, B., Kingdon, D., Startup, H., Molodynski, A., Rosebrock, L., . . . Bird, J.C. (2019). The revised Green et al., Paranoid Thoughts Scale (R-GPTS): Psychometric properties, severity ranges, and clinical cut-offs. *Psychological Medicine, 1*-10. https://doi.org/10.1017/S0033291719003155

Freeman, D., Sheaves, B., Waite, F., Harvey, A. G., & Harrison, P. J. (2020). Sleep disturbance and psychiatric disorders. *The Lancet Psychiatry, 7*(7), 628–637. https://doi.org/10.1016/S2215-0366(20)30136-X

Garvey, M. J., Mungas, D., & Tollefson, G. D. (1984). Hypersomnia in major depressive disorders. *Journal of affective disorders, 6*(3-4), 283–286. https://doi.org/10.1016/s0165-0327(84)80006-3

Gibson, E. S., Powles, A. C., Thabane, L., O'Brien, S., Molnar, D. S., Trajanovic, N., Ogilvie, R., Shapiro, C., Yan, M., & Chilcott-Tanser, L. (2006). "Sleepiness" is serious in adolescence: two surveys of 3235 Canadian students. *BMC public health, 6*, 116. https://doi.org/10.1186/1471-2458-6-116

Gold, P. W., Chrousos, G. P. (2002). Organization of the stress system and its dysregulation in melancholic and atypical depression: high vs low CRH/NE states. *Molecular psychiatry, 7*(3), 254–275. https://doi.org/10.1038/sj.mp.4001032

Gold, A. K., Sylvia, L. G. (2016). The role of sleep in bipolar disorder. *Nature and science of sleep, 8*, 207–214. https://doi.org/10.2147/NSS.S85754

Goldstein, T. R., Bridge, J. A., & Brent, D. A. (2008). Sleep disturbance preceding completed suicide in adolescents. *Journal of consulting and clinical psychology, 76*(1), 84–91. https://doi.org/10.1037/0022-006X.76.1.84

Grigolon, R. B., Trevizol, A. P., Cerqueira, R. O., Lee, Y., Mansur, R. B., McIntyre, R. S., & Brietzke, E. (2019). Hypersomnia and Bipolar Disorder: A systematic review and meta-analysis of proportion. *Journal of affective disorders, 246*, 659–666. https://doi.org/10.1016/j.jad.2018.12.030

Hagell, P., & Broman, J. E. (2007). Measurement properties and hierarchical item structure of the Epworth Sleepiness Scale in Parkinson's disease. *Journal of sleep research, 16*(1), 102–109. https://doi.org/10.1111/j.1365-2869.2007.00570.x

Hahn, D. L., Hoffmann, A. E., Felzien, M., LeMaster, J. W., Xu, J., & Fagnan, L. J. (2017). Tokenism in patient engagement. *Family practice, 34*(3), 290–295. https://doi.org/10.1093/fampra/cmw097

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review, 31*(2), 225–235. https://doi.org/10.1016/j.cpr.2010.04.003

Hawkins, D. R., Taub, J. M., & Van de Castle, R. L. (1985). Extended sleep (hypersomnia) in young, depressed patients. *The American journal of psychiatry, 142*(8), 905–910. https://doi.org/10.1176/ajp.142.8.905

Hein, M., Lanquart, J. P., Loas, G., Hubain, P., & Linkowski, P. (2019). Prevalence and risk factors of excessive daytime sleepiness in major depression: A study with 703 individuals referred for polysomnography. *Journal of affective disorders, 243*, 23–32. https://doi.org/10.1016/j.jad.2018.09.016

Helmus, T., Rosenthal, L., Bishop, C., Roehrs, T., Syron, M. L., & Roth, T. (1997). The alerting effects of short and long naps in narcoleptic, sleep deprived, and alert individuals. *Sleep, 20*(4), 251–257. https://doi.org/10.1093/sleep/20.4.251

Hong, Q. N., Gonzalez-Reyes, A., & Pluye, P. (2018). Improving the usefulness of a tool for appraising the quality of qualitative, quantitative and mixed methods studies, the Mixed Methods Appraisal Tool (MMAT). *Journal of Evaluation in Clinical Practice, 24*(3),

459–467. https://doi.org/10.1111/jep.12884

Huang, J. F., Chen, L. D., Lin, Q. C., Chen, G. P., Yu, Y. H., Huang, J. C., & Zhao, J. M. (2016). The relationship between excessive daytime sleepiness and metabolic syndrome in severe obstructive sleep apnea syndrome. *The clinical respiratory journal, 10*(6), 714–721. https://doi.org/10.1111/crj.12276

INVOLVE. (2018). National Standards for Public Involvement. Available at: https://drive.google.com/file/d/1MRzOnq_dXTU96vWfwpe5-wkYBa8mUu3o/view

Izci, B., Ardic, S., Firat, H., Sahin, A., Altinors, M., & Karacan, I. (2008). Reliability and validity studies of the Turkish version of the Epworth Sleepiness Scale. *Sleep & breathing = Schlaf & Atmung, 12*(2), 161–168. https://doi.org/10.1007/s11325-007-0145-7

Iovieno, N., van Nieuwenhuizen, A., Clain, A., Baer, L., & Nierenberg, A. A. (2011). Residual symptoms after remission of major depressive disorder with fluoxetine and risk of relapse. *Depression and anxiety, 28*(2), 137–144. https://doi.org/10.1002/da.20768

Jacobson, N. S., Martell, C. R., & Dimidjian, S. (2001). Behavioral activation treatment for depression: Returning to contextual roots. *Clinical Psychology: Science and Practice, 8*(3), 255–270. https://doi.org/10.1093/clipsy.8.3.255

Jamovi project. (2023). Jamovi (Version 2.3) [Computer Software]. Retrieved from https://www.jamovi.org.

Jha, M. K., Fava, M., Freeman, M. P., Thase, M. E., Papakostas, G. I., Shelton, R. C., Trivedi, M. H., Dirks, B., Liu, K., & Stankovic, S. (2020). Effect of Adjunctive Pimavanserin on Sleep/Wakefulness in Patients with Major Depressive Disorder: Secondary Analysis From

CLARITY. *The Journal of clinical psychiatry, 82*(1), 20m13425. https://doi.org/10.4088/JCP.20m13425

Johns, M. W. (1992). Reliability and factor analysis of the Epworth Sleepiness Scale. *Sleep, 15*(4), 376-381. https://doi.org/10.1093/sleep/15.4.376

Kaplan, K. A., & Harvey, A. G. (2009). Hypersomnia across mood disorders: A review and synthesis. *Sleep Medicine Reviews, 13*(4), 275–285. https://doi.org/10.1016/j.smrv.2008.08.001

Kaplan, K. A., Gruber, J., Eidelman, P., Talbot, L. S., & Harvey, A. G. (2011). Hypersomnia in inter-episode bipolar disorder: does it have prognostic significance? *Journal of affective disorders, 132*(3), 438–444. https://doi.org/10.1016/j.jad.2011.03.013

Kaplan, K. A., McGlinchey, E. L., Soehner, A., Gershon, A., Talbot, L. S., Eidelman, P., Gruber, J., & Harvey, A. G. (2015). Hypersomnia subtypes, sleep and relapse in bipolar disorder. *Psychological medicine, 45*(8), 1751–1763. https://doi.org/10.1017/S0033291714002918

Kaplan, K. A., Harvey, A. G. (2009). Hypersomnia across mood disorders: a review and synthesis. *Sleep medicine reviews, 13*(4), 275–285. https://doi.org/10.1016/j.smrv.2008.09.001

Kaplan, K. A., Plante, D. T., Cook, J. D., & Harvey, A. G. (2019). Development and validation of the Hypersomnia Severity Index (HSI): A measure to assess hypersomnia severity and impairment in psychiatric disorders. *Psychiatry research, 281*, 112547. https://doi.org/10.1016/j.psychres.2019.112547

Keetharuth, A. D., Brazier, J., Connell, J., Bjorner, J. B., Carlton, J., Taylor Buck, E., Ricketts, T., McKendrick, K., Browne, J., Croudace, T., & Barkham, M. (2018). Recovering Quality of Life (ReQoL): a new generic self-reported outcome measure for use with people experiencing

mental health difficulties. *The British journal of psychiatry: The journal of mental science, 212*(1), 42–49. https://doi.org/10.1192/bjp.2017.10

Kessing, L. V. (2007). Epidemiology of subtypes of depression. *Acta psychiatrica Scandinavica. Supplementum,* (433), 85–89. https://doi.org/10.1111/j.1600-0447.2007.00966.x

Kessler, R. C., & Bromet, E. J. (2013). The epidemiology of depression across cultures. *Annual review of public health, 34*, 119–138. https://doi.org/10.1146/annurev-publhealth-031912-114409

Kluge, M., Himmerich, H., Wehmeier, P. M., Rummel-Kluge, C., Dalal, M., Hinze-Selch, D., Kraus, T., Dittmann, R. W., Pollmächer, T., & Schuld, A. (2012). Sleep propensity at daytime as assessed by Multiple Sleep Latency Tests (MSLT) in patients with schizophrenia increases with clozapine and olanzapine. *Schizophrenia research, 135*(1-3), 123–127. https://doi.org/10.1016/j.schres.2011.12.017

Konuk, N., Atasoy, N., Atik, L., & Akay, O. (2006). Open-label study of adjunct modafinil for the treatment of patients with fatigue, sleepiness, and major depression treated with selective serotonin reuptake inhibitors. *Advances in therapy, 23*(4), 646–654. https://doi.org/10.1007/BF02850053

Kraepelin, E. (1919). Dementia praecox and paraphrenia (E. S. Barclay, Trans.). Livingston. (*Original work published 1913 in Textbook of Psychiatry,* 8th ed.)

