Digital phenotyping and interventions for sleep & circadian rhythms in borderline personality disorder

Julia Lindsay

St Cross College

University of Oxford

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Abstract

Borderline personality disorder (BPD) is a mental health condition associated with severe psychological distress, considerable reductions to life expectancy, and significant increases to treatment cost relative to other psychiatric disorders. A growing body of evidence suggests that sleep disturbance is characteristic of the disorder and may be driven by circadian rhythm dysfunction.

This thesis aimed to understand whether digital interventions may be appropriate for treating sleep and circadian rhythm disturbances in individuals with BPD. This was undertaken first by using observational methods to first consider personality as a predisposing factor for insomnia (Chapter 2), and then by phenotyping sleep, rest-activity patterns, and mood instability in a BPD cohort and control group (Chapter 3). These findings motivate the remainder of the thesis, which focuses on clinical applications of digital interventions. Chapter 4 assesses the feasibility and preliminary efficacy of bright light therapy (BLT) and the first known study of digital cognitive behavioural therapy for insomnia (dCBT-I) in BPD. Chapter 5 explores participant experiences with these interventions and considers factors contributing to treatment adherence. Chapter 6 increases the generalisability of this thesis by systematically reviewing digital interventions treating symptoms of BPD, presenting meta-analyses of treatment effect, and considering the impact of interface design and implementation on the efficacy of digital interventions.

Findings suggest that maladaptive personality may contribute to sleep problems which in turn exacerbate mood instability, one of the core symptoms of BPD. Preliminary research on dCBT-I and BLT in BPD suggested improvements to sleep and an anti-

depressant effect, though results require replication. Participants reported that treatment was acceptable but highlighted difficulties with motivation and implementation that were reflected in adherence data; these concerns may be addressed by implementing participant feedback and employing interface features associated with treatment efficacy. This thesis motivates regular clinical assessment of sleep in BPD and suggests that digitally delivered sleep and chronotherapeutic interventions may be feasible in this group with appropriate modifications.

Declarations

This thesis was completed between October 2020 and October 2024 and was supervised by Professor Kate Saunders and Dr Niall McGowan. The work has not been submitted for any other degree, in this or any other university or learning institute. Some parts of the thesis have been published, and details are given below and in the relevant chapters. In accordance with the guidelines, the body of this thesis does not exceed 50,000 words (excluding references, appendices, figures, and tables) and contains fewer than 150 figures.

Contributions

Various collaborators have contributed to this thesis; these are detailed below.

JL: Julia Lindsay (candidate)
KEAS: Prof. Kate Saunders (supervisor)
NMM: Dr. Niall McGowan (supervisor)

Chapter 2: Effects of personality-related factors on insomnia symptoms & treatment

Portions of this chapter have been adapted from a previously published manuscript:

> **Lindsay, J. A. B,** McGowan, N. M., King, N., Rivera, D., Li, M., Byun, J., Cunningham, S., Saunders, K.E.A & Duffy, A. (2022). Psychological predictors of insomnia, anxiety and depression in university students: potential prevention targets. *BJPsych open*, *8*(3), e86.

Data was collected by DR, ML, JB & SC (research assistants) under the supervision of Prof. Anne Duffy and Dr. Nathan King (collaborators). Data cleaning was conducted by NK. This analysis was designed and conducted by JL with support from NMM, KEAS, NK & AD. The original manuscript was written by JL with support from NMM, KEAS, NK & AD.

Chapter 3: Phenotyping sleep and circadian rhythms in BPD

KEAS & NMM developed the study protocol. NMM conducted all recruitment and data collection, and sleep-scored the actigraphy files in MotionWare. JL cleaned the ecological momentary assessment data, designed and conducted all further analysis.

Chapter 4: Feasibility study of sleep & circadian interventions in BPD

KEAS & NMM developed the study protocol. JL conducted recruitment, data collection, designed and conducted the analysis.

Chapter 5: Participant experience of sleep & circadian interventions in BPD

JL & NMM developed the interview schedule. JL conducted and transcribed interviews. JL & Serena Guillemard (research assistant) developed themes in discussion with KEAS.

Chapter 6: Design and implementation of digital interventions for symptoms of BPD

This chapter has been adapted from a published manuscript:

> **Lindsay, J. A. B**, McGowan, N. M., Henning, T., Harriss, E., & Saunders, K. E. (2024). Digital Interventions for Symptoms of Borderline Personality Disorder: Systematic Review and Meta-Analysis. *Journal of medical Internet research*, *26*, e54941.

JL & Eli Harriss (research librarian) developed the search strategy. JL & Thomas Henning (medical school student) screened abstracts and papers. JL conducted data extraction. JL designed and conducted the analysis and wrote the original manuscript with support from NMM and KEAS.

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1 Chapter 1: Introduction

1.1 Borderline Personality Disorder

Overview

Borderline personality disorder (BPD) is a serious mental health condition that is typically diagnosed in early adulthood and is associated with emotional distress and functional impairment, often causing considerable disturbance to others. BPD is characterized by instability in identity, interpersonal relationships, and mood. Individuals with BPD frequently develop maladaptive coping mechanisms, which may lead to hospitalization due to impulsive actions, suicidal behaviours and non-suicidal self-injury (NSSI). Consequently, individuals with BPD are overrepresented in acute psychiatric care: some estimates place BPD patients as 25% of inpatient admissions (Zimmerman et al., 2008), and the cost of care for these patients is considerably higher relative to those with other mental health disorders (van Asselt et al., 2007). The disorder is associated with significant mortality and decreases to life expectancy (Fok et al., 2012), although most individuals achieve remission without recurrence (Gunderson et al., 2011; Zanarini et al., 2003).

Genetic vulnerability is thought to predispose BPD (Reichborn-Kjennerud et al., 2013) and overlap with several other psychiatric disorders including major depressive disorder, bipolar disorder, and schizophrenia (Witt et al., 2017). However, the manifestation of specific BPD symptoms, which varies greatly between individuals, may be shaped by environmental factors. A substantial body of research indicates that these factors are frequently linked to invalidating early life experiences with caregivers (Hallquist et al., 2015; Winsper, Hall, et al., 2017). Such experiences may hinder the development of emotion regulation skills and the ability to understand mental states (mentalisation), leading to deficiencies that can precipitate

maladaptive coping mechanisms. Additionally, many individuals with BPD report having faced adverse experiences (Golier et al., 2003) and the trauma associated with these experiences may also contribute to the development of BPD by increasing the likelihood of maladaptive behaviours and intensifying affective symptoms (Trull, 2001).

Current treatment guidelines for BPD do not indicate pharmacological intervention, although many individuals may be prescribed antidepressant, antipsychotic, or mood stabilising medication for comorbid disorders (NICE, 2019; Stoffers-Winterling & Lieb, 2015). Two psychotherapies developed for BPD have empirical support: these are dialectical behavioural therapy (DBT) and mentalisation-based treatment (MBT) (Bateman & Fonagy, 2010; M. Linehan, 1993). The former aims to improve emotion regulation skills through cognitive and behavioural skill development; the latter targets mentalisation ability. Both therapies are effective (Cristea et al., 2017; Storebø et al., 2020) but require extended courses of treatment and are resource-intensive, contributing to high rates of service closure (King et al., 2018; Swales et al., 2012).

Historical Context of Borderline Personality Disorder

Much of our modern classification of psychiatric illness is based on the work of Emil Kraepelin, who relied on longitudinal observation of patients to develop distinctions between disorders (Kraepelin, 1883). One of his most important contributions was the distinction between “dementia praecox” and manic-depressive illness, which we now know as schizophrenia and bipolar disorder, respectively. He and his contemporaries considered these disorders “major psychoses” since they drastically affected patients’ experience of reality and virtually always required institutional care, unlike neuroses (our modern affective and

obsessive-compulsive disorders) (Kraepelin, 1915). However, the major psychoses excluded a considerable number of individuals with abnormal mental states and disruptive behaviours but largely intact functioning. Kraepelin described this group as inhabiting a *Zwischengebiet*: literally, in his native German, an “intermediate area” or borderland (Kraepelin, 1909). This concept of an intermediary disorder was adapted by numerous psychiatrists over the following century, typically to describe individuals with mild forms of either dementia praecox or manic-depressive illness (Moore, 1921; Schmideberg, 1947) or symptoms of both psychoses (Oberndorf, 1930). The resultant group of patients included in this "borderland" was far broader than what is today recognized as borderline personality disorder (BPD), encompassing conditions such as phobias and panic disorders (Stone, 2005).

This early concept of the borderland laid the groundwork for later developments in personality disorder theory. One of these was Kernberg’s three tiers of personality organization: neurotic, borderline, and psychotic (Kernberg, 1981). The borderline level was characterized by an intact capacity for reality testing, distinguishing it from the psychotic level, but a distorted or weak sense of self, differentiating it from the neurotic level. Kernberg’s “borderline personality organization” encompassed many features now associated with modern borderline personality disorder (BPD), such as heightened emotional reactivity and impulsivity (1967). However, it was also broad enough to include other personality disorders and eating disorders. Much of Kernberg’s definition focused on behaviour and functional impairment rather than personality traits per se.

The term "borderline personality disorder" (BPD) was first proposed by Gunderson and Singer, who outlined criteria including diminished work capacity, impulsivity, suicidal

threats, brief psychotic episodes, and disturbances in personal relations (Gunderson & Singer, 1975). BPD was subsequently included in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), drawing criteria from both Kernberg and Gunderson's proposals. This conceptualisation of BPD successfully distinguished the disorder from schizophrenia and affective disorders, but showed considerable overlap with histrionic and antisocial personality disorders (Pope et al., 1983). Few of the DSM-III criteria were directly related to personality traits, reflecting a broader trend in psychiatric and psychoanalytic literature, where the conceptualization of personality disorders evolved with minimal input from the field of personality theory (Widiger & Frances, 1985).

Diagnosis of Borderline Personality Disorder

This context resulted in a series of diagnostic criteria published in the DSM and International Classification of Diseases (ICD) which rely on the categorical presence of multiple traits and behaviours, none of which in isolation are sufficient or necessary for diagnosis (Livesley, 2001). Critics have argued that these categorical models are both conceptually flawed and not supported by empirical evidence (Tyrer et al., 2015). The categorical models divide personality disorders into three clusters without theoretical rationale (Livesley, 2001) and require an arbitrary number of features for diagnosis (Widiger & Lowe, 2007). Categorical models also have low consistency and reliability compared to dimensional models of personality (Bornstein, 2003; Durbin & Klein, 2006). The resulting personality disorder diagnoses have both significant within-group heterogeneity and between-group overlap (Widiger et al., 1991). Finally, the categorical models imply a partition between normal and pathologic personality, contributing to the "othering" of patients and resulting resentment towards the diagnosis of personality disorder (Stalker et al., 2005). These criticisms have led

to the development of alternative diagnostic models, which postulate personality disorder as extreme variants of normal dimensions of personality.

These dimensional diagnostic models of personality disorder have been included in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) alternative model and the International Classification of Diseases, 11th revision (ICD-11). Although these dimensional models of personality still garner criticism for issues such as low agreement between self- and other-ratings (J. D. Miller et al., 2005), many psychiatrists and academics argue that they are preferable to the preexisting diagnostic models for personality disorder (Hopwood et al., 2018). Both the DSM-5 and ICD-11 models require functional deficiency directed towards the self or others and additionally rate individuals along dimensions five maladaptive traits: negative affectivity, detachment, antagonism, disinhibition and psychoticism/anankastia (Bach & First, 2018; Krueger et al., 2012). These five traits were selected based on extensive literature reviews and psychometric analysis of maladaptive personality traits. Four of the traits bear strong resemblance to those included in the Five-Factor Model (FFM), which is arguably the prevailing theory in personality psychology. Both the ICD-11 and DSM-5 include a fifth trait: anankastia as defined in the ICD-11 relates to rigidity and control, and may be relevant to the conceptualisation of obsessive-compulsive personality disorder (Bach & First, 2018). Psychoticism as defined by the DSM-5 alternative model indicates frequency and severity of cognitive or perceptual abnormalities, this may be relevant to what was previously labelled schizotypal personality disorder (Krueger et al., 2012). The ICD-11 also includes a borderline trait specifier, which is used to indicate instability and impulsivity. The links between the DSM-5, ICD-11 and FFM traits are shown in Table 1.1 below.

Table 1.1. Trait associations between personality models. (-) indicates an inverse correlation with the corresponding DSM-5/PID-5 trait.

DSM-5/PID-5	ICD-11	FFM
Negative Affectivity	Negative Affectivity	Neuroticism
Detachment	Detachment	Extraversion (-)
Antagonism	Dissociality	Agreeableness (-)
Disinhibition	Disinhibition /Anankastia (-)	Conscientiousness (-)
Psychoticism		Openness

Unique constellations of these five traits may account for the difference in presentation of personality disorder between individuals. There have also been suggestions that certain personality disorders arise from specific combinations of traits. For instance, people with BPD tend to endorse high levels of negative affect (high FFM neuroticism) and, to a lesser extent, antagonism (low FFM agreeableness) and disinhibition (low anankastia/FFM conscientiousness) (Samuel et al., 2013; Samuel & Widiger, 2008). Neuroticism has been suggested as the underlying feature linking the diverse presentations of BPD, whether the primary manifestation is affective instability, impulsivity, suicidal ideation, or self-injury, with the latter three likely representing maladaptive coping behaviours developed in response to negative affect (Clarkin et al., 1983; Samuel et al., 2013). These findings underscore the dimensionality of personality and the relevance of general personality research to personality disorder.

Differential Diagnosis & Lived Experience of BPD

Negative patient response to diagnosis is frequent in BPD; many individuals report feeling unsupported and uninformed during the diagnostic process (Tedesco et al., 2024). Other common reports include facing stigma, blame, and exclusion associated with the diagnosis (Horn et al., 2007; Tedesco et al., 2024). Objective data is consistent with these claims and

suggests that people with personality disorder receive substandard medical care: in the UK, their basic needs are more frequently left unmet (Hayward et al., 2006) and their physical health conditions are undertreated (Sanatinia et al., 2015) relative to people with other psychiatric diagnoses. These factors likely contribute to the drastic 18-year reduction in life expectancy ascribed to people with personality disorder (Fok et al., 2012)

Perhaps because of these experiences, there are many alternative diagnostic labels which patients may see as preferrable. These have also been considered in the academic discourse: at various points, BPD has been proposed as a variant of major depressive disorder (Akiskal et al., 1985), bipolar disorder (Perugi et al., 2003), posttraumatic stress disorder (Gunderson & Sabo, 2013; Herman et al., 1989) and autism spectrum disorder (Dell'Osso et al., 2023; Rydén et al., 2008). One reason for the abundance of these suggestions is that the diagnostic criteria in the categorical model of BPD rely primarily on symptoms rather than personality traits, and all the aforementioned disorders share some symptoms with BPD. Most of these disorders are also associated with increased likelihood of adverse early life experiences, offering a tidy solution to the aetiology of these symptoms (Haruvi-Lamdan et al., 2018; K. L. Lewis & Grenyer, 2009). However, BPD has persisted as a diagnostic label because its comorbidity with other disorders is frequent but varied, and it has a distinct phenomenology, family history, longitudinal course and response to treatment (Gunderson & Phillips, 1991; Paris et al., 2007; Stoffers-Winterling & Lieb, 2015; Storebø et al., 2020; White et al., 2003; Zanarini et al., 2003).

Sleep & BPD

Given the heterogeneity of symptom presentation in BPD (Maffei, 2005) and discourse surrounding diagnosis, the identification of biological and physiological markers are of interest. One possible marker is sleep disturbance, which is commonly reported in BPD (Wood et al., 2015). These complaints have been validated by the study of neural and physiological markers of sleep, a process called polysomnography (Winsper, Tang, et al., 2017). Meta-analytic evidence suggests that key measures of sleep continuity are worsened in BPD compared to healthy controls, and that people with BPD appear to have reduced slow-wave, or restorative, sleep (Winsper, Tang, et al., 2017).

These findings, which are independent of medication use and comorbid psychiatric disorders, have been collectively suggested as a sleep phenotype of BPD. This is not merely an artefact of the diagnostic models for BPD; robust trait-based findings have linked elevated neuroticism with sleep problems, particularly insomnia (Engel & Engel-Sittenfeld, 1980; Freedman & Sattler, 1982; Mendelson et al., 1984; Niemcewicz et al., 2001; Wang et al., 2001). The internalisation hypothesis suggests that this occurs because the physiological arousal accompanying negative affect impairs sleep, suggesting an interaction with the regulatory processes of sleep (van de Laar et al., 2010). There is some evidence to support an association between emotion dysregulation and low self-reported sleep quality and symptoms of insomnia (Fitzpatrick et al., 2023; Jenkins et al., 2022). Furthermore, symptoms of emotion dysregulation including waking stress and “emotional cascades” (escalations of negative affect) predict the frequency of self-reported nightmares in BPD, suggesting that daytime arousal from negative affect may spill over into elevated nighttime cognitive activity (Selby et al., 2013). Parasomnias, especially nightmares, are much more common in BPD

than in either the general population or other psychiatric groups, with estimates of nightmare prevalence converging on 50% in BPD (Hafizi, 2013; Kessler & Merikangas, 2004; Semiz et al., 2008). Polysomnographic evidence suggests that nightmares contribute to worsened subjective sleep quality but do not affect sleep architecture or duration in BPD (Paul et al., 2015).

In summary, the extant literature suggests that BPD is associated with both objective and subjective sleep disturbance. In healthy controls, sleep disturbance has also been linked with emotion dysregulation, a key manifestation of BPD, but this link has not been demonstrated with objective sleep measures in BPD, aside from a single study linking elevated time in bed and low emotional awareness (Jenkins et al., 2022). The directionality of the association between sleep disturbance and emotion regulation has not been established although a cyclical process in which heightened affect impairs sleep and sleep disruption reduces capacity for emotion regulation has been proposed (Selby, 2013), with increased amygdala reactivity in BPD as a putative mechanism (New et al., 2007). Further research using objective measures of sleep is necessary to elucidate the relationship between emotion regulation and sleep in BPD.

1.2 Sleep & the Circadian System

Two independent processes regulate sleep: homeostatic sleep drive (Process S) and circadian rhythm (Process C, Borbély, 1982). Process S refers to the build-up of sleep pressure that increases during the wakeful period. Process C is a history-independent oscillator on a near-24-hour cycle (Tobler et al., 1983). When aligned, the two processes alternately promote sleep and wakefulness, as shown in Figure 1.1 below. For instance, even if homeostatic sleep

pressure (Process S) is high, an opposing circadian alerting signal (Process C) can delay sleep onset, as commonly observed in the evening hours when individuals feel alert despite prolonged wakefulness (Borbély et al., 2016).

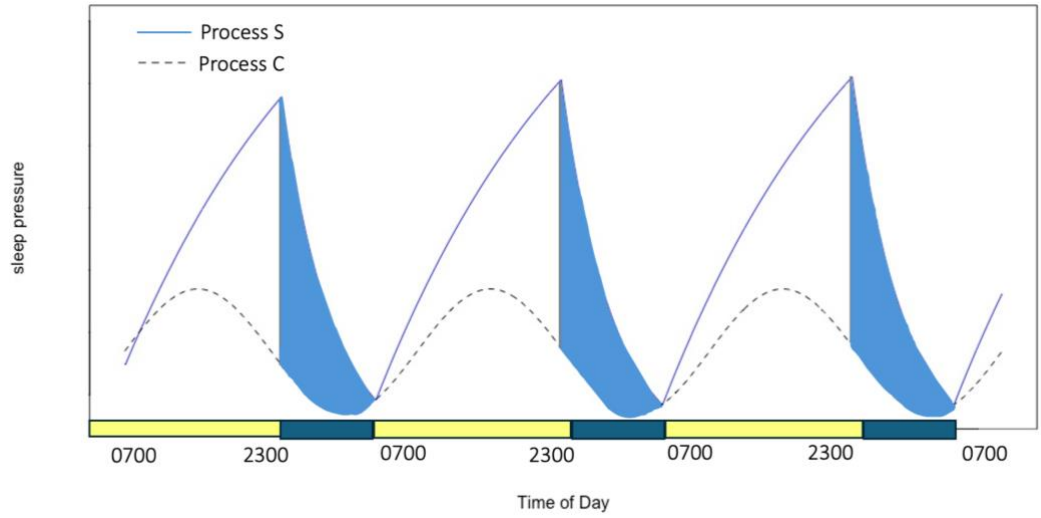

Figure 1.1. Process S and C counteract each other to promote daytime wakefulness during the subjective day (depicted by yellow shading) and align to induce sleep during the night (blue shading).

The central pacemaker for the circadian system is the suprachiasmatic nucleus (SCN), located in the hypothalamus. The SCN regulates a variety of bodily functions: sleep, but also body temperature, endocrine levels, and metabolism fluctuate periodically, relying on signals directly from the SCN and clock cells in peripheral tissues (Aschoff, 1983; Green et al., 2008; Slat et al., 2013). The molecular oscillation of the SCN occurs through the CLOCK and BMAL1 genes, which transcribe PER and CRY proteins (Gekakis et al., 1998). The PER and CRY proteins dimerise, inhibiting CLOCK and BMAL1 from transcribing further proteins, and creating a transcriptional-translational negative feedback loop. The existing PER and CRY proteins eventually degrade, thus allowing further protein transcription by CLOCK and BMAL1 (Edery et al., 1994). The degradation status of PER and CRY determines circadian

phase, while their rate of degradation determines the circadian rhythm's period; in humans this is approximately 24.2 hours (S. P. Fisher et al., 2013). Under constant conditions the SCN maintains this period whilst circadian phase gradually shifts later relative to clock time. However, light exposure can entrain the SCN to the external light-dark schedule: intrinsically photosensitive retinal ganglion cells (ipRCGs) send neural signals to the SCN, upregulating PER proteins and advancing or delaying circadian phase dependent on the timing (Ashton et al., 2022). The relationship between time of light exposure and the resulting phase shift is the "phase response curve" (PRC) which has three portions. Exposure to light in the early subjective morning (prior to or just after typical rise time) increases PER1 transcription, which has the effect of advancing circadian phase. Evening light extends the transcription of PER2, inhibiting CLOCK and BMAL longer and thus delaying circadian phase. Daytime light does not appear to have a phase-shifting effect (Ashton et al., 2022). Phase shift may also occur in response to non-photic cues: the social zeitgeber theory suggests the circadian system may entrain to social interaction demands, and food consumption may have a similar effect (Grandin et al., 2006; Pickel & Sung, 2020). Phase shift may also be induced through melatonin, which is a naturally occurring hormone secreted in darkness that signals circadian phase to peripheral tissues. Ingestion of exogenous melatonin when endogenous melatonin is low (ie. not during the subjective night) results in a phase shift. The PRC for exogenous melatonin is roughly opposite to the light PRC such that subjective evening melatonin advances phase and subjective morning melatonin produces phase delays (Burgess et al., 2008). Exogenous melatonin is typically used to extend or promote sleep whereas light exposure may be used to promote wakefulness.

Circadian phase affects sleep through several mechanisms including regulation of body temperature and endocrine release. Core body temperature declines in the evening, promoting sleepiness (Kräuchi, 2007). Cortisol also reaches its lowest levels in the evening, having peaked in the morning to promote wakefulness (Gamble et al., 2014). Simultaneously, melatonin secretion increases during the dark hours, indicating circadian phase to peripheral tissues and promoting sleepiness through vasodilation (Dawson & Armstrong, 1996; Kräuchi & Wirz-Justice, 2001). Misalignment of these endocrine profiles has been suggested as a cause of circadian rhythm sleep wake disorders (Zisapel, 2001). Circadian rhythm sleep wake disorders occur in individuals unable to entrain to the external environment, instead expressing their intrinsic period, or else consistently expressing an advance or delay relative to the environment (Reid & Zee, 2011). The former circumstance, called Non-24 Hour Sleep-Wake Disorder (N24SWD), results in arrhythmic rest-activity patterns and is typically seen in subjects for whom ocular damage prevents normal signalling from environmental light. Meanwhile, chronic phase advances relative to the external environment are termed Advanced Sleep Wake Phase Disorder (ASWPD) and associated with early sleep offset or sleep maintenance insomnia, while Delayed Sleep Wake Phase Disorder (DSWPD) causes sleep onset insomnia and difficulty with morning awakenings (Reid & Zee, 2011).

Circadian Associations with Psychiatric Symptoms

Beyond sleep problems, there is accumulating evidence linking CRSWDs with mood symptoms. For example, mood disorder patients often exhibit circadian irregularities, such as reduced amplitude in body temperature, melatonin, and cortisol rhythms, which resemble patterns observed in blind subjects (Souêtre et al., 1989). These irregularities can be mitigated with antidepressant treatment, suggesting an association between circadian function and

mood symptom severity. In bipolar disorder, circadian instability is associated with the onset of manic episodes (Malkoff-Schwartz et al., 1998). Moreover, circadian genes have been implicated in multiple mental illnesses, indicating a genetic component to these circadian-mood interactions (Mansour et al., 2006).

There is evidence for CRSWDs in BPD: N24SWD and particularly DPSWD are found in BPD at an elevated prevalence relative to the general population, and vice versa (Dagan et al., 1996, 1998), which may contribute to the frequency of insomnia (Winsper, Tang, et al., 2017) and irregular rest-activity patterns (Wood et al., 2015). Further evidence also points towards phase delays: questionnaire measures suggest that people with BPD tend to have later chronotypes, indicating a preference for later bedtimes and rise times (Fitzpatrick et al., 2020). Data derived from wearable activity trackers (actigraphs) indicates delayed rise time (Huỳnh et al., 2016). Studies of cortisol in BPD further suggest circadian irregularities, including a more rapid increase upon awakening (Lieb et al., 2004; Rausch et al., 2015) and higher overnight levels (Wingenfeld et al., 2007). However, the sole study of salivary melatonin profiles in BPD reported no difference relative to controls (Bromundt et al., 2013).

To date, the strongest evidence for circadian phase delay in BPD is from the Automated Monitoring of Symptom Severity (AMoSS) study, which indicated that sleep and heart-rate variability lagged significantly behind rest-activity patterns in BPD, but not in bipolar disorder (BD) or healthy controls (Carr, Saunders, Bilderbeck, et al., 2018). Variability in circadian rhythms generally and delayed circadian phase specifically also covaried with BPD symptoms of impulsivity and mood instability (Carr, Saunders, Tsanas, et al., 2018; McGowan et al., 2020). This association, which was significantly stronger in BPD than in

bipolar disorder, suggests people with BPD may be highly reactive to circadian disruption. The link between circadian disruption and psychopathology could arise from reduced capacity for behavioural control and emotion regulation due to chronic physiological stress and conflicting neural signals (Wulff et al., 2010). The deleterious effects of circadian rhythm disruption have been posited in the context of BD, leading to the development of interpersonal and social rhythm therapy (IPSRT) (Frank, 2007). Although IPSRT can be effective in the treatment of BD, it seems to be insufficient for individuals with comorbid BD and BPD (Swartz et al., 2005). However, preliminary research on the inclusion of IPSRT in standard BPD psychotherapy has begun, in the hopes that stabilisation of circadian rhythm could prove an effective adjunct (Bailey et al., 2023).

If evidence of circadian phase delay in BPD is replicated, it could constitute an objective phenotype of BPD which may improve specificity of the current diagnostic models. Associations with symptom severity, particularly mood instability, also suggest that treating circadian rhythm dysfunction might affect BPD pathology, in addition to reducing symptoms of insomnia. However, the direction of association is currently unclear: circadian phase delay could be a symptom of BPD, or the resulting physiological strain might be a contributing factor to the symptom presentation of the disorder. Further phenotyping of sleep, circadian rhythms, and mood instability in BPD is warranted.

Sleep and Chronotherapeutic Interventions in BPD

Sleep and chronotherapeutic interventions may also be especially pertinent in BPD given limited existing treatment options. Currently pharmacotherapy is not indicated (Kendall et al., 2009; Stoffers-Winterling et al., 2020), with evidence instead favouring dialectical

behavioural therapy (DBT) and mentalisation-based treatment (MBT) (Stoffers-Winterling et al., 2012). Though both psychotherapies have demonstrated efficacy, they are resource-intensive, require multi-year time commitments from service users, and are oversubscribed, particularly in international contexts (Iliakis et al., 2019a). Additionally, services offering DBT and MBT are difficult to maintain due to clinician turnover and financial strain: fewer than half of DBT services in the UK have remained active for more than decade (King et al., 2018; Swales et al., 2012).

Circadian rhythm and sleep interventions could be used as adjunctive treatment or stopgaps for people with BPD who commonly experience these symptoms, while service users wait for psychotherapy. Circadian rhythm function can be supported by bright light therapy (BLT), which has been found to reduce mood symptoms (Prasko et al., 2010) and advance circadian phase in BPD (Bromundt et al., 2013). This intervention is discussed in more detail in Chapter 3. The gold standard sleep intervention is cognitive behavioural therapy for insomnia (CBT-I) and is highly effective, both from cost (Kyle et al., 2023) and symptom-reduction perspectives (Edinger & Means, 2005). Evidence also suggests that CBT-I reduces severity of some psychiatric symptoms (Cunningham & Shapiro, 2018; D. J. Taylor & Pruiksma, 2014). This may be attributable to the antidepressant effect of sleep restriction therapy, a component of CBT-I which increases homeostatic sleep pressure (Process S) (Germain & Kupfer, 2008). CBT-I has successfully been adapted to scalable digital formats (Batterham et al., 2017; Cheng et al., 2019; Espie et al., 2019), which are accessible regardless of geographic location or diagnostic status. Digital sleep interventions hold potential to be effective, accessible treatments for people with BPD. They are also transdiagnostic and thus may remain applicable regardless of diagnostic model or label employed. However, digital interventions,

here defined as interventions delivered asynchronously via the internet or smartphone apps, are not without disadvantages. A major obstacle to the efficacy of digital interventions is user attrition, estimated at over 50% in clinical trials of digital interventions for depression (Lakhtakia & Torous, 2022; Torous et al., 2020). User engagement with digital interventions typically exhibits an exponential decay pattern, which may be more pronounced in unsupervised settings compared to clinical trials, from which most published data is derived (Eysenbach, 2005; Lakhtakia & Torous, 2022). Despite their potential, the efficacy, acceptability, and usability of digital interventions remain largely untested in BPD.

1.3 Thesis Objectives

Although evidence to date suggests associations between insomnia and personality traits, particularly neuroticism, most of this literature is cross-sectional and thus it is unclear whether personality traits may predispose insomnia or are merely associated factors. Additionally, little is known about the characteristics of individuals who choose to enrol for digital insomnia treatment outside of clinical trial settings. Accordingly, the objectives of Chapter 1 are:

a. To use epidemiological methods to examine personality factors as predisposing for sleep/circadian issues, especially insomnia, in a large non-clinical group

b. To determine user characteristics associated with the decision to enrol for an unsupervised digital insomnia intervention

Previous research has identified insomnia and circadian phase delay in BPD. However, these findings have yet to be replicated, and it is not clear whether some BPD symptoms,

particularly mood instability, are driven by sleep and circadian rhythms or vice versa. Chapter 2 has the following aims:

c. To further phenotype the sleep, circadian, and mood instability profiles of BPD.
d. To explore associations between sleep, circadian rhythm, and mood instability in BPD

There is limited extant research on sleep interventions in BPD. Chapter 3 is a pilot evaluation of an intervention in a small sample of seven participants, which aims

e. To determine feasibility and effects of a dual sleep and circadian intervention in BPD.

Adherence is a major obstacle to digital interventions, which may be exacerbated by systems which do not meet users' needs. None of the limited work on sleep interventions in BPD has presented data on acceptability, adherence over time, or user preferences. This gives rise to the aims of Chapter 4:

f. To qualitatively analyse factors impacting adherence to a digital sleep and circadian intervention in BPD, including user concerns and preferences.

The thesis concludes with a systematic review and meta-analysis of digital interventions targeting symptoms of BPD. This Chapter aims:

g. To characterise digital interventions currently available which might be used to treat symptoms of BPD
h. To identify specific digital intervention features that may be associated with increased treatment effect for symptoms of BPD

Chapter 2: Longitudinal associations between personality-related factors, insomnia symptoms & treatment

2.1 Introduction

Motivating Epidemiological Study of Insomnia in Students

The dimensional model of personality disorder posits a spectrum of personality from functional to maladaptive (Hopwood et al., 2018; Samuel et al., 2013), implying that healthy and subclinical personality function may provide insight to personality disorder (Trull, 2001). Subclinical populations may be of particular importance to determining the factors of personality which lead to functional impairment. One of these may be low self-esteem, a mental schema in which the self is viewed as unworthy or inferior. This affects the way that new information, including from social interactions, is integrated, and may cause marked reactivity to stimuli compared to people with stronger self-esteem (Pyszczynski et al., 2004). In healthy controls, reduced self-esteem is linked to characteristic BPD behaviours including heightened reactivity, angry outbursts, and maladaptive coping (Winter et al., 2017), and has been associated with most personality pathologies (D. C. Watson, 1998). A major component of self-esteem arises from self-criticism (Heatherton & Wyland, 2003), which in excess is considered a maladaptive form of perfectionism (Dickie et al., 2012; Dunkley et al., 2006). Self-critical perfectionism is hypothesised to be caused by a weak sense of identity, a feature of BPD (Blatt, 2008). Lowyck et al. supported this hypothesis in a clinical sample by showing that psychodynamic treatment reduced self-critical perfectionism and that the decrease covaried with reductions in symptomatic distress (2017). Another facet of perfectionism is elevated personal standards, but this is less frequently associated with

negative health outcomes (Dunkley et al., 2006). Given that self-critical perfectionism and low self-esteem are elevated in personality disorder but also frequently present in healthy individuals, these factors could be indicators of maladaptive personality.

Personality as a Predisposing Factor for Insomnia

Low self-esteem and self-critical perfectionism are associated with poor emotion regulation and heightened reactivity (Dunkley et al., 2003; Hope et al., 2018; Winter et al., 2017), both of which are characteristic of BPD and also thought to contribute to insomnia. The internalisation hypothesis suggests that heightened physiological arousal caused by these states impairs sleep and predisposes insomnia disorder (van de Laar et al., 2010). Insomnia is characterised by chronic difficulty falling or staying asleep, and resulting daytime impairment (Morin et al., 2015). Symptoms of low sleep efficiency, fatigue, and extended sleep onset latency are frequently reported by people with BPD (Winsper, Tang, et al., 2017; Wood et al., 2015), but insomnia is widely prevalent, including in university students (D. J. Taylor et al., 2013). The prevailing '3P' model of insomnia theorises that the disorder is predisposed by biological and psychological factors, precipitated by acute stressors, and perpetuated by cognitive and environmental factors (Spielman et al., 1987). University students constitute a large population centred around the typical age of BPD diagnosis and including individuals with subclinical personality features (Fonseca-Pedrero et al., 2011; Liu et al., 2016; Meaney et al., 2016) and extensive acute stressors: sudden increases in academic and social pressures coupled with shared accommodation may all precipitate insomnia in predisposed individuals (D. J. Taylor & Bramoweth, 2010).

Personality is thought to be a predisposing factor for insomnia: all of the FFM traits save agreeableness have been associated (negatively or positively) with insomnia (Ellis et al., 2021), particularly neuroticism (Engel & Engel-Sittenfeld, 1980; Freedman & Sattler, 1982; Mendelson et al., 1984; Niemcewicz et al., 2001; van de Laar et al., 2010; Wang et al., 2001). However, there is currently a deficit of longitudinal studies of personality and insomnia, which are necessary to determine whether personality is a predisposing or co-occurring factor (Akram et al., 2023; van de Laar et al., 2010). There are no known longitudinal studies of self-esteem as a predisposing factor for insomnia and a single longitudinal study of perfectionism, which found that it was not a significant predictor of insomnia when symptoms of depression and anxiety were accounted for (Jansson-Fröjmark & Linton, 2007). This is consistent with a general trend in the literature which suggests that insomnia is predisposed by negative affective symptoms, or in personality terms, by neuroticism (Akram et al., 2023; van de Laar et al., 2010). In this study we sought to replicate Jansson-Fröjmark & Linton's analyses with the addition of self-esteem to determine whether these factors have any predisposing effect beyond their associations with negative affect.

Cognitive Behavioural Therapy for Insomnia

Insomnia is associated with a host of negative outcomes including physical illness, reduced well-being and worsened academic performance (Duffy et al., 2020; Khurshid, 2018; H. G. Lund et al., 2010). Fortunately, there is a gold-standard treatment: cognitive behavioural therapy for insomnia (CBT-I). The intervention has several components: cognitive restructuring, stimulus control, sleep hygiene, relaxation techniques and sleep restriction, the latter of which is considered the most effective (Kyle et al., 2023; C. B. Miller et al., 2014; Rossman, 2019). The intervention has been adapted to several digital formats, notably

Sleepio which has been shown to reduce insomnia symptoms in numerous clinical trials (Espie et al., 2012, 2019). The intervention also appears to reduce symptoms of other mental health problems, such as depression and psychosis (Cheng et al., 2019; Freeman, Sheaves, Goodwin, Yu, Nickless, Harrison, et al., 2017). Treatment efficacy has previously been demonstrated in university students as well as in the workplace (Barnes et al., 2017; Bostock et al., 2016; Freeman, Sheaves, Goodwin, Yu, Nickless, Harrison, et al., 2017).

Given the extensive evidence for dCBT-I, it could be an effective resource for reducing symptoms of insomnia and other mental health problems in universities' student welfare services. However, the simplest implementation of dCBT-I would be fully self-guided and unsupervised, possibly increasing risk of intervention non-completion, also referred to as attrition. Notably the largest study of dCBT-I in university students had significant attrition, with only half of participants completing two of the six treatment sessions (Freeman, Sheaves, Goodwin, Yu, Nickless, Harrison, et al., 2017). A review of mental health interventions offered in university services has found that this attrition is common to most interventions and likely reduces their impact (Becker & Torous, 2019). The characteristics of students choosing to enroll for self-guided digital interventions may be important to optimising their deployment. For example, personality factors could affect insomnia treatment, both at the stage of enrolment and longer-term treatment efficacy. Perfectionism is negatively associated with help-seeking in psychologically distressed students (Ey et al., 2000). Although to our knowledge there is no literature on the effects of self-esteem on insomnia treatments, willingness to admit personal flaws and shortcomings was associated with lower efficacy of behavioral insomnia treatment (Edinger et al., 1988). Psychopathology is associated with worsened outcomes for cognitive behavioural insomnia interventions in

people without comorbid psychiatric disorders (Lacks & Powlishta, 1989). Determining whether personality factors act as barriers to treatment-seeking may help optimize resources for insomnia treatment in community and clinical settings, perhaps by making personality factors an explicit target for insomnia treatment.

Accordingly, we have conducted two studies analysing the associations between personality, insomnia symptoms, and treatment uptake in a cohort of undergraduate students. The objectives, methods, results and brief discussions of each study are outlined separately below.

2.2 Study I: Personality-Related Predictors of Insomnia in University Students

Objectives

1. To determine whether perfectionism and low self-esteem predict future insomnia symptoms, accounting for negative affective symptoms.