Krupp, L. B., LaRocca, N. G., Muir-Nash, J., & Steinberg, A. D. (1989). The fatigue severity scale. Application to patients with multiple sclerosis and systemic lupus erythematosus. *Archives of Neurology, 46,* 1121–1123.

Krystal, A. D, Goforth, H. W., Roth, T. (2008). Effects of antipsychotic medications on sleep in schizophrenia. *Int Clin Psychopharmacol, May;23*(3):150-60. https://doi.org/10.1097/YIC.0b013e3282f39703

Krystal, A. D., Mittoux, A., Meisels, P., & Baker, R. A. (2016). Effects of Adjunctive Brexpiprazole on Sleep Disturbances in Patients with Major Depressive Disorder: An Open-Label, Flexible-Dose, Exploratory Study. *The primary care companion for CNS disorders, 18*(5), 10.4088/PCC.15m01914. https://doi.org/10.4088/PCC.15m01914

Lammers, G. J., Bassetti, C. L. A., Dolenc-Groselj, L., Jennum, P. J., Kallweit, U., Khatami, R., Lecendreux, M., Manconi, M., Mayer, G., Partinen, M., Plazzi, G., Reading, P. J., Santamaria, J., Sonka, K., & Dauvilliers, Y. (2020). Diagnosis of central disorders of hypersomnolence: A reappraisal by European experts. *Sleep medicine reviews, 52*, 101306. https://doi.org/10.1016/j.smrv.2020.101306

Laskemoen, J. F., Simonsen, C., Büchmann, C., Barrett, E. A., Bjella, T., Lagerberg, T. V., Vedal, T. J., Andreassen, O. A., Melle, I., & Aas, M. (2019). Sleep disturbances in schizophrenia spectrum and bipolar disorders - a transdiagnostic perspective. *Comprehensive psychiatry, 91*, 6–12. https://doi.org/10.1016/j.comppsych.2019.02.006

Lawrence, D., Hancock, K. J., & Kisely, S. (2013). The Gap in Life Expectancy from Preventable Physical Illness in Psychiatric Patients in Western Australia: Retrospective Analysis of Population Based Registers. *BMJ, 346*, f2539. https://doi.org/10.1136/bmj.f2539

Leibenluft, E., Turner, E. H., Feldman-Naim, S., Schwartz, P. J.,Wehr, T. A., Rosenthal, N. E. (1995). Light therapy in patients with rapid cycling bipolar disorder: preliminary results. *Psychopharmacology Bulletin, 31*(4):705–710.

Lindenmeyer, A., Hearnshaw, H., Sturt, J., Ormerod, R., & Aitchison, G. (2007). Assessment of the benefits of user involvement in health research from the Warwick Diabetes Care Research User Group: a qualitative case study. *Health expectations: an international journal of public participation in health care and health policy, 10*(3), 268–277. https://doi.org/10.1111/j.1369-7625.2007.00451.x

Liu, X., Uchiyama, M., Kim, K., Okawa, M., Shibui, K., Kudo, Y., Doi, Y., Minowa, M., & Ogihara, R. (2000). Sleep loss and daytime sleepiness in the general adult population of Japan. *Psychiatry research, 93*(1), 1–11. https://doi.org/10.1016/s0165-1781(99)00119-5

Lopes, J. M., Dantas, F. G., & Medeiros, J. L. (2013). Excessive daytime sleepiness in the elderly: association with cardiovascular risk, obesity and depression. *Revista brasileira de epidemiologia = Brazilian journal of epidemiology, 16*(4), 872–879. https://doi.org/10.1590/s1415-790x2013000400007

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales* (2nd ed.). Psychology Foundation of Australia.

Martin, J., Jeste, D. V., Caliguiri, M. P., Patterson, T., Heaton, R., & Ancoli-Israel, S. (2001). Actigraphic estimates of circadian rhythms and sleep/wake in older schizophrenia patients. *Schizophrenia research, 47*(1), 77–86. https://doi.org/10.1016/s0920-9964(00)00029-3

Maruani, J., Boiret, C., Leseur, J., Romier, A., Bazin, B., Stern, E., Lejoyeux, M., & Geoffroy, P. A. (2023a). Major depressive episode with insomnia and excessive daytime sleepiness: A more homogeneous and severe subtype of depression. *Psychiatry Research, 330*, 115603. https://doi.org/10.1016/j.psychres.2023.115603

Maruani, J., Molière, F., Godin, O., Yrondi, A., Bennabi, D., Richieri, R., El-Hage, W., Allauze, E., Anguill, L., Bouvard, A., Camus, V., Dorey, J. M., Etain, B., Fond, G., Genty, J. B., Haffen, E., Holtzmann, J., Horn, M., Kazour, F., Nguon, A. S., … Geoffroy, P. A. (2023b). Diurnal symptoms of sleepiness and dysfunction predict future suicidal ideation in a French cohort of outpatients (FACE-DR) with treatment resistant depression: A 1-year prospective study about sleep markers. *Journal of affective disorders, 329*, 369–378. https://doi.org/10.1016/j.jad.2023.02.106

Matza, L. S., Revicki, D. A., Davidson, J. R., & Stewart, J. W. (2003). Depression with atypical features in the National Comorbidity Survey: classification, description, and consequences. *Archives of general psychiatry, 60*(8), 817–826. https://doi.org/10.1001/archpsyc.60.8.817

Miller, D. D. (2004). Atypical antipsychotics: sleep, sedation, and efficacy. *Primary care companion to the Journal of clinical psychiatry, 6*(Suppl 2), 3–7.

Miller, M. A. (2011). Association of inflammatory markers with cardiovascular risk and sleepiness. *Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine, 7*(5 Suppl), S31–S33. https://doi.org/10.5664/JCSM.1356

Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G., & PRISMA Group (2009). Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS medicine, 6*(7), e1000097. https://doi.org/10.1371/journal.pmed.1000097

Monti, J. M., & Monti, D. (2005). Sleep disturbance in schizophrenia. *International Review of Psychiatry, 17*(4), 247-253. https://doi.org/10.1080/09540260500104516

Morin, C. M., Belleville, G., Bélanger, L., & Ivers, H. (2011). The Insomnia Severity Index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep, 34*(5), 601–608. https://doi.org/10.1093/sleep/34.5.601

Mullington, J., Broughton, R. (1993). Scheduled naps in the management of daytime sleepiness in narcolepsy-cataplexy. *Sleep, 16*(5), 444–456. https://doi.org/10.1093/sleep/16.5.444

Mume, C. O. (2010). Excessive daytime sleepiness among depressed patients. *The Libyan journal of medicine, 5*(1), 4626. https://doi.org/10.3402/ljm.v5i0.4626

Murru, A., Guiso, G., Barbuti, M., Anmella, G., Verdolini, N., Samalin, L., Azorin, J. M., Angst, J. J., Bowden, C. L., Mosolov, S., Young, A. H., Popovic, D., Valdes, M., Perugi, G., Vieta, E., Pacchiarotti, I., & BRIDGE-II-Mix Study Group (2019). The implications of hypersomnia in the context of major depression: Results from a large, international, observational study. European neuropsychopharmacology: the journal of the European College of *Neuropsychopharmacology, 29*(4), 471–481. https://doi.org/10.1016/j.euroneuro.2019.02.011

Nofzinger, E.A, Thase, M.E., Reynolds, 3rd C.F., Himmelhoch, J.M., Mallinger, A., Houck, P. et al. (1991). Hypersomnia in bipolar depression: a comparison with narcolepsy using the multiple sleep latency test. *The American journal of psychiatry, 148*(9), 1177–1181. https://doi.org/10.1176/ajp.148.9.1177

Ong, J.C., Schuiling, M. D. (2022). CBT-I for patients with hypersomnia disorders. In S. Nowakowski, S. N., Garland, M. A. Grandner, J. Leisha, L. J. Cuddihy (Eds). *Adapting Cognitive Behavioural Therapy for Insomnia. Academic Press.* https://doi.org/10.1016/B978-0-12-822872-2.00018-9

Ohayon, M. M., & Roth, T. (2003). Place of chronic insomnia in the course of depressive and anxiety disorders. *Journal of Psychiatric Research, 37*(1), 9–15. https://doi.org/10.1016/S0022-3956(02)00052-3

Ohayon, M. M. (2013). Narcolepsy is complicated by high medical and psychiatric comorbidities: a comparison with the general population. *Sleep medicine, 14*(6), 488–492. https://doi.org/10.1016/j.sleep.2013.03.002

Okruszek, Ł., Jernajczyk, W., Wierzbicka, A., Waliniowska, E., Jakubczyk, T., Jarema, M., & Wichniak, A. (2014). Daytime sleepiness and EEG abnormalities in patients treated with second generation antipsychotic agents. *Pharmacological Reports, 66*, 1077–1082

Ong, J. C., Dawson, S. C., Mundt, J. M., & Moore, C. (2020). Developing a cognitive behavioural therapy for hypersomnia using telehealth: a feasibility study. *Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine, 16*(12), 2047–2062. https://doi.org/10.5664/jcsm.8750

Pallesen, S., Nordhus, I. H., Omvik, S., Sivertsen, B., Tell, G. S, Bjorvatn, B. (2007). Prevalence and risk factors of subjective sleepiness in the general adult population. *Sleep. May;30*(5):619-24. https://doi.org/10.1093/sleep/30.5.619

Patarroyo-Rodriguez, L., Singh, B., Pazdernik, V., & Vande Voort, J. L. (2024). Hypersomnia as a predictor of response to intravenous ketamine/intranasal esketamine in treatment-resistant depression. *Journal of Affective Disorders, 349*, 272–276.

Patterson, S., Trite, J., Weaver, T. (2014). Activity and views of service users involved in mental health research: UK survey. *British Journal of Psychiatry, 205*, 68-75.

Pérez-Carbonell, L., & Leschziner, G. (2018). Clinical update on central hypersomnias. *Journal of thoracic disease, 10*(Suppl 1), S112–S123. https://doi.org/10.21037/jtd.2017.10.161

Plante, D. T., Landsness, E. C., Peterson, M. J., Goldstein, M. R., Wanger, T., Guokas, J. J., Tononi, G., & Benca, R. M. (2012). Altered slow wave activity in major depressive disorder with hypersomnia: a high-density EEG pilot study. *Psychiatry research, 201*(3), 240–244. https://doi.org/10.1016/j.pscychresns.2012.03.001

Plante, D. T. (2015). Hypersomnia in Mood Disorders: A Rapidly Changing Landscape. *Current sleep medicine reports, 1*(2), 122–130. https://doi.org/10.1007/s40675-015-0017-9

Plante, D. T., Birn, R. M., Walsh, E. C., Hoks, R. M., Cornejo, M. D., & Abercrombie, H. C. (2018). Reduced resting-state thalamostriatal functional connectivity is associated with excessive daytime sleepiness in persons with and without depressive disorders. *Journal of affective disorders, 227*, 517–520. https://doi.org/10.1016/j.jad.2017.11.054

Popay, J., Roberts, H., Sowden, A., Petticrew, M., Arai, L., Rodgers, M., Britten, N., Roen, K., & Duffy, S. (2006). Guidance on the conduct of narrative synthesis in systematic reviews: A product from the ESRC Methods Programme. *Economic and Social Research Council (ESRC)*. Retrieved from https://www.lancaster.ac.uk/shm/research/nssr/research/dissemination/publications/NS_guidance_v1.pdf

Poulin, J., Chouinard, S., Pampoulova, T., Lecomte, Y., Stip, E., & Godbout, R. (2010). Sleep habits in middle-aged, non-hospitalized men and women with schizophrenia: a comparison with healthy controls. *Psychiatry research, 179*(3), 274–278. https://doi.org/10.1016/j.psychres.2009.08.009

Reeve, S., Sheaves, B., & Freeman, D. (2015). The role of sleep dysfunction in the occurrence of delusions and hallucinations: A systematic review. *Clinical psychology review, 42*, 96–115. https://doi.org/10.1016/j.cpr.2015.09.001

Reeve, S., Emsley, R., Sheaves, B., & Freeman, D. (2018). Disrupting Sleep: The Effects of Sleep Loss on Psychotic Experiences Tested in an Experimental Study with Mediation Analysis. *Schizophrenia bulletin, 44*(3), 662–671. https://doi.org/10.1093/schbul/sbx103

Reeve, S., Nickless, A., Sheaves, B., Hodsoll, J., Roberts, A., & Freeman, D. (2019). Sleep loss as a trigger of severe mental illness: A population-based case-crossover study. *Sleep, 42*(10), zsz150. https://doi.org/10.1093/sleep/zsz150

Reeve, S., Sheaves, B., & Freeman, D. (2019). Sleep Disorders in Early Psychosis: Incidence, Severity, and Association with Clinical Symptoms. *Schizophrenia bulletin, 45*(2), 287–295. https://doi.org/10.1093/schbul/sby129

Reeve, S., Sheaves B., Freeman, D. (2021). Excessive sleepiness in patients with psychosis: An initial investigation. *PLoS ONE 16*(1): e0245301. https://doi.org/10.1371/journal.pone.0245301

Rehman, A., Waite, F., Sheaves, B., Biello, S., Freeman, D., & Gumley, A. (2017). Clinician perceptions of sleep problems, and their treatment, in patients with non-affective psychosis. *Psychosis, 9*(2), 129–139. https://doi.org/10.1080/17522439.2016.1206955

Risco, J., Ruiz, P., Mariños, A., Juarez, A., Ramos, M., Salmavides, F., Vega, J., Kruger, H., & Vizcarra, D. (2013). Excessive sleepiness prevalence in public transportation drivers of a developing country. *Traffic injury prevention, 14*(2), 145–149. https://doi.org/10.1080/15389588.2012.692493

Roberts, R. E., Shema, S. J., Kaplan, G. A., & Strawbridge, W. J. (2000). Sleep complaints and depression in an aging cohort: A prospective perspective. *The American journal of psychiatry, 157*(1), 81–88. https://doi.org/10.1176/ajp.157.1.81

Rogers, A. E., Aldrich, M. S., & Lin, X. (2001). A comparison of three different sleep schedules for reducing daytime sleepiness in narcolepsy. *Sleep, 24*(4), 385–391. https://doi.org/10.1093/sleep/24.4.385

Ronald, A., Sieradzka, D., Cardno, A. G., Haworth, C. M. A., McGuire, P., & Freeman, D. (2014). Characterization of psychotic experiences in adolescence using the Specific Psychotic Experiences Questionnaire: Findings from a study of 5000 16-year-old twins. *Schizophrenia Bulletin, 40*(4), 868–877. https://doi.org/10.1093/schbul/sbt106

Rosenbaum, S., Morell, R., Abdel-Baki, A., Ahmadpanah, M., Anilkumar, T. V., Baie, L., Bauman, A., Bender, S., Boyan Han, J., Brand, S., Bratland-Sanda, S., Bueno-Antequera, J., Camaz Deslandes, A., Carneiro, L., Carraro, A., Castañeda, C. P., Castro Monteiro, F., Chapman, J., Chau, J. Y., Chen, L. J., … Ward, P. B. (2020). Assessing physical activity in people with mental illness: 23-country reliability and validity of the simple physical activity questionnaire (SIMPAQ). *BMC psychiatry, 20*(1), 108. https://doi.org/10.1186/s12888-020-2473-0

Sharma, P., Dikshit, R., Shah, N., Karia, S., & De Sousa, A. (2016). Excessive Daytime Sleepiness in Schizophrenia: A Naturalistic Clinical Study. *Journal of clinical and diagnostic research: JCDR, 10*(10), VC06–VC08. https://doi.org/10.7860/JCDR/2016/21272.8627

Sheaves, B., Isham, L., Bradley, J., Espie, C., Barrera, A., Waite, F., Harvey, A. G., Attard, C., & Freeman, D. (2018). Adapted CBT to Stabilize Sleep on Psychiatric Wards: a Transdiagnostic Treatment Approach. *Behavioural and cognitive psychotherapy, 46*(6), 661–675. https://doi.org/10.1017/S1352465817000789

Shen, J., Hossain, N., Streiner, D. L., Ravindran, A. V., Wang, X., Deb, P., Huang, X., Sun, F., & Shapiro, C. M. (2011). Excessive daytime sleepiness and fatigue in depressed patients and therapeutic response of a sedating antidepressant. *Journal of affective disorders, 134*(1-3), 421–426. https://doi.org/10.1016/j.jad.2011.04.047

Simpson, A., Jones, J., Barlow, S., Cox, L. (2014). Adding SUGAR service user and carer collaboration in mental health nursing research. *Journal of Psychosocial Nursing and Mental Health Services, 52*, 22-30.

Soehner, A. M., Kaplan, K. A., & Harvey, A. G. (2014). Prevalence and clinical correlates of co-occurring insomnia and hypersomnia symptoms in depression. *Journal of affective disorders, 167*, 93–97. https://doi.org/10.1016/j.jad.2014.05.060

Stern, C., Lizarondo, L., Carrier, J., Godfrey, C., Rieger, K., Salmond, S., Apóstolo, J., Kirkpatrick, P., & Loveday, H. (2020). Methodological guidance for the conduct of mixed methods systematic reviews. *JBI evidence synthesis, 18*(10), 2108–2118. https://doi.org/10.11124/JBISRIR-D-19-00169

Strassnig, M., Caceda, R., Newcomer, J., Harvey, P. D. (2012). Cognitive deficits, obesity and disability in schizophrenia. *Translational Neuroscience, 3*(4), 345–354. https://doi.org/10.2478/s13380-012-0046-5

Stubbs, B., Williams, J., Gaughran, F., & Craig, T. (2016). How sedentary are people with psychosis? A systematic review and meta-analysis. *Schizophrenia research, 171*(1-3), 103–109. https://doi.org/10.1016/j.schres.2016.01.034

Thase, M. E., Fava, M., DeBattista, C., Arora, S., & Hughes, R. J. (2006). Modafinil augmentation of SSRI therapy in patients with major depressive disorder and excessive sleepiness and fatigue:

a 12-week, open-label, extension study. *CNS spectrums, 11*(2), 93–102. https://doi.org/10.1017/s1092852900010622

Thompson, J., Barber, R., Ward, P. R., Boote, J. D., Cooper, C. L., Armitage, C. J. (2009). Health researchers' attitudes towards public involvement in health research. *Health Expectations, 12*, 209-220.