Methods

Procedure

All procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. All procedures involving human subjects were approved by the Queen's University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board (HSREB, PSIY-608-18). Informed consent *was obtained from all subjects prior to survey completion.*

The U-Flourish Student Well-being study is a longitudinal repeat-measure study of students at Queens' University, Canada. The survey is sent to all incoming first-year students each

September and follow-up surveys are sent to responders each March and September thereafter, until they fail to respond or leave the university. September surveys are sent two weeks into the first academic term and March follow-up surveys are sent at the conclusion of academic teaching, prior to final examinations. Student-led campus engagement campaigns encourage participation amongst first-year students at the start of each academic year the survey is conducted. As incentive to participate, students are entered into a draw for one of five iPads for each survey they respond to. Additionally, all students who complete the fall survey are emailed a voucher for a free drink at a café on campus. Further details of sampling and recruitment for the study have been published (Duffy et al., 2020; S. M. Goodday et al., 2019).

Participants

Data were obtained from first-year (n = 871) and second-year (n = 412) students responding to both the September 2019 survey and March 2020 follow-up. These analysis groups represent 17% and 8% of students in each year, respectively. The reduced number of responses in second-year students is due to loss to follow-up since initial contact in September 2018. There was insufficient statistical power to include transgender, gender-fluid and other gender diverse participants (<1%) in these statistical analyses.

Measures

Gender, self-reported diagnostic history, personality-related factors and symptom severity were measured in the September survey at the beginning of the academic year. Symptom severity was measured again at the conclusion of the academic year in March (Table 2.1).

Table 2.1 Timing of questionnaire measures

Measure	Entry (September)	End of Year (March)
Gender	x	
Self-reported sleep disorder diagnosis	x	
Sleep Condition Indicator (SCI)	x	x
Patient Health Questionnaire (PHQ-9)	x	x
Generalised Anxiety Screener (GAD-7)	x	x
Rosenberg's Self-Esteem (RSE)	x	
Clinical Perfectionism Questionnaire (CPQ)	x	

September-Only Measures

Self-Reported Diagnosis History: Participants who selected "sleep disorder (e.g. Insomnia)" in response to the question "Have you ever been diagnosed with any of the following mental health conditions or learning problems?".

Clinical Perfectionism Questionnaire (CPQ): The CPQ is a 12-item scale self-report measure of a clinically driven definition of perfectionism (Shafran et al., 2002). Scores range from 12 to 48, with higher scores indicating higher clinical perfectionism. The CPQ was split into two factors as proposed by Dickie: personal standards (range: 6 to 24) which correspond to the setting, checking and upwards trending of self-imposed ideals, and self-critical concerns (range: 4 to 16) which captures distress as a response to perceived failure (2012). Internal consistency (Cronbach's α) for personal standards was calculated as 0.73 (95% CI 0.70 to 0.57), and for self-critical concerns it was $\alpha = 0.64$ (95% CI: 0.60-0.67).

Rosenberg Self-Esteem Scale (RSE): A 10-item self-report measure of self-esteem that was originally designed for use with high school students but has since been validated in a variety

of populations (Rosenberg, 1965). Scores range from 10-40 with higher scores indicating higher self-esteem.

September & March Measures

Sleep Condition Indicator (SCI): An eight-item screening tool for insomnia symptoms based on the DSM-5 criteria with scores ranging from 0-32 and lower scores indicating worsened insomnia symptoms. In the analysis, SCI score at the conclusion of the academic year was used as a continuous outcome, though logistic regression reported in the appendix used a clinical symptom threshold of scores ≤16 (Espie et al., 2014). Questions address factors such as sleep continuity, severity of insomnia and daytime impairment symptoms. The SCI has strong internal consistency (Cronbach's $\alpha = 0.86$) and convergence with other insomnia screening tools.

Patient Health Questionnaire 9 (PHQ-9): A screening tool measuring depressive symptoms using nine items (rated 0= "Not at all" to 3= "Nearly every day"). Scores range from 0 to 27 with a clinically significant threshold of ≥ 10. The PHQ-9 has demonstrated strong reliability and validity in a variety of samples (Kroenke et al., 2001; Martin et al., 2006).

Generalised Anxiety Disorder Screener 7 (GAD-7): A screening tool measuring generalised anxiety symptoms with seven items (rated 0= "Not at all" to 3= "Nearly every day") resulting in total scores ranging from 0-21. A clinically significant threshold of score ≥ 10 was used. The GAD-7 has strong validity and reliability (Löwe et al., 2008; Spitzer et al., 2006)

Statistical Analysis

Person-mean imputation was used for scale data when a single item was missing. If more than one item was missing from a scale, the entire scale was coded as missing. As none of the variables of interest had more than 20% of the scale results missing, a complete case analysis was used. Complete case analysis was chosen because data tended to be either complete or missing for all scales (rather than a single scale) due to students not responding to the end-of-year questionnaire. In this situation, complete case analysis is preferred (Hughes et al., 2019).

For the purposes of descriptive analysis, comparisons between proportions of students were made using Chi-squared independence tests (Agresti, 2018). Comparisons between continuous measures were made using Welch's independent sample t-tests, as they have lower Type 1 error rates than Student's *t*-tests when variances differ between comparison groups and maintain performance when variances are equal (Delacre et al., 2017).

Associations between psychological constructs measured at entry to university and end-of-year SCI scores were examined with hierarchical regressions, meaning known covariates were entered in a separate step ahead of predictors of interest. This method controlled for the effects of sex, pre-existing sleep disorder diagnoses, and symptoms of insomnia and affective disorders at baseline (M. Lewis, 2007). Item three in the PHQ-9 asks about sleep problems ("Over the past month, how often have you been bothered by trouble falling/staying asleep, or sleeping too much?"); this item was removed from the scale for use in the regression model only. The model reported below uses this "PHQ-8" however a replication using the PHQ-9 can be found in the Appendix, Section 2. The baseline model was compared to the final model using an ANOVA (Field et al., 2012). The objective of this analysis was to

determine whether continuous measures of personality-related factors predicted insomnia severity rather than binary clinical status, so linear regression was used instead of logistic regression. This approach is also less pathologizing, an important consideration in both student mental health and the study of personality disorder.

All statistical analyses were conducted using R version 4.3.3. Results were reported as statistically significant where a p-value of <0.05 was detected. Linear regressions used the *lm()* function and models were compared using *anova()*.

Results

Descriptive Analysis

The analysis group had a mean age of 18.38 (SD = 1.25). They were also more likely to identify as female than the group of all eligible students (76% vs 58%, $\chi^2 = 178.192$, $p < .001$). Mean SCI scores for insomnia at conclusion of the academic year were not significantly different between first- and second-year students, 20.18 (SD = 7.42) *vs.* 20.87 (7.66), $t(783) = 1.50$, p = .13 . Significantly more students screened positive for insomnia, depression, and anxiety disorders at the completion versus beginning of the academic year (Table 2.2).

Table 2.2. Distribution of students in the Sleepio analysis group, including proportions of students self-reporting a sleep disorder diagnosis and reaching thresholds on clinical screening questionnaires

Outcome Measure	Beginning of academic year, n (%)	End of academic year, n (%)	x^2	p†
Analysis Group	1283 (100.0)	-	-	-
Female	980 (76.4)	-	-	-
Lifetime sleep disorder*	42 (3.3)	-	-	-

SCI	288 (22.4)	350 (27.3)	360.96	< .001
PHQ-9	420 (32.7)	481 (37.5)	323.43	< .001
GAD-7	431 (33.6)	481 (37.5)	260.53	< .001

† Values from Chi-squared test. * Lifetime student-reported diagnoses.

SCI: insomnia symptoms, PHQ-9: depressive symptoms, GAD-7: generalised anxiety symptoms

Comparative Results

There were significant differences in personality-related factors between the student groups who screened positive for insomnia (above the SCI threshold score of 16) compared to those who did not (Table 2.3). Students screening positive for insomnia had significantly lower self-esteem and more self-critical perfectionism and higher personal standards. Self-esteem was negatively correlated with both subscales of the CPQ: strongly with self-critical perfectionism (Spearman's *rho* = -0.68, $p < .001$) and weakly with personal standards (Spearman's *rho* = -0.13, $p < .001$).

Table 2.3. Comparisons of personality-related factors between groups screening positive and negative for insomnia at baseline.

	Normal Sleep Group [a] Mean (SD)	Insomnia Group [b] Mean (SD)	t-test Statistic†	Mean difference, [95% CI]†	*p*†
Self-Esteem [c]	19.63 (5.36)	15.04 (5.31)	13.65	4.59 [3.93, 5.25]	< .001
Self-Critical Perfectionism [e]	10.16 (2.35)	11.87 (2.29)	-11.69	-1.71 [-1.99, -1.42]	< .001
Personal Standards [e]	17.20 (3.33)	17.87 (3.59)	- 3.00	-0.67 [-1.11, -0.23]	< .01

SD: standard deviation. CI: confidence interval. † Value from Welch's *t*-test.
[a] Negative screen for insomnia (SCI > 16). [b] Positive screen for insomnia (SCI ≤ 16).
[c] Rosenberg Self-Esteem scale. [d] Subscales of Clinical Perfectionism Questionnaire

Longitudinal Hierarchical Linear Regression Model

In Table 2.4, adjusted R^2 values indicate that 52% of variation in insomnia symptoms at completion of first year was explained by both the baseline and final models. Adding personality-related predictors significantly improved model fit (ANOVA: F(3, 1283) = 3.30, p = .02.). The self-critical concerns dimension of perfectionism was a significant predictor of increased insomnia symptoms whereas personal standards and self-esteem were not. Variance inflation factor was < 3.2 for all covariates, suggesting acceptable collinearity (Myers, 1990).

Table 2.4. Multivariable linear regression predicting associations between risk factors measured at entry to university and insomnia symptoms measured at completion of first year.

	ΔR²	*B*	*SE B*	*t*	*p*
Baseline Model	0.52				
Intercept		5.93	1.85	3.21	<.01
Female		0.23	0.36	0.64	.52
Lifetime sleep disorder [a]		0.83	0.84	0.98	.33
Baseline SCI		0.68	0.03	24.86	< .001
Baseline PHQ-8[b]		-0.13	0.04	-3.12	< .01
Baseline GAD-7		-0.08	0.04	-1.92	.05
Final Model	0.00				
Intercept		7.16	2.42	2.96	< .01
Female		0.32	0.36	0.90	.37
Lifetime sleep disorder [a]		0.74	0.84	0.88	.38
Baseline SCI		0.68	0.03	25.34	< .001
Baseline PHQ-8 [b]		-0.07	0.05	-1.52	.13
Baseline GAD-7		-0.07	0.04	-1.68	.09
Perfectionism Subscales					
Self-Criticism [c]		-0.22	0.09	-2.50	.01
Personal Standards [c]		0.01	0.05	0.26	.80
Self Esteem [d]		0.01	0.04	0.34	.73
Adj. R^2 for final model:	0.52				

[a] Self-reported diagnoses at entry to university. [b] PHQ-9 at entry to university, item 3 removed.
[c] Subscale of the CPQ [d] Rosenberg Self-Esteem Scale.
Predictors significant at $\alpha < .05$ are highlighted in yellow.

Study I Discussion

Findings

The major contribution of this project was the finding that personality-related factors longitudinally predispose insomnia, as most existing research on predisposing factors is cross-sectional (van de Laar et al., 2010). Self-critical perfectionism was a unique predictor of insomnia even accounting for the effects of baseline affective symptoms, despite some suggestions that self-critical perfectionism may be a generic indicator of psychological distress (Jansson-Fröjmark & Linton, 2007; van de Laar et al., 2010). Additionally, students screening positive for insomnia were significantly more self-critical, had higher personal standards, and lower self-esteem than normal sleepers. In contrast, low self-esteem may stem from affective symptoms or general psychological distress, since it was elevated in students with insomnia but was not a significant predictor of symptoms. This study also replicated previous findings that high personal standards were not associated with insomnia (Jansson-Fröjmark & Linton, 2007; Vincent & Walker, 2000), lending further support to the notion of adaptive perfectionism (Dunkley et al., 2003).

In this model, insomnia was prospectively predicted by self-critical perfectionism. Though not universally considered a feature of personality, perfectionism does reflect habitual behaviours and perspectives, and as such self-critical perfectionism has been associated with all of the ICD-11 maladaptive trait measures as well as the borderline pattern specifier (Stricker et al., 2022). As previously mentioned, the role of self-critical perfectionism in predisposing insomnia has been attributed to generic psychological distress (Jansson-

Fröjmark & Linton, 2007; van de Laar et al., 2010). However, in this model self-critical perfectionism remained a significant predictor of insomnia despite controlling for baseline affective symptoms, which did not significantly contribute to the model. This may indicate that in university students, self-critical perfectionism has unique predisposing features not entirely explained by negative affective symptoms. The differing role of perfectionism in this model and the one reported by Jansson-Fröjmark & Linton may be attributable to participant age differences: perhaps being younger exacerbates the effects of self-criticism. A possible mechanism of action is that self-critical perfectionism may impair sleep due to the accompanying hyperarousal. Edinger (1988) hypothesised that insomnia associated with self-criticism and hyperarousal is distinct from insomnia associated with neuroticism, and that this distinction also determines treatment response. He found that sleep onset latency was not responsive to treatment (elements of modern CBT-I including psychoeducation, stimulus control therapy, and progressive relaxation) in a self-critical group but was in a neurotic group. These findings were based on cluster analysis, which may have resulted in an arbitrary division to create types of insomnia; nonetheless Edinger's findings are consistent with the notion of self-criticism as a unique contributor to insomnia.

Study I Implications

These findings suggest that a personality-related feature, self-critical perfectionism, may represent an important intervention target in help-seeking students with symptoms of insomnia. Results also support dissemination of evidence-based sleep guidance amongst university students. Descriptive analysis of this sample demonstrated that sleep disorders may

not be readily recognized, given that only 3% of participants reported a sleep disorder diagnoses, yet about a quarter of students reported clinically significant insomnia symptoms. Insomnia is persistent if left untreated (Espie, 2002), but fortunately effective treatment programmes have already been developed (Espie et al., 2019; Rossman, 2019).

Study I Limitations

This analysis was reliant on self-report measures, which only have moderate accuracy particularly for self-reported insomnia (A. G. Harvey & Tang, 2012). The analysis group was limited in scope as it was self-selecting, much more likely to be female and only included cisgender students; further research is required to confirm the effects of these psychological constructs on the mental health of transgender, genderfluid and non-binary students.

This study measured two facets of perfectionism using subscales of the CPQ which have been previously found to have acceptable reliability (Dickie et al., 2012). In this sample the self-critical perfectionism measure had moderate reliability $\alpha = 0.64$ (95% CI: 0.60-0.67), but these findings should be replicated using an alternative measure of self-critical perfectionism such as Frost's Multidimensional Perfectionism Scale (Frost et al., 1990).

Finally, the COVID-19 pandemic may also have affected student responses to the March 2020 questionnaire. Queens' University cancelled in-person classes on 13 March 2020 and the follow-up survey was open from 16 - 30 March. Students were likely preoccupied, and no in-person engagement events were possible. However, the effects of remote learning may not have been captured in this dataset due to the compressed timeline. Furthermore, there were no significant differences in proportions of end-of-year positive disorder screens between the

data analysed in the present study and the previous year's cohort responses in March 2019 (Lindsay et al., 2022).

2.3 Study II: Unsupervised Digital Cognitive Behavioural Therapy for Insomnia in University Students – Uptake, Adherence & User Characteristics

Objectives

1. To understand undergraduate students' attrition from a self-guided, unsupervised dCBT-I intervention over time.
2. To describe the characteristics of undergraduate students who self-select for a self-guided digital cognitive behavioural therapy intervention for insomnia symptoms.
3. To evaluate the association between personality-related factors and enrolment with the aforementioned digital intervention whilst controlling for demographic differences and psychiatric symptoms at baseline, including insomnia.

Methods

Participants

This analysis uses data from the U-Flourish September 2019 study. All incoming first-year students and second-year students who had previously completed the March 2019 survey were invited to participate in the survey, with 58% and 54% of each cohort responding, respectively. Engagement events catered to first-year students during their first weeks on campus were likely the cause of higher response rates in that group.

Materials

U-Flourish Questionnaire Measures

All data in this analysis arose from the September 2019 baseline study. All measures have been described above in Study I apart from a brief self-rating of current mental health on a scale ranging from one (very poor) to five (very good). This analysis used the full PHQ-9 including the sleep question since treatment uptake was the outcome of interest rather than insomnia symptoms.

Sleepio

Sleepio is a mobile application-based digital cognitive behavioural therapy intervention for insomnia (dCBT-i). Sleepio consists of a series of six automated weekly sessions, led by an animated narrator ('The Prof') and covering topics such as sleep hygiene, sleep restriction therapy, and a variety of cognitive and relaxation techniques (Espie et al., 2012). Users are offered tailored guidance on sleep timing and appropriate cognitive and behavioural techniques based on their responses to baseline questionnaires and daily sleep diaries. Sleepio's efficacy in clinical trial settings has been well-established (Bostock et al., 2016; Espie et al., 2012; Freeman et al., 2017).

Procedure

Recruitment followed the same procedures detailed in Study I above. After completing the baseline survey at the beginning of the academic year, all responders were offered free access to Sleepio.

Analysis

Missing Data

Person-mean imputation was used for scales with missing items, as described in Study I. Some students enrolled for dCBT-I without using their university email, thus preventing linkage to any of their survey data. As there is no survey data on which to base imputation, these participants have been excluded from the between-group comparisons.

Between-Group Comparisons

The sample was split into "enrolled" and "non-enrolled" groups depending on whether participants had created Sleepio accounts. Between-group comparisons were conducted using Fisher's Exact tests for categorical variables, Welch's *t*-tests for normally distributed continuous variables and Mann-Whitney U tests for all other continuous variables (Delacre et al., 2017; Kim, 2017; Nachar, 2008). Shapiro-Wilk tests and visual inspection of histograms were used to determine whether continuous variables were normally distributed (Razali & Wah, 2011).

Group Matching & Marginal Risk Differences

After initial between-group comparisons, a propensity score matching algorithm was used to create a non-enrolled comparison group with equal distributions of gender, self-reported sleep disorder diagnostic status, self-rated general mental health and baseline affective symptom severity (PHQ-9 & GAD-7). This matched group was used to determine whether personality factors impacted the decision to enroll for dCBT-I while controlling for the aforementioned factors. Using subset selection to match the groups based on these covariates prior to fitting a logistic model improves robustness and reduces risk of bias due to incorrect model

specification (Ho et al., 2007). Several matching algorithms were executed using the *MatchIt* package in *R* and compared to optimize the balance between the groups (Ho et al., 2007, 2023). Matched samples were assessed holistically based on both statistical results (number of unmatched "enrolled" participants, and standard mean difference, variance ratio, and empirical cumulative distribution function statistics for each variable) and visual assessment (density, love, and empirical quantile-quantile plots) (Austin, 2009; Belitser et al., 2011; Greifer, 2022; Ho et al., 2007). Nearest neighbour and optimal full matching algorithms were attempted, but optimal pair matching with Mahalanobis distances provided superior balance (Hansen & Klopfer, 2006). The optimal pair algorithm matched each member of the dCBT-I study group with an individual from the larger cohort (1:1 matching) optimally minimizing the sum of Mahalanobis distances between all pairs. The Mahalanobis distance is the pooled covariance matrix of all covariates in the matching algorithm, in this case gender, lifetimes reported sleep disorder, and scores on the SCI, PHQ-9, GAD-7 and current self-rated mental health (Rubin, 1980). Figure 2.1 shows the standardised mean differences for each matched covariate before and after optimal pair matching. The unadjusted sample showed worsened symptoms of insomnia, depression, and anxiety in the group of enrollers as compared to the rest of the sample, as well as increased likelihood of a lifetime sleep disorder diagnosis and an increased proportion of female students. These differences were reduced after matching.

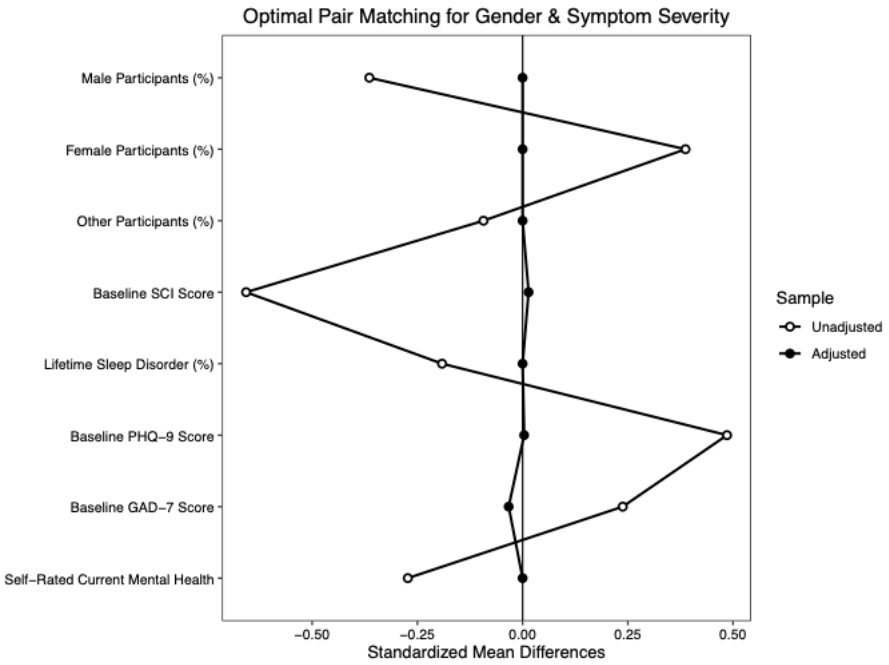

Figure 2.1. Standardised mean differences in covariates between enrollers and non-enrollers before and after exact matching for gender, lifetime sleep disorder diagnosis, and baseline insomnia, depression & anxiety symptom severity. Note: unlike the PHQ-9 and GAD-7, lower SCI scores indicate increased symptom severity.

After matching, continuous covariates were scaled to facilitate comparison and then the matched groups and their associated sampling weights were extracted and used to fit a logistic regression. The sampling weights are created as part of the matching algorithm to ensure that covariate distribution is balanced between the two groups. Following recommendations, all matched covariates as well as self-esteem and both perfectionism subscales were included in the model to increase precision and robustness in the effect estimates (Greifer, 2024). G-computation was then used to determine the marginal risk difference, or the effect of each variable on the probability of enrolling for dCBT-I for self-esteem and the two CPQ subscales (Greifer, 2022; Snowden et al., 2011).

All analyses were conducted using *R* v.4.3.3 (R Core Team, 2024). Between-group tests were implemented using the *stats* package v.4.3.3 (R Core Team, n.d.). Matching algorithms were

implemented with the *MatchIt* package v.4.5.5. (D. Ho et al., 2023). The "glm()" function in *stats* was used to fit the logistic regression model and the "avg_comparisons()" function from the *marginaleffects* v.0.19.0 package was used to perform g-computation to estimate marginal risk differences for each covariate (Arel-Bundock, 2024).

Results

2,947 out of 5,123 (58%) of first-year students and 1,063 out of 1,984 (54%) second-year students responded to the September 2019 U-Flourish survey. Of the baseline responders in both cohorts, 102 (2.5%) enrolled for dCBT-I, 77 of whom had linked U-Flourish survey data. Of these, 76 responded to all questions of interest and are included in the study group. Individuals in the study group were not significantly different in age from the larger sample but were significantly more likely to be female and have a sleep disorder as well as report significantly worse symptoms of insomnia, depression, and anxiety (Table 2.4). dCBT-I enrollers also reported significantly lower self-esteem and increased perfectionism compared to non-enrollers. The mean SCI score of dCBT-I enrollers was 16 (SD = 7.36), which is the threshold screening score for possible clinical insomnia. Figure 2.2 shows the declining number of participants who completed each treatment session: of the 102 students who enrolled, 28 (27%) completed their first session, 11 (11%) of enrollers completed two or more sessions, 6 (6%) completed Session 3, and 2 (2%) of students completed all dCBT-I sessions. For the first session only, a third (33%) of participants initiating the session did not complete it. Table 2.6. shows the results of a logistic regression model fitted to the matched groups. Increased self-esteem and self-critical concerns were associated with higher likelihood of enrolment (3% and 9%, respectively), while higher personal standards were

associated with lower likelihood of enrolment (< 0.2 %), however none of these associations were significant.

Table 2.5. Comparisons of demographic variables, psychopathology, and personality factors between the dCBT-I study group and the overall U-Flourish cohort.

Variable	dCBT-I Enrollers (n = 76)	Non-Enrollers (n = 3,344)	Test Statistic	p
Age, mean (SD)	18.46 (1.62)	18.31 (1.44)	W = 148372	.79
Gender				
Female, n (%)	64 (84%)	2344 (70%)	*Fisher's Exact*	.01
Male, n (%)	12 (16%)	972 (29%)	*Fisher's Exact*	.01
Other [a], n (%)	0 (0%)	28 (1%)	*Fisher's Exact*	.01
Sleep Disorder[b], n (%)	7 (9%)	123 (4%)	*Fisher's Exact*	.02
SCI, mean (SD)	16.00 (7.36)	20.84 (6.78)	W = 189188	< .001
PHQ-9, mean (SD)	10.80 (7.11)	7.35 (6.10)	W = 97816	< .001
GAD-7, mean (SD)	8.95 (5.97)	7.53 (5.67)	W = 1191216	.02
Current Mental Health, mean (SD)	3.05 (1.11)	3.35 (1.00)	W = 161318	.04
RSE[c], mean (SD)	16.86 (6.53)	18.69 (5.71)	W = 0.9872	< .001
CPQ[d], mean (SD)	34.89 (5.21)	33.21 (5.40)	W = 0.99476	< .001
Personal Standards	17.49 (3.42)	17.05 (3.44)	W = 0.98647	< .001
Self-Critical Concerns	11.36 (2.40)	10.47 (2.40)	W = 0.98102	< .001

[a] "Other" category includes transgender, non-binary, and genderfluid participants.
[b] Reported lifetime history of any sleep disorder e.g., insomnia, sleep apnea, restless leg syndrome
[c] Rosenberg Self-Esteem scale [d] Clinical Perfectionism Questionnaire

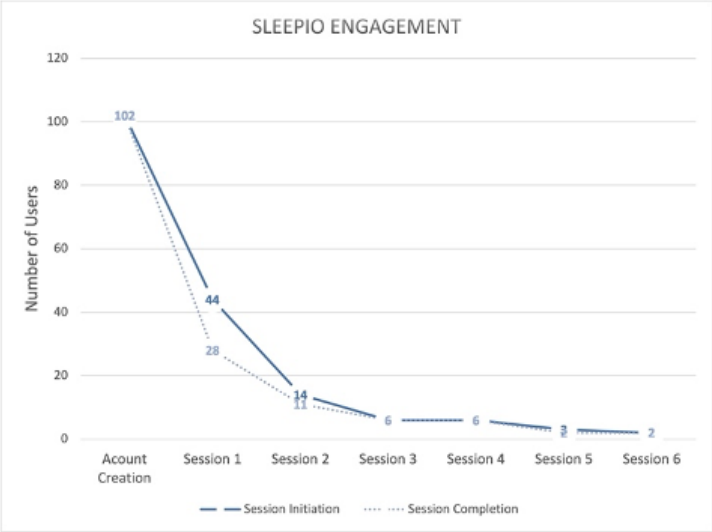

Figure 2.2. Sleepio dCBT-I engagement over time follows a decay trend.

Table 2.6. Logistic regression coefficients of psychopathology & personality-related factors on dCBT-I enrolment. Values are derived from a logistic regression model fitted to data from matched groups (n = 76 in each).

Covariate	Estimate[a]	Std. Error	95% CI	*p*
RSE	0.03	0.08	-0.12, 0.19	.68
CPQ-EC	0.09	0.06	-0.02, 0.21	.12
CPQ-PS	-1.79e-03	0.04	-0.08, 0.08	.97

[a] Fractional increase in likelihood of enrolment per standardised unit increase in covariate. After standardisation, all covariates have a mean of 0 and a standard deviation of 1. RSE = Rosenberg Self-Esteem Scale, CPQ-PS = Clinical Perfectionism Questionnaire – Personal Standards, CPQ-EC = Clinical Perfectionism Questionnaire –Self-Critical Concerns

Study II Discussion

The uptake of a self-guided CBT-based digital intervention for insomnia in a population of first- and second-year undergraduate students found low initial enrolment with rapid non-usage attrition: fewer than half of users creating accounts initiated the first treatment session and only 6% of participants reached the third treatment session, which introduces Sleep Restriction Therapy, hypothesised as a key component of CBT-I (L. Harvey et al., 2002; Kyle et al., 2023; C. B. Miller et al., 2014). This pattern of attrition has been observed in symptomatic university student populations using digital self-guided supports both in general (Becker & Torous, 2019; Pankow et al., 2024) and specifically with regards to this dCBT-I intervention (Freeman, Sheaves, Goodwin, Yu, Nickless, Harrison, et al., 2017). Although students enrolling for dCBT-I had significantly worse baseline SCI scores compared to non-enrollers, 43% of enrolled students did not meet the clinical screening threshold for insomnia, thus potentially reducing their observable advantage of completing treatment and increasing their risk of attrition (Eysenbach, 2005). A variety of factors hypothesised to increase attrition from digital interventions also apply in this setting: ease of enrolment in the treatment programme, no consequences of dropout, no access cost incurred by the user, no clinician

contact during treatment, limited introduction and expectation management prior to enrolment, few reminders and notifications sent by the app, considerable time commitment and external schedule demands experienced by university students in their first weeks of the academic term. Plausibly these factors, combined with mild symptom severity at baseline, contributed to attrition from the intervention, although this is common for digital health interventions (Eysenbach, 2005).

Students who chose to enrol for dCBT-I were significantly more likely to be female, report a lifetime diagnosis of sleep disorder, and experience worsened symptoms of insomnia, depression, and anxiety relative to their non-enrolling peers. These characteristics are likely intercorrelated regardless of dCBT-I enrolment: presumably uptake was motivated by some degree of insomnia, which often presents alongside psychological distress. Both these experiences are reported more frequently and severely by women (Morin et al., 2015; Riecher-Rössler, 2017).. Additionally, females are more likely than males to seek treatment for insomnia (Liu et al., 2016; Morin et al., 2006). Age could be associated with enrolment but the limited range (Interquartile range = 1 year) of this sample did not facilitate this analysis.

Students enrolling in dCBT-I also reported significantly lower self-esteem and increased perfectionism relative to their peers; however, these factors were not significant predictors of dCBT-I enrolment when gender and symptom severity were controlled for. This suggests that self-esteem and perfectionism are factors more likely to be associated with being female and experiencing insomnia rather than specifically with the decision to enrol and engage in a dCBT-I sleep intervention. However, it remains possible that there is an effect of personality-

related factors on the likelihood of enrolment for dCBT-I, but that this sample was underpowered to detect such an effect.

Study II Implications

Sleep interventions could support students' cognitive function and help them avoid the feedback loop between insomnia and other mental health difficulties (Palagini et al., 2022). However, this analysis of self-guided, unsupervised dCBT-I has identified attrition over time as a challenge to such interventions, a phenomenon well-documented in the extant literature (Becker & Torous, 2019; Eysenbach, 2005). Based on these results, it seems unlikely that offering dCBT-I as an unsupervised, open-access or non-targeted support for university students is an effective strategy. As previously discussed, the conditions of this study may not have optimally promoted adherence to the treatment programme. It remains unclear what conditions might best support treatment adherence in users with personality disorder, although a more targeted approach with signposting based on individual need would be an important step forward. A proof-of-concept study of a different dCBT-I intervention successfully used treatment response after three weeks to identify participants at risk of treatment failure, and found that increasing clinician contact at that point attenuated the risk of failure and led to a greater symptom reduction compared to participants whose clinician contact did not increase (Forsell et al., 2019). This effect necessitated only an increase of 14 minutes per week in clinician contact, which may be feasible in some university wellbeing settings.

This study tentatively suggests that the user characteristics associated with treatment uptake were primarily to do with symptom severity, and that the measured personality factors did not

encourage or deter treatment uptake. However, replication with a larger sample and more measures of personality would improve understanding of the relationship between personality, treatment uptake, and adherence.

Study II Limitations

Several limitations apply to this analysis. The U-Flourish sample is a self-selected group already biased towards female students, and these factors were exacerbated in the group who enrolled for dCBT-I. As a result, the findings are not reflective of university students in general but rather help-seeking students. This may have relatively inflated initial dCBT-I uptake. Additionally, the group of dCBT-I enrollers was relatively small, meaning the logistic regression was likely not adequately powered to identify effects of personality while controlling for baseline symptomatology. Further, as discussed in Study I, while self-critical perfectionism predisposed the development of insomnia, this only represents one factor contributing to personality. A broader personality measure, for example the Personality Inventory for DSM-5 may provide more insight to the effects of personality on adherence to treatment (Krueger et al., 2012). In particular, conscientiousness has been associated with adherence to medication, and further research may indicate whether this also applies to self-guided digital interventions (Molloy et al., 2014).

2.4 Overall Conclusions

Insomnia is highly prevalent in university students, but elective, self-guided treatment faces several obstacles, namely the small proportion of help-seekers and high attrition from treatment. In this non-clinical sample, self-critical perfectionism was associated with future insomnia symptoms while controlling for affective symptoms, suggesting that maladaptive

perfectionism may independently predispose the development of insomnia in university students and could form an important treatment target for insomnia. Self-critical perfectionism and self-esteem were not associated with treatment uptake over and above symptom severity, although this sample may have been underpowered to detect such an effect. Instead, self-guided insomnia treatment uptake was associated with female sex, an existing sleep disorder, and worsened affective and insomnia symptoms. Attrition from the intervention was high, with only 2% enrollers completing the intervention. Future research in a larger sample should investigate the effects other personality factors on adherence to digital interventions.

Chapter 3: Phenotyping Sleep, Circadian Rhythm, and Mood Instability in Borderline Personality Disorder

3.1 Introduction

Mood Instability in BPD

Borderline personality disorder (BPD) is partially characterised by high frequency fluctuations in valence and intensity of mood states (Bohus et al., 2021). Though mood instability is a transdiagnostic symptom, in the context of BPD it has been theorised to be the product of biological vulnerabilities, particularly limbic dysfunction, and invalidating early life emotional experiences (Broome et al., 2015; M. M. Linehan, 1993). More recent extensions of Linehan's theory have proposed trait impulsivity as an additional predisposing factor for mood instability in the context of BPD (Crowell et al., 2009). Linehan also suggested that mood instability might underpin other symptoms of BPD such as interpersonal problems, and it has also been postulated to underpin self-harm and suicidal ideation (Koenigsberg et al., 2002; Peters et al., 2016; Rizk et al., 2019). Traditional retrospective assessment of mood instability in BPD is hampered by recall biases regarding recency and intensity of mood, especially negative mood (Ebner-Priemer et al., 2006; Solhan et al., 2009), but mobile phones have created an opportunity for in situ assessment of mood instability via Ecological Momentary Assessment (EMA). In EMA studies, participants are asked to complete brief questionnaire measures at high frequencies, typically multiple times per day. Responses can be paired with other ambulatory data, for example physical activity, heart rate variability, social activity, or location data creating a richer and more accurate representation of daily experience (Yim et al., 2020). The value of EMA in studying BPD has already been recognised and used to demonstrate elevated mood instability as compared to healthy

participants, as well as greater differences in successive measures and increased time to return to baseline after a period of altered mood state (Ebner-Priemer et al., 2007; Stiglmayr et al., 2005; Trull et al., 2008). A more recent EMA study, the Automated Monitoring of Symptom Severity (AMoSS) study piloted a brief questionnaire called MoodZoom by following participants with BPD, participants with bipolar disorder and healthy volunteers for a period of at least three months. Not only did MoodZoom data correlate with weekly questionnaire measures of mania, depression and anxiety, its variability could be used to differentiate participants with BPD from the other groups (Tsanas et al., 2016).

Circadian Rhythm Disturbance in BPD

The AMoSS study also linked mood EMA responses with data from an accelerometer patch to identify a circadian phase delay in BPD participants' estimated sleep phase as compared to their activity and diurnal heart rate variations. This phase delay, which covaried with BPD symptom severity, was not present in healthy volunteers or participants with bipolar disorder, suggesting potential internal desynchrony in circadian function in people with BPD (Carr, Saunders, Bilderbeck, et al., 2018; Carr, Saunders, Tsanas, et al., 2018). In further analysis of this sample, the rest-activity pattern phase delay was also captured by wrist-worn accelerometers called actigraphs which showed circadian phase delay in BPD rest-activity patterns relative to other participants (McGowan et al., 2019a). Associations between personality disorder and circadian phase dysregulation have been suggested previously: Dagan et al. found consistently higher prevalence of all personality disorders (save obsessive-compulsive and paranoid personality disorders) in a sample of people with circadian sleep-wake phase disorders (CSWPDs) compared to healthy controls (1996). Similarly, people with BPD demonstrated increased prevalence of CSWPDs, particularly Delayed Sleep Wake

Phase Disorder (DSWPD) and Non-24 Hour Sleep-Wake Disorder (N24SWD) (Dagan et al., 1998). DSWPD is associated with difficulties falling asleep and late awakenings, resulting in a phase delayed rest-activity pattern relative to typical working hours. N24SWD occurs due to inability to entrain to external environmental cues and results in irregular rest-activity patterns typically longer than 24 hours (Reid & Zee, 2011). Dagan's findings suggest a possible association between personality disorder and irregularities in circadian phase, further supported by the prevalence of late chronotypes (preferences for activity later in the day) in BPD and its associated personality factors of low conscientiousness and low agreeableness (Fitzpatrick et al., 2020; Lipnevich et al., 2017; McGowan & Saunders, 2021; Tsaousis, 2010). Circadian sleep wake phase disorders are also associated with desynchrony between the phase of the internal circadian clock and the external environment, particularly light exposure as the principal zeitgeber of the circadian system. As a result, CSWPDs result in misalignment between desired bedtime and physiological preparedness for sleep, including body temperature and hormone levels such as melatonin and cortisol (Gamble et al., 2014; Reid & Zee, 2009). Individuals with circadian phase delay may regularly experience extended sleep onset latency when attempting to sleep prior to reaching the appropriate physiological state. Maladaptive behaviours and beliefs frequently develop in response to extended sleep latency, for example spending excessive amounts of time in bed (Morin, 1994). These behaviours and beliefs are common in people with BPD, who tend to have increased preoccupations with the consequences of poor sleep, feelings of helplessness about sleep problems and beliefs that medication is the only solution for insomnia (Philipsen et al., 2005; Plante et al., 2013).