Trotti, L. M. (2017). Waking up is the hardest thing I do all day: Sleep inertia and sleep drunkenness. Sleep medicine reviews, 35, 76–84. https://doi.org/10.1016/j.smrv.2016.08.005

Tsai, S. C. (2010). Excessive sleepiness. *Clinics in chest medicine, 31*(2), 341–351. https://doi.org/10.1016/j.ccm.2010.02.007

Tsuno, N., Besset, A., & Ritchie, K. (2005). Sleep and depression. *Journal of Clinical Psychiatry, 66*(10), 1254–1269. https://doi.org/10.4088/JCP.v66n1008

Vancampfort, D., Firth, J., Schuch, F. B., Rosenbaum, S., Mugisha, J., Hallgren, M., Probst, M., Ward, P. B., Gaughran, F., De Hert, M., Carvalho, A. F., & Stubbs, B. (2017). Sedentary behavior and physical activity levels in people with schizophrenia, bipolar disorder and major depressive disorder: a global systematic review and meta-analysis. *World psychiatry: official journal of the World Psychiatric Association (WPA), 16*(3), 308–315. https://doi.org/10.1002/wps.20458

van der Heide, A., van Schie, M. K., Lammers, G. J., Dauvilliers, Y., Arnulf, I., Mayer, G., Bassetti, C. L., Ding, C. L., Lehert, P., & van Dijk, J. G. (2015). Comparing Treatment Effect Measurements in Narcolepsy: The Sustained Attention to Response Task, Epworth Sleepiness Scale and Maintenance of Wakefulness Test. *Sleep, 38*(7), 1051–1058. https://doi.org/10.5665/sleep.4810

Vorontsova, N., Garety, P., & Freeman, D. (2013). Cognitive factors maintaining persecutory delusions in psychosis: The contribution of depression. *Journal of Abnormal Psychology, 122*(4), 1121–1131. https://doi.org/10.1037/a0034952

Waite, F., Myers, E., Harvey, A. G., Espie, C. A., Startup, H., Sheaves, B., & Freeman, D. (2016). Treating Sleep Problems in Patients with Schizophrenia. *Behavioural and cognitive psychotherapy, 44*(3), 273–287. https://doi.org/10.1017/S1352465815000430

Waite, F., Sheaves, B., Isham, L., Reeve, S., & Freeman, D. (2020). Sleep and schizophrenia: From epiphenomenon to treatable causal target. *Schizophrenia research, 221*, 44–56. https://doi.org/10.1016/j.schres.2019.11.014

Walker, E. R., McGee, R. E., & Druss, B. G. (2015). Mortality in mental disorders and global disease burden implications: a systematic review and meta-analysis. *JAMA psychiatry, 72*(4), 334–341. https://doi.org/10.1001/jamapsychiatry.2014.2502

Wasilewska, J., Jarocka-Cyrta, E., & Kaczmarski, M. (2007). Narcolepsy, metabolic syndrome and obstructive sleep apnea syndrome as the causes of hypersomnia in children. Report of three cases. *Advances in medical sciences, 52*(Suppl 1), 207–211.

Wescott, D. L., Franzen, P. L., Hasler, B. P., Miller, M. A., Soehner, A. M., Smagula, S. F., Wallace, M. L., Hall, M. H., & Roecklein, K. A. (2023). Elusive hypersomnolence in seasonal affective disorder: actigraphic and self-reported sleep in and out of depressive episodes. *Psychological medicine, 53*(4), 1313–1322. https://doi.org/10.1017/S003329172100283X

Wirz-Justice, A., Benedetti, F., & Terman, M. (2013). *Chronotherapeutics for affective disorders: A clinician's manual for light and wake therapy (*2nd rev. ed*.).* Karger

Wulff, K., Gatti, S., Wettstein, J. G., & Foster, R. G. (2010). Sleep and circadian rhythm *disruption in psychiatric and neurodegenerative disease. Nature Reviews Neuroscience, 11*(8), 589–599. https://doi.org/10.1038/nrn2868

Wulff, K., Dijk, D. J, Middleton, B., Foster, R. G., Joyce, E. M. (2012). Sleep and circadian rhythm disruption in schizophrenia. *Br J Psychiatry,200*(4):308-16. https://doi.org/10.1192/bjp.bp.111.096321

Wykes, T., Huddy, V., Cellard, C., McGurk, S. R., & Czobor, P. (2011). A meta-analysis of cognitive remediation for schizophrenia: Methodology and effect sizes. *American Journal of Psychiatry, 168*(5), 472–485.

YoEargon, M. Y., Kara, H., & Solmaz, M. (1997). Sleep disturbances and suicidal behavior in patients with major depression. *The Journal of clinical psychiatry, 58*(6), 249–251. https://doi.org/10.4088/jcp.v58n0602

Zhang, Y., Folarin, A. A., Sun, S., Cummins, N., Bendayan, R., Ranjan, Y., Rashid, Z., Conde, P., Stewart, C., Laiou, P., Matcham, F., White, K. M., Lamers, F., Siddi, S., Simblett, S., Myin-Germeys, I., Rintala, A., Wykes, T., Haro, J. M., Penninx, B. W., … RADAR-CNS Consortium (2021). Relationship Between Major Depression Symptom Severity and Sleep Collected Using a Wristband Wearable Device: Multicenter Longitudinal Observational Study. *JMIR mHealth and uHealth, 9*(4), e24604. https://doi.org/10.2196/24604

Zhao, J., Liu, H., Wu, Z., Wang, Y., Cao, T., Lyu, D., Huang, Q., Wu, Z., Zhu, Y., Wu, X., Chen, J., Wang, Y., Su, Y., Zhang, C., Peng, D., Li, Z., Rong, H., Liu, T., Xia, Y., Hong, W., … Fang, Y. (2021). Clinical features of the patients with major depressive disorder co-occurring insomnia and hypersomnia symptoms: a report of NSSD study. *Sleep medicine, 81*, 375–381. https://doi.org/10.1016/j.sleep.2021.03.005

Zimmerman, M., McGlinchey, J. B., Posternak, M. A., Friedman, M., Boerescu, D., & Attiullah, N. (2005). Differences between minimally depressed patients who do and do not consider themselves to be in remission. *The Journal of clinical psychiatry, 66*(9), 1134–1138. https://doi.org/10.4088/jcp.v66n0908

Appendices

Appendix A

Demographic and Medical Background

About You:

Please complete the following questions about yourself to help us understand more about the people completing this survey.

Age:

Gender:

Female

I identify as another gender

Prefer not to say

Please provide further details if you identify as another gender.

Ethnicity:

Asian (any background)

Black (any background)

Mixed or Multiple ethnic groups

White (any background)

Other

Prefer not to say

If other, please specify below:

Occupational status:

Full-time employed (over/equal to 35 hours per week, inc. self-employed)

Part-time employed (under 35 hours per week, inc self-employed)

Unemployed

Student

Homemaker/carer

Retired

Highest level of education completed:

Did not complete secondary education

Primary School

High / Secondary School or equivalent [e.g. GCSE, NVQ]

Sixth Form / College or equivalent [e.g. A-Level; Bac; BTEC]

Higher Education Diploma [e.g. Certificate of Higher Education; Level 4 Diploma]

Undergraduate university degree [e.g. BA; BSc]

Postgraduate university degree [e.g. MA; MSc; PGCE]

Doctorate degree [e.g. PhD]

Mental health information:

Please select your psychotic disorder diagnosis from the below list :

First episode psychosis

Schizophrenia

Schizotypal disorder

Delusional disorder

Schizoaffective disorder

Psychotic disorder

Psychotic disorder NOS (not otherwise specified)

Not sure

No diagnosis

Other

If other diagnosis, please specify below:

Do you take medication for your mental health?

Yes

No

Mental health medication question

Can you provide the name and dosage of your mental health medication?

Yes

No

I don't take any mental health medication

Please provide below the name and dosage of your mental health medication.

Mental health team question

What CPFT mental health team are you seen under?

Not under CPFT NHS care (recruited from social media)

CAMEO North (Peterborough)

CAMEO South (Cambridge)

Cambridge adult locality team (CALT)

Fenland adult locality team (FALT)

Huntingdon adult locality team (HALT)

Peterborough adult locality team (PALT)

Not sure

Other team

If other team, please specify below:

Appendix B

Sleepiness Questions

1. Over the last month, did you typically sleep over 11 hours in a 24- hour period? (including overnight sleep and any daytime sleep) (Y/N)
2. Over the last month, did you typically sleep over 9 hours at night? (Y/N)
3. Over the last month, how long did you typically sleep each night? (HH:MM)
4. Over the last month, did you feel excessively sleepy during the day, to the extent it was difficult to stay awake or take part in activities? (Y/N)
5. If yes – how many days in a typical week? (0-7)

Appendix C

Outcome Measures

Epworth Sleepiness Scale (ESS)

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired?

This refers to your usual way of life in recent times. Even if you haven't done some of these things recently try to work out how they would have affected you.

Use the following scale to choose **the most appropriate number** for each situation.

0 = would **never** doze

1 = **slight chance** of dozing

2 = **moderate chance** of dozing

3 = **high chance** of dozing

It is important that you answer each question as best you can.

Situation	**Chance of Dozing (0-3)**
Sitting and reading	
Watching TV	
Sitting, inactive in a public place (e.g. a theatre or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	

Hypersomnia severity index (HSI)

1. For these next few questions, please consider your SLEEP IN THE PAST MONTH. To what extent do you think that you:

	Not at All	**A Little**	**Somewhat**	**A Lot**	**Very Much**
Sleep too much at night?	0	1	2	3	4
Have difficulty waking up in the morning or from naps?	0	1	2	3	4
Sleep during the day?	0	1	2	3	4
Feel sleepy during the daytime?	0	1	2	3	4

2. How SATISFIED/dissatisfied are you with your current sleep pattern?
   - Very satisfied 0
   - Satisfied 1
   - Moderately satisfied 2
   - Dissatisfied 3
   - Very dissatisfied 4

3. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g., daytime fatigue, ability to function at work/daily chores, concentration, memory, mood, etc.)?

   Not at all 0 | A little 1 | Somewhat 2 | Much 3 | Very much 4

4. How NOTICEABLE to others do you think your sleeping problem is in terms of impairing the quality of your life?

   Not at all Noticeable 0 | Barely 1 | Somewhat 2 | Much 3 | Very much Noticeable 4

5. How WORRIED/DISTRESSED are you about your current sleep problem?

   Not at all 0 | A little 1 | Somewhat 2 | Much 3 | Very much 4

6. Do you ever have “sleep attacks,” defined as unintended sleep in inappropriate situations?

- Not at all 0
- A little 1
- Sometimes 2
- Often 3
- All the time 4

The Integrated tiredness index (ITI)

This questionnaire assesses the impact of tiredness. In the last week, how much of an issue have you had with….					
(circle as appropriate)	**0 (none)**	**1 (slight)**	**2 (moderate)**	**3 (very much)**	**4 (extreme)**
1. Feeling worn out	0	1	2	3	4
2. Feeling dozy	0	1	2	3	4
3. Feeling half awake	0	1	2	3	4
4. Feeling drained	0	1	2	3	4
5. Feeling sleepy	0	1	2	3	4
6. Feeling low in energy	0	1	2	3	4
7. Feeling fatigued	0	1	2	3	4
8. Feeling drowsy	0	1	2	3	4

Items 1,4,6,7 – fatigue

Items 2,3,5,8 – sleepiness

Fatigue Severity Scale (FSS)

Please circle the number between 1 and 7 which you feel best fits the following statements. This refers to your usual way of life within the last week. 1 indicates “strongly disagree” and 7 indicates “strongly agree.”

Read and circle a number	**Strongly Disagree -> Strongly agree**						
My motivation is lower when I am fatigued	1	2	3	4	5	6	7
Exercise brings on my fatigue	1	2	3	4	5	6	7
I am easily fatigued	1	2	3	4	5	6	7
Fatigue interferes with my physical functioning	1	2	3	4	5	6	7
Fatigue causes frequent problems for me	1	2	3	4	5	6	7
My fatigue prevents sustained physical functioning	1	2	3	4	5	6	7
Fatigue interferes with carrying out certain duties and responsibilities	1	2	3	4	5	6	7
Fatigue is among my most disabling symptoms	1	2	3	4	5	6	7
Fatigue interferes with my work, family, or social life	1	2	3	4	5	6	7

Insomnia severity index (ISI)

The following questions ask about your sleep in the past two weeks. For each question, please CIRCLE the number that best describes your answer.

Please rate the following in relation to your CURRENT sleep (**in the past *two weeks)***.

Sleep problem	None	Mild	Moderate	Severe	Very Severe
1. Difficulty falling asleep	0	1	2	3	4
2. Difficulty staying asleep	0	1	2	3	4
3. Problem waking up too early	0	1	2	3	4

4. How satisfied/ dissatisfied are you with your current sleep pattern?

- Very satisfied 0
- Satisfied 1
- Moderately satisfied 2
- Dissatisfied 3
- Very dissatisfied 4

5. To what extent do you consider these sleep problems to interfere with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood etc.).

Not at all interfering	A little	Somewhat	Much	Very much interfering
0	1	2	3	4

6**.** How noticeable to others do you think these sleep problems are in terms of impairing the quality of your life?

Not at all noticeable	Barely	Somewhat	Much	Very much noticeable
0	1	2	3	4

7. How worried/distressed are you about these sleep problems?

Not at all worried	A little	Somewhat	Much	Very much worried
0	1	2	3	4

Depression Anxiety Stress Scale (DASS)

Please read each statement and select the option that best indicates how much the statement applies to you. There are no right or wrong answers. Try not to spend too much time on any statement.			
Did not apply to me at all	**Applied to me to some degree, or some of the time**	**Applied to me to a considerable degree, or a good part of the time**	**Applied to me very much or most of the time**
0	**1**	**2**	**3**
1. I was aware of dryness of my mouth			
0	1	2	3
2. I couldn't seem to experience any positive feeling at all			
0	1	2	3
3. I experienced breathing difficulty (e.g. excessively rapid breathing, breathlessness in the absence of physical exertion)			
0	1	2	3
4. I found it difficult to work up the initiative to do things			
0	1	2	3
5. I experienced trembling (e.g. in the hands)			
0	1	2	3
6. I was worried about situations in which I might panic and make a fool of myself			
0	1	2	3
7. I felt that I had nothing to look forward to			
0	1	2	3
8. I felt downhearted and blue			
0	1	2	3
9. I felt I was close to panic			
0	1	2	3
10. I was unable to become enthusiastic about anything			
0	1	2	3
11. I felt I wasn't worth much as a person			
0	1	2	3
12. I was aware of the action of my heart in the absence of physical exertion (e.g. sense of heart rate increase, heart missing a beat)			
0	1	2	3
13. I felt scared without any good reason			
0	1	2	3
14. I felt that life was meaningless			
0	1	2	3

The Revised Green et al. Paranoid Thoughts Scale (R-GPTS)
R-GPTS Part A (Reference) scale

Please read each of the statements carefully. They refer to thoughts and feelings you may have had about others over the last month. Think about the last month and indicate the extent of these feelings from 0 (Not at all) to 4 (Totally). (Please do not rate items according to any experiences you may have had under the influence of drugs.)

	Not at all		Somewhat		Totally
1. I spent time thinking about friends gossiping about me.	0	1	2	3	4
2. I often heard people referring to me.	0	1	2	3	4
3. I have been upset by friends and colleagues judging me critically.	0	1	2	3	4
4. People definitely laughed at me behind my back.	0	1	2	3	4
5. I have been thinking a lot about people avoiding me.	0	1	2	3	4
6. People have been dropping hints for me.	0	1	2	3	4
7. I believed that certain people were not what they seemed.	0	1	2	3	4
8. People talking about me behind my back upset me.	0	1	2	3	4

R-GPTS Part B (Persecution) scale

Please read each of the statements carefully. They refer to thoughts and feelings you may have had about others over the last month. Think about the last month and indicate the extent of these feelings from 0 (Not at all) to 4 (Totally). (Please do not rate items according to any experiences you may have had under the influence of drugs.)

		Not at all		Somewhat		Totally
1.	Certain individuals have had it in for me.	0	1	2	3	4
2.	People wanted me to feel threatened, so they stared at me.	0	1	2	3	4
3.	I was certain people did things in order to annoy me.	0	1	2	3	4
4.	I was convinced there was a conspiracy against me.	0	1	2	3	4
5.	I was sure someone wanted to hurt me.	0	1	2	3	4
6.	I couldn't stop thinking about people wanting to confuse me.	0	1	2	3	4
7.	I was distressed by being persecuted.	0	1	2	3	4
8.	It was difficult to stop thinking about people wanting to make me feel bad.	0	1	2	3	4
9	People have been hostile towards me on purpose.	0	1	2	3	4
10	I was angry that someone wanted to hurt me.	0	1	2	3	4

SPEQ hallucinations scale

Please read each statement and click the number that best indicates how frequently you have each of these experiences. There are no right or wrong answers. Try not to spend too much time on any statement.					
Not at all 0	**Rarely** 1	**Once a month** 2	**Once a week** 3	**Several times a week** 4	**Daily** 5
1. Hear sounds or music that people near you don't hear?					
0	1	2	3	4	5
2. See things that other people cannot?					
0	1	2	3	4	5
3. Feel that someone is touching you, but when you look nobody is there?					
0	1	2	3	4	5
4. Hear noises or sounds when there is nothing around to explain them?					
0	1	2	3	4	5
5. Detect smells which don't seem to come from your surroundings?					
0	1	2	3	4	5
6. See shapes, lights, or colours even though there is nothing really there?					
0	1	2	3	4	5
7. Notice smells or odours that people next to you seem unaware of?					
0	1	2	3	4	5
8. Experience unusual burning sensations or other strange feelings in or on your body that can't be explained?					
0	1	2	3	4	5
9. Hear voices commenting on what you're thinking or doing?					
0	1	2	3	4	5
Overall, how distressed are you by these experiences?					
0 (not distressed)		**1 (a bit distressed)**	**2 (quite distressed)**		**3 (very distressed)**

Recovering quality of life (REQOL)

For each of the following statements, please tick one box that best describes your thoughts, feelings and activities over the last week

Over the last week	None of the time	Only occasionally	Sometimes	Often	Most or all of the time
I found it difficult to get started with everyday tasks					
I feel able to trust others					
I felt unable to cope					
I could do the things I wanted to do					
I felt happy					
I thought my life was not worth living					
I enjoyed what I did					
I felt hopeful about my future					
I felt lonely					
I felt confident in myself					
	No problems	Slight problems	Moderate problems	Severe problems	Very severe problems
Please describe your physical health (problems with pain, mobility, difficulties caring for yourself or feeling physically unwell) over the last week					

None of the time = 0, Most or all of the time =4

Items 1, 3, 6, 9 and the physical health item are reverse scored

Physical activity questions (adapted SIMPAQ)

We are interested in your physical activity levels over the past seven days. These questions are optional, so you can skip them by selecting the appropriate option.