In addition to insomnia, it has been hypothesized that the conflicting neural signals caused by circadian rhythm dysfunction may contribute to mood instability (Wulff et al., 2010). Actigraphic data from 21 individuals with BPD monitored for 28 days identified associations between circadian dysfunction and BPD symptoms: both rhythm stability and amplitude of activity levels were correlated with mood instability and impulsivity in BPD but not in healthy controls or people with bipolar disorder (BD), a disorder also partially characterised by mood instability (McGowan et al., 2020). Within-day variability in activity, daytime activity onset and the magnitude of nighttime activity also correlated with mood instability in the BPD group but not in healthy controls or BD. Symptoms of BPD have been previously linked with sleep disturbance, but this literature is scarce and mostly relies on self-reported sleep parameters which tend to be inaccurate (A. G. Harvey & Tang, 2012; Selby, 2013). These preliminary findings, in conjunction with numerous associations between mood symptoms and sleep disturbance in other psychiatric groups, lend further support to the hypothesis that circadian rhythm dysfunction, sleep disturbance, and mood instability are linked in BPD (Konjarski et al., 2018; Murray & Harvey, 2010).

Generally, the relationship between mood instability and sleep disturbance is thought to be reciprocal rather than unidirectional, but much of the evidence is cross-sectional and thus cannot disentangle this relationship (Konjarski et al., 2018; Zohar et al., 2005). Mood instability may be underpinned by heightened emotional reactivity (D'Aurizio et al., 2023), which can be measured in laboratory settings using visual stimuli paradigms. Evidence for sleep disturbance driving emotional reactivity includes an fMRI study which showed that sleep deprivation increased following-day amygdala reactivity to aversive stimuli by over 60% compared to individuals who had slept normally (Yoo et al., 2007). The primary role of

amygdala is processing emotionally salient stimuli; this is thought to be regulated by the orbital frontal cortex (OFC) (Izquierdo et al., 2005; H. Kim et al., 2004). People with BPD show increased amygdala activity in response to emotionally salient or aversive stimuli (Donegan et al., 2003; Herpertz et al., 2001; Minzenberg et al., 2007), and unlike healthy controls, the amygdala and OFC appear to be uncoupled in BPD (New et al., 2007). This neural pathway may also be implicated by mild sleep deprivation, which has been shown to increase emotional reactivity and intensify negative mood in non-clinical groups (James & Gregg, 2004; Zohar et al., 2005), though mild sleep deprivation driving emotional reactivity has not thus far been demonstrated in BPD.

There is also evidence for the inverse relationship of mood inhibiting sleep: pre-sleep anxiety and distress are often associated with insomnia (Vandekerckhove et al., 2011), as is trait anger, a core symptom of BPD (Shin et al., 2005). High-arousal positive emotion has been associated with reduced sleep time and reduced proportion of time spent asleep during the attempted sleep window (Tavernier et al., 2016). Mania, a period of extremely elevated affect and energy, is typically associated with decreased need for sleep (Harvey, 2008).

Several studies have attempted to disentangle the relationship between sleep and mood by examining associations between a night's sleep and the following day's mood or vice-versa. One such study found that higher self-reported sleep quality significantly improved EMA mood ratings on the following day, with this effect being larger than the reverse association (Triantafillou et al., 2019). This finding was consistent between healthy controls and participants with affective disorders. However, self-reported sleep quality is a single parameter which is often decoupled from objective sleep measures in both clinical and

community samples (A. G. Harvey & Tang, 2012). An actigraphy study of individuals with BD did not find significant associations between sleep and mood, but did report that daytime activity impacted the following day's mood, further suggesting lagged associations between rest-activity patterns and mood (Merikangas et al., 2019). To our knowledge, no studies have yet explored such lagged associations between sleep, mood, or mood instability in BPD populations.

Objectives

The objectives of this study are:

1. To replicate previous findings of delayed phase in rest-activity patterns and associations between objective measurements of rest-activity and mood instability.

2. To employ lagged models to investigate two hypotheses: (i) that sleep disturbance leads to increased mood instability the following day in individuals with BPD, and (ii) that mood instability contributes to sleep disturbance the following night. Comparisons will also be made with the control group.

3.2 Methods

Participants

As part of Stage 1 of the Sleep in BPD study, 64 female participants aged 18-35 were recruited as healthy volunteers (n = 31) or based on a pre-existing diagnosis of BPD (n = 33). Participants were recruited through online advertisements on the University of Oxford Department of Psychiatry website and local community bulletins, as well as through flyers

and announcements at local outpatient mental health services. Interested participants completed an online webform in which they self-reported exclusion criteria diagnoses of bipolar disorder, schizophrenia, neurodevelopmental disorders, sleep apnoea, restless leg syndrome or other sources of chronic sleep disturbance. Remaining participants were invited to an in-person study visit which began with screening for active suicide risk and re-screening for bipolar disorder and schizophrenia using the Mini-International Psychiatric Interview (MINI) version 5.0.0 (Sheehan et al., 1998). At this point BPD diagnoses were confirmed using the International Personality Disorder Examination (IPDE) interview (Loranger et al., 1997). Additionally, volunteers who reported emergency psychiatric hospital admissions within the month prior to enrolment were not eligible to participate. The study protocol was approved by the NRES Committee Oxford B - South Central (Reference no. 18/SC/0366) and all participants gave written informed consent.

Measures

Questionnaire Measures

Self-reported sleep quality and disturbance were measured at study entry and final visit using the self-report Pittsburgh Sleep Quality Index (PSQI). Seven component scores (subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbance, use of sleeping medications and daytime dysfunction) on 0-3 scales are combined to form a global score with a possible range of 0-21 and a threshold score of ≥ 5 indicating significant sleep disturbance (Buysse et al., 1989).

Upon awakening each morning, participants completed the Consensus Sleep Diary (Carney et al., 2012) which was used in parallel with the actigraphy to derive sleep parameters (Carney

et al., 2012). The CSD is a structured form on which participants record clock times for getting into bed, trying to start sleeping, final awakening, and getting out of bed. The form also includes spaces to record number of awakenings, time spent awake, and subjective sleep quality.

Insomnia symptom severity was measured at study entry and conclusion with the Sleep Condition Indicator (SCI), an eight-item self-report questionnaire with scores ranging from 0-32 and scores ≤ 16 indicating clinically significant insomnia symptoms (Espie et al., 2014).

Chronotype was assessed at the first study visit using the Morningness-Eveningness Questionnaire (MEQ), a self-report 19-item questionnaire with a possible score range from 16-86 and scores > 58 indicating early chronotype (Horne & Ostberg, 1976).

Maladaptive cognitions about sleep were measured at study entry and conclusion with the self-report abbreviated Dysfunctional Beliefs and Attitudes about Sleep (DBAS-16) questionnaire. Participants rate their degree of conviction on a scale from 0-10 for each of 16 statements about their sleep and associated daytime impairment, and their total score is the average value of these responses. Total scores ≥ 4 represent unrealistic expectations about sleep (Morin et al., 2007a).

Affective lability, or frequency and range of affective states, was measured at study entry and conclusion with the self-report short-form Affective Lability Scale (ALS-SF). Scores range from 0 to 54, with higher scores denoting increased affective lability. The scale can be broken into three subscales: Anxiety/Depression, Depression/Elation, and Anger, which have been shown to have good internal consistency (Oliver & Simons, 2004).

Sleep & Circadian Measures

Rest-activity patterns were measured using wrist-worn MotionWatch 8 actigraphs, which are CE-marked Class 1 medical devices with FDA approval (K132764) manufactured by CamNTech Ltd (Fenstanton, UK). Device recording periods were set to 60 second epochs. Participants were asked to wear the actigraphs on their non-dominant wrists continuously during the 28-day study period, removing them only for bathing, swimming, and high-impact sport. To improve accuracy of sleep-scoring, participants were also instructed to press a button on the actigraph just before they began trying to sleep and just after their final awakening, which created 'Lights Out' and 'Got Up' activity markers, respectively, in their data. Sleep timings derived from actigraphy were confirmed using nightly self-report sleep diaries, which could be completed either on paper or on an online version emailed to participants each morning.

The following are definitions of the sleep ('MotionWare Sleep Analysis: Advanced Actigraphy Software Tools', n.d.) and non-parametric circadian variables (Van Someren et al., 1999) derived from actigraphy and sleep diaries:

- Time in Bed (TIB): The standard definition of TIB is time elapsed between getting into bed before sleep and getting out of bed after sleep. However, MotionWare automatically calculates TIB as time elapsed between 'Lights Out' and 'Got Up' activity markers and does not offer an alternative variable. This definition, which excludes time spent in bed before attempting to sleep, was used in this analysis.
- Total sleep time (TST): Number of minutes automatically scored as sleep within the window between first sleep onset and final awakening.
- Wake after sleep onset (WASO): Number of minutes automatically scored as awake within the window between first sleep onset and final awakening.
- Sleep onset latency (SOL): Time elapsed between 'Lights Out' and sleep onset.
- Sleep efficiency (SE): TST expressed as a percentage of MotionWare's defined TIB period.
- Least Active Five Hours (L5): Average activity level during the least active five hours of the day, typically during sleep. Considered an indicator of sleep quality.

- L5 Onset: the clock time at which the least active five hours of the day began which is an indicator of circadian phase.
- Most Active Ten Hours (M10): Average activity level during the day's most active period; an indicator of daytime activity intensity.
- M10 Onset: the clock time at which the most active ten hours of the day began. This is another indicator of circadian phase.
- Intradaily variability (IV): a measure of fragmentation of the rest-activity pattern on a [0,2] range. Normal rest-activity patterns present as one extended period each of rest and activity per day, while abnormal rest-activity patterns might include shorter and more frequent state changes, resulting in a greater IV value.
- Interdaily stability (IS): a measure of the regularity between days on a [0,1] range, with higher values representing more similarity between days. IS indicates the strength of association between activity patterns and external zeitgebers.
- Relative amplitude (RA): a measure of the difference in activity levels between the rest (L5) and active (M10) periods adjusted for total activity levels, resulting in a [0,1] range (derivation formula below). Higher values are desirable as they indicate greater differences between rest and activity.

$$RA = \frac{M10 - L5}{M10 + L5}$$

Mood Instability

Intensity of each mood state was measured using MoodZoom, an EMA tool with which participants rate their current mood on using six descriptors on 0-7 Likert scales: anxiety, sadness, energy, elation, anger, and irritability. Mood instability was conceptualised as the variability between successive ratings of intensity within each valence, an approach which has been previously validated and used to distinguish between people with BPD and both people with bipolar disorder and healthy controls, providing specificity beyond standard retrospective questionnaires (Ebner-Priemer et al., 2007; Tsanas et al., 2016).

Procedure

Interested volunteers were invited to complete an online screening questionnaire, then to attend an in-person study visit where they were screened for suicide risk and symptom

severity using validated questionnaire measures. Eligible participants were provided with a paper sleep diary and an actigraph and were enrolled with MoodZoom. The first three weeks of the study consisted of a low-intensity recording period (LIRP) in which participants were prompted via email to complete the MoodZoom questionnaire twice a day at 8:00 and 20:00. Late responses were permitted until the following MoodZoom prompt was sent. Before the fourth and final week of the study was a high-intensity recording period (HIRP) in which participants were prompted to complete MoodZoom ten times per day with prompts every 90 minutes between 7:00 and 20:30 PM. After the high-intensity period, participants attended a final study visit where they returned their actigraphs and paper sleep diaries (Figure 3.1).

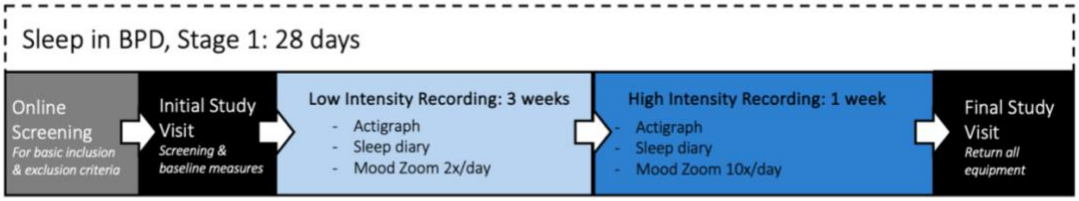

Figure 3.1. Sleep in BPD Stage 1 study protocol. Diagram adapted from the original study protocol written by Dr Niall McGowan.

Analysis

Actigraphy

Upon return of the actigraphs, data was downloaded and scored by an experienced sleep-scorer (Dr Niall McGowan) using CamNTech MotionWare software v.1.2.26, the algorithm of which has been validated in comparison with polysomnography, the gold-standard for measuring sleep variables (Elbaz et al., 2012; Morgan et al., 2012; *MotionWare*, n.d.). MotionWare uses epoch-by-epoch sleep/wake categorisation based on acceleration to

automatically determine the sleep period for each night, though this period was manually adjusted to match the 'Lights Out' and 'Got Up' activity markers and sleep diaries when necessary. Following visual inspection of participant actograms in MotionWare, in instances when the activity markers were absent or misaligned from the light and activity profiles by more than 15 minutes, then the sleep diary question "What time did you try to go to sleep?" was used instead of the 'Lights Out' marker and "What time did you get out of bed for the day?" was used instead of 'Got up'. If these sleep diary responses were also misaligned from light and activity profiles, the sleep diary was still used, as the discrepancy could be due to insomnia. If both the activity markers and sleep diary entry were absent, then the definitive ending of light and activity profiles were used. If this was indiscernible, the night was excluded from the analysis. MotionWare then calculates nightly and average values for TIB, TST, SOL, WASO and SE, as defined above. Nights with missing actigraphy data were excluded from the calculation of sleep variables. Non-parametric circadian rhythm analysis (NPCRA) variables, including L5, M10, IS, IV, and RA were calculated using the raw accelerometer data processed by the *nparACT* version 0.8 package in *R* version 4.2.3, as this package provide more precise values for L5 and M10 than MotionWare, which only estimated these parameters to the closest hour. The following steps were taken to handle missing actigraphy data before NPCRA variables were calculated: missing active periods less than three hours long were replaced with the average activity level of that day in MotionWare. Missing rest periods under three hours were not imputed given the risk of missing sleep and wake times. After this imputation, only periods with greater than five days of continuous data were included in the calculation of NPCRA variables. Five days is the period required for stable actigraphy-derived estimates of NPCRA variables in adolescents

and thus seemed like a reasonable cutoff (Acebo et al., 1999; Sadeh, 1996). Four participants (3 BPD, 1 HV, $p = .61$ via Fisher's Exact test) had insufficient actigraphy data to be included in analysis, which included one loss to follow-up and three cases of non-adherence to actigraph wear.

MoodZoom Responses

Missing data was imputed for LIRP days with at least one response (out of two) and HIRP days with at least three responses (out of ten). Imputation was done using the Kalman Filter function from the *imputeTS* package (v.3.3) in *R*. Kalman filtering has been shown to outperform multiple imputation for handling missing ecological momentary assessment data (Hamaker & Grasman, 2012; Mansueto et al., 2022; Slipetz et al., 2023). Variability between successive MoodZoom ratings was calculated as the root mean squared successive difference (RMSSD) via the *varian* (v.0.2.2) package in *R*. RMSSD has been previously used as a measure of mood instability in psychiatric populations (Ebner-Priemer et al., 2007; Gershon & Eidelman, 2015; Tsanas et al., 2016). Confirmatory factor analysis verified whether the previously identified three-factor structure of MoodZoom was present in this dataset (Tsanas et al., 2017). Each of the three factors was derived by taking the mean RMSSD of its components, as follows: the negative factor from the anxious and sad responses, the positive factor from the elation and energy responses and the irritability factor from the anger and irritability responses. Factor loadings > 0.5 were interpreted as strong (T. A. Brown, 2006).

Statistical Analysis

In order to compare demographic, occupational, sleep, circadian, and mood instability variables between the BPD group and the healthy volunteers, Welch's *t*-tests were used for

normally distributed variables, Fisher's exact test for proportions of categorical variables, Watson-Williams' tests for L5 start times, which were circular due to distribution around 24:00, and Mann-Whitney U-tests for all other variables (Delacre et al., 2017; H.-Y. Kim, 2017; Nachar, 2008; G. S. Watson & Williams, 1956). The tests were implemented in the *stats* v.4.3.1 package in *R* (R Core Team, n.d.). Correlations between variables were calculated with Pearson's *r* also from *stats* v.4.3.1(R Core Team, n.d.). The *circular* v.0.5 package in *R* was used to calculate the means and standard deviations of L5 start times (U. Lund et al., 2023).

HIRP NPCRA Linear Models Predicting Mood Instability

Separate linear regression models were fitted for the clinical and control groups to determine the associations between mean RMSSD of the three mood factors (negative, positive, and irritable), and sleep and circadian variables across the week-long HIRP period. Separate models were made for each group, as the associations between mood instability and sleep were hypothesised to be different in BPD compared to the healthy control group. Due to limited sample sizes, separate models were fitted for sleep and NPCRA variables to reduce model complexity. Covariates were age and employment status, with the latter selected based on previous work (Gillett et al., 2021) and entered as a binary categorical variable (0 = unemployed, 1 = employed). Forced entry was used to insert the remaining predictor variables, as this method improves replicability of findings (Studenmund & Cassidy, 1987). Sleep variables included TST, SE & SOL. TST was included as it is the simplest quantification of sleep and can indicate both insomnia and hypersomnia (Hirshkowitz et al., 2015). SE is associated with subjective sleep quality but also provides an indication of TIB and WASO (Åkerstedt et al., 1994; Reed & Sacco, 2016). SOL was included as an indicator

of sleep-onset insomnia, which has been associated with BPD (Selby, 2013). NPCRA models included standard predictors: IS, IV, L5 and M10 activity, L5 and M10 start times. RA was not included as it was highly correlated with L5 and M10 activity levels. All linear regression models were fitted using *stats* v.4.3.1 in *R* (R Core Team, n.d.).

Day-lagged Prospective Models

To evaluate the effect of sleep on following-day RMSSD of mood ratings, multilevel linear models predicting each MoodZoom factor (negative, positive and irritable) were fitted using data from the HIRP period. Level 1 predictors were age, employment status, and weekend/weekday status coded using a binary dummy variable. Level 2 predictors were TST, SE, and SOL. Non-parametric circadian variables were not included in the day-lagged models as they had higher levels of missingness compared to the sleep variables (TST, SE, SOL), because participants were more likely to remove the actigraphs during the day than overnight. Daytime removal did not impact calculation of sleep variables but did restrict non-parametric circadian rhythm analysis. Including NPCRA variables in the models may have reduced statistical power of the models and potentially compromised their robustness.

Models were gradually increased in complexity, with initial models only including fixed effects of forced entry sleep and demographic variables. Random effects were then added: random intercepts to model between-participant differences in mean RMSSD of mood intensity, and random slopes to model the different effects of TST on RMSSD of mood intensity for each participant. Finally, autoregressive covariance terms were added to account for within-person correlations in mood instability. First-order autoregressive terms account for correlations with the previous and following day's mood instability. The addition of a

"moving average" accounts for further correlations between residuals (Pinheiro & Bates, 2006). The models typically failed to converge when second-order autoregressive terms accounting for correlations with mood instability with the two previous and following days, likely due to insufficient sample size (Zuur et al., 2009). The decision to include an autoregressive term was based on comparisons between the random slopes models and i) a model with a first-order autoregressive term and ii) a model with a first-order regressive term and first-order moving average. Final models are reported with autoregressive covariance structures only when ANOVA testing suggested that they improved model fit. Multilevel linear models were fitted using the *nlme* v.3.1-164 package in *R* (Pinheiro et al., n.d.).

3.3 Results

Demographic Variables

Data from 60 participants was analysed; their demographic information is shown in Table 3.1. There were no significant between-group differences in age. Differences in employment status were not significant (p = .08). Only participants in the BPD group were taking psychotropic medications, most commonly antipsychotics and antidepressants. There were not enough participants in part-time study or employment (n = 3) to justify entering it as a separate covariate in statistical models.

Table 3.1. Comparative Demographic & Occupational Data

Demographics	BPD (n = 30)	HC (n = 30)	Test Statistic	*p*
Age, mean (SD)	26.10 (4.96)	25.73 (3.14)	t = 0.34	.73
Unemployed, no. (%)	8 (27%)	2 (7%)	Fischer's Exact 2x2 test	.08
Part- or full-time employment or study, no. (%)	22 (73%)	28 (93%)	Fischer's Exact 2x3 test	.08

Medications				
Anticonvulsant, no.	0	0		
Antipsychotic, no.	11	0		
Antidepressant, no.	12	0		
Anxiolytic, no.	4	0		
Hypnotic, no.	4	0		

Self-Report Data

Self-reported sleep measures were significantly worse in the clinical group compared to controls. The BPD participants reported significantly worsened insomnia symptom severity (SCI, $W = 925.5$, $p < .001$), dysfunctional beliefs about sleep (DBAS, $t = -7.35$, $p = .001$), global affective lability (ALS-SF, $W = 13$, $p < .001$) and global sleep quality (PSQI, $W = 66$, $p < .001$), as shown in Table 3.2 below. There were significant between-group self-reported differences in PSQI subscales of: subjective sleep quality, sleep onset latency, sleep duration sleep disturbance, use of sleep medication and daytime dysfunction (all $p < .001$), again with BPD > HC. There were no significant between-group self-reported differences in sleep efficiency. Chronotype as measured by the MEQ was significantly later in the BPD group (MEQ, $t = 2.95$, $p < .01$).

Table 3.2. Mean questionnaire response scores, by group.

Measure, Timepoint	BPD mean (SD)	HC mean (SD)	Test statistic	*p*
MEQ	48.00 (12.70)	56.03 (8.57)	$t = 2.95$	<.01
SCI	15.56 (7.78)	27.90 (3.35)	$W = 925.5$	< .001
DBAS	5.67 (1.39)	3.31 (1.15)	$t = -7.35$	< .001
PSQI Global	11.13 (3.59)	5.68 (1.76)	$W = 66$	< .001
PSQI Subjective Sleep Quality	1.47 (0.67)	0.71 (0.53)	$W = 219$	< .001
PSQI Sleep Latency	1.75 (0.98)	0.61 (0.67)	W = 189	< .001
PSQI Duration	1.03 (1.06)	0.16 (0.45)	$W = 264.5$	< .001
PSQI Sleep Efficiency	2.81 (0.74)	2.81 (0.75)	$W = 495$	.99
PSQI Sleep Disturbance	1.50 (0.57)	0.90 (0.40)	$W = 245$	<.001

PSQI Sleep Medication	1.03 (1.40)	0.00 (0.00)	*W* = 310	< .001
PSQI Daytime Dysfunction	1.53 (0.67)	0.48 (0.51)	*W* = 135	< .001
ALS-SF Global Score	50.47 (9.46)	23.42 (5.22)	*W* = 13	< .001
ALS-SF: Anxiety/Depression	15.43 (2.97)	6.58 (1.89)	*W* = 13.5	<.001
ALS-SF: Depression/Elation	22.09 (4.35)	11.32 (3.38)	*W* = 33.5	<.001
ALS-SF: Anger	12.94 (4.22)	5.52 (1.23)	*W* = 35	<.001

Actigraphy-derived Sleep & Circadian Variables

Table 3.3 below shows the BPD group had significantly lower sleep efficiency (77 vs. 81%,, $p < .01$) driven by increased time spent in bed (mean difference in minutes: 49, $p < .001$) more wake after sleep onset (mean difference in minutes: 14, $p < .01$). Total sleep time and onset latency were not significantly different between groups. Non-parametric measures of circadian rhythm were not significantly different between groups save intradaily variability, which indicated worsened fragmentation in healthy volunteers. Interdaily stability was not significantly better in the BPD group (0.46 versus 0.41, $t = -1.83$, $p = .07$).

Table 3.3. Sleep and non-parametric circadian rhythm analysis variables, by group.

Sleep Variables	BPD (n = 30)	HC (n = 30)	Test Statistic	*p*
TST, mean min (SD)	422.46 (56.25)	409.05 (37.77)	*t* =-1.08	.28
TIB, mean min (SD)	552.98 (59.67)	504.19 (29.96)	*t* =-4.00	<.001
SOL, mean min (SD)	12.33 (13.66)	8.51 (4.55)	*t* =-1.45	.15
WASO, mean min (SD)	85.01 (25.43)	70.88 (17.13)	t =-2.52	.01
SE, % (SD)	76.71 (6.82)	81.18 (5.43)	t = 2.812	<.01
NPCRA Variables				
Rhythm Structure				
Interdaily Stability	0.46 (0.09)	0.41 (0.10)	*t* =-1.83	.07
Intradaily Variability	0.81 (0.21)	0.95 (0.19)	*W* = 627	< .01
Relative Amplitude	0.89 (0.07)	0.91 (0.05)	*W* = 508	.27

Activity Levels				
L5 Activity	16.80 (12.71)	13.65 (8.83)	W = 352.5	.21
M10 Activity	292.41 (81.27)	275.79 (65.99)	W = 378.5	.40
Rhythm Timing				
L5 Start time, clock time (SD in min)	1:08 (20)	1:21 (16)	F = 0.54	.47
M10 Start time, clock time (SD in min)	10:18 (133)	10:04 (140)	W = 388.5	.49

TST: total sleep time, TIB: time in bed, SOL: sleep onset latency, WASO: wake after sleep onset, SE: sleep efficiency, NPCRA: non-parametric circadian rhythm analysis, L5: least active five hours, M10: most active ten hours.

MoodZoom Data

Table 3.4 shows the percentage of missing MoodZoom responses in the LIRP (twice daily prompts) and HIRP (10 times daily prompts) periods. In the LIRP period, there were fewer missing responses from the BPD group than the healthy volunteers on average, though the difference was insignificant (3.13 versus 4.03, p = .06). In the HIRP period this was reversed with significantly more responses were missing from the BPD group (21.97 versus 18.67, p < .001). Tables 3.5 and 3.6 show mean MoodZoom scores during the LIRP and HIRP periods after Kalman filtering. During both periods, mean scores for negative and irritable moods were significantly higher in the BPD group and mean scores for elation and energy were significantly lower in the BPD group, with all differences significant at p < .001. Data without imputation are presented in the Appendix, Section III, Tables 3A.1 - 3A.4.

Table 3.4. Missingness in MoodZoom data, by group and period.

	BPD	HC	(p) Fisher's Exact Test between groups
LIRP, mean no. missing responses per participant (%)	3.13 (7.46)	4.03 (9.60)	.06
HIRP, mean no. missing responses per participant (%)	21.97 (31.38)	18.67 (26.67)	< .001

Table 3.5. Mean MoodZoom scores during the LIRP period (three weeks), with Kalman filtering.

	Anxiety	Elation	Sadness	Anger	Irritability	Energy
BPD	2.18 (1.60)	1.35 (1.52)	1.97 (1.84)	0.88 (1.30)	2.00 (1.70)	1.68 (1.50)

HC	0.93 (1.24)	2.15 (1.86)	0.61 (1.21)	0.30 (0.87)	0.82 (1.25)	2.67 (1.63)
Statistic	W = 41290	W = 978987	W = 409001	W = 558820	W = 447090	W = 1062919
p	<.001	<.001	<.001	<.001	<.001	<.001

Table 3.6. Mean MoodZoom scores during the HIRP period (one week), with Kalman filtering.

	Anxiety	Elation	Sadness	Anger	Irritability	Energy
BPD	2.22 (1.46)	1.49 (1.47)	1.65 (1.63)	0.86 (1.27)	2.05 (1.59)	1.99 (1.39)
HC	0.91 (1.17)	2.26 (1.86)	0.55 (1.05)	0.23 (0.76)	0.63 (1.02)	2.98 (1.44)
Statistic	W = 973058	W = 2590212	W = 1143709	W = 1365990	W = 948698	W = 2868340
p	<.001	<.001	<.001	<.001	<.001	<.001

Table 3.7 shows significantly greater mean RMSSD of MoodZoom ratings in the BPD group during the three-week long LIRP period, aside from mean RMSSD of energy ratings which was not significantly different between groups. The difference in mean RMSSD of anxiety, sadness, anger, and irritability ratings were all significant at $p < .001$. The mean RMSSD of elation ratings was greater in the BPD group at $p = .03$. During the HIRP period, all moods had significantly greater mean RMSSD in the BPD participants compared to the control group (Table 3.8). The difference in mean RMSSD of anxiety, sadness, and irritability was significant at $p < .001$. The difference in mean RMSSD of anger was significant at $p < .01$, and the differences in mean RMSSD of elation and energy were both significant at $p = .02$.

Table 3.7. RMSSD of MoodZoom scores during the LIRP period (two daily ratings for three weeks), after Kalman filtering the raw MoodZoom scores.

	Anxiety RMSSD	Elation RMSSD	Sadness RMSSD	Anger RMSSD	Irritability RMSSD	Energy RMSSD
BPD	1.69 (0.57)	1.57 (0.62)	1.90 (0.61)	1.32 (0.65)	1.74 (0.43)	1.80 (0.72)
HC	1.12 (0.55)	1.25 (0.48)	0.96 (0.61)	0.68 (0.59)	1.14 (0.60)	1.57 (0.46)
Statistic	*t* =-4.04	*t* =-2.25	*t* =-6.07	*W* = 224.5	*W* = 230.5	*W* = 401

p	< .001	.03	<.001	< .001	< .001	.20

Table 3.8. RMSSD of MoodZoom scores during the HIRP period (ten daily ratings for one week), after Kalman filtering the raw MoodZoom scores.

	Anxiety RMSSD	Elation RMSSD	Sadness RMSSD	Anger RMSSD	Irritability RMSSD	Energy RMSSD
BPD	1.20 (0.31)	1.07 (0.45)	1.00 (0.36)	0.92 (0.45)	1.22 (0.41)	1.15 (0.39)
HC	0.69 (0.40)	0.78 (0.45)	0.53 (0.41)	0.39 (0.37)	0.71 (0.39)	0.91 (0.38)
Statistic	*t* =-5.55	*t* =-2.47	*W* = 194	*W* = 0.94	*t* =-4.94	*W* = 298
p	< .001	.02	<.001	< .01	<.001	.02

Confirmatory Factor Analysis of MoodZoom Data

Visual inspection of the Scree plot (Figure 3.2) found limited increases in explained variance in models with greater than three factors, indicated by the shallower slope of the line segments between points representing models with more than three components. Confirmatory factor analysis (Table 3.9) using the three previously identified MoodZoom factors (i. Energy & Elation, ii. Anger & Irritability, iii. Anxiety & Sadness) found that all parameter estimates demonstrated strong and significant associations between the observed MoodZoom data and three hypothesised latent variables. Table 3.10 shows a comparison of two- and three-factor models resulting in similar comparative fit indices (CFI) and Bayesian information criterion (BIC) values, but ANOVA comparison found the three-factor model was significantly superior to the two-factor model, in both the BPD group ($\Delta\chi2 = 132.39$, $p < .001$) and the control group ($\Delta\chi2 = 36.35$, $p < .001$).

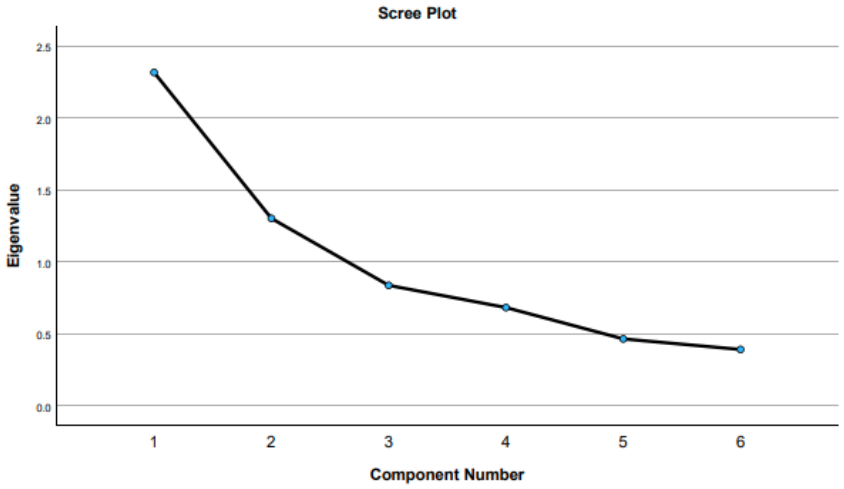

Figure 3.2. Scree plot showing variance of MoodZoom data explained by each additional component.

Table 3.9. Confirmatory factor analysis parameter estimates showing weights of each MoodZoom item to latent factors.

BPD Group	Items	Estimate	Std. Err.	*z*	*p*
Negative	Sadness	0.59	0.03	20.00	< 0.001
	Anxious	0.52	0.03	18.64	< 0.001
Irritable	Irritable	0.79	0.03	26.10	< 0.001
	Anger	0.62	0.03	22.77	< 0.001
Positive	Elation	0.98	0.04	23.22	< 0.001
	Energy	0.59	0.03	19.00	< 0.001
HC Group	**Items**	**Estimate**	**Std. Err.**	***z***	***p***
Negative	Sadness	0.51	0.03	17.70	< 0.001
	Anxious	0.44	0.03	15.80	< 0.001
Irritable	Irritable	0.61	0.03	21.90	< 0.001
	Anger	0.53	0.02	22.17	< 0.001
Positive	Elation	0.72	0.04	20.28	< 0.001
	Energy	0.59	0.03	18.32	< 0.001

Table 3.10. ANOVA comparison of two- versus three-factor confirmatory factor analysis models for MoodZoom data.

BPD	**χ2 (df)**	**RMSEA [95% CI]**	**CFI**	**BIC**
Two-Factor Model	237.60	0.12 [0.11 – 0.13]	0.99	32252.05
Three-Factor Model	105.22	0.09 [0.08 – 0.11]	0.96	32134.89
HC				
Two-Factor Model	60.73	0.06 [0.05 – 0.07]	0.97	29514.81

Three-Factor Model	24.38	0.04 [0.02 – 0.06]	0.99	29493.58

RMSEA: root mean square error of approximation, CFI: comparative fit indices, BIC: Bayesian information criterion.

Linear Models of Concurrent Sleep & Mood Instability

Results for linear models predicting average positive, negative, and irritable mood instability over one week based on concurrent sleep and circadian variables are reported in the Appendix, Section III, Tables 3A.5 - 3A.10. In brief, none of the sleep or circadian variables significantly predicted average mood instability in either group.

Day-lagged Models: Prospective Effects of Sleep on Following-Day Mood Instability

Multilevel models predicting mood instability from previous night's sleep and vice versa were built in stages starting with fixed effects only. Random intercepts varied significantly between participants and were therefore added to all models. Random slopes were added to account for the between-participant effects of TST, and finally autoregressive error terms were added when they significantly improved model fit. The final models for each group and mood factor are reported below, while preliminary models can be found in the appendix, in following with recommendations (Field et al., 2012).

Negative Mood Instability

Table 3A.5 (Appendix) presents the coefficients of a fixed effects-only model predicting negative mood instability from previous night's sleep in the BPD group. Random intercepts varied significantly, indicating different baseline levels of instability in negative mood between participants (SD = 0.50 (95% CI: 0.16, 1.57), $\chi 2(3) = 43.98$, $p < .0001$). Random slopes accounting for the varied effects of TST also varied significantly across participants (SD = 1.55e-05, 95% CI: 3.24e-05, 7.47e-05). Random slopes and intercepts were negatively

but not significantly correlated, cor = -0.86 (95% CI: -0.99, 0.22). This model does not include an autoregressive error term as it did not significantly improve model fit, suggesting within-participant ratings of mood instability were not correlated ($\chi2(2) = 4.34$, $p = .11$). Table 3.11 below shows the coefficients and significance of fixed effects in the final model, which found that increased sleep efficiency significantly reduced following-day instability of negative mood in people with BPD (b = -0.01, 95% CI: -0.02, -1.36e-03, $p = .03$). Instability in negative mood was also reduced on weekends (b = -0.12, -0.27, -0.01, $p = .04$). Additionally, the intercept of the model suggests that instability in negative mood is significantly greater than zero even when controlling for age, weekend days, and sleep variables (b = 1.26, 95% CI: 0.30, 2.22, $p = .01$).

Table 3.11. The fixed effects of the final model for instability in negative mood in the BPD group, after inclusion of a random intercept for each participant and a random slope for the effect of TST.

Term	*b*	*SE b*	95% CI of *b*	*p*
Intercept	1.26	0.49	0.30, 2.22	.01
Age	0.01	0.01	-0.01, 0.03	.34
Employed	-0.13	0.13	-0.39, 0.12	.31
Weekend Day	-0.12	0.06	-0.27,-0.01	.04
TST	1.41e-05	8.1e-06	-1.68e-06, 2.98e-05	.09
SE	-0.01	4.32e-03	-0.02,-1.36e-03	.03
SOL	-3.83e-05	2.44e-05	-8.56e-05, 9.06e-06	.12

SE: standard error, CI: confidence interval, TST: total sleep time, SE: sleep efficiency, SOL: sleep onset latency.

Table 3.A6 (appendix) shows the coefficients and significance of a fixed effects-only model predicting negative mood instability from previous night's sleep in the healthy control group. The addition of a random intercept indicated significant variance in baseline negative mood between participants, SD = 0.07 (95% CI: 3.09e-17, 1.46e14), $\chi2(1) = 66.17$, $p < .0001$. Additionally, random slopes accounting for the effects of TST varied significantly across participants, SD = 1.03e-05 (95% CI: 2.41e-08, 4.36e-03) and the slopes and intercepts were

negatively but not significantly correlated (cor = -0.34 95% CI: -0.99, 0.99). The final model includes a first-order autoregressive covariance structure with a moving average accounting for time which significantly improved model fit $\chi2(1) = 45.45$, $p < .0001$ and the resulting Φ value of 0.53 (95% CI: 0.07, 0.80) suggests strong positive within-participant correlations between observations. $\Theta = 0.11$ (-0.21, 0.41). Fixed effects for the final model are shown in Table 3.12, which found that baseline instability in negative mood was significantly greater than zero, $b = 1.39$ (95% CI: 0.61, 2.17), $p < .001$. No other covariates were significant predictors of negative mood instability.

Table 3.12. The fixed effects of the final model predicting instability in negative mood in the HC group, after inclusion of random intercepts for each participant, random slopes for the effect of TST, and a first-order autoregressive covariance structure.

Term	*b*	*SE b*	95% CI of *b*	*p*
Intercept	1.39	0.40	0.61, 2.17	<.001
Employed	-0.31	0.26	-0.83, 0.21	.25
Weekend Day	-0.06	0.06	-0.16, 0.05	.31
TST	-3.0e-06	7.2e-06	-1.71e-05, 1.11e-05	.68
SE	-5.93e-03	-5.93e-03	-0.01, 2.89e-03	.19
SOL	-4.3e-06	3.27e-05	-6.78e-05, 5.92e-05	.89

Positive Mood Instability

Table 3A.7 (appendix) reports a fixed effects-only model predicting instability in positive mood from previous night's sleep in the BPD group. The addition of a random intercept showed significant variance in baseline positive mood between participants, *SD* = 0.45 (95% CI: 0.20, 1.45), $\chi2(1) = 60.33$, $p < .0001$. Additionally, random slopes accounting for the effects of TST varied across significantly participants, *SD* = 1.09e-05 (95% CI: 1.94e-06, 6.11e-05) and the slopes and intercepts were negatively but not significantly correlated, cor = -0.84 (95% CI: -0.99, 0.70). The fixed effects for the final model are reported in Table 3.13 below; this model does not include an autoregressive covariance structure as it did not improve model fit, $\chi2(2) = 0.22$, p = .90. Improved sleep efficiency significantly reduced

positive mood instability, b = -0.01 (95% CI: -0.02, -1.81e-03), p = .02. Increased sleep onset latency also significantly reduced positive mood instability b = -7.01e-05 (95% CI: 1.22e-04, -1.79e-05), p = .01, and baseline variability in positive mood was significantly greater than zero, b = 2.00 (95% CI: 1.26, 2.74), p < .001.

Table 3.13. The fixed effects of the final model predicting instability in positive mood in the BPD group, after inclusion of random intercepts for each participant and random slopes for the effect of TST.

Term	*b*	*SE b*	95% CI of *b*	*p*
Intercept	2.00	0.38	1.26, 2.74	<.001
Employed	-0.02	0.16	-0.33, 0.30	.92
Weekend Day	-0.07	0.06	-0.19, 0.05	.24
TST	-2.4e-06	8.4e-06	-1.87e-05, 1.39e-05	.77
SE	-0.01	4.80e-03	-0.02,-1.81e-03	.02
SOL	-7.01e-05	2.68e-05	-1.22e-04,-1.79e-05	.01

Table 3A.8 (appendix) reports a fixed effect-only model predicting variability in positive mood based on previous night's sleep in the healthy control group. The addition of a random intercept showed significant variance in baseline positive mood between participants, SD = 0.33 (95% CI: 0.05, 2.16), $\chi2(1) = 141.60$, $p < .0001$. Random slopes accounting the effects of TST varied significantly across participants, SD = 8.12e-06 (95% CI: 1.07e-07, 6.18e-04). Correlations between random slopes and intercepts was highly uncertain, cor = -0.12 (95% CI: -0.99, 0.99), suggesting that baseline variability in positive mood was completely uncoupled with the individual effects of TST on positive mood instability. The model includes a first-order autoregressive structure, which significantly improved model fit: $\chi2(1) = 7.87$, p = .01. The resulting Φ value of 0.29 (95% CI: 0.13, 0.53) suggests moderate positive autoregressive effect between days. Table 3.14 below shows the fixed effects of the final model; none of the covariates were significant predictors of instability in positive mood but baseline instability was significantly greater than zero, b = 1.42 (95% CI: 0.63, 2.21), p < .001.

Table 3.14. The fixed effects of the final model predicting instability in positive mood in the control group, after inclusion of random intercepts for each participant, random slopes for the effect of TST and a CAR structure accounting for time.

Term	*b*	*SE b*	95% CI of *b*	*p*
Intercept	1.42	0.41	0.63, 2.21	<.001
Employed	-0.10	0.29	-0.68, 0.49	.74
Weekend Day	-0.02	0.04	-0.11, 0.06	.64
TST	-1.00e-05	6.3e-06	-2.23e-05, 2.22e-06	.11
SE	-3.53e-03	4.07e-03	-0.01, 4.39e-03	.39
SOL	-2.84e-05	2.87e-05	-8.42e-05, 2.74e-05	.32

Irritable Mood Instability

Table 3A.8 (appendix) reports a fixed effect-only model predicting variability in irritable mood based on previous night's sleep in the healthy control group. Adding a random intercept showed significant variance in baseline irritable mood between participants, *SD* = 0.34 (95% CI: 0.24, 1.83), $\chi 2(1) = 37.49$, $p < .0001$. Random slopes accounting for the effects of TST varied significantly across participants, *SD* = 6.48e-07 (95% CI: 3.44e-16, 1219.57). Correlations between random slopes and intercepts were highly uncertain, cor = -0.05 (95% CI: -0.88, 0.86), suggesting that baseline variability in irritable mood was completely uncoupled with the effects of TST on positive mood instability. Adding an autoregressive covariance structure accounting for time significantly improved model fit, $\chi 2(1) = 8.14$, $p < .01$ and resulted in significant moderate positive autoregressive effect, $\Phi = 0.30$ (95 CI%: 0.14, 0.54), indicating within-participant correlation in irritable mood instability over time. The fixed effects of the final model are shown in Table 3.15. None of the covariates were significant predictors of instability in irritable and angry mood, but baseline instability was significantly greater than zero, intercept $b = 1.09$, (95% CI: 0.33, 1.84), $p = .01$.

Table 3.15. The fixed effects of the final model predicting instability in irritable mood in the BPD group, after inclusion of random intercepts for each participant, random slopes for the effect of TST and an autoregressive error structure.

Term	*b*	*SE b*	95% CI of *b*	*p*

Intercept	1.09	0.39	0.33, 1.84	.01
Employed	0.05	0.16	-0.28, 0.38	.75
Weekend Day	0.12	0.08	-0.03, 0.28	.13
TST	-4.02e-06	9.3e-06	-2.20e-05, 1.40e-05	.67
SE	-1.11e-03	5.32e-03	-0.01, 9.24e-03	.84
SOL	-3.56e-05	3.03e-05	-9.46e-05, 2.33e-05	.24

Table 3A.9 (appendix) reports a fixed effect-only model predicting variability in irritable mood based on previous night's sleep in the healthy control group. The addition of a random intercept showed significant variance in baseline irritable mood between participants, *SD* = 0.71 (95% CI: 0.21, 2.83), $\chi 2(1) = 32.15$, $p < .0001$. Random slopes accounting for the effects of TST varied significantly across participants, *SD* = 2.09e-05 (95% CI: 2.67e-06, 1.64e-04). Random slopes and intercepts were significantly and strongly negatively correlated, cor = -0.96 (95% CI: -0.99, -0.66), suggesting that increased baseline instability in irritable mood was associated with reduced effects of total sleep time on mood instability. Adding an autoregressive covariance structure improved model fit $\chi 2(1) = 3.78$, p = .0 and is thus included in the final model reported in Table 3.16. The covariance structure demonstrated a small but significant positive autoregressive effect, $\Phi = 0.19$ (95 CI%: 0.06, 0.42), indicating within-participant correlation in irritable mood instability. Improved sleep efficiency significantly reduced instability in irritable and angry mood, *b* = -0.01 (95% CI: -0.02, -2.44e-03), *p* = .02. Baseline irritable instability was significantly greater than zero, *b* = 1.72 (95% CI: 0.77, 2.67), $p < .001$.

Table 3.16. The fixed effects of the final model predicting instability in irritable mood in the control group, after inclusion of random intercepts for each participant, random slopes for the effect of TST and an autoregressive error structure.

Term	*b*	*SE b*	95% CI of *b*	*p*
Intercept	1.72	0.49	0.77, 2.67	<.001
Employed	-0.35	0.22	-0.79,-0.09	.12
Weekend Day	-0.03	0.06	-0.15, 0.09	.60
TST	6.78e-06	1.00e-5	-1.26e-05, 2.62e-05	.50

SE	-0.01	0.01	-0.02,-2.44e-03	.02
SOL	-1.58e-05	4.27e-05	-9.87e-05, 6.72e-05	.71

Day-lagged Models: Mood Instability Predicting Following Night's Sleep

Table 3A.10 (appendix) reports a fixed effects-only model predicting total sleep time (TST) of each night based on the preceding day's mood instability. The addition of a random intercept showed significant variance in baseline TST between participants, *SD* = 2830.73 (95% CI: 1322.15, 6060.58), $\chi 2(1) = 38.20$, $p < .0001$. The model would not converge with random slopes for RMSSD of negative or irritable mood, but random slopes accounting for instability in positive mood varied significantly across participants: SD of random slopes = 1752.76 (95% CI: 280.47, 10953.56). Correlation between random intercepts and slopes was insignificant, cor = -0.28 (95% CI: -0.95, 0.86). Adding an autoregressive covariance structure significantly improved model fit, $\chi 2(2) = 8.36$, $p < .02$, but suggested insignificant moderate negative autoregressive effect, $\Phi = -0.21$ (95 CI%: -0.70, 0.41), $\Theta = -0.13$ (-0.71, 0.55), suggesting that within-person correlation of TST is present but was not significantly significant. Table 3.17 shows the fixed effects for the final model, which did not find that instability in any of the mood factors was a significant predictor of sleep duration, although participants who were employed had non-significant reductions in sleep duration, *b* = -2522.17 (95% CI: -5261.00, 216.66), *p* = .07.

Table 3.17. The fixed effects of the final model predicting total sleep time in the BPD group, after inclusion of random intercepts for each participant, random slopes for the effect of instability in positive mood and an autoregressive error structure.

Term	*b*	*SE b*	95% CI of *b*	*p*
Intercept	26366.80	1588.56	23280.52, 29453.08	<.0001
Employed	-2522.17	1360.46	-5261.00, 216.66	.07
Weekend Day	125.18	560.93	-964.60, 1214.96	.82
Instability in Negative Mood	-1532.46	923.68	-3327.00, 262.09	.10
Instability in Positive Mood	1245.23	887.78	-479.57, 2970.03	.16

Instability in Irritable Mood	1103.19	669.52	-197.58, 2403.96	.10

Table 3A.11 (appendix) below shows a fixed effects-only model predicting total sleep time (TST) of each night based on the preceding day's mood instability. The addition of a random intercept showed significant variance in baseline TST between participants, SD = 2032.75 (95% CI: 20778.00, 28071.96), $\chi2(1) = 15.81$, $p < .0001$. Random slopes accounting for instability in positive mood varied significantly across participants: SD of random slopes = 947.39 (95% CI: 2.58, 348029.80). Correlation between random intercepts and slopes was insignificant, cor = -0.44 (95% CI: -0.99, 0.99). The final model shown in Table 3.18 does not include an autoregressive error term as it did not significantly improve model fit: $\chi2(1) = 5.75e-07$, $p < .99$. The final model did not find that instability in any of the mood factors was a significant predictor of sleep duration.

Table 3.18. The fixed effects of the final model predicting total sleep time in the healthy control group, after inclusion of random intercepts for each participant and random slopes for the effect of instability in positive mood.

Term	*b*	*SE b*	95% CI of *b*	*p*
Intercept	24424.98	1877.94	20777.99, 28071.96	<.0001
Employed	1008.48	1759.04	-2542.52, 4559.47	.57
Weekend Day	1090.21	569.91	-16.55, 2196.98	.06
Instability in Negative Mood	1087.74	885.74	-632.38, 2807.85	.22
Instability in Positive Mood	-1741.96	958.56	-3603.48, 119.57	.07
Instability in Irritable Mood	-955.03	784.74	-2479.01, 568.95	.23

3.4 Discussion

This study analysed the relationship between sleep, rest-activity patterns, and mood instability in people with BPD compared to healthy controls. Although linear model results suggested that average sleep over a week was not associated with contemporaneous mood instability, day-lagged models found that reduced sleep efficiency on a given night increased following-day instability in positive and negative mood for people with BPD, but not for

healthy controls. Furthermore, actigraphy revealed that the BPD group had significantly worse sleep efficiency than healthy volunteers. This difference was likely driven by increased time spent in bed in BPD rather than reduced sleep duration, for which there was no significant difference between groups. Significant autoregressive effects were observed in positive and negative mood instability in the healthy control group, suggesting that mood instability on one day predicts instability on the following day, reflecting predictable temporal trends. In contrast, these autoregressive effects on positive or negative mood were not significant in the clinical group, indicating that mood instability in BPD follows a less predictable temporal pattern. The only mood instability factor with significant autoregressive effects for BPD participants was irritability, which was not predicted by demographic, occupational, or sleep covariates. The results of this analysis suggest that reduced sleep efficiency amplifies instability in positive and negative mood in BPD, while variation in irritability shows temporal trends and may be unrelated to sleep. Conversely, sleep does not seem to impact instability in positive or negative mood in healthy volunteers, but worsened sleep efficiency is associated with greater variability in irritable mood. Finally, the effect of sleep duration on mood instability varied significantly between participants in both groups, suggesting individual differences in the strengths of these associations; increasing sleep duration may not affect mood instability in all individuals.

Comparative Circadian Results

Actigraphy results did not suggest significant between-group differences in circadian rhythm, aside from significantly greater rhythm fragmentation (IV) in healthy controls. This result contradicted the typical association between psychiatric pathology and increased IV, however the AMoSS study also found significantly greater IV in the control group as compared to the

bipolar group, possibly due to increased occupational or caring commitments in the control group (McGowan et al., 2019a). The control group in the SBPD sample had more employed participants than the BPD group, which might lead to more sedentary daytime periods and resulting higher rhythm fragmentation. This analysis also did not find evidence for the circadian phase delays or increased variability in sleep onset times in BPD (Carr, Saunders, Bilderbeck, et al., 2018; McGowan et al., 2019a), or associations between BPD symptoms and circadian rhythm dysfunction suggested by previous research (Huỳnh et al., 2016; McGowan et al., 2019, 2020). However, compared to the AMoSS study, the SBPD participant group was younger, (mean age 38.09 vs. 25.92 respectively, Welch's $t = -8.76$, $p < .001$). Adolescents and young adults have consistently been shown to exhibit the latest chronotypes of all age groups (Fischer et al., 2017; Roenneberg et al., 2004); it may be that the chronotypes of young women with BPD do not differ significantly from those of their peers. Most individuals will experience increasing circadian phase delays throughout childhood and adolescence until reaching an inflection point around age 20, at which point phase begins to advance continually throughout the rest of the lifespan (Roenneberg et al., 2004). Discrepancy from this pattern has been associated with circadian rhythm disorders, particularly delayed phase sleep wake disorder (DPSWD), in which phase delays can persist well into middle age (Micic et al., 2016). The "social jetlag" effect of delayed circadian phase, in which morning occupational commitments cause individuals to begin their days during their biological night, affects people with DPSWD in addition to otherwise healthy adolescents and young adults (Fischer et al., 2017). Young adults with BPD may demonstrate greater reactivity to the experience of social jet lag than their peers, contributing to reduced

capacity for emotion regulation and increased mood instability. This aligns with previous reports of increased reactivity to circadian disruption in BPD (McGowan et al., 2020).

Hypothesised persistence in phase delay in adults with BPD could be attributable to several factors. One possibility is weak entrainment by social zeitgebers, which are theorised to provide non-photic cues to the SCN (Grandin et al., 2006). Reduced occupational commitments have previously been shown to increase phase delay in DPSWD (Otsuki et al., 2022). The present study identified reduced employment and elevated TIB in the BPD cohort; if these phenomena continued into later adulthood, they might be indicative of weakened social zeitgebers. Another possibility is that adults with BPD may exhibit longer circadian periods as compared to healthy individuals, driving gradual delays in physiological preparedness for both sleep and wake which accumulate to create significant phase delay. Adolescents tend to have longer circadian periods compared to adults, possibly contributing to their relative phase delays (Carskadon et al., 1999). These longer circadian periods might be maintained into adulthood in BPD; indeed, one study has reported irregular circadian period in BPD correlated with symptom severity (Verkes et al., 1996), although these findings may reflect low interdaily variability rather than abnormal circadian period. Finally, phase delay in DSWPD is sometimes attributed to altered light sensitivity (Micic et al., 2016); this may also be plausible in BPD. Hypersensitivity to evening light or hyposensitivity to morning light could each cause inappropriate photic entrainment of the SCN, resulting in circadian phase delay. There is some evidence linking DSWPD with abnormal melatonin suppression and increased phase delay in response to evening light (Aoki et al., 2001; L. A. Watson et al., 2018), although these studies were conducted in small samples. No known studies of light sensitivity have been conducted in BPD.

Comparative Sleep Results

The BPD group did not differ significantly from the control group in sleep duration but spent more time in bed and exhibited lower sleep efficiency and increased nighttime awakening. These factors likely contributed to the significantly worsened subjective experience of sleep quality and insomnia symptoms reported by the BPD participants. Excess time in bed directly reduces sleep efficiency, and also contributes to nighttime awakening by eroding the stimulus pairing between bedtime and sleep (Morin, 2004), as well as impeding the accumulation of fatigue from daytime activity. Time in bed may be elevated due to dysfunctional beliefs about sleep. Generalised dysfunctional beliefs, for example about interpersonal relationships or self-perception, are transdiagnostic mechanisms which perpetuate psychological distress and are common in BPD (Beck, 2005; Plante et al., 2013). Sleep-specific dysfunctional beliefs encourage the development of counterproductive bedtime behaviours which perpetuate insomnia, including excess time in bed (A. G. Harvey, 2002; Morin et al., 2007a). Given that dysfunctional beliefs about sleep were significantly greater in the BPD group, they seem a likely mechanism driving the increased time in bed, decreased sleep efficiency, and equivalent sleep duration recorded in the BPD group as compared to the healthy volunteers. Addressing these dysfunctional beliefs, reducing time in bed, and increasing sleep efficiency are primary treatment goals of cognitive behavioural therapy for insomnia (CBT-I), but may require differential treatment for people with BPD given the worsened perception of sleep in this group (Espie et al., 2012; Morin & Benca, 2012). For example, additional treatment time could be spent on the reasons why perceptions of sleep can be distorted, both in healthy sleepers and in people with chronic insomnia. Some CBT-I platforms integrate users' questionnaire responses to illustrate this point, and the addition of data from actigraphs or

other commercially available wearables might provide patients with further clarity about their own sleep.

A second possible factor contributing to excess time in bed is unstructured daily routine, perhaps due to reduced occupational commitment. This sample had a larger proportion of unemployed people in the BPD group as compared to the healthy volunteers, and previous research suggests low rates of employment in BPD as well as occupational impairment (Sansone & Sansone, 2012; Skodol et al., 2005; Wood et al., 2015). Irregular daily routines have been hypothesised as disruptors to circadian rhythm function leading to downstream somatic symptoms and mood instability in vulnerable individuals (Frank, 2007; Haynes et al., 2016). For example, inconsistent social and occupational engagements may lead to irregularities in sleep-wake times and light exposure, which disrupts circadian function and activates the stress axis, altering neurotransmitter release and thus increasing the risk of pathology including mood instability (Wulff et al., 2010). This pathway was first theorised as the social rhythms hypothesis of depression, which was the basis for social rhythm therapies (SRTs) (Ehlers et al., 1988; Frank, 2007; Haynes et al., 2016). Despite the likelihood of instability in daily routines in BPD, there is a paucity of SRT studies this group; however, some research in comorbid BPD and bipolar disorder has been published (Swartz et al., 2005). This study found that participants with comorbid BPD were slower to respond to SRT than those with just bipolar disorder, suggesting that SRT is not a sufficient monotherapy for BPD and would likely need to be integrated into another treatment programme. In this vein, Bailey et al. (2023) have proposed a new treatment for comorbid BPD and bipolar called Dialectical Behaviour and Social Rhythm Therapy (DBSRT), which blends SRT and

dialectical behaviour therapy, the gold-standard psychological treatment for BPD. The present findings of elevated time in bed in the BPD group further motivate DBSRT.

Finally, psychotropic medication usage in the BPD group is likely to have affected sleep relative to the non-clinical group. Time in bed generally increases in response to antidepressants, antipsychotics, anxiolytics, and hypnotics (Mihic & Harris, 2011; Robillard et al., 2016), and this may have contributed to the between-group difference in time spent in bed. About a third of the BPD group were using selective serotonin reuptake inhibitors or serotonin-norepinephrine reuptake inhibitors, which are associated with increased time in bed but not sleep duration, resulting in reduced sleep efficiency (Gillin et al., 1997; Rush et al., 1998). However, this effect on sleep efficiency may have been partially balanced by another third of the BPD group using antipsychotics, which may improve sleep duration and efficiency (Robillard et al., 2016). Antipsychotic medication may also have had some effect on circadian phase: quetiapine, which is frequently prescribed in BPD (Stoffers-Winterling et al., 2020), has been shown to cause circadian phase delays in bipolar disorder (Hwang et al., 2017). If this effect is also present in BPD, quetiapine may have contributed to the phase delay previously identified in the AMoSS study (Carr, Saunders, Bilderbeck, et al., 2018)

Disentangling Temporal Relationships in Sleep & Mood Instability

This study did not find evidence of a significant association between mood instability and sleep or circadian rhythm over a week-long period, implying that the cumulative effects of low sleep efficiency were not associated with average mood instability in either the clinical or healthy group. However, as hypothesised there were some significant effects of sleep efficiency on following-day mood instability. In the BPD group, low sleep efficiency was

associated with increased following-day instability in both positive and negative mood. This is aligned with evidence that total sleep deprivation drastically increases amygdala reactivity (Yoo et al., 2007) and that mild sleep deprivation may reduce capacity for emotion regulation (James & Gregg, 2004; Zohar et al., 2005). This effect was not observed in irritability/anger instability for BPD participants. This difference may be attributed to a lower likelihood of experiencing anger compared to other mood states, such as positive or negative emotions. Consistent with the findings from the AMoSS study (Tsanas et al., 2016), participants in both the control and clinical groups were least likely to report anger among the monitored mood states (Tables 3.5 - 3.6), and it was also the least variable mood (Tables 3.7 - 3.8). This could indicate a lower frequency of encountering stimuli that elicit anger compared to those that trigger other emotional responses. Additionally, participants may have been less willing to endorse anger than other mood states, perhaps seeing it as less acceptable or more shameful than, for example, sadness or elation. Individuals with BPD may be aware that "inappropriate anger" is a criteria for DSM-V diagnosis (American Psychiatric Association, 2000); no qualifications of propriety are made for other affective states mentioned in the categorical diagnostic criteria.

An alternative explanation is that the effects of sleep disturbance on emotion regulation are not uniform across all moods. This may be due to differences in neural regulation of these moods. Gray's reinforcement sensitivity theory (1982) suggests that positive, negative, and angry mood are regulated by joint motivational subsystems, all of which appear to be more reactive in people with BPD than healthy individuals. Normal functioning of the behavioural activation system (BAS) is thought to instigate action towards appetitive, reward-signaling stimuli, driving positive affect; extreme BAS reactivity is associated with impulsive

behaviours (Bijttebier et al., 2009). Indeed, elevated BAS sensitivity has been noted in BPD (Pastor et al., 2007), possibly contributing to impulsive behaviour characteristic of the group. The behavioural inhibition system (BIS) regulates avoidant responses to aversive stimuli and has been associated with anxious mood (Campbell-Sills et al., 2004; Segarra et al., 2007). Although the Cluster B personality disorders (BPD in addition to histrionic, narcissistic, and antisocial) are each associated with high BAS sensitivity, only BPD appears to be additionally associated with high BIS sensitivity (J. Taylor et al., 2006). This combination of overreactivity to both appetitive and aversive stimuli may contribute to instability in positive and negative affect in BPD. Our findings suggest that low sleep efficiency may further contribute to mood instability in people with BPD, perhaps by exacerbating BIS and BAS reactivity. This implies that sleep efficiency could form a treatment target for mood instability and emotion dysregulation in BPD. We did not find evidence of sleep efficiency contributing to following-day mood instability in healthy individuals. Instead, mood instability in the control group appeared to auto-correlate, demonstrating continuity in both stable and unstable periods rather than short-term reactivity to reduced sleep efficiency.

Anger, irritability, and aggression are externalising behaviours thought to be co-regulated by the BIS and the flight/flight/freeze system (FFFS), which is hypothetically engaged when the threat of a stimulus outweighs the potential reward (Bijttebier et al., 2009). In healthy individuals, the BIS should inhibit the FFFS whenever the potential reward outweighs the perceived threat, halting aggressive or fearful responses. However, this inter-system regulation is thought to be reduced in individuals with extreme personality, resulting in unchecked FFFS responses (Corr, 2002). Lack of BIS regulation on the FFFS may be enacted by failure of the orbital frontal cortex (OFC) to regulate the amygdala in BPD (Donegan et

al., 2003; Herpertz et al., 2001; New et al., 2007), resulting in aggressive responses to perceived threat. Frequent exposure to perceived threat may increase FFFS sensitivity over time, and thus it may become more reactive in individuals with history of adverse life experiences (Blair, 2012), which is applicable to many people with BPD (Wertz et al., 2020). Heightened baseline reactivity may account for the lack of further exacerbation in irritable mood instability following sleep disturbance within the BPD group. This is consistent with the significantly higher baseline ratings of anger and irritability in BPD, as well as the greater instability observed in these mood states (Tables 3.5–3.8). The "always on" nature of anger and aggression pathways may render people with BPD less sensitive to short-term fluctuations in sleep efficiency, resulting in fewer day-to-day changes in anger reactivity. Instead, longer term temporal trends in irritability and anger may be present, as indicated by the significance of autoregressive structures in the day-lagged models. In other words, irritability and anger are high-frequency trait behaviours in BPD rather than abnormal states triggered by an event such as poor sleep. In contrast, in healthy individuals, normal regulation of the amygdala by the OFC appears be compromised by poor sleep (Yoo et al., 2007), possibly leading to increased instability in irritability when sleep efficiency is low.

EMA Adherence

Ecological momentary assessment (EMA) proved an effective method of capturing mood instability in the BPD group: even at the lower twice-daily frequency significant between-group differences were apparent. These findings are in alignment with the extant EMA literature which includes repeated evidence of increased variability in negative mood (Ebner-Priemer et al., 2007; Jahng et al., 2008; Trull et al., 2008). MoodZoom response rates were high at the twice-daily frequency but decreased as frequency increased, as did frequency of

sleep diary responses, possibly due to response fatigue. The BPD group seems to have been especially affected by this, as they missed significantly more prompts than the healthy volunteers in the HIRP period. EMA sampling requires optimising the balance between accuracy and response fatigue; in BPD there may be a lower threshold of overwhelm than in other groups. Given relevance of short-term, within-day mood instability in BPD, future studies employing EMA should sample at least twice daily but perhaps not much more since instability is detectable even at low frequencies. The duration of the study and timing of prompt frequency should also be considered: increased frequency of sampling may be more tolerable for shorter periods of time, or at the very beginning of a study before cumulative response fatigue develops.

Limitations

This work has several limitations, the first arising from the sample, which was entirely people assigned female at birth. This was an intentional decision based on the elevated prevalence of BPD in females and sex-based sleep differences, but nonetheless limits applicability of findings to people assigned female at birth. The analysis also preferably would have included more granular information about working versus non-working days – weekend days were entered as covariates in the day-lagged models, but this would not have accounted for participants who worked over weekends or had continuous caring responsibilities, likely reducing the accuracy of findings. Ideally future work will also include more participants in part-time work or study to check for differences in effects at ordinal levels of occupational commitment. Another limitation is the reliance on self-report to exclude chronic sleep disturbances like sleep apnoea or restless leg syndrome; however, as these conditions are not known to be more prevalent in BPD, they are unlikely to significantly skew between-group

comparisons. Finally, there were significantly more missing responses in the BPD group than in the healthy volunteers during the HIRP period. Kalman filtering was used to correct this, but imputation inherently introduces error into the data, which may have reduced the accuracy of findings.

3.5 Conclusion

This study found support for the hypothesis that sleep efficiency has significant effects on following-day mood instability in BPD, as opposed to mood predicting following-night's sleep. Objective measures suggest that sleep efficiency is significantly worse in BPD than healthy controls, possibly due to worsened dysfunctional beliefs about sleep and excessive time in bed. These factors can be treated by the cognitive and sleep restriction components of CBT-I, respectively, motivating a pilot study in BPD. Inconsistent with the extant literature, this analysis did not find evidence of circadian phase delayed in young women with BPD relative to their peers. Circadian phase in BPD should be further investigated across all age groups.

4 Chapter 4: Feasibility study of bright light therapy & digital cognitive behavioural therapy for insomnia in borderline personality disorder

4.1 Introduction

Bright Light Therapy

Sleep disturbance is common in BPD, and presents similarly in unipolar depression, irrespective of comorbidity between the two disorders, suggesting that sleep disturbance may have a direct association with BPD rather than being merely a symptom of concomitant depression (Harty et al., 2010; Winsper, Tang, et al., 2017). The similarities in sleep disturbance between BPD and unipolar depression in conjunction with elevated prevalence of delayed circadian phase in BPD motivate the use of bright light therapy (BLT) in this group (Dagan et al., 1998; Fitzpatrick et al., 2020; McGowan et al., 2019b). BLT was originally developed as a treatment for seasonal affective disorder (SAD), which manifests as a period of depressive symptoms occurring circannually most years, typically beginning in the fall or winter and spontaneously resolving in the springtime (Pjrek et al., 2020). The etiology of SAD is still disputed, hypothesised for example as an energy conservation response to low environmental light levels experienced at high or low latitudes during the winter months (Levitan, 2007). The more widely-held phase shift hypothesis suggests that SAD is driven by circadian phase shift resulting in a misalignment between an individual's internal circadian phase and desired rest-activity pattern (Lewy et al., 2007). This may result in daily fluctuations of hormonal secretions, body temperature, and rest-activity patterns to be offset from their optimal phase, resulting in a disarray like that experienced during travel across time zones. This hypothesis is supported by findings that circadian phase alignment explained

65% of the variance in SAD symptom severity, and that circadian phase shift through exogenous melatonin accounted for 35% of the resulting change in SAD symptoms (Lewy et al., 2007). Both the phase shift and energy-conserving hypotheses have motivated the study bright light therapy (BLT), in which subjects sit in front of a high-intensity light, typically in the morning. BLT entrains the suprachiasmatic nucleus (SCN), and therefore the body's peripheral clocks, to the desired rest-activity pattern (Wirz-Justice, 2009).

BLT is also indicated for the treatment of delayed sleep-wake phase disorder (DSWPD) with the objective of advancing circadian phase (Auger et al., 2015; Wilson et al., 2019), although a recent review found weak evidence of efficacy (Gomes et al., 2021). The mechanism of action of BLT begins with photosensitive retinal ganglion cells (pRGCs) transmitting neural signals to the suprachiasmatic nucleus (SCN) upon ocular light exposure, which ultimately upregulates transcription of PER proteins. The specific effect on PER is contingent on light exposure timing: morning light increases transcription of PER1, advancing circadian phase, while evening light reduces degradation of PER2, delaying physiological preparedness for sleep and thus circadian phase (Ashton et al., 2022). This pattern of phase response to light, or phase response curve (PRC), has been consistently replicated (Khalsa et al., 2003; Van Cauter et al., 1994) and used to inform guidelines for the timing of BLT administration (M. Terman & Terman, 2005b). Light early in the subjective morning consistently produces both the largest circadian phase advance and the greatest antidepressant effects (Khalsa et al., 2003; J. S. Terman et al., 2001). As previously mentioned, BLT has an antidepressant effect, which has been demonstrated in SAD (Pjrek et al., 2020), subsyndromal SAD (Kasper et al., 1989), and major depressive disorder (Tao et al., 2020). BLT has also been found to improve mood in healthy individuals during the winter (Partonen & Lönnqvist, 2000). The exact

mechanism by which BLT influences mood remains unclear, though it may involve the stabilization of serotonin transporters (Harrison et al., 2015), which, when disrupted, are implicated in circadian rhythm dysregulation (Daut & Fonken, 2019). This indicates potential circadian regulation of mood, which is further supported by associations between circadian phase delay and the severity of mood symptoms in major depressive disorder (Emens et al., 2009). As noted in the previous chapter, the extant literature has identified circadian phase delay in BPD, (Carr, Saunders, Tsanas, et al., 2018; McGowan et al., 2019a) and mood instability is a characteristic feature of the disorder which has previously been associated with increased circadian rhythm disruption, particularly with interdaily stability, intradaily variability, reduced amplitude and delayed phase (McGowan et al., 2020). This association, which was significantly stronger in BPD than in bipolar disorder, suggests people with BPD may be highly reactive to circadian disruption. Collectively, these findings motivate study of BLT in BPD.

To date, two open-label studies report the effects of BLT in BPD-specific groups. A single-arm study of six weeks of bright light therapy at 10,000 lux intensity and a fixed time for all participants (06:30 – 07:30) concurrent with antidepressant medication resulted in significant reductions to depressive and anxious symptoms relative to baseline. However, the study did not measure any sleep or circadian outcomes, making it unclear whether BLT resulted in a circadian phase advance (Prasko et al., 2010). The second study was an open-label cross-over trial which evaluated a three-week course of morning bright light therapy at 8,000 lux, was found to advance circadian phase, reduce time spent in bed and sleep duration, and attenuate SAD symptoms, supporting its use as an adjunctive treatment for the disorder (Bromundt et al., 2013). BLT did not appear to improve symptoms of depression or any other mood

symptoms; however, the authors note that a longer treatment course may have been necessary to attenuate mood symptoms. An additional open-label, single-arm study of BLT in psychiatric inpatients, one-third of whom had BPD diagnoses, reported significant antidepressant effects and increases in functioning after 30-minute 10,000 lux BLT sessions with variable morning start times. No measures of sleep or circadian rhythm were taken (Trinh et al., 2021).

Cognitive Behavioural Therapy for Insomnia

As discussed in Chapter 2, circadian phase delays may also contribute to sleep onset insomnia (Flynn-Evans et al., 2017). Although not a core symptom of BPD, subjective reports and objective evidence of sleep disturbance suggest that insomnia is a prevalent symptom in those with the disorder (Winsper, Tang, et al., 2017). Widely supported models of insomnia suggest that it can be predisposed by psychological distress and precipitated by acute stressors, both of which are characteristic of BPD (Conway et al., 2018; Ellis et al., 2021). Insomnia is further perpetuated by maladaptive behaviours and negative cognitions about sleep which, as shown in the previous chapter, appear to be significantly more prevalent in BPD than in healthy controls (Ellis et al., 2021). The gold-standard treatment for insomnia is cognitive behavioural therapy for insomnia (CBT-I), which is supported by extensive evidence in both primary insomnia and in other psychiatric groups (Hertenstein et al., 2022). CBT-I has also been successfully adapted into several digital formats (Batterham et al., 2017; Cheng et al., 2019; Espie et al., 2012). However, to our knowledge neither face-to-face nor digital CBT-I (dCBT-I) has been formally evaluated in BPD. The prevalence of insomnia in BPD, in conjunction with the aforementioned association between circadian

rhythm dysfunction and mood instability, suggests the need for further study of interventions to stabilise rest-activity patterns and discern secondary effects on mood symptoms.

Interventions such as dCBT-I and BLT have the advantages of scalability and accessibility regardless of location, unlike current gold-standard psychotherapies for BPD which are resource-intensive and typically oversubscribed (Iliakis et al., 2019a; Swales et al., 2000). The scalable interventions are also lower cost than face-to-face interventions and may be accessible without formal diagnosis, which is important for people with BPD, many of whom reject the diagnostic label (Stalker et al., 2005). However, both BLT and dCBT-I require considerable time commitments, and user adherence difficulties have been noted in both therapies, likely reducing treatment efficacy (Freeman, Sheaves, Goodwin, Yu, Nickless, Harrison, et al., 2017; Michalak et al., 2007a). To date, adherence trajectories of people with BPD to dCBT-I and BLT have not been reported in the extant literature.

Objectives

The objectives of this study were:

1) To determine the effects of a dual CBT-I and bright light therapy intervention on sleep and circadian rhythm disturbance in BPD.
2) To compare mood instability before and after the intervention.
3) To determine adherence to self-guided at-home sleep interventions in this group.

4.2 Methods

Participants

Participants were recruited as part of the Sleep in Borderline Personality Disorder – Stage II study via mailing lists from Stage I, online advertisements on the University of Oxford

Department of Psychiatry website, and local community and charity bulletins. Participants were screened online for the same criteria as Stage I (reported in Chapter 2), with the additional requirements of meeting the clinical threshold for insomnia (score ≤ 16) on the Sleep Condition Indicator (SCI) and having an evening or intermediate chronotype (score 16 – 58, inclusive) on the Morningness-Eveningness Questionnaire (MEQ) to ensure that the treatment targets of the interventions being studied were appropriate for the participants. Participation was timed to avoid daylight savings time shifts and travel to other time zones. The original recruitment target was 20 participants, which would have been sufficient to detect a pre- versus post-intervention effect size of Cohen's $d = 0.66$ with a conventional power (1-β) of 0.8 at significance $\alpha = 0.05$ (G*Power 3.0.10; Faul et al., 2007). This effect size was deemed reasonable as the primary outcome measure of the study was L5 onset time, and the estimated effect size of BLT in advancing midsleep phase is large at $d = 0.94$ (Fargason et al., 2017). Estimates suggest the therapeutic effect of BLT in treating symptoms of non-seasonal depression is also moderate-to-large ($d = 0.62$; Al-Karawi & Jubair, 2016).

These 20 participants were to be primarily recruited via re-contacting participants from Stage I of the study. However, there was a two-year gap between Stages 1 & 2 due to the COVID-19 pandemic during which many of the Stage 1 participants left the area or no longer met eligibility criteria. Recruitment efforts were also hindered by daylight savings-related constraints: we chose not to have participants enrolled over the beginning or ending of daylight-savings time (DST), as the clock change may cause sudden delays or advances in sleep and rest-activity patterns, which risked confounding the BLT treatment effect (Kantermann et al., 2007), particularly shifts in circadian phase which were an outcome of interest in this study. This decision resulted in 16 weeks (8 weeks prior to every clock

change) in which participants could not be enrolled, which also hampered recruitment. These factors, as well as withdrawals due to burden and major life disruptions, resulted in a reduced sample of seven participants. The study protocol was approved by the NRES Committee Oxford B - South Central (Reference no. 18/SC/0366) and all participants gave written informed consent.

Materials

Self-Report Measures

The following self-report measures were used:

Insomnia symptoms were assessed using the SCI (Espie et al., 2014) and the Insomnia Severity Index (ISI) (Bastien et al., 2001). The SCI has eight items and scores range from 0-32 with a threshold of ≤ 16 indicating clinically significant insomnia, while the ISI has seven items and scores ranging from 0-28 with a threshold of ≥ 15 indicating clinically significant insomnia.

Subjective sleep quality was measured with the Pittsburgh Sleep Quality Index (PSQI), a series of subscales providing a global score with a possible range of 0-21 and a threshold score of ≥ 5 indicating significant sleep disturbance (Buysse et al., 1989).

Maladaptive beliefs about sleep were measured with the Dysfunctional Beliefs and Attitudes about Sleep (DBAS-16) questionnaire (Morin et al., 2007b). The final score is equivalent to mean of all responses, falling on a scale from 0-10 with greater scores indicating increased dysfunctional beliefs and scores ≥ 4 representing unrealistic expectations for sleep.

The MEQ measured sleep timing preferences and was used as a proxy measure of circadian phase. It is a 19-item questionnaire with scores ranging from 16-86 and scores > 58 indicating early chronotypes (Horne & Ostberg, 1976). MEQ scores were also used to determine start timings for bright light therapy during the intervention period, following guidelines proposed by Terman & Terman (2005b).

Symptoms of depression were measured with the Quick Inventory of Depressive Symptomology – Self Report (QIDS-SR) (Rush et al., 2003). Scores range from 0-27, with scores ≥ 6 indicating mild depression, scores ≥ 10 indicating moderate depression, and scores ≥ 16 indicating severe depression.

The short Affective Lability Scale (ALS-18) measures changeability in affect between euthymia and affective states including anxiety, depression, elation, and anger (Oliver & Simons, 2004). Scores range from 18-72 with higher scores indicating greater lability.

The short Affect Intensity Measure (AIM-20) measures intensity of emotional responses (R. J. Larsen, 1985). Twenty questions provide a total score ranging from 0-6, with higher scores indicating greater affect intensity.