Over the past week, how long did you spend...

Sleeping or in bed (hours and minutes) e.g. 8 hours would be "8", 9 and a half hours would be "9.5"

Sitting or lying down (e.g. napping, eating, reading, watching TV, using electronic devices (hours and minutes)

Walking (hours and minutes)

Doing active exercise (e.g. jogging, running, swimming, bike riding, going to the gym, yoga) (hours and minutes)

Appendix D

Participant Information Sheet

Participant Information Sheet (Survey) Version 3:

19/06/2023

Sleepiness in Psychosis

Introduction

We would like to invite you to take part in a research project. Before you decide you need to understand why the research is being done and what it will involve for you. Please take the time to read the following information carefully and ask questions about anything you do not understand. Talk to others about the study if you wish.

If you need further information our contact details are at the end of this page.

What is the purpose of this study?

The project will aim to investigate the frequency, causes, and effects of excessive sleepiness among people who live with psychosis. This is a project being undertaken by a trainee on the Doctorate in Clinical Psychology at University of East Anglia (UEA), who are the sponsor organisation for this project. Where 'we' is written, reference is to the researchers who are conducting this study (Ioana Marinescu and Kate Robbins) who study at UEA.

Why have I been invited to take part in the study?

You have been invited to take part in this study because you are aged between 18-65 and have a diagnosis of a psychotic disorder (including schizophrenia, schizoaffective disorder, first episode psychosis, or psychotic disorder not otherwise specified), and have either responded to a social media advertisement or are under Cambridgeshire and Peterborough

NHS Foundation Trust mental health services (including CAMEO North, CAMEO South, Peterborough Adult Locality Team (PALT) , Fenland Adult Locality Team (FALT), Huntingdon Adult Locality Team (HALT) and Cambridge Adult Locality Team (CALT)).

What will happen to me if I take part?

We will ask you to complete an online survey which will take between 30-45 minutes. The survey will include questionnaires about your sleep, medication, mood, quality of life and physical activity.

At the end of the survey, you will be asked if you are willing to enter a prize draw for one of ten £10 amazon vouchers. This is optional and you will need to provide your email address in order to be contacted - the winners will be chosen randomly once the study is complete (latest March 2024).

You will also be asked if you are willing to take part in a follow-up online interview (maximum 1hr) about your sleep. Again, this is optional, and you will need to provide your email address for us to contact you with if you are eligible based on your survey responses. If you have questions about the interview, you can contact Kate Robbins (k.robbins@uea.ac.uk) who can provide further information. If you do take part in the interview, you will receive a £10 amazon voucher as a thank you for your time.

Do I have to take part?

No. It is up to you whether you wish to take part in the survey. If you are willing to take part, please complete the consent form at the bottom of this page to progress to the survey. To leave the study you can navigate away from this page. After you have consented you can leave the survey at any time by navigating away from the page. Only complete responses will be used for analysing the data. All the incomplete responses will be removed.

What are the benefits of taking part?

We hope that this work will benefit our understanding of and potential treatment options for excessive sleepiness, which will ultimately improve quality of life for people living with psychosis. Participants will also be thanked for their time via a prize draw for one of ten £10 Amazon vouchers, plus additional£10Amazon vouchers available for interview participants.

Will there be any risks or other health implications of taking part in this study?

We are not aware of any risks or health implications to you by taking part in this research. However, if going through the survey does make you feel distressed, we have provided links to support services at the end of the survey. You can contact your GP for mental health support or your clinical team if you are supported by mental health services.

In the event that you are harmed or wish to complain about the survey, please approach the researcher Ioana Marinescu i.marinescu @uea.ac.uk or supervisor Dr. Sarah Reeve (sarah.reeve@uea.ac.uk), or you can contact [independent PALS service].

How will we use information about you?

We will need to use information from you for this research project. If you consent to provide your contact details for the prize draw or for contact about the follow-up interview and the study findings, this information will include your contact details. People who do not need to know who you are will not be able to see your contact details. Your data will have a code number instead. We will keep all information about you safe and secure.

Only complete responses will be used for analysing the data. All the incomplete responses will be removed. We will write our reports in a way that no-one can work out that you took part in the study.

In line with UEA's research and data management policies, this data will remain accessible for at least 10 years, following which it will be archived. This data may be used for other ethically approved research during this time period.

What are your choices about how your information is used?

You can withdraw your participation at any time without giving a reason and without it affecting any benefits that you are entitled to. If you wish to withdraw your data from the study, please contact Ioana Marinescu (i.marinescu@uea.ac.uk) within two weeks of participation – after this point we will no longer be able to remove your data. We need to manage your records in specific ways for the research to be reliable. This means that we won't be able to let you see or change the data we hold about you.

Where can you find out more about how your information is used?

You can find out more about how we use your information:

- At www.hra.nhs.uk/information-about-patients/
- By asking one of the research team:
    - Ioana Marinescu (Researcher – survey lead), i.marinescu@uea.ac.uk
    - Kate Robbins (Researcher – interviews lead), k.robbins@uea.ac.uk
    - Dr Sarah Reeve (Supervisor, Chief Investigator), sarah.reeve@uea.ac.uk
- By viewing the UEA data protection policy at:

  https://www.uea.ac.uk/about/university-information/statutory-and-legal/data-protection

What will happen to the results of the study?

We will write our reports in a way that no one can work out that you took part in the study. The research will be published in a peer-reviewed scientific journal and will be incorporated into the doctoral theses of the researchers (Ioana Marinescu and Kate Robbins) for their doctorate in clinical psychology qualifications. The findings will also be disseminated

through social media and local services. No participants will be identifiable in any published reports on the study. If you wish to read about the findings of the study, you will need to provide your e-mail address.

Who is organising and funding the study?

This research is organised and funded by the University of East Anglia.

Who has reviewed the research?

Ethical approval for this study has been sought from NHS Research Ethics Committee (REC) to protect the rights, safety, dignity and wellbeing of research participants.

Contacts for further information:

Ioana Marinescu (Researcher – survey lead), i.marinescu@uea.ac.uk

Kate Robbins (Researcher – interviews lead), k.robbins@uea.ac.uk

Dr Sarah Reeve (Supervisor, Chief Investigator), sarah.reeve@uea.ac.uk

For further information on data protection please see:

https://www.uea.ac.uk/about/university-information/statutory-and-legal/data-protection