In addition to the self-report measures, the Zanarini Rating Scale for BPD (ZAN-BPD) was administered at baseline and at the final study visit (Zanarini, 2003). The ZAN-BPD a nine-item scale measuring the intensity and frequency of BPD symptoms with scores ranging from 0-36 and higher scores indicating increased symptom severity.

Daily Questionnaire Measures

Each morning, participants were prompted to respond to the Consensus Sleep Diary (Carney et al., 2012), recording the clock times at which they got in bed, began trying to fall asleep, their final awakening, and getting out of bed. The form also records frequency and duration of awakenings as well as subjective sleep quality.

MoodZoom is an ecological momentary assessment (EMA) platform for regular mood monitoring used during Stage I and described in detail in Chapter 2. Similarly, in Stage II participants responded to MoodZoom at two frequencies: ten times per day in the High Intensity Recording Periods (HIRPs) and twice daily in the Low Intensity Recording Period (LIRP). Figure 4.1 below shows MoodZoom sampling frequencies during each Stage of the study.

Actigraphy

As in Stage I, participants were given CamNTech MotionWatch 8 actigraphs and instructed to wear them as continuously as possible between the two study visits (Figure 3.1). Actigraph settings, sleep scoring procedures, and derived variables were consistent with Stage I, as described in Chapter 2.

Bright Light Therapy

Participants were provided with Diamond 5 SAD Light Boxes (Litepod Company) and instructed to sit facing the light box at arms-length, approximately 55 cm, each morning during the intervention period. The resulting dosage of approximately 10,000 lux for 30 minutes is the standard recommended efficacious dose (M. Terman & Terman, 2005b; Wirz-Justice & Terman, 2022). The timing of morning BLT was individualised for each participant

based on their MEQ score, which is an estimate of when BLT would appropriately interact with the phase response curve to produce a phase advance (M. Terman & Terman, 2005a). Wake times for MEQ score ranges were assigned based on previously established guidelines, such that individuals with later presumed circadian phase were assigned later wake times (M. Terman et al., 2001). The antidepressant effect of bright light therapy appears to be maximised when the light is used between 7.5 and 9.5 hours after melatonin onset time, which varies between individuals (J. S. Terman et al., 2001). Participants indicating that their recommended start time was too early were instructed to start at their typical wake time and shift their bright light therapy session earlier by 15 minutes each day until the recommended window was reached (Wirz-Justice & Terman, 2022). Participants were also asked to complete a paper log of their adherence to BLT.

Sleepio

CBT-I was delivered via Sleepio, accessed either on a mobile phone application or online (https://www.bighealth.co.uk/sleepio). The intervention has been described in more detail in Chapter 1. Participants were instructed to complete one session per week for the six-week intervention period and also to log each night's sleep diary on the platform at least once per week since this data is used to provide feedback and adjust treatment recommendations, particularly for assigning bed and wake times (Espie et al., 2012).

Procedure

At the initial study visit participants underwent the same screening procedure as in Stage I, completed the battery of questionnaires listed above, and were provided with a bright light therapy box and an actigraph to wear for the duration of the study. The following day they

began a one-week baseline HIRP, followed by the six-week LIRP in which they were instructed to use the bright light therapy box each morning as well as completing one Sleepio session each week. In the eighth week participants refrained from using Sleepio or the bright light therapy box, and ten-times daily MoodZoom monitoring resumed. At the final study visit participants repeated all questionnaire measures and returned their equipment (Figure 4.1). Upon completion or withdrawal, participants were compensated £60 for attending the first study visit and a further £60 if they had completed the final study visit, in addition to reimbursement of travel costs. Participants were invited to contact the research team if they had difficulties with any component of the study; and were all contacted via email at the study midpoint to help maintain engagement.

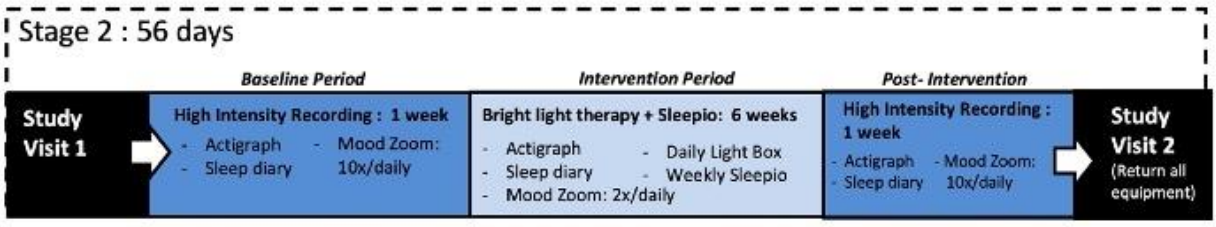

Figure 4.1. SBPD - Stage II study protocol and timeline.

Statistical Analysis

The original primary outcome measure of this analysis was actigraphy-derived change in L5 onset time between the baseline and post-intervention weeks. However, the reduced sample size of the trial limited the statistical power available for comparisons of aggregate data. Although statistical test results are presented, this analysis primarily focuses on individual change trajectories in questionnaire responses, actigraphy, and MoodZoom data, as well as

discussing adherence to the study protocol. Although the small sample size limits the interpretations that can be made about treatment efficacy, recent analysis of physiological signals from wearable devices has found high risk of misleading aggregate results due to within-person heterogeneity, and instead suggests focusing on change relative to individual baseline (S. Goodday et al., 2024). Adherence to Sleepio was gathered from application usage data. An estimate of adherence to bright light therapy was formed using daily self-reports from a paper diary, but responses were often missing. Light intensity readings from the actigraphs were used to supplement self-report adherence data; days with 20-40 minutes of sudden onset and offset light exposure > 400 lux concurrent with low activity levels shortly after waking were coded as compliant even if self-report data was missing. This threshold was chosen because it was well above the light intensity registered on the MotionWatch from indoor electrical light or computer lighting (Falck et al., 2021). In some cases, actograms clearly demonstrated light exposure meeting these criteria (*eg.* Appendix, Section IV, Figures 4A12-13). However, this method likely had low accuracy as adherence could be underestimated for participants who routinely covered their actigraphs with long sleeved clothing, or overestimated for any participants who were exposed to bright environmental light immediately after waking. Thus, the BLT adherence data presented in this report should only be considered a rough estimate and should be reviewed with caution.

Questionnaire Data

Baseline and post-intervention scores were compared via paired Welch's *t*-tests for normally distributed data, Watson-Williams tests for L5 onset time data (due to circularity), and Wilcoxon matched-pairs signed-rank tests for all other data (Delacre et al., 2017; G. S. Watson & Williams, 1956; Wilcoxon, 1945).

MoodZoom Data

To examine changes in affect intensity, median mood ratings of each mood for each participant during the baseline and post-intervention were compared using Wilcoxon matched-pairs signed-rank tests. Mood instability was calculated using within-week and within-day tRMSSD for each mood and again compared between baseline and post-intervention periods using Wilcoxon matched-pairs signed-rank tests. Although Kalman filtering was considered to handle missing values, the degree of missingness prevented imputation of 6% of baseline responses and 29% of post-intervention responses ($\chi 2 = 96.74$, $p < .001$). This difference in missing data meant that the time elapsed between measurements was more irregular in the post-intervention period than the baseline period. To facilitate comparison between these periods despite this difference, time-adjusted Root Mean Successive Squared Differences (tRMSSDs) were calculated for each mood and participant instead of using Kalman filtering. tRMSSD is calculated according to the following formula (Taquet et al., 2023):

$$tRMSSD = \sqrt{\frac{1}{N}\sum_{i=1}^{N-1}\left(\frac{x_{i+1} - x_i}{t_{i+1} - t_i}\right)^2}$$

In which N represents the number of measurements taken, x_i represents the value of the i_{th} data point, and t_i represents the timestamp at which the i_{th} measurement was taken. The subscript i+1 indicates the subsequent variable. Therefore, increased time elapsed between measures (as occurs when consecutive data points are missing) results in a smaller tRMSSD, reflecting that differences in consecutive measures are less indicative of instability as time elapsed between measurements increases. For each participant and mood, tRMSSD was

calculated within each HIRP week as well as within each day. Within-day tRMSSD was only used to graph changes in mood instability over the course of each week. Both types of tRMSSD were compared between baseline and post-intervention periods using Wilcoxon matched-pairs signed-rank tests; for within-day tRMSSD this was done using the median value for each participant. Comparisons between baseline and post-intervention tRMSSD were made with Wilcoxon matched pairs signed-rank tests (Wilcoxon, 1945).

Actigraphy Data

The actigraphy data was cleaned and analysed following the same procedure as in Chapter 2, except that JL conducted the sleep scoring in this instance. Of note, time in bed (TIB) was calculated as the period between the first sleep attempt and getting out of bed, not including time spent in bed prior to attempting to sleep. This is not the standard variable definition but is in alignment with the procedure followed in Chapter 2 and the values automatically generated by CamNTech's MotionWare software. Comparisons between mean values in the baseline and post-intervention weeks were made using Watson-Williams tests for L5 onset time data (due to circularity), and Wilcoxon matched-pairs signed-rank tests for all other data, since the mean values were not normally distributed (Watson & Williams, 1956; Wilcoxon, 1945). For L5 and M10, clock times were converted to decimal values prior to statistical comparison. L5 values were further converted to circular data using the *circular* package v.0.5-0 in R (U. Lund et al., 2023). For some of the figures presented in this chapter, min-max normalisation was used to scale data to a [0, 1] range. This was done by subtracting the minimum value from each value and dividing it by the difference between the minimum and maximum values.

4.3 Results

Recruitment & Uptake

Eleven participants attended in-person screening visits, one of whom was ineligible to participate due to psychiatric history and head injury. Of the remaining ten participants, one withdrew after three weeks, one withdrew between the screening visit and beginning the baseline period the following day, and one was lost to follow-up (Figure 4.2). Both participants who withdrew cited study burden in conjunction with stressful life events. Figure 4.3 shows that five participants completed the protocol in the winter (November – March), one in the spring (May – July), and one in the autumn (September – October).

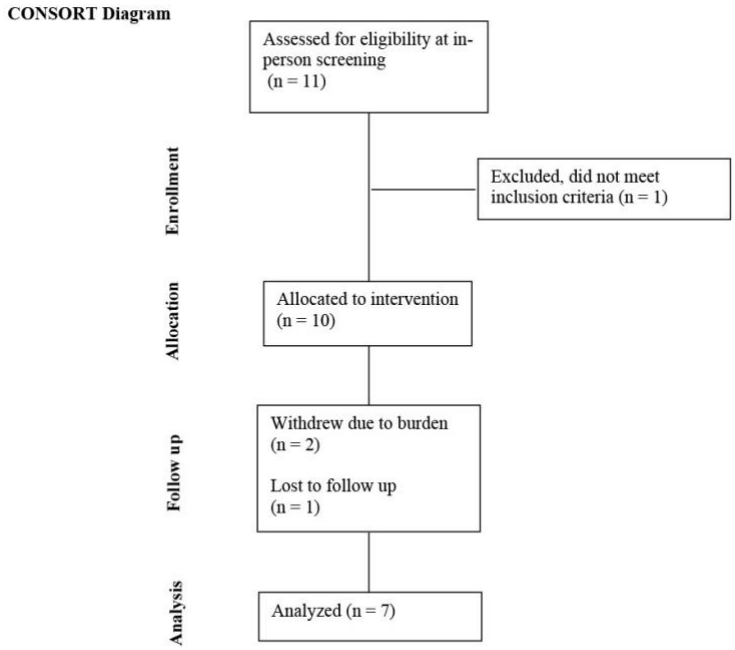

Figure 4.2.CONSORT diagram showing the flow of participants through recruitment and the study.

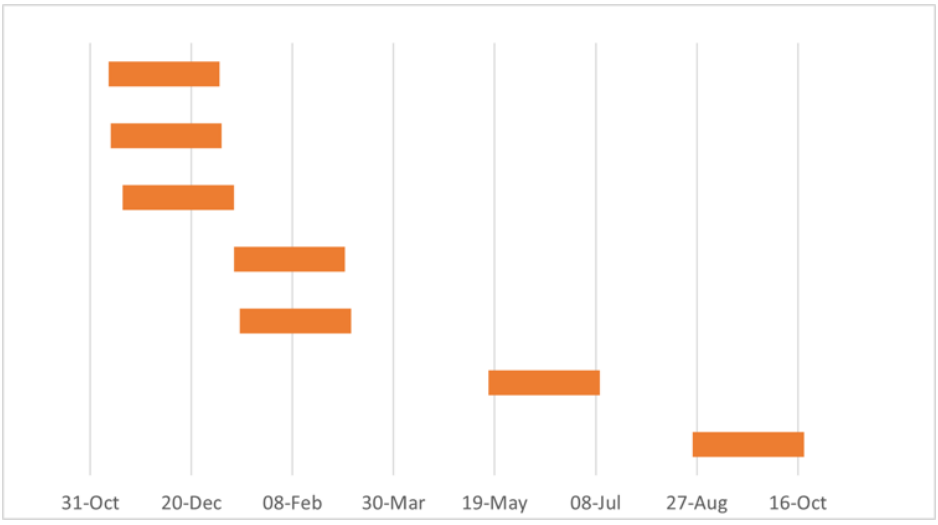

Figure 4.3. Seasonal distribution of participants in Stage II of the Sleep in BPD Study.

Patient Demographics

Participants (n = 7) ranged in age from 20 to 27 years (mean = 24.71, SD = 2.37). Five participants reported full-time study (n = 3) or employment (n = 2), one was studying part-time in addition to part-time carer responsibilities, and one was unemployed. Lifetime diagnoses of major depressive disorder, anxiety disorders, and current antidepressant medication were common in the sample (Table 4.1)

Table 4.1. Self-reported lifetime diagnoses of concomitant disorders and self-reported current psychotropic medication use.

Concomitant Disorders	Major Depressive	Anxiety	Eating	cPTSD		
No. Participants	5	5	1	1		
Psychotropic Medication	Antidepressant	Anxiolytic	Antipsychotic	Hypnotic	Beta-Blocker	None
No. Participants	5	2	2	2	2	2

Self-Report Measures

At the final study visit, there were significant improvements in mean values of all subjective sleep measures, along with significant improvements in depressive symptoms and social functioning (SCI Wilcoxon Signed-Rank test: V(1), $p = .03$; ISI: t(4.45), $p < .01$; DBAS: t(4.04), $p = .01$; PSQI: t(3.08), $p = .02$; QIDS: t(3.06), $p < .02$; SFQ: t(2.97), $p = .02$), full results in Table 4.2. All other measures showed non-significant improvements (or non-significant increased morningness, indicated by an increase in MEQ score). Figures 4.5 - 4.7 below show reduced self-reported insomnia symptoms, improved sleep quality and less dysfunctional beliefs about sleep across nearly all participants.

Table 4.2. Mean questionnaire scores at baseline versus post-intervention

Questionnaire	Mean Baseline [a] Visit Score	Mean Final Study Visit [b] Score	Test Statistic [c]	*p*
Sleep/Circadian				
SCI	9.14 (3.76)	21.1 (7.84)	V = 1	.03*
ISI	15.29 (2.98)	7.43 (4.20)	t = 4.45	<.01*
DBAS	5.93 (1.66)	3.37 (1.19)	t = 4.04	.01*
PSQI	13.14 (4.06)	8.86 (2.79)	t = 3.08	.02*
MEQ	40.43 (9.07)	46.57 (15.58)	t =-1.83	.12
Symptom Measures				
ZAN-BPD	10.57 (7.76)	7.29 (6.99)	t = 1.32	.23
QIDS	15.00 (3.51)	8.57 (6.08)	t = 3.06	.02*
ALS	46.57 (18.13)	43.57 (14.89)	t = 0.61	.56
AIM	77.75 (11.62)	75.71 (9.41)	t =-0.53	.60
Other				
SFQ	14.43 (4.47)	10.43 (5.65)	t = 2.97	.02*

[a] Measure taken on Day 0. [b] Measure taken on Day 56. [c] V: Paired Wilcoxon-Signed Rank Test, t: Paired Welch's t-test. *Significant at $\alpha < .05$

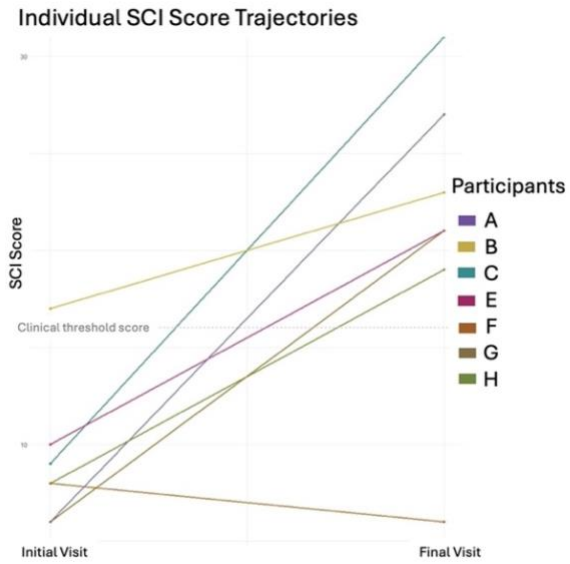

Figure 4.5. Change in SCI score between the first and final study visits. Higher SCI scores indicate reduced insomnia symptoms. Clinical threshold score is indicated by the dotted line.

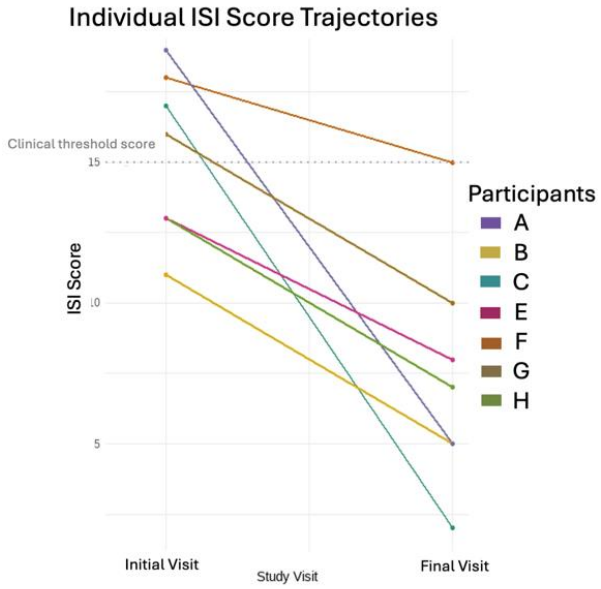

Figure 4.4. Change in ISI scores between the first and final study visits. Lower ISI scores indicate reduced insomnia symptoms. Clinical threshold score is indicated by the dotted line.

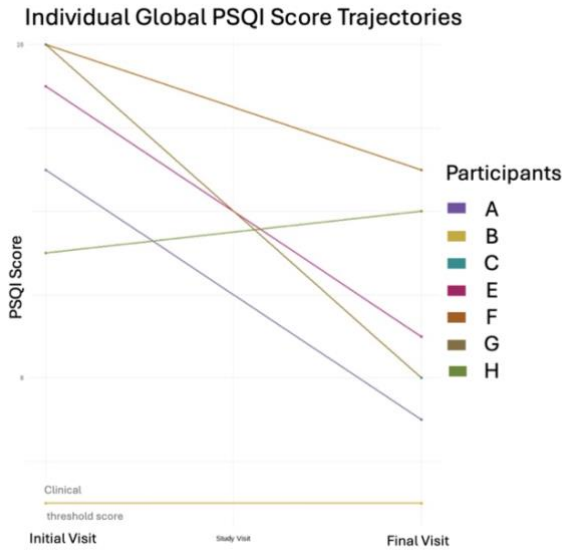

Figure 4.6. (above). Change in PSQI score between the first and final study visits. Lower PSQI scores indicate improved sleep quality. The threshold score of 5 is obscured by the data.

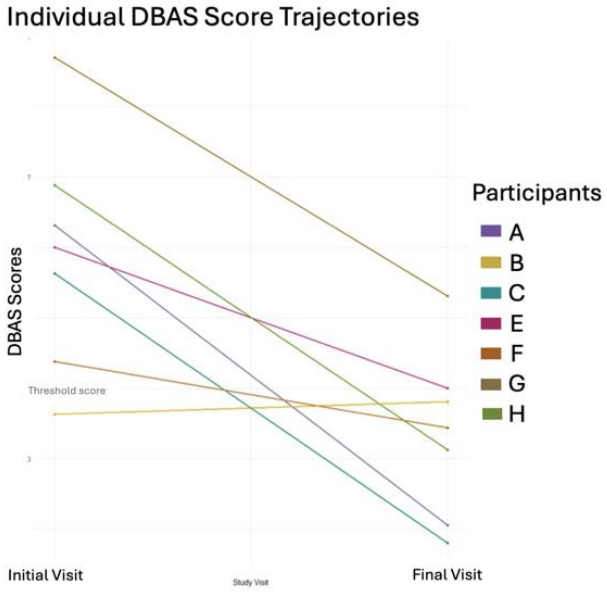

Figure 4.7. (above) Change in DBAS scores between the first and final study visits. The DBAS has threshold score of 4.

Adherence & Engagement

Figure 4.8 below shows the attrition of participants from Sleepio over the six-week treatment course. Five out of seven (71%) of participants reached session three, which introduces sleep restriction therapy, although only two of seven (29%) completed Sleepio. Figure 4.9 shows estimated adherence to bright light therapy in number of sessions, which ranged from 1-42 (2-100%), mean = 27.43 (64%), SD = 12.51.

Figure 4.8. Number of participants completing each Sleepio session.

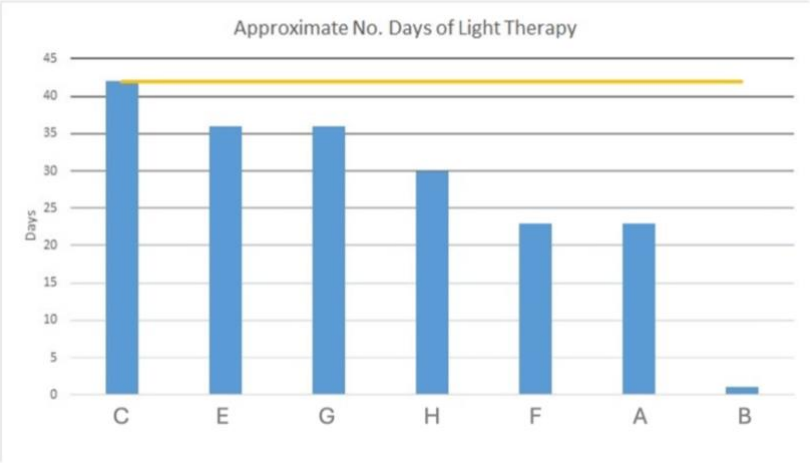

Figure 4.9. Estimated number of completed bright light therapy sessions per participant, with yellow line indicating the maximum possible number of sessions (42).

EMA Missingness

Figure 4.10 shows the average number of daily MoodZoom responses decreasing from 80.4% of prompts during the baseline monitoring period to 48.8% in the post-intervention period ($\chi 2 = 107.77$, $p < .001$). The low-intensity period mean response rate was high (85.7%) and relatively stable throughout that six-week period. Figure 4.11 illustrates three MoodZoom adherence trajectories throughout the duration of the study: Participant C was highly responsive to throughout the study, Participant F was moderately responsive during the baseline period but began to miss responses during the intervention period and did not adhere to the high-intensity post-intervention monitoring. Participant B exhibited a common response pattern of strong early engagement, reasonable engagement throughout the low-intensity period but then a clear response fatigue in the post-intervention period. Although Kalman filtering was considered to handle missing values, the degree of missingness prevented imputation of 6% of baseline responses and 29% of post-intervention responses ($\chi 2$

= 96.74, $p < .001$). To facilitate comparison between the baseline and post-intervention period despite this difference, the daily and weekly tRMSSD were calculated instead.

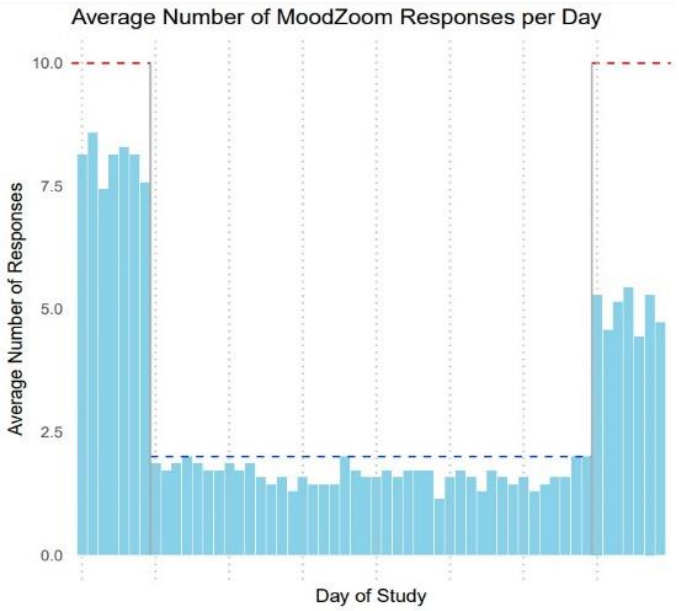

Figure 4.10. Average Number of MoodZoom Responses per Day. Dashed lines represent the number of prompts sent on each day (10 during the HIRP and 2 during the LIRP).

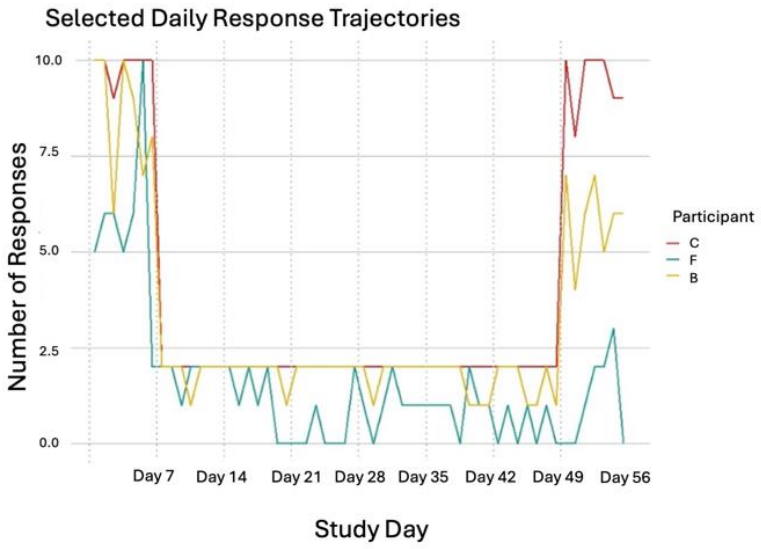

Figure 4.11. Selected Daily MoodZoom Response Trajectories

Mood Ratings & Instability

Table 4.3 below compares median mood ratings across all participants in the baseline and post-intervention weeks and presents the results of paired Wilcoxon Signed-Rank tests comparing within-participant values. There were no significant differences in raw mood ratings between the baseline and post-intervention periods. Table 4.4 below shows tRMSSD. There were no significant differences in mood instability between the baseline and post-intervention weeks. Median values are presented in all tables since data was not normally distributed.

Table 4.3. Median MoodZoom rating during the baseline versus post-intervention weeks.

Mood	Median Baseline[a] MoodZoom rating [IQR]	Median Post-Intervention[b] MoodZoom rating [IQR]	Test Statistic[c], (*p*)
Anxiety	1 [0,3]	1 [0, 3]	V = 6 (.86)
Elation	0 [0, 1]	0 [0, 2]	V = 1.5 (.27)
Sadness	1 [0, 3]	0 [0, 1]	V = 8 (.36)
Anger	0 [0, 0]	0 [0, 0]	V = 1 (.99)
Irritability	1 [0, 2]	0 [0, 2]	V = 16 (.28)
Energy	1 [0, 3]	2 [1, 4]	V = 8.5 (.39)

[a] Measure taken on Day 0. [b] Measure taken on Day 56. [c] Paired Wilcoxon Signed-Rank test.

Table 4.4. Median tRMSSD before versus after the intervention.

Mood	Median Baseline [a] tRMSSD [IQR]	Median Post-Intervention [b] tRMSSD [IQR]	Test Statistic[c], (*p*)
Anxiety	3.39e-04 [2.44e-04, 5.26e-04]	1.85e-04 [1.56e-04, 2.38e-04]	V = 14 (.13)
Elation	1.43e-04 [6.31e-04, 2.95e-04]	1.25e-04 [8.95e-05, 2.65e-04]	V = 10 (.63)
Sadness	2.17e-04 [1.47e-04, 4.28e-04]	8.72e-05 [6.88e-05, 2.57e-04]	V = 11 (.44)
Anger	3.04e-04 [1.18e-05, 5.51e-04]	5.01e-05 [0.00, 1.27e-04]	V = 14 (.13)
Irritability	5.15e-04 [2.22e-04, 5.92e-04]	1.86e-04 [1.70e-04, 2.09e-04]	V = 11 (.44)
Energy	3.22e-04 [1.69e-04, 3.37e-04]	1.81e-04 [1.66e-04, 2.66e-04]	V = 9 (.81)

[a] tRMSSD over the baseline week (Days 1 – 7). [b] tRMSSD over the post-intervention week (Days 50 – 56) [c]Paired Wilcoxon Signed-Rank test.

Actigraphy Results

Table 4.5, Figures 4.12 & 4.13 below show no significant differences in TST or TIB following intervention. SOL was significantly reduced from baseline (Median = 10.29 min) following intervention (Median = 2.6 min, V = 28, p = .02). Participant had greater sleep onset latency at baseline relative to the other participants (Figure 4.14). In order to determine whether the change in her SOL was singularly responsible for the significant post-intervention reductions, another Wilcoxon-Signed rank test was repeated without her data, but the difference remained significant (V =21, p = .03).

Figure 4.15 below shows normalised values for TST, TIB, and SOL over the study period for each participant. Participants C, H, and G show reductions in SOL coinciding with the introduction of sleep restriction therapy (SRT). There were no significant differences in non-parametric circadian rhythm analysis variables between the baseline and post-intervention weeks (Table 4.6). However, there were numerical changes in median L5 start time, which advanced by 35 min and the median M10 start time which advanced by 105 minutes. Figures comparing the distribution of non-parametric circadian variables at baseline and post-intervention are in Section IV of the Appendix.

Actograms for the most and least bright light therapy-adherent participants are presented in the Appendix, Section IV, Figures 4A.12 and 4A.13, respectively. Figure 4A.12 shows stabilisation of wake times and period of high-intensity light exposure each morning with eventual reduction in variability of bedtimes. These changes appear to endure in the post-intervention week despite discontinuation of bright light therapy. Figure 4A.13 shows activity from a participant who was non-adherent to her prescribed wake time. Rest-activity patterns

appear unstable, with frequent nighttime activity and light exposure, and some nights without a rest period.

Table 4.5. Comparisons of actigraphy-derived sleep variables in the baseline versus post-intervention weeks. Median values are presented since data distribution was not normal (likely due to small sample size).

Variable	Baseline Week Median [IQR]	Post-Intervention Week Median [IQR]	Test Statistic [a], (*p*)
Total Sleep Time (min)	417.64 [383.29, 430.29]	414.5 [369.32, 437.60]	V = 15, (.94)
Time in Bed (min)	515.21 [499.93, 548.86]	502.33 [487.67 , 523.21]	V = 20, (.38)
Sleep Efficiency (%)	82.00 [71.74, 82.99]	82.89 [77.76, 84.11]	V = 2, (.05)
Sleep Onset Latency (min)	10.29 [6.79, 15.5]	2.6 [1.57, 4.79]	V = 28, (.02)
Sleep Onset Latency, outlier removed* (min)	8.57 [6.75, 13.71]	3.23 [1.83, 5.25]	V =21, (.03)
Wake After Sleep Onset (min)	79.21 [75.43, 144.00]	84.20 [77.46, 107.27]	V = 21, (.30)

[a] Paired Wilcoxon-Signed Rank Test. * Participant C (see Figure 4.13).

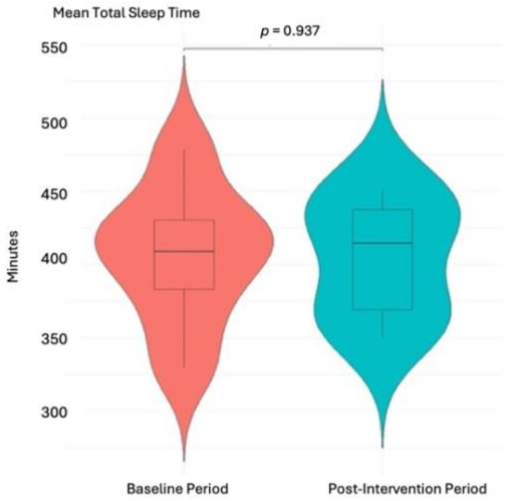

Figure 4.12. Actigraphy-derived total sleep time (TST) during the baseline versus post-intervention weeks.

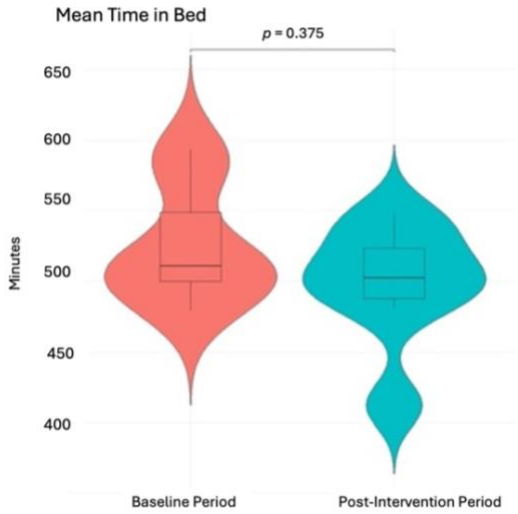

Figure 4.13. Actigraphy-derived time in bed (TIB) during the baseline versus post-intervention weeks.

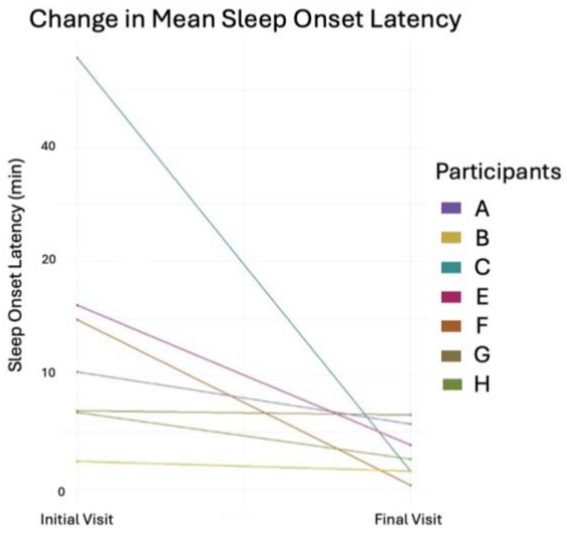

Figure 4.14. Individual change in sleep onset latency (SOL) in minutes during the baseline versus post-intervention week.

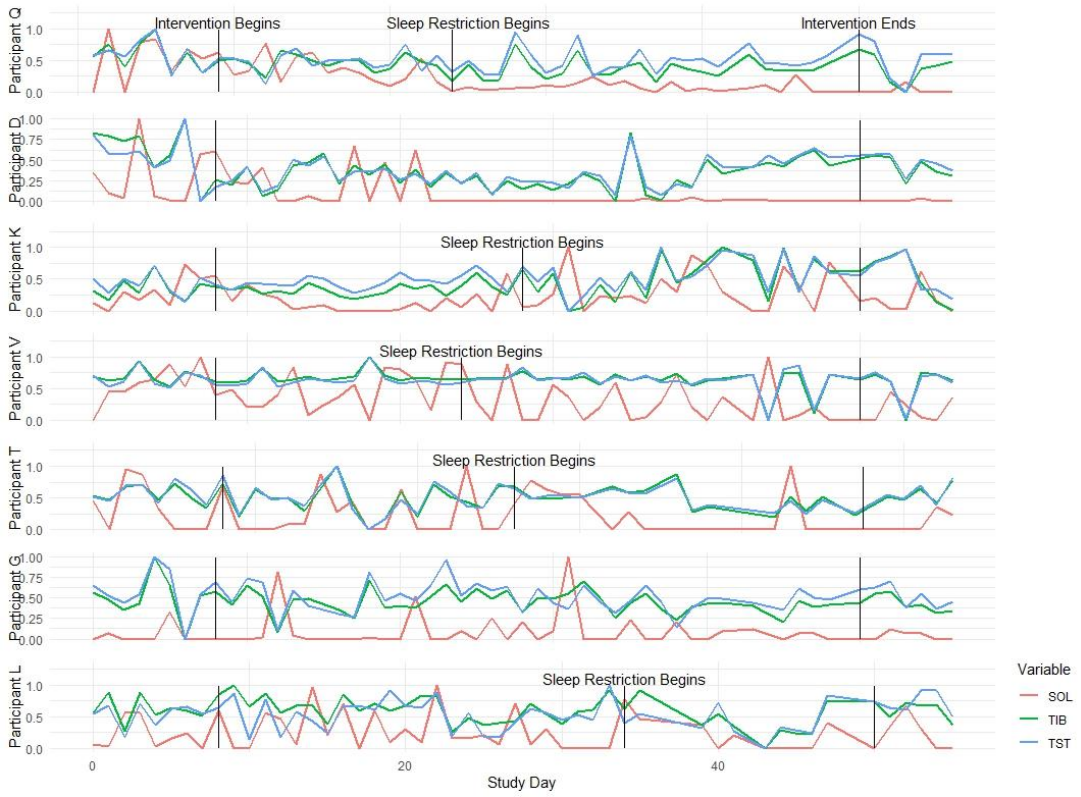

Figure 4.15. Normalised values for total sleep time, time in bed, and sleep onset latency over the study period for all study participants. "Sleep restriction begins" indicates the study day on which the third Sleepio session was completed. This label is absent for participants who did not initiate Session 3 (the introduction to sleep restriction therapy).

Table 4.6. Comparisons of actigraphy-derived non-parametric circadian rhythm variables during the baseline versus post-intervention weeks.

Variable	Baseline Week Median [IQR]	Post-Intervention Week Median [IQR]	Test Statistic [a], (*p*)
Rhythm Structure			
Interdaily Stability	0.47 [0.40, 0.55]	0.52 [0.41, 0.58]	V = 2 (.18)
Intradaily Variability	1.04 [0.72, 1.11]	0.89 [0.79, 1.04]	V = 16 (.81)
Relative Amplitude	0.88 [0.84, 0.92]	0.91 [0.87, 0.93]	V = 2 (.09)
Activity Levels			
L5 Activity	13.61 [11.66, 26.43]	14.72 [10.66, 19.03]	V = 25 (.08)
M10 Activity	256.16 [175.17, 323.01]	258.64 [205.03, 283.85]	V = 15 (.94)
Rhythm Timing			
L5 Start time, clock time	02:12 [00:56, 3:30]	01:37 [1:16, 3:08]	F = 0.21 (.65)
M10 Start time, clock time	10:41 [9:54, 14:00]	08:55 [08:06, 13:00]	V = 24.5 (.09)

Median values are presented since data distribution was not normal (likely due to small sample size).
[a] V: Paired Wilcoxon-Signed Rank test. F: Watson-Williams test.