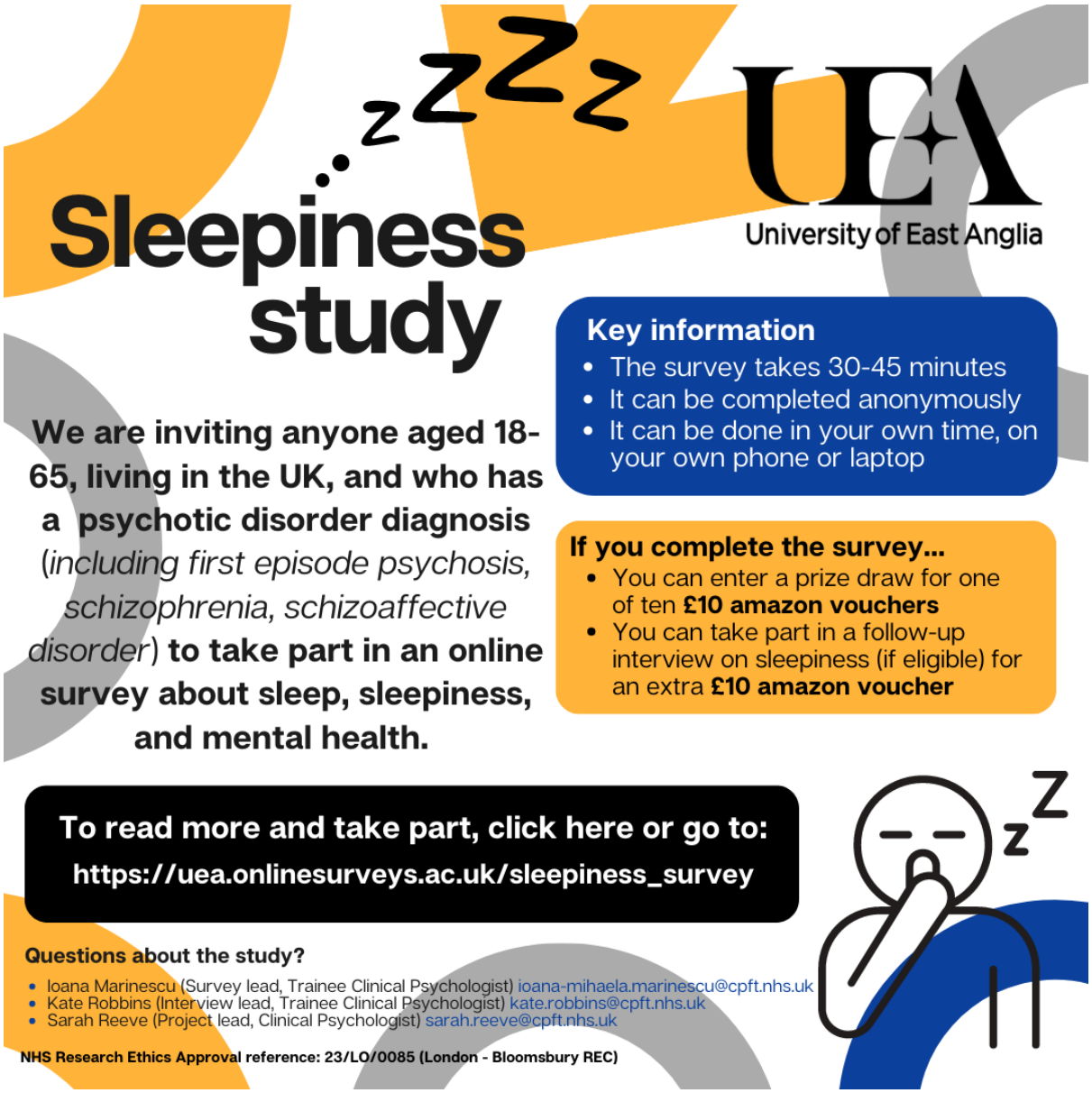
www.hra.nhs.uk/information-about-patients/

Thank you for reading this information sheet.