4.4 Discussion

Summary of Findings

After a six-week course of bright light therapy and cognitive behavioural therapy for insomnia, participants with BPD reported significant improvements relative to baseline in all subjective measures of insomnia symptoms and in actigraphy-derived sleep onset latency. Despite low statistical power, increases in actigraphy-derived sleep efficiency, circadian rest-activity phase, and relative amplitude were also recorded, although total sleep time, time in bed, circadian rhythm structure, and mean activity levels did not change significantly. Results suggest that treatment may have improved participants' cognitions about sleep and advanced their circadian rest-activity phase, motivating further study of CBT-I and bright light therapy in BPD. The effect of treatment on mood and mood instability remains unclear as this analysis was underpowered, although instability in angry mood was significantly reduced relative to baseline. Participants also reported significant improvements in depression symptoms and social functioning. Adherence to bright light therapy and CBT-I was moderate in this study but might be improved with more frequent clinical contact.

Observed changes in actigraphy-derived measures of sleep and circadian phase, particularly the significant reduction in sleep onset latency, align with the extant literature on BLT in BPD (Bromundt et al., 2013). Reductions to sleep latency in the present study likely contributed to the improvement in sleep efficiency. Reduction in onset latency and increase in sleep efficiency without significant changes to total sleep time, time in bed, or mean activity levels suggest that these changes may have been driven by circadian phase advances and improved sleep cognitions, rather than behavioural changes from sleep restriction therapy.

Proposed Mechanisms

Self-reported dysfunctional beliefs about sleep were significantly improved at the final study visit. Stage I of the SBPD study suggested that people with BPD may experience increased dysfunctional sleep cognitions, and CBT-I research in other groups has suggested that sleep cognitions are tractable treatment targets (Edinger et al., 2001; Plante et al., 2013). Resolving dysfunctional beliefs about sleep may reduce sleep-disruptive behaviours and physiological arousal at bedtime, thus making it easier to fall asleep, reducing sleep onset latency and in turn improving efficiency. Advancing circadian phase could also contribute to shorter onset latency by reducing lag between bedtime and physiological preparedness for sleep, particularly by inducing earlier melatonin release and reduction in core body temperature via heat dissipation from core to the periphery (Kräuchi & Wirz-Justice, 2001). Previous research found that people with BPD had a significant phase delay in rest-activity patterns relative to healthy controls, which has been suggested as a cause of subjective sleep disturbance reported in BPD (Carr, Saunders, Bilderbeck, et al., 2018; Winsper, Tang, et al., 2017). Our current results suggest that light therapy may be effective in advancing circadian phase in women with BPD experiencing insomnia, although this effect should be further investigated in a larger sample and ideally with endocrine and physiological measures of circadian phase, such as dim light melatonin onset and core body temperature, respectively. Although circadian phase delay has previously been associated with mood instability in BPD (Carr, Saunders, Tsanas, et al., 2018; McGowan et al., 2020), this analysis was underpowered to find evidence that CBT-I or bright light therapy had a secondary effect on mood instability.

Participants did not report significant change in their overall BPD symptom severity (measured via the ZAN-BPD), but depressive symptoms were significantly reduced

following treatment, suggesting BLT may have an antidepressant effect in people with BPD. Meta-analyses have identified reductions in both seasonal and non-seasonal depressive symptoms following BLT, although heterogeneity and risk of bias were noted in both reviews (Pjrek et al., 2020; Tao et al., 2020). BLT may attenuate depressive symptoms by reducing amygdala reactivity to negative stimuli: one study established a significant negative dose-response relationship between BLT and amygdala reactivity (P. M. Fisher et al., 2014), which is of interest to the treatment of BPD given that the disorder is associated with elevated amygdala reactivity, thought to drive mood instability in the group (Donegan et al., 2003; Herpertz et al., 2001; New et al., 2007). An alternative hypothesis for the mechanism of action for BLT is that the resulting advance in circadian phase aligns processes under circadian control to the desired sleep-wake cycle, reducing physiological strain and in turn depressive symptoms (Lewy et al., 2007). This phase shift hypothesis is particularly relevant to seasonal affective disorder (SAD), since exposure to natural light is typically abbreviated in the winter which may result in circadian phase delays.

Indeed, a dose-response relationship between BLT-driven circadian phase advance and depressive symptoms has been demonstrated in SAD (J. S. Terman et al., 2001). Taken together, these findings suggest that the antidepressant effects of BLT may act through multiple mechanisms; replications of both dose-responses would elucidate this. CBT-I also appears to have antidepressant effects which may have contributed to the reduction in depressive symptoms (Cheng et al., 2019; Henry et al., 2021). Notwithstanding the negative finding of BLT and dCBT-I on mood instability, reduction in depressive symptoms is clearly important to quality of life and may have a secondary effect of reducing mood instability which this sample was not powered to detect.

Intervention Adherence

The lack of significant change to total sleep time and time in bed is inconsistent with both the SRT component of CBT-I and previous findings of reduced sleep time after bright light therapy (Bromundt et al., 2013). This discrepancy may be due to moderate adherence to assigned wake times coupled with the small sample size of this cohort. Adherence is highly relevant to both interventions: the mechanism of bright light therapy relies on regularity to stabilise circadian phase and most components of CBT-I also rely on consistency at least during treatment (Wirz-Justice & Terman, 2022). Omitting Participant B, who was non-adherent to assigned wake times (Figure 3.9), the others had an estimated mean adherence of 75% to bright light therapy sessions. While this is reasonable, late awakenings on the remaining days may have produced enough variability to render the difference insignificant. Figures 3.15 and 3.16 illustrate the range in rest-activity patterns between participants, which may be related to adherence. Participant C had relatively more stable rest-activity patterns during the baseline week and was able to adhere to her prescribed wake time, which eventually also stabilised her bedtime. She experienced a reduction in sleep onset latency, as discussed above. By comparison, Participant B demonstrated highly variable bedtimes and sleep durations in the baseline week and was not adherent to prescribed wake times. There may be a requisite level of stability for feasible unmonitored bright light therapy in BPD given the propensity for variability in rest-activity patterns and that prescribed wake times may be unpleasantly early for participants with suspected circadian phase delays (Wood et al., 2015). In theory, adherence becomes easier as treatment continues and circadian phase advances, but this process requires initial consistency and may prove difficult for people with severe rest-activity pattern instability (Kräuchi & Wirz-Justice, 2001). Adherence challenges

also arose with Sleepio: only five participants reached the third session of Sleepio introducing sleep restriction techniques, so at least two participants were not instructed to reduce their sleep window beyond their bright light therapy assigned wake times. Additionally, only two participants completed the final two sessions. This pattern of decay in adherence, dubbed the "Law of Attrition" is widespread in digital interventions, and a similar response fatigue was observed in the final days of MoodZoom monitoring (Eysenbach, 2005).

Implications

The observed changes in sleep onset latency, sleep efficiency, circadian phase measures and subjective sleep experience warrant further research on CBT-I and bright light therapy in BPD, particularly since they reached statistical significance despite a small sample size. However, the results of this study should be interpreted with caution as changes observed in a single-arm, open-label study design could be due to participant expectation or regression to the mean. Future research should evaluate these treatments in a randomised controlled trial with a larger sample size, and ideally use an endocrine measure of circadian phase such as dim light melatonin onset. Although there is no consensus instrument for measuring mood instability, the EMA approach used in this study effectively distinguished between clinical and control groups and demonstrated high levels of adherence. This makes it a promising tool for application in future RCTs. Future study design should account for the possibility of treatment discontinuation prior to sleep restriction content delivery and consider making contact with participants just ahead of SRT introduction to ensure that this key component of CBT-I is relayed to participants, since its standalone efficacy has been demonstrated (Kyle et al., 2023). Previous work has suggested that small increases in clinical contact can effectively attenuate treatment failure in dCBT-I (Forsell et al., 2019). Secondary effects of CBT-I and

BLT on mood instability, proposed in light of previously identified associations with circadian phase lag, might also be identified with further statistical power.

Alongside objective improvements in sleep and probable advances in circadian phase, participants reported significant and consistent improvements in subjective sleep experience, which further motivates investigation of these treatments. Although some previous research has suggested inaccuracy in self-reported sleep duration and quality in BPD relative to polysomnographic measures (Philipsen et al., 2005), these findings suggest that changes to subjective measures tracked with objective measures of sleep onset latency and sleep efficiency. This implies that sleep latency and efficiency may influence subjective experience more than sleep duration or nighttime awakenings, which were both slightly worsened in the post-intervention week. Improvements in subjective sleep experience are notable given that people with BPD are often characterised as having rigid dysfunctional beliefs that impact treatment efficacy (Beck et al., 2001; Wenzel et al., 2008). These results suggest that not only are sleep problems tractable in this group, people with BPD are also attuned to changes in their sleep. Successful sleep interventions may improve general wellbeing in BPD as well as attitudes towards psychological interventions more broadly.

Non-adherence posed difficulties to evaluation of both interventions in this study, which was emphasized by differences between participants' actigraphy-derived trajectories. Both interventions require major time commitments, and some participants struggled with the assigned wake times of bright light therapy. Rest-activity pattern instability prior to treatment may contribute to difficulty adhering to bright light therapy wake times, and likely also bedtime sleep restriction. More frequent clinical support might help maintain accountability

to treatment, and data visualization from activity trackers could also support this dynamic, particularly if it could be linked with other symptom-monitoring data.

Limitations

As previously mentioned, this study was not powered to detect significant changes in mood instability after intervention or to construct predictive models. However, recent work in other participant groups suggests that aggregate data from wearable devices can be irrelevant or even misleading when predicting a single person's data, such as in clinical contexts (S. Goodday et al., 2024). Instead, the authors recommend within-person comparisons to baseline data, as reported in this analysis. Several further factors may have impacted our findings: this was an open-label trial without a control group and therefore subjective ratings of sleep and mood could be inflated by expectation. Participants were also selected based on elevated insomnia symptoms and delayed circadian phase; it is possible that changes following intervention are attributable to regression to the mean rather than treatment effect. Adherence to both interventions was moderate which likely also reduced true treatment efficacy. Nonetheless, significant changes to objective sleep parameters motivate further research into these interventions in people with BPD. Another limitation is the 'time in bed' variable was defined as time from first sleep attempt through to getting out of bed in the morning. This does not include time spent in bed prior to sleep. Although participants may have followed SRT guidelines by reducing this time in bed, those changes were unfortunately not detected in this analysis. Additionally, six of the seven participants completed the study during the autumn and winter. Shorter winter daylight in England may have increased the observed efficacy of the bright light therapy relative to use in summer months, although

previous research with bright light therapy has not detected a significant seasonal difference in effect (Chojnacka et al., 2016; Martiny et al., 2005). This study shares a limitation with Stage I of the SBPD study: reliance on self-reported sleep disorders, meaning it is possible that sleep disturbance at baseline was due to sleep apnoea, restless leg syndrome, or other disorders as opposed to insomnia or irregularities in circadian rhythm. Thus, it is possible that the interventions employed in this study were not addressing the source of sleep disturbance in this group and efficacy was thus underestimated. Finally, this study did not employ exclusion criteria related to eye disease or photosensitivity, which was an oversight as these conditions may limit the efficacy of BLT and, more importantly, pose a safety concern as they may increase susceptibility to light-induced changes, particularly ocular lesions (Remé et al., 1996).

4.5 Conclusions

A six-week period of dual morning BLT and dCBT-I was efficacious at improving sleep quality and tended to advance circadian phase in a small sample of women with BPD. Both objective and subjective measures of sleep were significantly improved, perhaps due to reduced maladaptive cognitions about sleep. The interventions did not appear to change mood instability, aside from reducing instability in anger, but did seem to produce a significant antidepressant effect. However, these results are limited by the potential effects of expectation and regression to the mean and thus require replication in a randomised controlled trial.

5 Chapter 5: Participant Experience of the Sleep in BPD Intervention

5.1 Introduction

Patient and public involvement and engagement (PPIE) is the practice of seeking feedback on research methods or health services from people with a specific lived experience, with the objective of optimising research and healthcare and ensuring that these processes are conducted appropriately (Crawford et al., 2002). In the context of piloting interventions, PPIE helps to determine whether the proposed interventions and their delivery are seen as effective, appropriately delivered, and worthwhile by the intended audience. This is particularly important in a patient group with BPD because of the ongoing discourse surrounding diagnosis and by extension treatment of the disorder. PPIE may provide an opportunity for individuals with symptoms of BPD to contribute to the development of non-stigmatising care. The practice of PPIE also returns some agency to a group of individuals who often report feeling disregarded or excluded by the medical community (NICE, 2019; Stalker et al., 2005).

Incorporating PPIE in preliminary studies of an intervention also helps to avoid the pitfall of designing an intervention for an idealised patient instead of the actual users. This is the principle behind the human-centred design (HCD) movement (Melles et al., 2021), which suggests that systems implemented without realistic understanding of the end user will not be used (or will be mis-used). Just as drivers will adjust their route to avoid a poorly designed intersection, patients will not sustain engagement with an intervention that does not seem to be a worthwhile use of their time. There are, of course, many other factors which affect engagement and retention, such as users' intrinsic motivation, their degree of functional

impairment, their availability or intervention side-effects. However, none of these are as easily modifiable as intervention design, and the HCD movement suggests that the responsibility for this design lies with the people who inform, create, and disseminate these interventions.

This may be especially important for self-guided or automated interventions accessed via the internet or smartphone applications, since these typically lack the completion accountability of face-to-face care. This is increasingly relevant after the COVID-19 pandemic, in which a "decade of innovation in two months" rushed many psychotherapy services into digital or remotely-accessible formats, leaving some systems fragmented, particularly in terms of service user experience (Roland et al., 2020). HCD proposes that the solution to this problem lies in iterative consultation with service users, reducing user difficulties to ensure that quality of care is not compromised by delivery format (Vial et al., 2022). This approach should similarly be applied to the monitoring components of studies to optimise data accuracy and reduce missingness.

This study employed ecological momentary assessment (EMA) to collect mood data and actigraphy to monitor sleep patterns. Although both tools have been utilised in previous BPD research, the extended duration of this study compared to most others may influence the acceptability and feasibility of these monitoring methods. EMA offers the advantage of providing granular data without recall bias but requires frequent responses from participants. Meta-analytic evidence suggests that an average response rate to EMA prompts of about 80% in BPD cohorts, though none of the included studies were over a month in duration (Davanzo et al., 2023). Given the decrease in response rates between baseline and post-intervention

periods reported in the previous chapter, response fatigue appears to be likely. In contrast to EMA, actigraphy allows for passive data collection, though it requires participants to wear the device almost continuously, possibly causing discomfort or embarrassment since the device is outwardly visible. The increasing popularity of consumer wearables may affect participants' perception of wearing an activity tracker for a research study. Understanding whether these tools are acceptable and appropriate for extended monitoring in BPD may be helpful in determining whether these may be appropriate in non-research contexts, such as for clinical monitoring.

User attrition is a frequent obstacle to digital interventions, typically following a pattern of exponential decay (Eysenbach, 2005; Lakhtakia & Torous, 2022). Factors generally associated with attrition from digital interventions include lack of completion accountability, lack of expectation management, low initial access effort, and poor interface usability (Eysenbach, 2005). Digital interventions also do not provide the same therapeutic alliance as face-to-face care, which may further increase attrition in people with BPD (Meehan, 2007; Spinhoven et al., 2007). Furthermore, both the interventions assessed in the Sleep in BPD study are associated with attrition or poor adherence. Previous trials have noted major attrition from dCBT-I (Freeman, Sheaves, Goodwin, Yu, Nickless, & Harrison, 2017), notably prior to sleep restriction guidance, which is the most effective therapeutic component (Kyle et al., 2023; Maurer et al., 2021). The work reported in this thesis has replicated this pattern in a non-clinical population (Chapter 1) and a small group of women with BPD (Chapter 3). Attrition may be preceded by periods of non-adherence or non-engagement. Little has been published about attrition and non-adherence in BLT, but studies reporting this data suggest that it is considerable, and furthermore that there is no association between self-

reported and actual adherence (Desan et al., 2004; Michalak et al., 2007b). Morning BLT primarily functions by shifting circadian phase earlier, while the primary mechanism of sleep restriction therapy is to use increase homeostatic sleep pressure to override maladaptive physiological arousal preventing sleep. Both these therapies therefore require consistent use to achieve their intended effects, particularly in the acute phase of treatment. Thus, adherence and premature attrition can drastically reduce treatment effect.

Understanding whether participants with BPD are willing to make the time commitments required of these therapies, and what factors impact that decision, is important to optimising the delivery of the interventions in future trials. Chapter 3 identified between-person differences in adherence trajectories over time. Qualitative analysis of PPIE interview data from the feasibility study of dCBT-I and BLT discussed in Chapter 3 provides some insight into the factors which contribute to these adherence trajectories. Participants were invited to share their thoughts or concerns related to the monitoring and intervention components of the study, and the resulting interview data is interpreted in the context of the following research questions.

Objectives

The goal of this analysis was to answer the following research questions:

1. Were the monitoring components (MoodZoom & actigraphs) of the SBPD study acceptable to this group of participants?
2. What factors impacted participants' adherence and engagement with the dCBT-I & BLT interventions? Were any of these factors particularly relevant to the treatment of people with BPD?

5.2 Methods

Design

This is an exploratory qualitative analysis taking an inductive, human-centered design approach to understand participants' unique experiences with the SBPD study monitoring tools and interventions. Melles suggests that key elements of the human-centered design approach are (1) Understanding the unique needs of the intended users, (2) Engaging with stakeholders early in the design process and (3) Understanding interactions between the intervention and other systems (2021). Anyone who engages with the tools or interventions is considered a stakeholder, resulting in a group that includes the intended users, researchers, and members of the care team. The present analysis engages people with BPD, who are both intended users and stakeholders, to determine what they want from a remotely accessible, self-guided intervention and how such an intervention might interact with other areas of their lives, such as their usual care, psychiatric symptoms, sleep, circadian rhythms, and their occupational and familial routines. Understanding these perspectives at this early stage allows for improvements to be made prior to further scientific evaluation, which may affect adherence and even treatment efficacy in future research (Kelders et al., 2012). Following from these principles, the interview schedule was devised by JL and NMM to emphasise the adherence and engagement with interventions as functions of design rather than user characteristics. Individual interviews were chosen over focus groups due to participant confidentiality, although this approach also allowed participants to share their unique needs and concerns without self-imposed need for conformity or fear of judgement from peers.

Participants

Participants (n = 7) are those who chose to participate in an optional interview at their final study visit for the Sleep in BPD Study - Stage 2 (n = 6), or upon early withdrawal (n = 1), if applicable. The full study protocol and their demographic information has been presented in detail in Chapter 3. In brief they were all women aged 20-27 years with a variety of occupational statuses ranging from unemployed to students (undergraduate, postgraduate, and vocational) to professionals. Ethical approval for this study was granted by the NHS South Central Oxford B Research Ethics Committee (ref. no. 18/SC/0366).

Interviews

Participants were interviewed by JL in a private room at the Department of Psychiatry at their final study visit or at their early withdrawal equipment return visit. Written consent to audio-recording of the interview and use of anonymised quotations was sought prior to interviews, which were semi-structured. Interviews began with a brief statement of purpose explaining that their opinions were being collected to inform further explorations of remote and digital interventions for BPD. Then participants were asked structured interview questions, shown below in Table 5.1, responses to which were used as starting points for further inquiry. After each of the structured questions was explored, participants were invited to share or revisit any other ideas.

Table 5.1. Structured interview questions, shown by intervention component to which they relate.

General	a. How would you describe your experience in the study overall? b. Did you have any difficulties during the study? c. Are there any components of the study that you might continue with? d. Is there anything else you've thought of that we haven't discussed yet?
Sleepio	a. Did you prefer to use Sleepio on your phone or on your desktop computer, for your daily sleep logging? b. How did you remember to use Sleepio every day? On a scale of 1 to 5, how easy or difficult was this? c. When you think about what you learned while using Sleepio, what jumps to mind? d. Did you ever have problems using Sleepio? e. Do you think you'll keep tracking your sleep, using Sleepio, sleep diaries or another method, now that you are no longer required to? Why or why not? f. Did you notice any changes in your sleep while you were using Sleepio? These could be positive, negative or neutral changes.
Light Therapy Box (aka 'Light Box')	a. Walk me through your usual morning routine from before you used your Light Box. It's ok if this changed from day to day, you can just tell me about a morning that felt normal to you. b. Walk me through your usual morning routine while you used your Light Box. What did you typically do while you used it? c. How difficult or easy was it to wake up at your assigned time? Did it get easier over time? d. On a scale of 1 to 5, how easy or difficult was it to make time to use your Light Box? How come? e. Would you consider getting a Light Box or similar product to use now that you are done using it for the study? f. While using the light box, did you notice any changes to how you felt during the day or slept at night? These could be positive, negative, or neutral changes. g. Have you noticed any changes since stopping the use of your Light Box?
MoodZoom	a. What was your experience of using MoodZoom twice a day compared to ten times a day? b. Did you notice any differences in your awareness of your own moods when you were using MoodZoom twice a day as opposed to ten times a day? (if relevant) Did you find that helpful?
Actigraph	a. What were your experiences of wearing an actigraph during the study? b. Have you ever worn an activity tracking watch or similar device in the past? Was the actigraph comparable?

Analysis

Interviews were transcribed verbatim by JL and only the transcriptions were used in the analysis for participant privacy. Participants were each assigned a random initial from A to H to distinguish transcripts and quotes for the purposes of analysis and reporting. Braun & Clarke's thematic analysis process was chosen for its clarity, simplicity, and replicability (2006). Accordingly, both JL and SG familiarised themselves with the transcripts and generated inductive codes individually before discussing them collaboratively. Both then returned to the transcripts to generate themes individually which were finalised in collaboration with KEAS. Themes are defined here as concepts relevant to the aforementioned research questions and referenced in multiple utterances. All collaborators were blind to the quantitative study results reported in Chapter 4 until the "Results" section of this qualitative report was written.

5.3 Results

Tolerability of Monitoring Components

MoodZoom

Most participants felt that MoodZoom tracking was a productive exercise that increased self-awareness:

> C: *"Not doing this, I don't really stop and think about how I'm feeling, so it's quite interesting to do that, and then look at what's causing that. Uhm so yeah just tracking, it's quite helpful to kind of keep it in mind, like how you're feeling and why."*
>
> F: *"I liked being more aware and just having that like, thinking space about it."*

Some participants found that the process of tracking changed their understanding of their mood.

> G: *"I'd start to question like am I actually angry or am I just irritable? I found that I was more irritable out of any of the other emotions […] I used to think like oh I'm angry but I'm not really. Like I'm irritable, not angry."*
>
> A: *"It was quite helpful in a way to sort of see as I was rating it, when it was asking "are you anxious" and really reflecting on actually no I wasn't."*

Although tracking was helpful for most participants, they tended to dislike the ten-times daily sampling during the high-intensity recording period and expressed a preference for the lower intensity monitoring:

> B: *"Twice a day check in, it's pretty good but ten times a day I feel like I just constantly have unfinished business."*
>
> C: *"Twice a day I had no problems with."*

One participant also suggested that the second high intensity recording period was less tolerable than the first:

> C: *"It was better the first week, because then I'd got used to sort of two times a day."*

While another found that neither sampling frequency was suitable:

> F: *"There's probably a happy medium somewhere in the middle. Like, 2 I didn't feel was capturing the changes enough. But 10 was obviously like, capturing too much [...] it felt like both like the number of times of day was suboptimal in a way."*

Actigraphy

Most of the participants had no remarks on the experience of wearing an actigraph, aside from commenting on how they forgot that they were wearing it.

> Interviewer: *"Would you say you were kind of able to forget about it or…?"*
> F: *"Entirely, yeah"*
>
> A: *"I've kept it on the whole time [laughs] I didn't find it annoying at all."*

One participant, however, experienced repeated skin irritation from the actigraph which resulted in several removals lasting over 24 hours.

> G: *"That's what I struggled with the most. Like generally speaking, it's not too bad. Like obviously I must have really sensitive skin […] it's happened like 2 or 3 times."*

Factors Affecting Adherence, Engagement & Perception of Interventions

Our analysis identified factors arranged into four themes: i) obstacles to proposed behavioural changes, ii) emotional experiences of users with BPD, iii) the perceived impact of the interventions and iv) the intervention design (Figure 5.1). Each theme includes several subthemes introduced below.

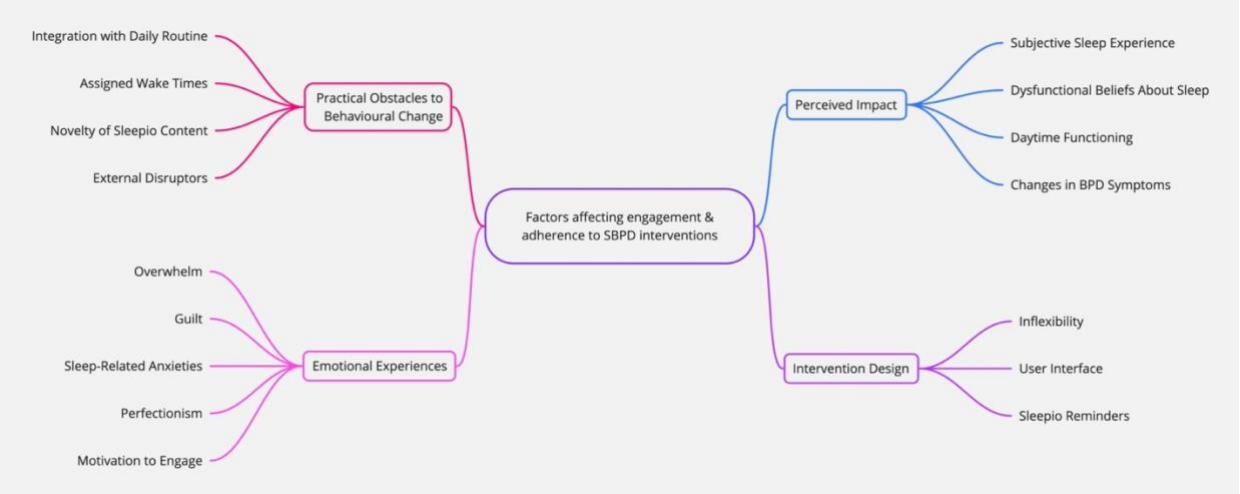

Figure 5.1. Factors impacting participants adherence & engagement with the SBPD interventions, arranged by theme.

Obstacles to Proposed Behavioural Changes

Integration with Daily Routine

Some participants found it straightforward to fit the interventions into their existing routines.

> A: *"I'd normally just have breakfast or something when I was using the light."*
>
> G: *"I would […] just sit like on the bed with it at arm's length in front of me and then I would do my makeup. And then I'd just scroll through my phone."*
>
> C: *"I would get up and use it, and then I would go downstairs for breakfast and coffee and everything."*

One of these participants further noted that she used the interventions concurrently and completed the paper sleep diary during this period:

> C: *"When I would plug the lightbox on, I would use that half hour to fill out all the sleep diaries. I would start with the paper one, cos I didn't get the email about the other- the MoodZoom one- until... I think it was like 8 AM. So, I would just fill out the paper one and the Sleepio one. And then when I had [Sleepio] sessions available, I would do them then."*

Another suggested that differences between working and non-working days changed the way that interventions were integrated in her daily routine:

> D: *"It kind of depended whether I was like working or not. So, when I was working I would kind of do it during my skincare and stuff. But if I wasn't working it was more like I would wake up and then immediately turn it on and sit"*

Other participants reported that finding time to use the intervention was difficult. This was true both as a university student with flexible working hours (redaction for privacy):

> F: *"It was kind of like imposing this artificial schedule on a [student] world in which there is not very much schedule which yeah, didn't work as well as I'd hoped it might."*

And remained difficult later in the study when she was working a more structured job:

> F: *"I've been in different places at different times, and commuting and stuff, it's been really […] So then it's become like an 'oh god, when do I fit this into my schedule?'"*

Assigned Wake Times

Adherence was affected by the difficulty of implementing behavioural changes suggested by the interventions. Many participants found the assigned wake time of the light therapy box to be a limiting factor in adhering to the study protocol.

> G: *"I think it's just the whole waking up so early thing that I felt I struggled with a lot of the time, sticking to that. You'll see a pattern you know, late by 15 minutes."*

> F: *"Really difficult, yeah. Um cos… I really wasn't in a habit of getting up at that sort of time."*

One participant found it so difficult to adhere to the assigned wake time that she chose to wake up, use the light therapy box, and then go back to sleep for several hours until her habitual wake time.

> B: *"I still don't feel like I am emotionally-- or my body is ready to be awake at that time that I was assigned to use it and then just like use it and go onto the day… So I make an effort to go back to my bed and then get out of my bed again."*

Novelty of Sleepio Content

Conversely, participants expressed frustration when faced with behavioural changes that were not novel. Nearly all participants commented on their pre-existing knowledge of the Sleepio content that they encountered:

> F: *"I think a lot of it was kind of stuff that I was familiar with but maybe that's because I have had periods of insomnia before and had done kind of some of the basic reading and a lot of it was at that sort of level […] I don't feel like I really learnt anything."*

> G: *"I do know my bedtime routine and that I should sleep with the lights off, no distractions, that sort of thing. So, it just felt like it was repeating itself over quite logical things."*

> D: *"I think I knew quite a lot of the things it was saying."*

One participant reflected that the useful content was hidden behind early sessions of familiar content.

> H: *"I did find the information useful but also it took a little while to get to the useful stuff. Cos the sleep schedule stuff wasn't till like session four or five […] I know that like exposure stuff usually comes later in CBT but um there wasn't – in the first session there wasn't really anything sort of interesting and then you had to wait a whole week – for the first kind of sort of useful thing I guess."*

For another participant, this familiarity seemed to contribute to her decision to stop engaging with Sleepio, likely before she reached sessions introducing novel content.

> B: *I feel like a lot of the CBT stuff I had already seen before and a lot of content I'd seen before in those printouts for patients, like sleep hygiene and stuff. […]*
> Interviewer: *Did you finish the Sleepio – all six sessions of it?*
> B: *I don't think so actually… yeah I don't think so. Kind of halfway I just like ignore it.*

External Disruptors

Most participants reported changes in their schedule during the study, impacting their engagement with the interventions as well as their rest-activity patterns. In some cases, these disruptors were life events such as beginning full-time work or moving house (redactions for privacy):

> F: *"I was doing a [university course] in the first few weeks of the study, and then I started a full-time job [...] Like a couple of weeks in. So like, that's really disrupted my, like-- that's really changed my work pattern to one that's much more structured."*

> G: *"I had some time off to move houses and just the routine changed up [...] You'll see a little bit of a difference between bedtime and what time I'm sleeping."*

> D: *"I was going to bed earlier anyway because I had placement so I kind of need to go to bed earlier."*

For other participants, minor disruptors such as holidays or morning meetings created schedule irregularities that made it difficult to adhere to the study protocol consistently:

> B: *"There's a confounding variable because I start[ed] going to my professor's office at like 9 or 10 in the morning."*

> F: *"That made it quite hard, in that place, having just got over COVID to re-motivate myself to like, yeah let's get really engaged with this again and like get up at the time, in my like week off over Christmas."*

> A:*" There were a couple of times over the Christmas period where I wasn't home […]I think if I had a structured routine then it was quite easy, I think, but if it was a*

more difficult night's sleep, or if I woke up a bit later than I'd hoped or something like that, then it was a bit harder to like make the time."

Emotional Experiences of Users with BPD

Overwhelm

External factors in conjunction with the demands of the study left some participants feeling overwhelmed, stressed, or anxious.

F: *"It felt quite overwhelming quite quickly"*

B: *"I already have like 10,000 things on my to-do list and now it's another"*

B: *"I usually see the [Sleepio] notifications and with the ePRO and either feel anxious about it or be like oh, I haven't done that and then I would procrastinate."*

Guilt

Non-adherence to study tasks and behavioural recommendations also created feelings of guilt for several participants:

F: *"I started feeling quite guilty quite quickly as soon as I started like 'failing' the stuff they had been setting."*

B: *"I find [sleep restriction] logically helpful but like practically I'm not very good at following that. It makes me feel bad about myself."*

B: *"It makes me feel like oh my god I didn't do it, or I forgot to do it and it just makes me more anxious the next day which means I procrastinate"*

Sleep-Related Anxieties

In addition to stress about the study, one participant also reported nighttime anxiety preexisting her participation in the study:

> G: *"I always get intrusive thoughts like in the night I just start thinking about random horrible stuff."*
>
> G: *"I think it's more of the worry of what I've got to do the next day, and then it makes me wake up and then I question about how long I've got left to sleep."*

Sleep restriction therapy provoked further anxiety for her as she did not trust that she would achieve sufficient sleep duration in her assigned sleep window:

> G: *"Ok, it wants me to go to sleep at this sort of time and wake up at this sort of time, I would just think like that's not enough, because I'm used to either oversleeping or not sleeping, or napping too much."*

Perfectionism

Some participants were preoccupied by potential inaccuracies in their study data, particularly in their sleep diaries and manual activity markers in their actigraphy data.

> F: *"I never really know how long it takes me to fall asleep, because I'm then in the process of trying really hard not to look at my phone in case there's a notification that distracts me. So, it's a lot of guesswork on like those type of questions."*

These inaccuracies provoked anxiety, sometimes to the degree of impairing sleep:

> G: "*I was like worried about when to press the [actigraph] button and when to press it when I wake up. I would sort of then start thinking like am I definitely going to get*

to sleep now that I've pressed it? Like am I gonna fall asleep, am I gonna get up and decide not to sleep?"

B: "*In terms of the sleep diary part […] it makes me a little bit anxious; I feel like when I wake up in the middle of the night I should count and I usually like put it down on my phone like on my notes."*

Lack of Motivation to Engage

Participants reported difficulty remaining motivated to complete study tasks. Lack of motivation arose in response to daily study tasks, resulting in occasional non-adherence:

G: *"[…] certain nights it felt like I couldn't be bothered to open up the app*."

In other cases, the lack of motivation extended to the full experience of the study, leading to non-usage attrition:

F: *"that made it quite hard, in that place, to […] re-motivate myself to like, yeah let's get really engaged with [the study] again"*

One participant compared her experience with Sleepio to past experiences receiving in-person care, reporting that motivation was easier to regulate when a clinician was observing her progress and expressing a desire for someone else to compel her to engage with treatment.

B: *"I think I need someone to be there and force me... Or I just need to better regulate myself, I dunno, I need someone to make me do it."*

Beyond a preference for clinician engagement, she reported a general lack of motivation to engage with digital interventions despite acknowledging clinical evidence supporting digital interventions. She further suggested this was a specific and perhaps unusual trait:

> B: *"App[s] like Calm and all those that have really strong evidence base, I know people really like it but I never find any of those particularly appealing to me or that I want to follow through so I feel like it might just be me like… there might be a subset of people who need something else"*

Perceived Impact of Interventions

Over the course of the study, participants observed changes to their sleep and sleep-related beliefs as well as daytime functioning, and psychiatric symptoms including mood. Perceptions of these changes contributed to attitudes towards the interventions and decisions about continued engagement with them.

Subjective Sleep Experience

Some participants reported more consolidated sleep, particularly fewer and more brief nighttime awakenings.

> D: *"When I was asleep my quality of sleep was a bit better."*

This observation held even in a case when the participant did not fully adhere to the study protocol:

> C: *"I didn't always completely follow [the sleep restriction protocol], but like it did help in kind of reducing the amount of times I'd wake up and like sleep was disturbed."*

One participant attributed her reduction in night-time awakenings to decreased anxiety and rest-activity patterns shifted earlier.

> G: *"So I think it has decreased to, like, I'd wake up two, three times and it used to be more often…* [referring to pre-study] *It's more of the worry of what I've got to do the next day and then it makes me wake up […]I felt sort of more tired […]I don't know if it was because I had gotten up earlier the previous morning or whether it's because I've had a full day of being up since that time and probably going to bed at a bit of a better time."*

Others experienced decreased sleep onset latency:

> C: *"It was a bit easier to get to sleep. I noticed a drastic reduction in the amount of time trying to sleep because I would just fall asleep."* (attributed to the light therapy box)
>
> H: *"The sort of time that it took me to fall asleep like shrunk quite… cos it used to take me about an hour to sort of fall asleep whereas it was like shortening to like less than fifteen minutes which was like really good. So um yeah that was partly helped by like moving my phone away which was the... part of the Sleepio stuff."* (attributed to Sleepio)

Reduced Dysfunctional Beliefs About Sleep

Several participants noticed changes in their beliefs about sleep, particularly to do with normalising poor quality or abbreviated sleep:

> C: *"Accepting things and knowing that it's not gonna be the end of the world and like if you can't stop thoughts of worry over not being able to sleep, you just think well... nothing incredibly bad is going to happen if you don't sleep well."*
>
> A: *"You get the deepest sleep in the first few hours which I didn't really know that, and kind of it was a bit reassuring to know that if I have a bad night's sleep, or if there's a shorter amount of time […] I should still be getting a good sleep in that first few hours, and I quite liked the normalising of that and it calmed me down a bit."*

One noted that completing her sleep diary made her realise her previous misperception of her sleep, particularly that she had been overestimating the duration of her nighttime awakenings:

> B: *"I did realise as I type in the answer from day to day like it's not 5 hours I'm awake, it's like 30 minutes in between; it makes me feel good."*

Another realised that prior to the study, her time in bed window was excessively long.

> G: [post-intervention] *"I don't need to try to get to bed at 8 PM and wake up at 8 AM in order to have a full night's sleep. I think it's more a sense that like I shouldn't be going to bed any earlier than realistically 10 o'clock […] there's no need for me to in bed at that time because there's no need for me to be asleep at that time, really."*

Shortening her sleep window also allowed her to accomplish more each day:

> G: *"Now that I've started getting to bed later, I've noticed actually I can do so much more, I can tidy up, I can do a lot more in the day."*

Daytime Functioning

Several participants attributed changes in their daytime functioning, particularly their morning alertness, to the light therapy box:

> C: *"The first time I switched it on it immediately woke me up."*
>
> D: *"I definitely did feel more alert in the morning."*
>
> H: *"Slightly more energetic in the morning maybe. Maybe like quicker to wake up properly."*

One participant reported increased energy throughout the day:

> A: *"Overall a bit more energy, like for longer throughout the day, if that makes sense. So not necessarily so tired in the afternoon."*

For some, increased alertness improved their mood and perception of the day.

> A: *"I felt alert really quickly with it, and it really improved like my whole morning and my whole day [...] I'm kind of sad that it's ending."*

> G: *"I felt like I was more ready for the day and sort of like… I don't know, I just felt a bit better about the day even if the day was a bit gloomy, and just like a bit rubbish."*

> C: *"I felt less like a zombie doing it."*

One participant; however, reported no change to her daytime functioning during the intervention period:

> F: *"I didn't feel particularly awake after [using the light box] or anything. I would still feel really quite sleepy most of the time after it."*

This was in spite of her hopes for the light therapy box to change her rest-activity patterns.