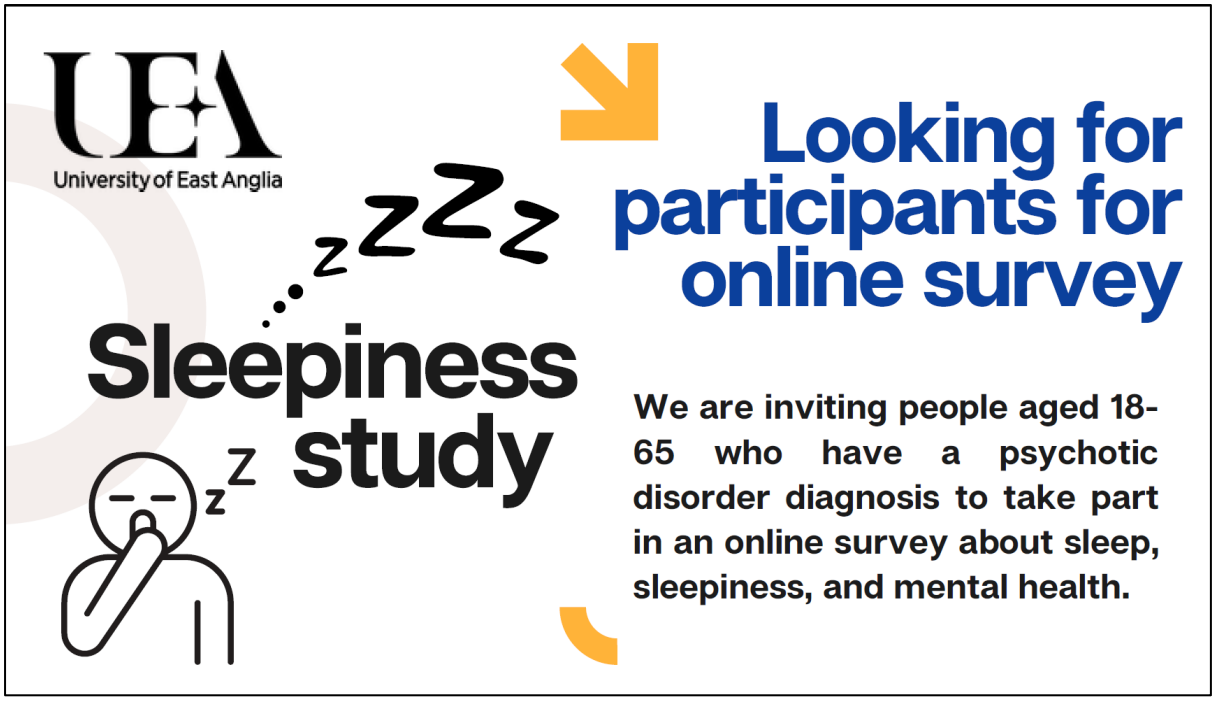
Appendix E

Study Poster and Cards

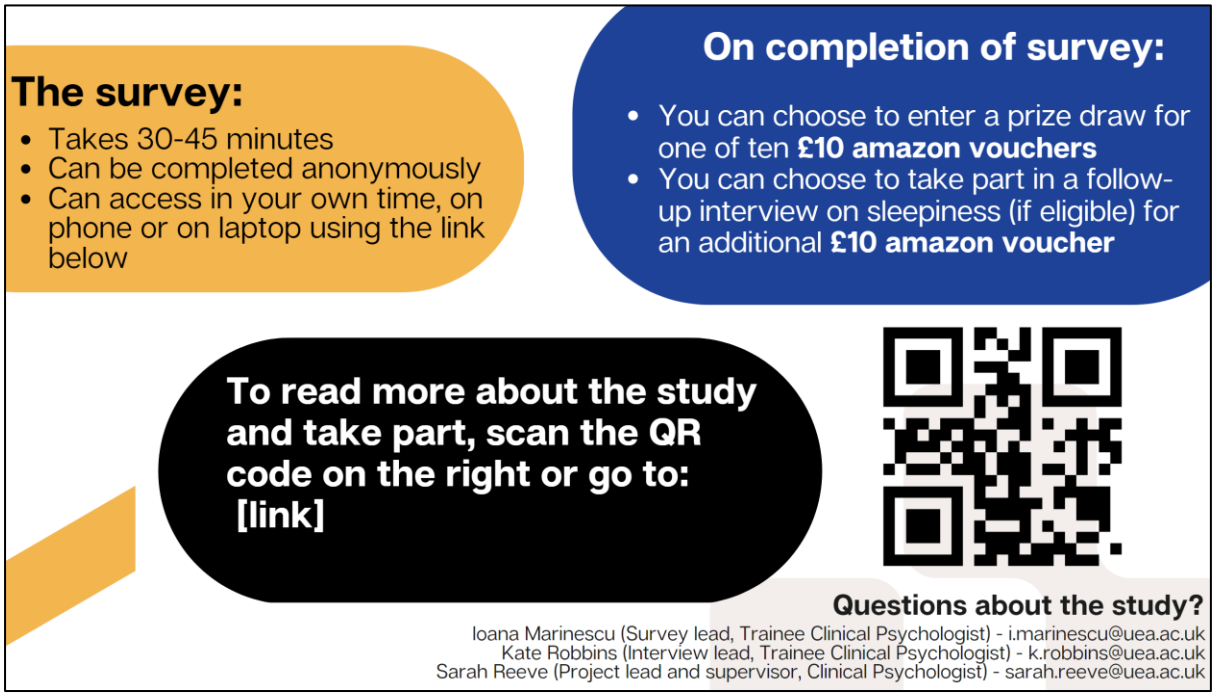
Poster



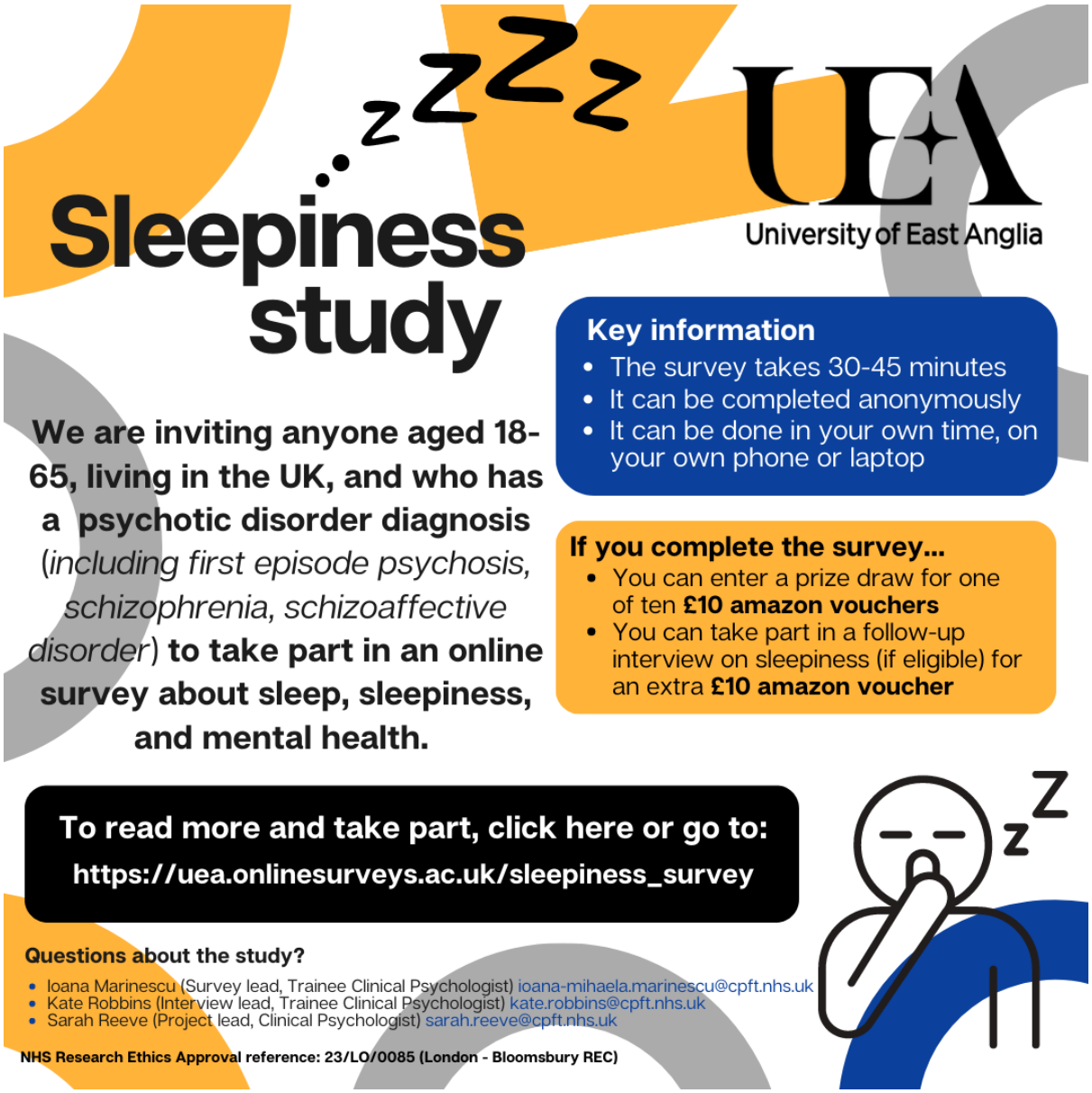
Business card front



Business card verso



Online advertisement text / image



Appendix F

Consent Form Version 3:19/06/2023

PART 1 – presented prior to the survey

I have read the information provided at the start of this survey on the current project (dated 19/06/2023, version 3).	I agree	I disagree
I understand the survey.		
I consent to participate in this survey.		
I understand how my information will be stored.		
I understand that the anonymised data from my survey will be used in future ethically approved research.		
I confirm I am aged between 18-65, have a diagnosis of a psychotic disorder and I am based in the UK.		

PART 2 – presented at end of the the survey

For each of the below, please select "Yes" if you are willing to be contacted for the below purposes or leave blank if you would not like to be contacted for that purpose.

Enter the prize draw for one of ten £10 Amazon vouchers.	
Be contacted about taking part in a follow-up interview (up to 1 hour, £10 amazon voucher) *	
Be contacted about the results of the study.	
If yes, please provide e-mail address here:	

* Your responses will be screened for eligibility, and we will contact you to let you know if you are eligible and to provide further information; interviewees will receive a £10 Amazon voucher.

Appendix G

REC Approval Letter

Ymchwil Iechyd a Gofal Cymru
Health and Care Research Wales

NHS
Health Research Authority

Dr Sarah Reeve
Norwich Medical School
University of East Anglia
Norwich
NR4 7TJ

Email: approvals@hra.nhs.uk
HCRW.approvals@wales.nhs.uk

10 March 2023

Dear Dr Reeve

HRA and Health and Care Research Wales (HCRW) Approval Letter

Study title:	**Sleepiness in psychosis: a mixed-methods exploration**
IRAS project ID:	**321546**
Protocol number:	**N/A**
REC reference:	**23/LO/0085**
Sponsor	**University of East Anglia**

I am pleased to confirm that HRA and Health and Care Research Wales (HCRW) Approval has been given for the above referenced study, on the basis described in the application form, protocol, supporting documentation and any clarifications received. You should not expect to receive anything further relating to this application.

Please now work with participating NHS organisations to confirm capacity and capability, in line with the instructions provided in the "Information to support study set up" section towards the end of this letter.

How should I work with participating NHS/HSC organisations in Northern Ireland and Scotland?
HRA and HCRW Approval does not apply to NHS/HSC organisations within Northern Ireland and Scotland.

If you indicated in your IRAS form that you do have participating organisations in either of these devolved administrations, the final document set and the study wide governance report (including this letter) have been sent to the coordinating centre of each participating nation. The relevant national coordinating function/s will contact you as appropriate.

Please see IRAS Help for information on working with NHS/HSC organisations in Northern Ireland and Scotland.

How should I work with participating non-NHS organisations?
HRA and HCRW Approval does not apply to non-NHS organisations. You should work with your non-NHS organisations to obtain local agreement in accordance with their procedures.

What are my notification responsibilities during the study?

The standard conditions document "*After Ethical Review – guidance for sponsors and investigators*", issued with your REC favourable opinion, gives detailed guidance on reporting expectations for studies, including:

- Registration of research
- Notifying amendments
- Notifying the end of the study

The HRA website also provides guidance on these topics, and is updated in the light of changes in reporting expectations or procedures.

Who should I contact for further information?
Please do not hesitate to contact me for assistance with this application. My contact details are below.

Your IRAS project ID is **321546**. Please quote this on all correspondence.

Yours sincerely,
Anna Martin
Approvals Specialist

Email: approvals@hra.nhs.uk

Copy to: *Polly Harrison*

List of Documents

The final document set assessed and approved by HRA and HCRW Approval is listed below.

Document	*Version*	*Date*
Contract/Study Agreement template [PIC]	1	19 December 2022
Copies of materials calling attention of potential participants to the research [Appendix A - Advertisements]	2	06 March 2023
Covering letter on headed paper [Response to Committee]	1	06 March 2023
Evidence of Sponsor insurance or indemnity (non NHS Sponsors only) [Insurance]		
Interview schedules or topic guides for participants [Topic Guide (Interview)]	2	06 March 2023
IRAS Application Form [IRAS_Form_20122022]		20 December 2022
Letter from sponsor [Sponsor Letter]		
Other [Public liability insurance]		
Participant consent form [Consent Form (Survey)]	2	06 March 2023
Participant consent form [Supplementary Consent Form (interview)]	2	06 March 2023
Participant information sheet (PIS) [Information Sheet (Survey)]	2	06 March 2020
Participant information sheet (PIS) [Supplementary information sheet (Interview)]	2	06 March 2023
Research protocol or project proposal [Protocol]	2	06 March 2023
Summary CV for Chief Investigator (CI) [Academic CV - Sarah Reeve]	1	19 December 2022
Summary CV for student [CV - Ioana Marinescu]	1	19 December 2022
Summary CV for student [CV - Kate Robbins]	1	19 December 2022
Summary, synopsis or diagram (flowchart) of protocol in non technical language [Appendix G - Study Flowchart]	2	06 March 2023
Validated questionnaire [Appendix H - Questionnaires (Survey)]	2	06 March 2023

IRAS project ID	321648

Information to support study set up

The below provides all parties with information to support the arranging and confirming of capacity and capability with participating NHS organisations in England and Wales. This is intended to be an accurate reflection of the study at the time of issue of this letter.

Types of participating NHS organisation	Expectations related to confirmation of capacity and capability	Agreement to be used	Funding arrangements	Oversight expectations	HR Good Practice Resource Pack expectations
Activities at NHS organisations will involve PIC activity only, including the identification of participants, database searches and the provision of recruitment cards.	Research activities should not commence at participating NHS organisations in England or Wales prior to their formal confirmation of capacity and capability to deliver the study in accordance with the contracting expectations detailed. Due to the nature of the activities involved, organisations will be expected to provide that confirmation to the sponsor Within **35 days** of receipt of the local information pack After HRA/HCRW Approval has been issued. If the organisation is not able to formally confirm capacity and capability within this timeframe, they must inform the sponsor of this and provide a justification. If the sponsor is not satisfied with the justification, then the sponsor may escalate to the National Coordinating Function where the participating NHS organisation is located.	The sponsor has provided the appropriate model commercial PIC agreement that it intends to use as a subcontract between participating organisations and NHS organisations acting as their Participant Identification Centres (PICs).	Sponsor is not providing funding to PICs.	The Chief Investigator will be responsible for all study activities performed at PICs.	The applicant has stated that local staff in participating organisations in England who have a contractual relationship with the organisation will undertake the expected PIC activities. No HR arrangements are needed as no research activity is occurring. Only members of the direct care team should have access to identifiable patient data.

Other information to aid study set-up and delivery

This details any other information that may be helpful to sponsors and participating NHS organisations in England and Wales in study set-up.
The applicant has indicated they do not intend to apply for inclusion on the NIHR CRN Portfolio.