> F: "*I'd hoped it would make me really a--- you know. Make me really a morning person, and it didn't."*

And ultimately contributed to her decision to stop engaging with the interventions prior to the study's conclusion:

> F: *"I don't feel this has worked well for me at all. To the point that I've given up on it because it really didn't seem to be making a difference."*

Changes in BPD Symptoms

Two participants reported changes to their BPD symptoms during the study. One of whom noticed improvements in her mood above and beyond additional awareness from using MoodZoom:

> Interviewer: *"Do you think there was a difference in your own awareness, or a difference in your actual mood [after tracking with MoodZoom]?"*
>
> A: *"A difference in my actual mood. Yeah, I know what you mean. I definitely noticed that […] my mood did improve quite quickly actually, using the light, which is quite shocking […] And I wasn't going into it thinking like 'Yes, it'll definitely work'."*

The other participant stated reductions in suicidal thoughts and self-harm during the study period, although she acknowledged that improved weather and increased daylight may have also contributed to this change:

> B: "*I didn't have suicidal thoughts for the past month which is good and probably only have one self-harm which is good, pretty much didn't really self-harm. I like feel brighter and happier but again I like the summer so, I feel like the weather might help a little bit, but I do feel happier."*

Intervention Design

Inflexibility

Some participants reported difficulty with the rigidity of assigned wake times for light therapy:

> G: *"It's just the whole waking up so early thing that I felt I struggled with a lot of the time, sticking to that. You'll see a pattern you know, late by 15 minutes. It's such a small, you know time difference but for some reason, the whole 6 o'clock just makes you think oh my god."*

> F: *"I don't know if the inconvenience was so much the lightbox as the getting up if that makes sense. Like working the lightbox in might have been easier if there was like flexibility about what time to use it."*

This participant highlighted the difference in her experience between weekday and weekend mornings as a situation where she would've preferred flexibility:

> F: *"The mornings that I was needing to be up at that sort of time were fine, mornings that I was getting up at that sort of time just for the sake, especially at weekends and stuff, felt like difficult."*

Another common complaint about lack of flexibility in the protocol was the unlocking of Sleepio sessions. Participants were frustrated by the sessions being restricted to exactly one week from completing the previous session, down to the minute.

> G: *"The sessions are locked so if I did it on a Wednesday and then the next Wednesday I didn't get a notification [...] so if I wanted to do it a week later on that same day, I couldn't. I had to wait for that specific hour and that specific minute.*
>
> F: *"Rather than just having a tiny bit of flexibility… it being like to the hour, seven days."*

The mandatory one week break between sessions proved especially frustrating in the circumstances reported below, which resulted in a two-week gap between sessions as the previous session had been closed during the Prof's outro[1]:

---

[1] "The Prof" is the animated, interactive "virtual therapist' who delivers Sleepio content (Espie et al., 2012).

> H: "*I had to wait a whole week which was how I ended up behind, because when he was doing his little goodbye thing I was like "mehh" but then because I hadn't finished the session it then delayed the like release [of the next session].*"

Participants also complained about the lack of control over Sleepio's pacing.

> B: *"I also got really used to watching my lecture like twice the speed so, and I watch some films twice the speed and I can't speed it up and I don't have control over that. And it's kind of slow, it just feels like you're watching a cartoon where the character intentionally speaks slowly to you."*

Multiple participants stated that they would have preferred to read the content rather than watch it:

> F: *"I kind of found that kind of annoying because it was kind of like moving very slowly […] you had to spend half an hour on something I could have read in 5 minutes. It was a bit frustrating."*

> D: *"It could be helpful for people but I just found it a bit annoying [...] when you have to watch all the things […] Yeah if it was just information and you could just kind of flick through."*

Another participant expressed annoyance about not knowing how long the sessions would take ahead of time.

> G: *"Out of everything, [Sleepio] was the thing that bothered me the most. I couldn't quite work out when was the best time to use it because some were like 20 minutes and some were like 40 so I wouldn't know up until it unlocks the session."*

Several others felt that the sessions were too long:

> B: *"I find some of the twenty minute [sessions] too long."*

D: *"The modules—I found it difficult to like sit through and do it all in one go."*
Interviewer: *"And was that just because they were a bit long?"*
D: *"Yeah."*

Interface

Two participants appreciated the Sleepio narrator, "the Prof's", voice.

B: *"[the Prof] ha[s] a very calming voice"*

F: *"It's a bit like David Tennant-esque and like that was actually quite nice. It's a bit like the element that sticks out as a good thing."*

One also remarked on the visual appeal of the app:

B: *"I like the app, it's really cute. And it's not too science... in a way that makes you feel radicalised or pathologised."*

She also suggested that users might benefit from more visible self-monitoring of progress.

B: "*I feel like if people could actually see the progress it might make them feel good."*

Sleepio Reminders

Finally, one participant stated that she would have preferred more frequent reminders to complete the weekly Sleepio sessions:

F: *"[Sleepio] would send you like one reminder to do the session [...] I wouldn't have minded a like "by the way, we told you this morning to do this". Because I think [Sleepio] just sent one, at like whatever the fixed time it reached that week was."*

5.4 Discussion

Participants found the study's remote monitoring components (MoodZoom and actigraphy) were acceptable and beneficial. None of the participants mentioned concerns about data

privacy or surveillance, despite evidence suggesting that suspicion and negative appraisals of trustworthiness are common in BPD (Masland et al., 2020). Both intervention components of the study (digital CBT-I and bright light therapy) exerted considerable demands on participants in terms of time commitment and changes to daily routine. Factors impacting adherence and engagement with these interventions ranged from practical obstacles regarding lifestyle integration and intervention delivery to attitudes towards the interventions, including beliefs about treatment efficacy.

Emotional Responses to Intervention

Some participants expressed concerns about the accuracy of their subjective sleep data, particularly their responses to the daily sleep diary. No similar concerns were reported about accuracy of mood logged via MoodZoom, suggesting that participants might see self-reported sleep data as more objective than mood data, and therefore possibly at greater risk of inaccuracy and perfectionist concerns. This preoccupation may also be due to expected comparisons between actigraphy-derived and self-reported measures, prompting a fear of being seen as mistaken or purposefully untruthful. Sleepio aims to resolve dysfunctional sleep beliefs with a personalised approach that prompts users to help a hypothetical person with insomnia amend their erroneous beliefs (Espie et al., 2012). Discrepancies between passively collected and self-reported data could be used therapeutically in a similar manner, for example to demonstrate to users that sleep latencies are shorter than they may think, or, as one participant in the presented study remarked, that sleep efficiency may be higher than expected. However, care must be taken such that these discrepancies are normalised rather than criticised since people with BPD may be sensitive to criticism (Kopala-Sibley et al., 2012).

Participants also reported feelings of guilt and failure when non-adherent to light therapy, Sleepio, or MoodZoom monitoring. These experiences have been previously reported in studies of active digital interventions (Richards et al., 2018) but were present here even during monitoring (MoodZoom) and passive (bright light therapy) interventions. Guilt related to non-adherence may be especially intense in people with BPD who tend to experience affect intensely and may be prone to self-criticism (Kopala-Sibley et al., 2012; Yen et al., 2002). People with BPD also may be avoidant of negative emotion (Berking et al., 2009), possibly increasing the risk of a brief period of non-adherence snowballing into full attrition from the interventions. Guilt may also have been intensified due to the research context, if participants felt that their non-adherence could hinder research activities; this may be less of a concern in clinical settings. Nonetheless, self-guided or blended-care (partially human supported) interventions for people with BPD should be cautious to avoid provoking guilt surrounding adherence, instead, using positive messaging to reinforce engagement and encouraging participants to think practically about reasons for non-adherence.

Interventions should also avoid prompting all-or-nothing mentalities with engagement, which could dovetail with the dichotomous thinking patterns reported in BPD (Veen & Arntz, 2000). Consistent engagement should be promoted over absolute accuracy of subjective measures, for example by ensuring that reminders employ forgiving and encouraging language. One potential feature promoting regular engagement without provoking guilt is gamification, in which engagement is presented as a challenge of sorts and virtually rewarded, for example through completion of ‘levels’ or collection of badges, trophies,

virtual gardens or the like. User feedback from the development of a different dCBT-I intervention supported the inclusion of gamification elements (Werner-Seidler et al., 2017).

Obstacles to Behavioural Changes

Practical issues for non-adherence included difficulty establishing consistent use of the intervention components, particularly the light therapy boxes. Daily routines in BPD can be irregular, in part due to instability in personal and work routines (Wood et al., 2015), impairing regular morning use of the light box. Some participants reported clashes between their occupational commitments and their assigned light therapy times on working days, and others struggled to wake at their assigned time on free days. Assigned wake times were frequently reported as an obstacle to adherence with several participants suggesting that their initial wake times could have been too early. Although wake times were assigned based on evidence-based recommendations from a large clinical sleep programme (M. Terman et al., 2001), in some cases participants struggled with consistency, likely blunting both the phase-shifting and antidepressant effects of wake-time stabilisation and the bright light therapy (Pail et al., 2011). Previous evidence suggests that bright light therapy delivered more than 9.5 hours after melatonin onset weakens the antidepressant effects (J. S. Terman et al., 2001). However, slightly delayed use is still preferrable to inconsistent use (Wirz-Justice & Terman, 2022). Additional guidance during the first weeks of light therapy might help participants create a realistic plan emphasising consistency. Future research and clinical implementations of these interventions in BPD should account for the likelihood of schedule disruptions and loss to follow-up in this group. The effects of schedule disruptions might be mitigated if study design accounts for them, for example by using portable light therapy devices for longer durations if participants expect to travel frequently.

Several participants highlighted perceived improvements to their sleep or morning alertness as factors encouraging engagement with bright light therapy. The initial phase of treatment could constitute a critical period whereby consistency is necessary to create treatment effect, which then motivates continued engagement. This also aligns with a critical period in habit acquisition (Lally & Gardner, 2013). This period is likely to be challenging for people with BPD, many of whom may exhibit delayed circadian phase and thus experience difficulty waking up in the mornings (Dagan et al., 1998). Indeed, several participants reported that their assigned morning wake times were too early to maintain continuously, especially on free days. For most participants, their assigned wake times slightly truncated the sleep window, effectively introducing a mild version of Sleep Restriction Therapy (SRT) which is also part of the third Sleepio session. However, when SRT is introduced by Sleepio, it is motivated by an explanation of sleep pressure and a discussion of possible side-effects. Ideally, participants would receive this information prior to beginning light therapy. Brief telehealth consultations focused on obstacles to adherence has previously been shown to attenuate treatment failure in CBT-I (Forsell et al., 2019). Given the instability in daily routines in BPD, participants may also have benefitted from a plan to stabilise other circadian zeitgebers during this critical period, particularly mealtimes and social contact (Carney et al., 2006; Monk et al., 2003).

Interface Design Considerations

Beyond increased clinical contact, the Sleepio interface could also offer more flexibility which might reduce frustration and promote engagement with CBT-I. Nearly all participants

reported frustration with the lack of novel content in the first two Sleepio sessions, which also fell in this critical period of habit acquisition. While a basic understanding of sleep, insomnia, and sleep hygiene is essential for implementing the other components of CBT-I, most participants in this study were already familiar with this information, having encountered it through prior clinical care, public health initiatives, or self-study in response to their insomnia symptoms. A possible compromise to avoid boredom while maintaining information exposure would be to use a quiz format to deliver basic sleep and psychoeducation content. Incorrect question responses could prompt further topical content delivery. This approach may be especially appropriate for BPD groups given that most individuals will have previous exposure to CBT principles. Participants also expressed frustrations about inflexible pacing of the intervention, particularly the playback speed of the narration. The prevalence of BPD is highest in young adulthood, a demographic which often elects to consume video media at increased playback speeds (Ominato & Gu, 2023). Doing so does not appear to affect content retention in this age group and may improve attention by reducing mind-wandering (Lang et al., 2020; Murphy et al., 2022, 2023; Ominato & Gu, 2023; Song et al., 2018). However, increased media playback speed may reduce comprehension in older adults (Murphy et al., 2023; Ominato & Gu, 2023). Allowing participants the option of personalising playback speed would have multiple benefits: reducing frustration, improving attention, and possibly shortening the session durations, which was another participant complaint. Multiple participants also reported difficulties with the session unlocking protocol as they expected to be able to complete their weekly Sleepio session on the same day each week but instead had to wait 168 hours (seven full days) from completion of their previous session, often resulting in a drift in their session timing. This

could be resolved by allowing participants to access sessions slightly sooner, perhaps after 144 hours (six days) after their previous session completion.

5.5 Conclusion

This group found passive activity monitoring, active mood monitoring and a dual intervention of dCBT-I and BLT to be largely acceptable but burdensome. Understanding participant experience and adherence patterns is important because both these interventions require consistent use to maintain treatment effect. Participants identified several factors affecting adherence, including perceived treatment efficacy, obstacles to behavioural change, interface design and emotional responses to the interventions. Schedule variability and high likelihood of disruption should be considered when people with BPD begin bright light therapy or sleep restriction therapy. Establishing a consistent wake time and BLT routine may be particularly critical in the first few weeks until circadian phase has shifted enough to facilitate the assigned wake time. Increased clinical support and the opportunity for personalisation of wake times may help mitigate non-adherence during this period (Forsell et al., 2019). Additionally, care should be taken to begin treatment in a period with minimal external disruption (*ie.* avoiding travel, changes in accommodation or personal life, and occupational commitments) such that appropriate routines can be established. Digital user interfaces should incorporate some flexibility to account for previous exposure to content and personal preferences, which may vary depending on age and occupational commitments. Intervention designers and clinicians should take care to avoid triggering feelings of guilt or inadequacy upon non-adherence, as this may result in further attrition. Consistency in daily routine should be emphasised to support stabilisation of rest-activity patterns.

6 Chapter 6: Digital Interventions for Symptoms of Borderline Personality Disorder, a Systematic Review & Meta-Analysis

6.1 Introduction

Digital Interventions for BPD

Currently the demand for BPD treatment vastly outstrips availability, with over 800 people seeking treatment for the condition for every evidence-based care provider in the United Kingdom, and even greater discrepancies internationally (Iliakis et al., 2019a). Furthermore, the cost per patient is estimated at more than double that of depression patients (Iliakis et al., 2019a; van Asselt et al., 2007). Possibly this treatment gap could be reduced by digital interventions, which inform users and support behaviour changes to reduce symptom severity and functional impairment (Barak et al., 2009; Lakhtakia & Torous, 2022). Though few digital interventions target BPD specifically, many address transdiagnostic symptoms of the disorder and thus may lend themselves to BPD treatment given the heterogeneity of symptom presentation. The dimensional model of personality disorder suggests that the disorder does not present a singular phenotype but rather as functional impairment co-occurring with a combination of pathological personality traits (Krueger & Markon, 2014). The combination of personality traits may vary between individuals, resulting in differing symptom presentations. Single-symptom interventions may therefore appropriately address only the relevant areas of impairment. For example, many digital interventions target emotion regulation, which is a central symptom in BPD but also mood, substance, and eating disorders (Sloan et al., 2017). People with BPD tend to use less effective emotion regulation strategies than healthy controls, contributing to mood instability in the disorder (Daros &

Williams, 2019). Poor emotion regulation is associated with behavioral control and drives maladaptive behaviours such as impulsive anger and NSSI, which are also symptoms of BPD (Axelrod et al., 2011; Briones-Buixassa et al., 2021; Neacsiu et al., 2010). Paranoia is another transdiagnostic symptom which can be treated effectively, although few interventions focus specifically on paranoia in BPD (Freeman, 2016).

Symptom-based care also provides treatment naïve to diagnosis which could reduce pathologisation, or unnecessary designation of illness, in people diagnosed with BPD, many of whom object to the label of personality disorder but acknowledge their experience of its symptoms (Stalker et al., 2005; Tedesco et al., 2024). Digital interventions offer advantages such as access without diagnosis, lower cost, continuous availability, and improved geographic accessibility, all of which are challenges for services offering specialised care for borderline personality disorder. Despite concerns about limited clinician contact, self-guided digital interventions can be as effective as traditional face-to-face alternatives (Cuijpers et al., 2010). However, several challenges remain, such as non-adherence, limited assessment of efficacy in controlled trials, and poor user experience (Eysenbach, 2005; M. E. Larsen et al., 2019).

Persuasive System Design

Non-adherence to treatment programmes is frequently cited as the primary obstacle to efficacy of digital interventions supporting behaviour change, including interventions for mental health problems (Baumel & Yom-Tov, 2018; Lakhtakia & Torous, 2022). In the context of digital health interventions, non-adherence is the failure to engage with the treatment programme at the intended frequency, which may ultimately lead to attrition from

the programme entirely. Oinas-Kukkonen and Harjumaa (2009) proposed a framework called Persuasive System Design (PSD) which can be used to identify and analyse elements of digital interventions that best support behaviour change. Systematic reviews of digital intervention studies suggests that persuasive system design elements, outlined in Table 6.1, contribute to increased adherence (Baumel & Yom-Tov, 2018; Kelders et al., 2012) and treatment efficacy (Orji & Moffatt, 2018). For example, reminders to engage with the intervention and opportunities to rehearse new skills may help users reach their treatment goals by maintaining their adherence to behavioural and cognitive interventions. Various elements of PSD are thought to have distinct effects: primary task reduction aims to reduce goal behaviours into smaller, manageable changes while dialogue support pertains to the interface between the user and the intervention, including features such as messages and reminders to the user, rewards, and the visual appeal of the system (Kelders et al., 2012). Persuasive system may also employ social elements encouraging collaboration or competition between users. Research on adherence typically compares characteristics between adherers and non-adherers, thereby putting the burden of adherence onto the user (Wildeboer et al., 2016). However, given the evidence for PSD, adherence may be partially a product of the interaction between user and intervention rather than merely a reflection of users' characteristics.

Existing reviews of digital interventions for BPD have not thoroughly considered the implementation and user experience of digital interventions, nor have they determined treatment effects for individual symptoms of BPD. Frias et al. conducted a scoping review of 15 studies of digital interventions in participants with BPD, which were primarily adjunctive to dialectical behaviour therapy (DBT) but did not calculate their treatment effect (Frías et al.,

2020). A meta-analysis of smartphone applications for BPD-related symptoms did not find a significant treatment effect. However, this meta-analysis pooled outcome measures across symptoms, potentially obscuring item-level changes for individual BPD symptoms (Ilagan et al., 2020). Other reviews have focused on user experience of these interventions, with one scoping review of eight interventions, primarily DBT tools, reporting positive user feedback while another found serious issues with functionality and interface design, such the exclusion of DBT diary cards, technical issues with enrolment, and difficulties with navigating the interface (Michaels et al., 2021; van der Boom et al., 2022). The current review provides a comprehensive evaluation of digital interventions which target both specific and transdiagnostic symptoms of BPD. Meta-analytic treatment effects for individual symptom measures are examined, along with an analysis of how user experience features and PSD elements influence treatment efficacy. Identifying characteristics associated with treatment efficacy may help optimize the design of future digital psychological interventions.

Objectives

Accordingly, this review aims:

1) To identify and describe the therapeutic approach, duration, frequency of use and user interface of digital interventions treating BPD symptoms in community and secondary care settings
2) To conduct a meta-analysis to determine the efficacy of digital interventions compared to active or passive control groups

3) To determine using subgroup analysis whether there were associations between treatment efficacy and characteristics of the intervention, such as the therapeutic modality, the degree of human support and the use of PSD elements.

6.2 Methods

Search Strategy

OVID Embase, Medline, and PsycINFO, and the Cochrane Central Register for Controlled Trials were searched for literature. The search strategy, further discussed in the first section of the supplemental material, was developed in consultation with co-author EH and consisted of three themes: the core symptoms of BPD, digital health, and intervention studies (*OVID Embase Search Strategy 2022*, n.d.). The digital health theme was based on a National Institute for Health and Care Excellence (NICE) validated filter for healthcare applications with additional terms added for web-based interventions (Ayiku et al., 2021). Automatic filters were used to remove non-English language studies and those published before the year 2000, since the technology of interest did not exist prior. The search was first executed on 19 July 2022 and was re-executed on 28 February 2023 to check for new literature. Further, we performed forward reference checking on the included publications to enhance the literature search.

Eligibility Criteria

All records were screened by co-authors JL and TH independently, and disagreements were settled in consultation with NMM. Rayyan was used to manage the screening process (www.rayyan.ai). Studies were eligible if they were peer-reviewed, published in the English language after the year 2000, and assessed an automated digital intervention with a treatment

target of BPD or one of its symptoms. “Automated” here indicates an intervention for which content is primarily delivered without human support. Given the possibility of preliminary studies in this area, both RCTs and non-randomised studies were eligible for inclusion in the descriptive analysis. All participants were aged ≥18 years and recruited either as healthy volunteers or based on a BPD diagnosis or its common comorbidities of depressive disorders, anxiety disorders, substance abuse disorders, posttraumatic stress disorder or complex posttraumatic stress disorder. Studies drawing on community samples were included since they tended to recruit participants with mild to moderate symptom severity, which corresponded with the degree of BPD symptom severity that would be reasonable to treat with an automated digital intervention. This aligns with NICE guidelines for digital interventions suggesting professional oversight should increase proportionally with clinical risk (National Institute for Health and Care Excellence, 2019). Exclusion criteria were: a) interventions without a primary digital component, b) non-automated interventions (e.g. therapist-centered treatment via telehealth), c) interventions requiring equipment inaccessible to the public (e.g. fMRI or professional-caliber virtual/augmented reality), d) participants recruited based on a mental health or neurodevelopmental disorder not mentioned in the inclusion criteria, or e) studies focusing on participants belonging to the following special groups: combat veterans, displaced persons, or law enforcement officers. These groups were excluded because their life experiences of these participants would be unlikely to be representative of a community sample of people with BPD.

Quality Assessment

The risk of bias was assessed with Cochrane's ROB2 for randomised controlled trials including cluster-randomised trials and the National Institute for Health's Quality Assessment for Pre-Post Studies for all single-arm studies (NHLBI, 2021; Sterne et al., 2019). All studies were separately evaluated by co-authors JL and TH, and discrepancies were settled by consensus.

Extracted Data

The following features were extracted for each intervention: method of delivery, therapeutic approach, overall intended treatment duration and frequency of use, degree of human support and any persuasive system design elements present. Data were extracted from the published literature and, where possible, supplemented by direct access to publicly available interventions and interviews with authors. A reliability check of the coding of PSD elements was carried out in a random sample of 5 papers, resulting in 91% interrater reliability. Therapeutic approaches were coded individually for the purposes of describing each study. To facilitate the subgroup analysis, therapeutic approaches were then classified into evidence-based treatments (EBTs) and non-EBTs. EBTs comprised dialectical behaviour therapy (DBT), cognitive behavioural therapy (CBT), mentalization-based treatment (MBT), acceptance & commitment therapy (ACT), schema therapy (ST), and transference-focused therapy (TFT). Degree of human support of the intervention was binary coded for either full automation or some degree of support based on the descriptions provided in the publications. Facilitated interventions ranged from technical support to therapeutic support to adjunct to in-person care.

Persuasive System Design

Table 6.1 below shows the coding scheme used for persuasive system design elements, adapted from Kelders et al. (2012) which itself was based on a framework developed by Oinas-Kukkonen and Harjumaa (2009). All interventions were coded dichotomously indicating the presence or absence of primary task, dialogue, and social support elements. In cases where the presence of PSD elements was unclear in the publication, we contacted the authors and/or accessed the intervention directly. Failing these means, the element was coded as absent.

Effect Sizes & Meta-Analysis

Treatment effects for the randomised controlled trials (RCTs) were calculated using between-group standardised mean differences at the study endpoint, with Hedges' small sample correction applied (Hedges, 1981). Hedges' *g* values of 0.0 – 0.2 were interpreted as small, 0.21 – 0.8 as moderate and 0.81 – 1.00 as large. Outcome data were extracted directly from the publications or requested from the study lead author. In cases (n = 2) where the authors did not reply or were unable to provide the requested data, the study was excluded from the meta-analysis (Drabu et al., 2022; Hasking et al., 2022).

Six separate meta-analyses were used to assess the effect of treatment for which there were at least two studies reporting these as outcomes. These involved the following outcomes: BPD symptom severity assessed via the Zanarini Rating Scale for Borderline Personality Disorder (ZAN-BPD, Zanarini, 2003) and Borderline Personality Disorder Severity Index (BPDSI, Arntz et al., 2003); suicidal ideation via the Beck Scale for Suicidal Ideation (BSSI) (Beck et al., 1979), Suicidal Ideation Attributes Scale (SIDAS, Van Spijker et al., 2014), Self-

Injurious Thoughts and Behaviours Interview – Suicidal Ideation (SITBI-SI, Nock et al., 2007), Columbia-Suicide Severity Rating Scale (C-SSRS, Posner et al., 2011), Suicidal Behaviours Questionnaire (SBQ, Osman et al., 2001) & Suicide Status Form (SSF, Conrad et al., 2009); non-suicidal self-injury via the Self-Injurious Thoughts and Behaviours Interview – Non-Suicidal Self-Injury Episodes (SITBI-NSSI, Nock et al., 2007); paranoia via the Paranoia Scale (PS, Fenigstein & Vanable, 1992) and Adapted Paranoia Checklist (APC, Schlier et al., 2016), and anger via the Modified Overt Aggression Scale (MOAS, Sorgi et al., 1991)& Trait Anger Scale (TAS, Spielberger, 2010). Since emotion regulation is considered a central mechanism in BPD, a meta-analyses was also conducted with the following outcome measures: the Difficulties with Emotion Regulation Scale in both long and short forms (DERS, DERS-SF) (Kaufman et al., 2016) & the German version of the Emotion Regulation Skills Questionnaire (SEK-27, Berking & Znoj, 2008). Following Cochrane Collaboration recommended procedures, in cases where multiple different outcome measures of the same symptom were used in a single study, the effect sizes of each measure were calculated and then aggregated before meta-analysis using the Borenstein et al. approach (Borenstein et al., 2021; Del Re & Hoyt, 2014; Higgins et al., 2019; Hoyt & Del Re, 2018).

Random-effects models were chosen over fixed-effects as considerable between-study heterogeneity was expected due to differences in control arms (Hedges & Vevea, 1998). I^2 was used to estimate between-study heterogeneity and restricted maximum likelihood (REML) was used to estimate heterogeneity variance τ^2 (Veroniki et al., 2016). The Knapp-Hartung adjustment was applied, given the small number of studies in some of the meta-analyses (IntHout et al., 2014). Funnel plots were visually inspected for risk of publication

bias. Egger's tests were conducted in meta-analyses with sufficient studies (Egger et al., 1997).

Subgroup Analysis

Following recommendations from Schwarzer (2015) and Fu (2011), in meta-analyses with ≥ 10 studies, subgroup analyses were conducted to determine the effects of specific intervention elements provided there were a minimum of four studies per subgroup. Intervention features assessed using subgroup analysis included persuasive system design elements, degree of human support, and use of EBT vs. non-EBT interventions. τ^2 was pooled across subgroups due to the small number of studies in some subgroups (Borenstein et al., 2021). Cochran's *Q* was used to evaluate significance in subgroup differences with $P < 0.1$ considered significant for the *Q*-test due to low event rates (Deeks et al., 2022; Harrer et al., 2021; West et al., 2010). The Benjamini-Hochberg false discovery rate was used to correct for multiple comparisons (Benjamini & Hochberg, 1995).

All meta-analytic calculations were made using the *R meta* v.6.2.1*, dmetar* v.0.1.0 and *Mad* v.0.8-3 packages (Del Re & Hoyt, 2014; Harrer et al., 2021; Schwarzer et al., 2015).

Registration

This systematic review was preregistered with PROSPERO (ID: CRD42022358270). Some deviations were made from the registered protocol: firstly, grey literature was not included. This decision was made upon examination of the grey literature because we felt that there was a risk of reducing the average quality of the included studies and increasing their overall risk of bias. Furthermore, most of the grey literature was not peer-reviewed and thus did not meet inclusion criteria for this review. Adherence was not used as an outcome measure of the

review because only 4 (9%) of the included publications described adherence as the number of participants who completed the intervention. Other adherence metrics were reported but these were inconsistent and could not be organized into a meaningful measure. Finally, some studies listed multiple outcome measures without identifying a primary measure. For the purposes of this review, all studies reporting at least one appropriate outcome measure were included. Studies of interventions with multiple treatment targets and distinct outcome measures were eligible to be included in multiple syntheses. For example, some interventions addressed both suicidal ideation and non-suicidal self-injury and thus are included in both syntheses.

6.3 Results

Search Results

Figure 6.1 illustrates the PRISMA flowchart of the initial and repeated searches. The first search, executed 19 July 2022, returned 7,531 records, of which 1,645 were duplicates. A further 428 were automatically removed due to their year of publication (before 2000) or initial publication in a language other than English. Of the remaining records, 69 were retrieved for full-text screening and 36 of these were included. A repeated search before analysis with publication dates restricted from the initial search date to 28 Feb 2023 returned 989 records, of which 665 were duplicates. The remaining 324 records were screened, 22 were selected for full-text screening, and 4 of those were included. The details of all included studies are provided in Tables 6A.1, 6A.2 and 6A.4-6A.6 in the Appendix, Section VI.

Search Outcomes

The most common reason for exclusion was an inappropriate outcome measure, including unvalidated measures and subscales. Other reasons for exclusion were lack of peer review, enrollment of participants <18 years of age, interventions that were delivered in-person or via telehealth meeting software, and research about combat veterans. Forty reports met the eligibility requirements of this review, one of which detailed three studies in the same report, resulting in a total of 42 unique studies screened in. Across these studies, 38 unique digital interventions were assessed and discussed in this review. Some studies assessed the same intervention, for example, translations of *Living with Deadly Thoughts* into multiple languages (De Jaegere et al., 2019; Eylem et al., 2021; Mühlmann et al., 2021; B. A. van Spijker et al., 2018; B. A. J. van Spijker et al., 2014). One study had three arms, each assigned a different intervention (Hooley et al., 2018). In total, 38 unique interventions are discussed in this review. Of the 42 studies included in the review, 32 were randomised controlled trials, 8 were single-arm (pre-post), one was an open-label trial, and one was a cluster-randomised controlled trial. Four studies reported BPD symptoms or key mechanisms as outcome measures, 20 reported suicidal ideation and/or suicidal thinking, five studies each reported paranoia and non-suicidal self-injury (NSSI), four reported emotion regulation, and four reported anger and/or hostility measures. Some interventions listed multiple outcome measures without indicating a primary. No interventions were identified targeting identity disturbance, impulsivity, feelings of emptiness, fears of abandonment, or relationship instability. Participants were recruited based on a variety of conditions: 51% of the 6,611 were recruited based on suicidal ideation, 10% based on NSSI, 6% on a diagnosis of BPD, 5% on trait anger or hostility and 3% on paranoia. Nineteen percent of participants were

recruited without specific baseline symptoms and 6% were recruited to studies with multiple possible symptom thresholds (*eg.* they met symptom thresholds for either suicidal ideation or NSSI).

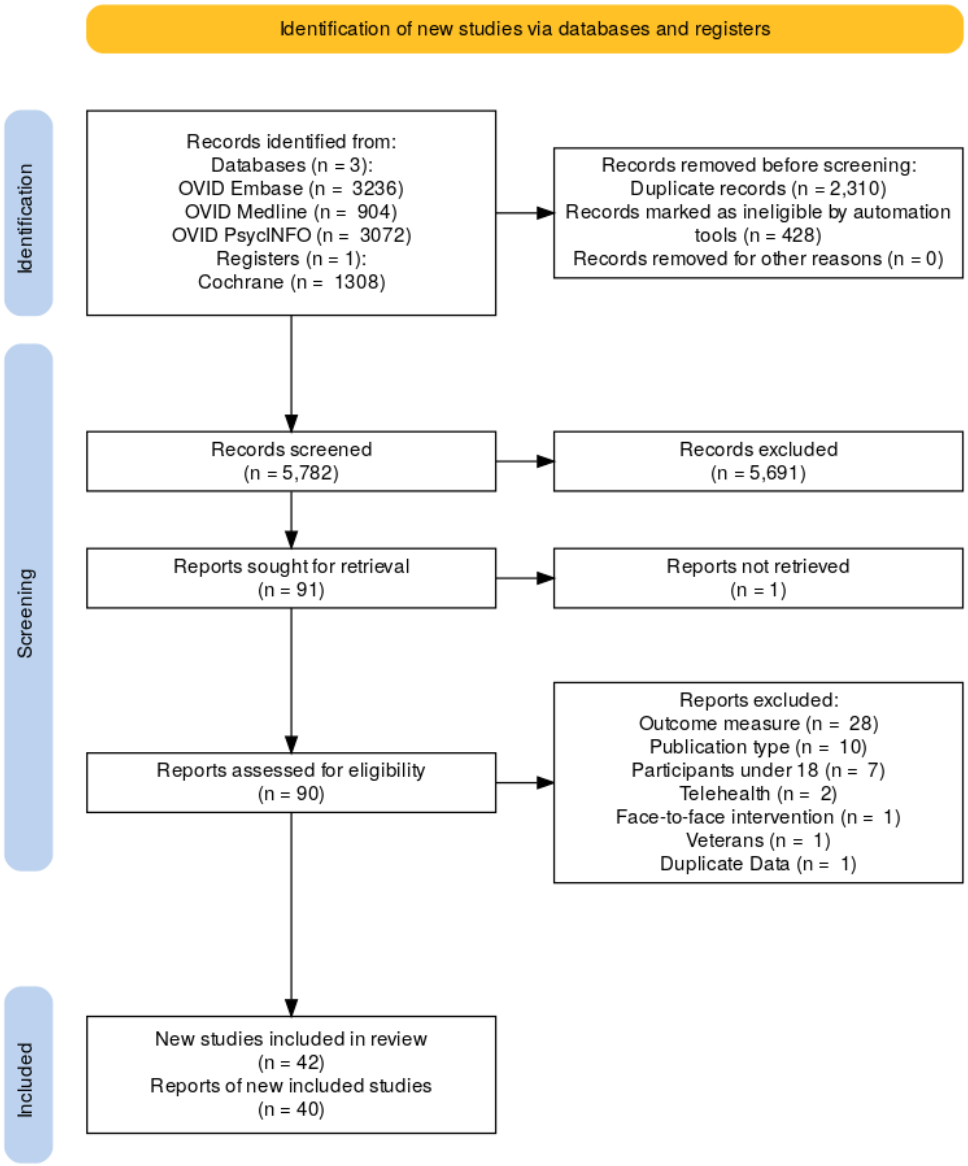

Figure 6.1. PRISMA flowchart showing the search & study selection process. For simplicity, the initial search and repeat search have been collapsed into a single diagram.

Risk of Bias

The results of the ROB 2.0 and NIH Quality Assessment for each study can be found in the Appendix, Section VI, Figures 6A.7-6A.11. In brief, there was a widespread risk of bias; specific results for each study design are discussed below.

Randomised Controlled Trials (RCTs)

There was widespread risk of bias amongst RCTs, with only 3 (9%) judged to have overall low risk, 22 (67%) for which authors had some concerns, and 8 (24%) at high risk of bias. Most of this risk arose in Domain 5: Bias in selection of the reported result, in which 22 (67%) of studies were judged to have some concerns or high risk. The most common reason for these concerns were lack of trial registration or registration without a pre-specified analysis plan. There were some concerns about bias due to deviations from the intended interventions (10 studies, 30%) and missing data (11 studies, 33%). However, very few studies were deemed at risk due to the randomisation process or measurement of the outcome (each 4 studies, 12%).

Cluster-Randomised Study

Some concerns for risk of bias emerged for the solitary cluster-randomised controlled trial, primarily due to the lack of a pre-registered analysis plan (Rodante et al., 2022). Other concerns arose because it was not specified whether the randomisation allocation sequence was concealed until allocation. Also, as with most studies of psychological interventions, some degree of risk arose due to participants not being blinded to their allocation and effectively acted as their own assessors, since outcome measures are self-reported.

Single-Arm & Open-Label Studies

These studies also carried considerable risk of bias, with just 1 (11%) deemed "Good" using the NIH Quality Assessment tool. All the studies were at risk of bias from Domain 8: Assessor Blinding, which as discussed above is difficult to avoid with psychological interventions. Most studies (8 studies, 89%) also failed each of Domain 9: Loss to Follow-Up and Domain 11, which rewards studies that took multiple baseline and follow-up measures of the primary outcome variable. We deemed Domain 11 to be overly stringent for our assessment since most of the studies in this group were pilot or exploratory work.

Intervention Features & System Design

The included studies were primarily conducted in the United States of America (n = 16), the United Kingdom (n = 6), or Australia (n = 5). The methods of intervention delivery included websites (n=28), mobile phone applications (n = 10), both (n = 3), and email (n = 2). The most common therapeutic approaches employed were based on DBT (n = 8), cognitive behavioural therapy (CBT, n = 6), both CBT & DBT (n = 5), mindfulness (n = 3), and acceptance and commitment therapy (ACT, n = 2). Nineteen interventions employed other or unspecified therapeutic approaches.

The mean course of treatment, unweighted by the number of participants, was 55 days (SD = 75), with a mean unweighted recommended frequency of use of 5.0 times per week (SD = 2.7). This frequency calculation excludes 11 (26%) interventions with open dosage or intended frequency of use not stated, as well as 4 (10%) single-session interventions.

Explanations for each PSD element and examples of relevant interventions are given in Table 6.1, and the frequency of PSD elements employed in each of the digital interventions is illustrated in Figure 6.2. The presence of PSD elements varied from *suggestion,* employed in 36 (86%) of the interventions to *praise*, for which we only found evidence in 4 (10%) of interventions (Figure 6.2). Elements of social support were not analyzed, as "e-motion" was the only intervention which allowed users to interact with one another (Salamin et al., 2019).

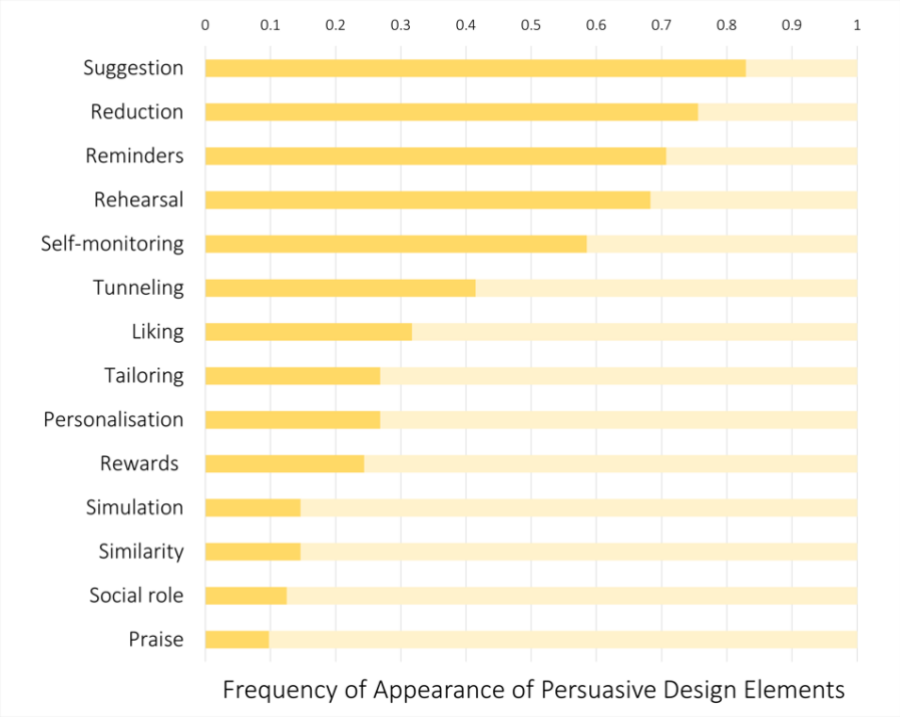

Figure 6.2. Frequency of appearance of persuasive design elements in the 42 included studies. Note: in this review, we coded compensation for participation in a study as a form of reward since compensation likely affected adherence to the intervention in question.

Table 6.1. Coding scheme for elements of persuasive system design which offer primary task and dialogue support. The scheme and table are adapted from Kelders et al., 2012.

Primary Task Support	Concept	Implementation Example
Reduction	Breaking goals down into smaller tasks, for example skills modules, makes them easier to achieve	DBT Coach: Presents the user with a series of tasks depending on the DBT skill being practiced in each session
Tunneling	Guiding the user through tasks in a predefined order reduces mental burden of learning by making it obvious what should be done next	Life Buoy: the user must complete each module in the sequence to unlock the next
Tailoring	The interventions' content and/or user interface is adapted to suit different groups of users. This content-matching means users receive more relevant information or guidance to help them reach their goals.	Man Therapy: external resources signposted depend on the U.S. state in which the user is located
Personalisation	The interventions' content and/or user interface is adapted for each user or provides options that users may choose between. Personalisation may increase engagement by helping the user feel seen.	Be a Mom: intervention uses the names of the user (a mother) and her baby
Self-monitoring	Providing users with either visual indicators of their progress or opportunities for self-reflecting on progress supports their motivation to reach their goals.	mDiary: 10 mood-related variables are logged daily and charts showing change over time are presented to the user.
Simulation	Estimating the cause-and-effect of different behavioural trajectories can motivate users to change or maintain their behaviour.	Sleep Scholar: shares information about the consequences of poor sleep strategies
Rehearsal	Practicing skills in a comfortable environment builds the habit of using them as needed.	*Johnson et al.*: Users are encouraged to practice relaxation skills and create an implementation intention to use their skills
Dialogue Support	**Concept**	**Implementation Example**
Rewards	Incentivizes engagement with the intervention. In this analysis, participant compensation is counted as a reward.	TEC: awards points for each TEC trial that the user completes. In the trial, participant compensation was based on the number of points earned.

Reminders	Prompting users may increase the likelihood of their sustained engagement with the intervention and prevent forgetful non-adherence. Reminders could be related to intervention use or the target behaviour.	priovi: users can choose to register for daily emails and/or SMS messages
Suggestion	Describing and endorsing target behaviour may encourage change in users.	*Bernstein et al.:* throughout the day, the user is prompted to use emotion-regulation skills towards any "current distressing emotions"
Similarity	Users may resonate more with interventions that feature people like the users or environments like their own	FitMindKit: modules are presented by characters who have the same mental health challenges as the user.
Liking	Appealing and/or cohesive user interfaces may promote engagement with the intervention	Life Buoy: features a sailing-themed interface in which each module is represented by an island. The interface was designed in consultation with a lived-experience group.
Social role	Interventions which take on a familiar anthropomorphic role (*eg*. coach, instructor, buddy) may be more naturally adopted by users	FitMindKit: features an expert narrator who takes on the role of instructor

BPD Interventions

i. Study Characteristics

Four studies, including three RCTs, with a BPD psychopathology outcome measure were identified, as outlined in supplementary Table 5.S1 (Jacob et al., 2018; Klein et al., 2021; Laursen et al., 2021; Zanarini et al., 2017). Published between 2017 and 2021, these four studies recruited 376 participants in total, of which 303 (74%) provided follow-up data. Three of the studies had moderate risk of bias (Jacob et al., 2018; Klein et al., 2021; Zanarini et al., 2017) while one was at high risk of bias (Laursen et al., 2021).

ii. RCT Effect Sizes (Post-treatment Standardized Mean Differences, Hedges' *g*)

Data from the three RCTs was pooled, and a random effects model fitted. Figure 6.3 shows a small and non-significant treatment effect for digital interventions on BPD symptoms as a whole: $N_c = 3$, $g = -0.17$, 95% CI: [-0.42; 0.10], $P = 0.11$. The between-study heterogeneity variance was estimated at $\tau^2 = 0.0$, 95% CI: [0.0 – 0.54] and $I^2 = 0\%$, 95% CI: [0.0; 89.6%]. The funnel plot does not show evidence of publication bias (Figure 6.4).

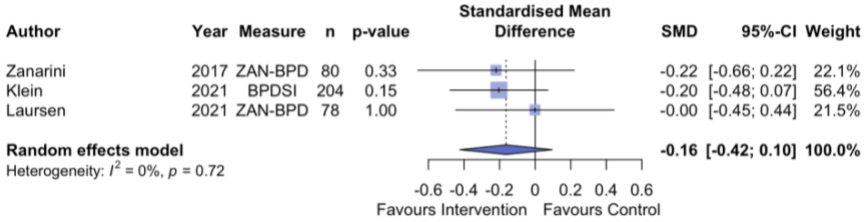

Figure 6.3. Forest plot of treatment effect on BPD symptom severity

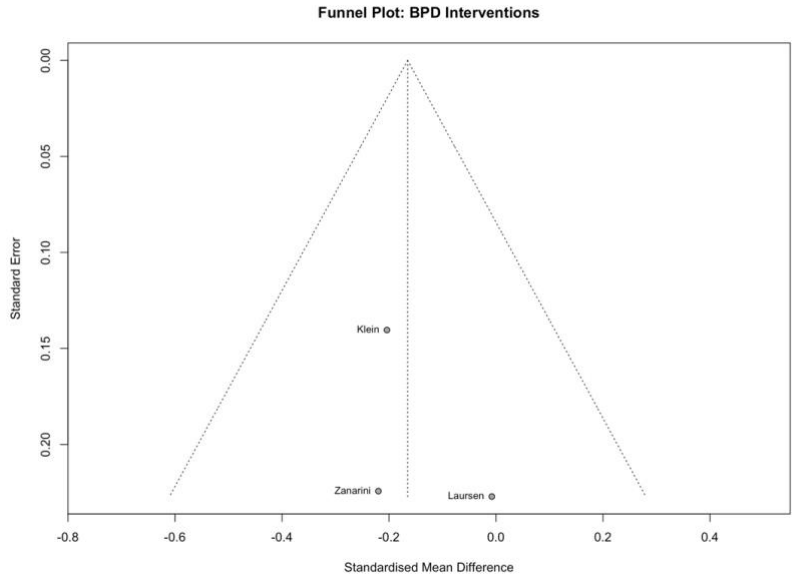

Figure 6.4. Funnel plot shows treatment effect and standard error of interventions treating BPD psychopathology.

Suicidal Ideation Interventions

i. Study Characteristics

Twenty studies (18 RCTs) of interventions for suicidal ideation symptoms were identified, with publication years ranging from 2014 to 2023 and enrolling a total of 3,983 participants (Appendix, Section VI, Table 6A.2). Of those, 2,623 (66%) provided post-treatment data. Two of the studies were assessed at low risk of bias (Tighe et al., 2017; B. A. J. van Spijker et al., 2014), 13 were at moderate risk of bias (Crosby & Witte, 2021; De Jaegere et al., 2019; Depp et al., 2023; Eylem et al., 2021; Franklin et al., 2016; Hooley et al., 2018; Mühlmann et al., 2021; Torok et al., 2022; B. A. van Spijker et al., 2018; Wilks et al., 2018), and five were at high risk of bias (Batterham et al., 2018; Frey et al., 2023; Laursen et al., 2021; O'Toole et al., 2019; Pauwels et al., 2017).

ii. Meta-Analysis

As shown in Figure 6.5, random effects model fitted to the data found that digital interventions elicited a small, significant reduction in suicidal ideation: $N_c = 18$, $g = -0.13$, 95% CI: [-0.25; -0.01], $P = 0.03$. Between-study heterogeneity variance was estimated at $\tau^2 = 0.3$, 95% CI: [0.01; 0.10]. Between-study heterogeneity was estimated at $I^2 = 64.7\%$, 95% CI: [41.6%; 78.6%]. As referenced in the Methods section, effect sizes in studies with multiple outcome measures of suicidal ideation were aggregated prior to meta-analysis. The funnel plot in Figure 6.6 does not show evidence of publication bias, nor did the Egger's test: intercept = 1.33 with 95% CI: [-0.2 to 2.86], $t = 1.70$, $P = 0.11$.

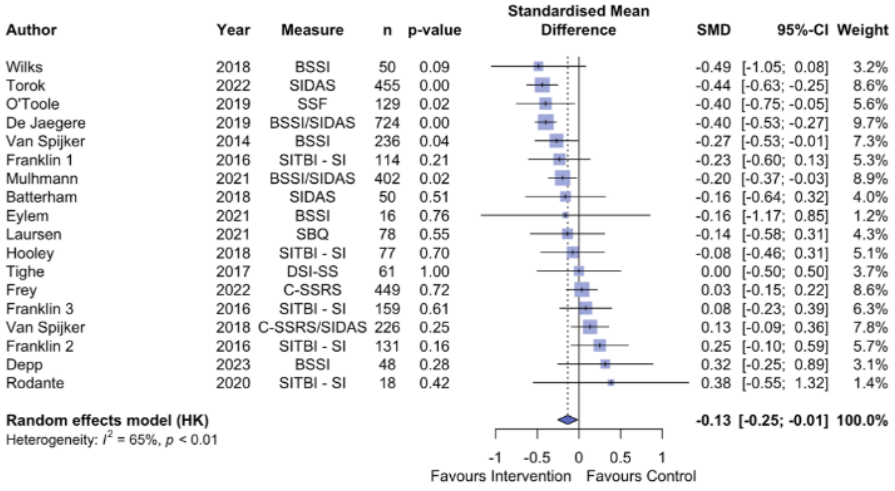

Figure 6.5. Forest plot of treatment effect for suicidal ideation interventions.

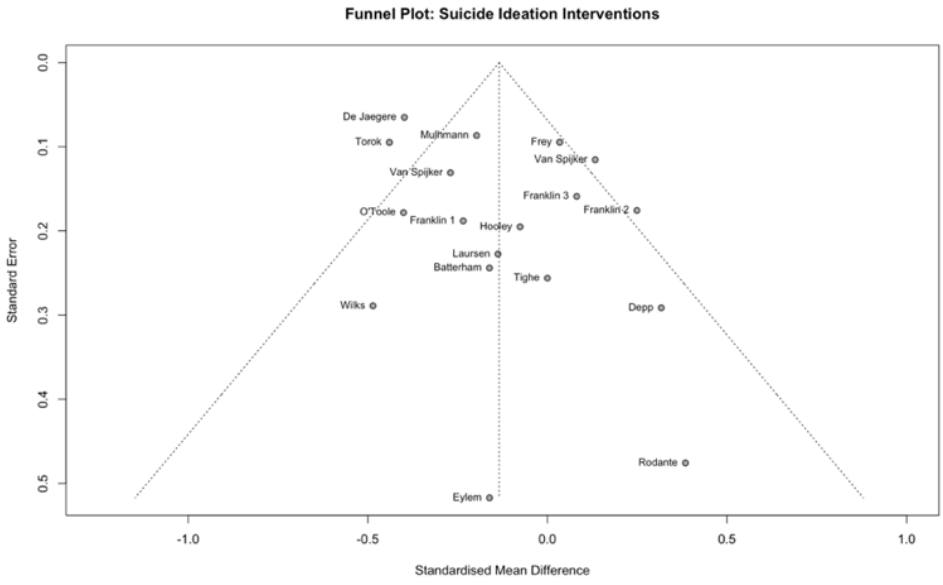

Figure 6.6. Funnel plot of treatment effect and standard error for suicidal ideation interventions.

Subgroup Analysis: Effects of Suicidal Ideation Intervention Features

We first compared interventions targeting suicidal ideation which employed EBTs (n = 12) to those which did not (n = 6). EBT interventions were associated with significantly greater treatment effect than non-EBT interventions (*SMD* = -0.21 vs. 0.00, Cochran's $Q = 4.87$, $P = 0.03$). We then compared suicidal ideation interventions which were fully automated to those which involved at least some degree of human support. There was not a significant difference in treatment effect between these two subgroups: (Automated SMD = -0.09 vs. Facilitated SMD = -0.16, Cochran's $Q = 0.37$, $P = 0.54$). Table 6.2 reports these results in detail, as well as sub-group analysis comparing treatment effect between interventions which did or did not employ each PSD feature. For statistical power reasons, this analysis was only conducted for PSD elements which were present and absent in four or more interventions. Interventions employing reminders, opportunities for self-monitoring, and opportunities for rehearsal were associated with significantly greater reductions in suicidal ideation than those without. No significant effects were identified for other persuasive design elements.

Table 6.2. Difference in treatment effects between interventions employing different therapeutic approaches, degrees of human support and PSD elements.

	n studies	SMD	95% CI	*P*	I^2	95% CI	p^a
Approach							**.03**
EBT	12	-0.21	-0.37 to -0.06	.01	61.4%	27.7 – 79.4%	
Non-EBT	6	0.00	-0.17 to 0.18	.98	0.0%	0 – 74.6%	
Support							.54
Human	12	-0.16	-0.32 to -0.01	.04	52.1%	7.67 – 75.2%	
None	6	-0.09	-0.35 to 0.17	.42	80.1%	56.8 – 90.8%	
Reduction							.18
present	14	-0.18	-0.32 to -0.04	.02	65.3%	33.8 – 80.3%	
absent	4	0.01	-0.31 to 0.34	.90	23.5%	0 – 88.3%	
Tunneling							.29
present	5	-0.26	-0.61 to 0.08	.10	33.0%	0 – 74.6%	
absent	13	-0.10	-0.24 to 0.04	.13	68.0%	43.0 – 82.1%	
Self-Monitoring							**.095**
present	13	-0.20	-0.35 to -0.06	.01	56.1%	24.9 –77.9%	
absent	5	0.01	-0.21 to 0.24	0.87	50.3%	0 – 80.4%	
Rehearsal							**.08**
present	13	-0.21	-0.36 to -0.07	.01	58.2%	22.7 – 77.4%	
absent	5	0.02	-0.19 to 0.23	.80	0%	0 – 79.2%	
Rewards							.21
present	6	-0.01	-0.29 to 0.28	.95	37.4%	0 – 75.1%	
absent	12	-0.19	-0.33 to -0.05	.01	66.8%	39.1 to 81.9%	
Reminders							**.08**
present	13	-0.21	-0.35 to -0.07	.01	58.0%	22.3 – 77.3%	
absent	5	0.03	-0.17 to 0.24	.67	0%	0 – 79.2%	
Suggestion							.18
present	14	-0.18	-0.32 to -0.04	.02	65.3%	38.8 –80.3%	
absent	4	0.01	-0.31 to 0.34	.90	23.5%	0 – 88.3 %	
Liking							.99
present	6	-0.13	-0.39 to 0.12	.23	65.6%	17.6 – 85.7%	
absent	12	-0.13	-0.29 to 0.03	.10	67.1%	39.6 – 82.0%	

[a] *p* indicates significance of differences in SMD between subgroups, corrected for multiple comparisons using the Benjamini-Hochberg method Note: the significance threshold for Cochran's *Q* is *P* <. 10 (Deeks et al., 2022).

Paranoia Interventions

i. Study Characteristics

Five RCTs of digital interventions for paranoia were identified, with publication years ranging from 2017 to 2021, totaling 413 participants enrolled (Appendix Section 6, Table 6A.4). Many of the paranoia interventions were single sessions, so loss to follow-up percentage is not applicable. Three were at moderate risk of bias (Muneghina et al., 2021; Newman-Taylor et al., 2021; Sood & Newman-Taylor, 2020) while two were at high risk (Newman-Taylor et al., 2018; Shore et al., 2018).

ii. Meta-Analysis

A random-effects model fitted to the data found a moderate, significant effect of digital interventions for paranoia: $N_c = 4$, $g = -0.52$ with 95% CI: [-0.86; -0.18], $P = 0.01$. The between-study heterogeneity variance was estimated at $\tau^2 = 0.04$, 95% CI: [0.0; 0.55] and between-study heterogeneity at $I^2 = 51.3\%$ [0.0%; 82.1%] (Figure 6.7). The funnel plot in Figure 6.8 shows possible evidence of publication bias, but there are too few studies to conduct Egger's test.

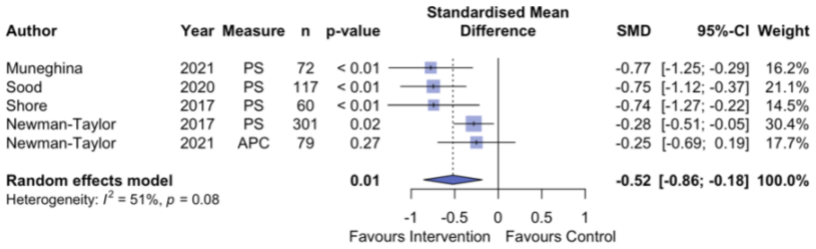

Figure 6.7. Forest plot showing standardised mean differences (SMDs) for interventions targeting paranoia.

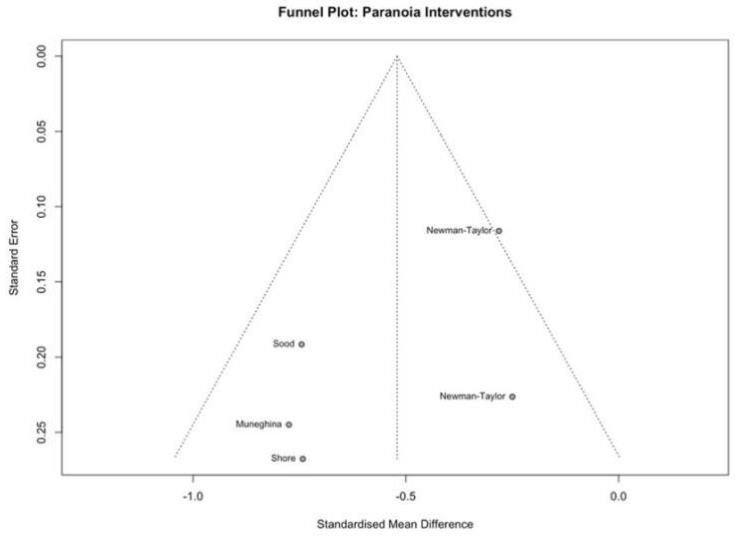

Figure 6.8. Funnel plot of digital interventions targeting paranoia.

Other BPD Symptoms

Meta-analysis of interventions targeting other symptoms (non-suicidal self-injury, emotion regulation and anger) did not suggest a significant treatment effect of digital interventions. Full results meta-analytic results for these interventions can be found in the Appendix, Section 6, Figures 6A.1-6.A6.

6.4 Discussion

Principal Results

The present review suggests that digital interventions may be efficacious in reducing BPD symptoms of paranoia and suicidal ideation. By contrast, digital interventions addressing emotional regulation, anger, and NSSI did not demonstrate a significant treatment effect. Amongst interventions targeting suicidal ideation, those based on evidence-based therapeutic modalities for BPD were associated with greater improvement, but treatment efficacy was not found to be dependent on degree of human support. Importantly, these findings identify which symptoms may be more suitable for support using digital interventions and may be candidates for further investigation and implementation in clinically confirmed BPD, where limited access to in-person therapy and service cost are barriers to receiving timely care.

A secondary objective of this review was to assess the effect of persuasive system design on treatment efficacy. There is overlap between the aims of psychological interventions and the PSD framework: both support change in behaviour and beliefs. At least some PSD element involvement was nearly ubiquitous amongst the studies reviewed; changes to thought and behaviour (*suggestion*) and reduction of these changes into component processes (*reduction*) were employed in 33 (76%) and 36 (86%) of the reviewed interventions, respectively. Importantly, our meta-analytic review of interventions targeting suicidal ideation found that the specific PSD elements *rehearsal, self-monitoring,* and *reminders* were associated with significantly greater treatment effect. Each element may confer different benefits to the design of digital interventions for suicidal ideation and per se parallel the therapeutic components of evidence-based treatments for suicidal ideation:

Rehearsal refers to practicing skills in a comfortable environment with the objective of building the habit of using them as needed. Rehearsing a response to periods of intense suicidal ideation may reduce the duration and risk of these events; this is a component in the Collaborative Assessment and Management of Suicidality approach (Jobes, 2012). Indeed, several evidence-based psychological interventions that address suicidal ideation such as DBT and CBT for suicide prevention (CBT-SP) involve rehearsal of applying the skills learned in therapy in advance of a challenging situation (Stanley et al., 2009; Zalewski et al., 2021).

Reminders are a design element that may help reduce forgetful non-adherence (Alkhaldi et al., 2016). Further work is necessary to understand which types of reminders are most effective for digital interventions. This PSD element is the most likely to be digital-intervention specific with no immediate comparative components in in-person evidence-based treatments, although automated reminders from digital interventions may confer a unique benefit of regular encouragement to practice cognitive skills or maintain behavioural changes.

Self-monitoring in the context of PSD refers to keeping users motivated by encouraging reflection on progress towards goals. Both processes may address feelings of hopelessness and difficulty maintaining a course of action which are associated with suicidal ideation and BPD (Loranger et al., 1997; Witte et al., 2006). Evidence from face-to-face interventions suggests that progress monitoring tends to improve treatment success rates (Shimokawa et al.,

2010) and self-monitoring, often with an emphasis on mood monitoring, is also a core aspect of interventions targeting emotional regulation and suicidality such as DBT and CBT-SP.

In the context of existing therapeutic frameworks, these PSD features could be leveraged in the development of future digital interventions for suicidal ideation for BPD. Due care should be taken in the design of digital mental health interventions; the inclusion of PSD elements is a good starting point but user experience should be evaluated and iterated upon, a process with established merits (Petrie & Bevan, 2009).

Quality of Included Studies

The overall quality of the included studies was moderate, primarily due to a lack of trial pre-registration and to primary outcome measures being self-reported by unblinded participants. The latter is a common source of bias in studies evaluating psychological interventions and may result in artificially inflated treatment effect due to participant expectations. Sham interventions can partially mitigate this and are easily deployed in digital formats, but differing participant expectations between the active and sham arms may still result in bias. RCTs of digital psychological interventions should also measure participant expectations during the study to check for associations between expectations and treatment effect in both arms (Boot et al., 2013). Only eight of the included studies (25% of RCTs) included active comparisons and no studies reported participant expectations (Franklin et al., 2016; Hooley et al., 2018; Klein et al., 2021; Stappenbeck et al., 2021; Torok et al., 2022; B. A. van Spijker et al., 2018). Employing an active control and measure of participant expectation should be

standard in RCTs of digital psychological interventions to improve replicability and make sure that research resources are not wasted.

Adherence

We hypothesised that persuasive design elements support the efficacy of digital interventions by increasing adherence to them: systems that are intuitive and appealing are more likely to be used regularly and thus produce a treatment effect. However, this chain of association could not be tested directly because of inconsistencies in the way that adherence was reported in the included studies. The need for standardised reporting of adherence and attrition in digital intervention studies has been under discussion for nearly two decades and yet continues to hinder research on BPD and beyond (Davanzo et al., 2023; Eysenbach, 2005). A more effective approach to reporting adherence would be to specify the proportion of participants who fully completed the intervention. Additionally, trial registration should include an estimated threshold of engagement beyond which a treatment response is expected (e.g., 10 hours of engagement or six weeks post-initial use), alongside the proportion of participants who reached this threshold. This approach is also applicable to interventions that do not have fixed endpoints.

Implications

As in face-to-face settings, digital EBT interventions for suicidal ideation such as CBT and DBT were more effective than alternative therapeutic approaches (D'Anci et al., 2019). EBTs appear to be effective for reasons which transcend delivery method, perhaps by introducing active components which directly facilitate treatment goals, for example behavioural activation in CBT, rather than general non-directive support (Jacobson & Gortner, 2000).

Despite the demonstrated efficacy of EBTs, some included interventions employed promising alternative therapeutic approaches, such as stimulus pairing or journaling (Franklin et al., 2016; Hooley et al., 2018).

Efficacy of digital intervention may also vary depending on treatment target. Included visualization interventions were effective, at least temporarily, in reducing symptoms of paranoia. Paranoia in BPD is transient, meaning immediate symptom reduction is relevant even if temporary. Recent work with virtual reality in community and psychosis samples has found that compassionate imagery, cognitive therapy, and mental relaxation exercises all appear to be effective in the reduction of paranoia symptoms (P. Brown et al., 2020; Freeman et al., 2023). The commonality between these approaches is reduced anxious cognitions, which likely underpin paranoia and form a suitable target for digital interventions (Contreras et al., 2022; Pauley et al., 2023). All included paranoia interventions were brief (ranging from one session to two weeks). The NICE guidance for BPD advises against brief interventions for BPD partially due to the lack of evidence-based short-term treatments available at that point, and partially due to the possibility of negative reaction upon withdrawal of therapist support (NICE, 2019). The latter issue is less applicable to automated digital interventions, particularly if they remain accessible post-treatment course. Given the efficacy of paranoia interventions in this review, and since brief interventions minimize the risk of user attrition over time, we suggest that further research should evaluate brief digital interventions for symptom relief and teaching specific skills. Such interventions could be deployed in adjunct or anticipation of in-person treatment.

Suicidal ideation also appears feasible as a digital treatment target. For example, *Living with Deadly Thoughts*, a CBT/DBT intervention that has been translated into multiple languages, has been found effective in several large RCTs. Collectively, the studies of *Living with Deadly Thoughts* involved 1,604 participants and contributed 35% of the summary suicidal ideation results. The intervention focuses reducing negative automatic thoughts, which are associated with suicidal ideation through several mediation pathways (Shen et al., 2023). Reduction in negative automatic thoughts after cognitive behavioural therapy is associated with reduced suicidal ideation (Coleman & Casey, 2007), a treatment pathway which appears to have been successfully digitally adapted in this series of interventions. For interventions in both paranoia and suicidal ideation, cognitive components of therapies may be the most suited to digital formats: by reducing anxious cognitions in paranoia and negative automatic thoughts in suicidal ideation.

Unlike paranoia and suicidal ideation, meta-analytic evidence did not show improvement in emotion regulation, anger, or NSSI after digital intervention. DBT, the gold-standard treatment for BPD, has been shown to improve these symptoms likely by improving behavioral control (Axelrod et al., 2011; Neacsiu et al., 2010). Behavioral control supports emotion regulation, including impulsive anger, which is common in BPD, and in turn predicts NSSI behaviours (Briones-Buixassa et al., 2021; Goodman & New, 2000; Neacsiu et al., 2010). DBT typically involves protracted membership in a therapeutic group, through which patients learn to maintain relationships with peers. This process of supervised exposure and interaction with others is not replicable in automated digital interventions but may be

essential to developing behavioral control. As a result, treatment targets of emotion regulation, anger, and NSSI may be less suited to the affordances of digital interventions than targets which can be effectively addressed through cognitive therapies, such as paranoia and suicidal ideation (Freeman, 2016; Hawton & Pirkis, 2024).

Previous research has found that human-supported digital mental health interventions are more effective than fully automated ones (Cuijpers et al., 2010; Werntz et al., 2023). This was not replicated in our subgroup analysis of suicidal ideation interventions; however, human support could deter engagement with suicidal ideation treatments due to stigma and shame, thus reducing treatment efficacy for human-supported interventions (Ebert et al., 2018). This may not be true for other treatment targets; nonetheless, the effects of anonymity and human support should be weighed carefully in the development of digital interventions.

Our results suggest that effective digital interventions focus on building specific skills through transdiagnostic evidence-based approaches or provide symptom management tools (Van Spijker et al., 2014). This review did not find evidence that digital interventions should attempt comprehensive BPD treatment, as these interventions likely require support from a trained clinician and repeated opportunities to build emotion regulation skills amongst peers. The affordances of digital interventions are likely better suited to targeted, single-symptom treatments which could be deployed adjunctively to in-person care. For people with BPD, this type of intervention may be less pathologising as the goal is to treat symptoms of the individual rather than the disorder. The single-symptom approach is increasingly relevant as the diagnosis of personality disorder becomes moves towards a dimensional model (Bach &

Sellbom, 2016). For users with and without personality disorders, the brevity and content relevance of transdiagnostic single-symptom interventions may improve adherence to digital interventions relative to extended courses of comprehensive treatment. Change in single-symptom severity may also be easier to measure, since single-symptom measures provide more granularity than composite diagnostic measures (Lindhiem et al., 2016).

Limitations

There are several limitations to this review, the first being limited applicability of findings across symptoms. For example, the association between PSD elements and treatment effect was only analyzed in interventions for suicidal ideation and may not hold for all treatment targets. Additionally, while the scope of this review includes interventions for BPD symptoms, many of the included studies draw on non-BPD samples. As a result, our findings address the optimization of digital interventions for symptom reduction, rather than BPD psychopathology specifically. These results remain relevant to BPD given that treatment mechanisms in the disorder are poorly understood, and there is little evidence to suggest that efficacy in existing treatments is specific to BPD rather than general reduction in psychiatric distress (Bateman et al., 2015) . This work may also have been restricted by lack of statistical power in some analyses, particularly the smaller meta-analyses of treatment effect and the subgroup analyses (Harrer et al., 2021). Additionally, the binary coding of interventions into human-supported and fully automated subgroups may have obscured some of the effect of human support. For example, interventions which only offered as-needed support were grouped with interventions that included regular support although the former might not

impact treatment effect as much as the latter. All negative subgroup results should be interpreted as an absence of evidence for efficacy rather than evidence of inefficacy. The meta-analyses and subgroup analyses also depend on the quality, completeness, and accuracy of the data in the included studies. As discussed, many studies were at risk of bias and had considerable loss to follow up. Finally, when intervention descriptions were ambiguous or absent and authors did not reply to our queries, PSD elements were coded conservatively. This may have lowered the frequency estimates of PSD elements (Fig. 2) and the accuracy of the sub-group analyses.

6.5 Conclusions

This review has found meta-analytic evidence that digital interventions, particularly cognitive interventions, are effective at reducing the BPD symptoms of paranoia and suicidal ideation. Included studies were at moderate risk of bias; future RCTs of digital psychological interventions should include active control arms and assess participant treatment expectations to better compare the experimental and control arms. The most effective suicidal ideation interventions were those which employed evidence-based therapies and certain features of persuasive system design. Automated digital interventions are unlikely to replace face-to-face care as primary interventions for BPD since they do not facilitate sufficient opportunities to develop emotion regulation skills. However, digital interventions still represent an opportunity for highly accessible treatment of specific BPD symptoms.

6.6 Abbreviations

ACT: acceptance & commitment therapy

APC: Adapted Paranoia Checklist

BPD: borderline personality disorder

BPDSI: Borderline Personality Disorder Severity Index

CBT: cognitive behavioural therapy

CBT-SP: Cognitive Behavioural Therapy for Suicide Prevention

C-SSRS: Columbia-Suicide Severity Rating Scale

DBT: dialectical behaviour therapy

DERS: Difficulties with Emotion Regulation Scale

DERS-SF: Difficulties with Emotion Regulation Scale – Short Form

EBT: evidence-based treatment

MBT: mentalization-based treatment

MOAS: Modified Overt Agression Scale

NICE: National Institute for Health and Care Excellence

NSSI: non-suicidal self-injury

PS: Paranoia Scale

PSD: persuasive system design

RCT: randomised controlled trial

SBQ: Suicidal Behaviours Questionnaire

SEK-27: Emotion Regulation Skills Questionnaire (German version)

SIDAS: Suicidal Ideation Attributes Scale

SITBI-SI: Self-Injurious Thoughts and Behaviours Interview – Non-Suicidal Self-Injury Episodes

SITBI-SI: Self-Injurious Thoughts and Behaviours Interview – Suicidal Ideation

SSF: Suicide Status Form

ST: Schema Therapy

TAS: Trait Anger Scale

TFT: transference-focused therapy

ZAN-BPD: Zanarini Rating Scale for Borderline Personality Disorder

Chapter 7: General Discussion

The overarching objective of this thesis was to understand whether digital interventions may be appropriate for treating sleep and circadian rhythm disturbances in individuals with borderline personality disorder (BPD). This treatment target was motivated by well-documented sleep disruption in BPD (Winsper, Tang, et al., 2017) and preliminary findings of circadian rhythm dysfunction covarying with BPD symptom severity (Carr, Saunders, Tsanas, et al., 2018; McGowan et al., 2020). This thesis contributes to the existing literature by investigating personality-related factors as predisposing factors for sleep problems, phenotyping sleep and circadian rhythm in the disorder, and determining associations between sleep and mood symptoms. This work also motivates the clinical assessment of sleep and circadian rhythms in BPD and identifies specific strategies and intervention features to improve adherence and tailor digital and remote-access sleep and circadian interventions for this group.

7.1 Summary of Findings

Sleep & Circadian Phenotype in BPD

This thesis found evidence for a bidirectional relationship between personality and sleep, with maladaptive personality factors predisposing insomnia (Chapter 2) and sleep disruption exacerbating symptoms of BPD (Chapter 3). The phenotyping study in Chapter 3 also used actigraphy to identify objective sleep difficulties in BPD, including significantly elevated time in bed and nighttime awakening which led to reduced sleep efficiency. These experiences were reflected in subjective measures of insomnia and sleep quality. Collectively, these results provide further validation to the sleep complaints frequently reported by individuals with BPD, consistent with polysomnographic measures of sleep in

BPD (Winsper, Tang, et al., 2017). Nights of low sleep efficiency were associated with increases in following-day instability in positive and negative mood in BPD, highlighting the importance of sleep in emotional regulation. Analysis of non-parametric circadian rhythm did not indicate that the sample of young women with BPD exhibited circadian phase dysfunction relative to their peers, including phase delay, which is inconsistent with the extant literature (Carr, Saunders, Bilderbeck, et al., 2018; McGowan et al., 2019a).

Sleep & Circadian Interventions

The findings of Chapter 3 motivated Chapter 4, which was a feasibility study of a dual-targeted intervention combining digital cognitive behavioural therapy for insomnia (dCBT-I) and bright light therapy (BLT) in individuals with BPD. This study is, to the best of available knowledge, the first reported investigation of CBT-I (whether digital or face-to-face) in BPD. Despite moderate adherence and a small sample size, participants experienced improvements in objective measures of sleep after the intervention period, including improvements in sleep efficiency and significant reductions to sleep onset latency. Additionally, participants reported subjective improvements in insomnia symptoms, sleep quality and depressive symptoms as well as social functioning. Results from this open-label, single-arm study must be interpreted with caution but warrant further study of these interventions in BPD, particularly given their advantages in terms of cost, accessibility, and rapid mechanisms of action relative to psychotherapies recommended for BPD. Several participants withdrew from the study citing burden; these experiences and factors contributing to the moderate adherence to the interventions were explored in qualitative interviews reported in Chapter 5. Participant

experience with the interventions was varied, and factors impacting adherence included perceived treatment efficacy, practical difficulties, negative emotional responses, and frustration with the intervention interface. Specific interface features associated with treatment effect in digital interventions for BPD symptoms were identified in the systematic review in Chapter 6. These included usage reminders, self-monitoring tools, and opportunities for rehearsing desired behaviours. Meta-analytic results suggest that tractable treatment targets for digital intervention include suicidal ideation and symptoms of paranoia; the review did not find evidence in favour of direct digital intervention for comprehensive BPD treatment, emotion regulation, non-suicidal self-injury or anger.

7.2 Implications

There are three overarching implications that can be drawn from this research: i) Sleep disturbance may contribute to mood instability in BPD, ii) Sleep and chronotherapeutic interventions are feasible in BPD and iii) Digital and remote-access interventions can be tailored to better treat symptoms of BPD. These implications are discussed below.

Theoretical Implications

Sleep disturbance & mood instability in BPD

The findings from Chapters 2 and 3 reinforce previous research suggesting that personality factors are associated with sleep disturbance. The differential impact of sleep efficiency on positive and negative mood instability between the BPD and control groups suggests that the

disorder may be associated with increased reactivity of internalising problems in response to sleep disruption. A potential source of sleep disruption is circadian phase delay, which has been previously identified in BPD (Carr, Saunders, Bilderbeck, et al., 2018) and linked to mood instability (Carr, Saunders, Tsanas, et al., 2018; McGowan et al., 2020). Although self-reported chronotype in Stage I of the Sleep in BPD study was significantly delayed in the BPD group relative to the control group, there was no objective evidence of significant phase delay in young women with BPD compared to their peers. Given that previous research reporting phase delay in BPD used samples with wider age ranges, it is possible that circadian phase delay does not differentiate BPD from the wider population until later in adulthood, though further research would be needed to verify this. Possible reasons for this hypothesised persistent phase delay in BPD include reduced entrainment by social zeitgebers, longer circadian period, or abnormal light sensitivity. Overall, the findings of Chapter 3 align with the extant literature implicating sleep disturbance in mood instability (Gillett et al., 2021; Konjarski et al., 2018) but little evidence was found for the hypothesised circadian phenotype in the disorder (Gillett et al., 2021; McGowan & Saunders, 2021).

The BPD cohort studied in Chapter 3 also exhibited important differences in sleep relative to the control group, namely reduced sleep efficiency driven by elevated time in bed and nighttime wakefulness. In the context of the aforementioned associations between sleep efficiency and mood instability, these findings imply that restricting time in bed may be clinically important in the treatment of BPD. Sleep restriction therapy (SRT) is a central component of cognitive behavioural therapy for insomnia (CBT-I) proven to be efficacious as a standalone therapy (Kyle et al., 2023), and should be prioritised when CBT-I is undertaken

in BPD. Scheduling of regular daytime and evening activity may also help reduce time in bed in this group.

Clinical Implications

Feasibility of sleep and chronotherapeutic interventions in BPD

This thesis found evidence of low sleep efficiency associated with mood instability in BPD, underscoring the importance of assessing sleep and rest-activity patterns in clinical care. Identification of insomnia disorder is a major barrier to its treatment (Morin, 2004); this could be achieved using a questionnaire screening tool at regular intervals during routine care for BPD. Actigraphy may also be useful for clinical determination of sleep problems or circadian rhythm disorders. In the near future, data from consumer wearables may also be incorporated in clinical care, though most devices have yet to be validated against polysomnography, and their integration in public healthcare depends on the resolution of issues such as data privacy and financial accessibility (de Zambotti et al., 2024; Depner et al., 2020). This thesis also provides preliminary evidence that digital interventions targeting sleep and rest-activity patterns are feasible and acceptable in BPD, though blended-care interventions including occasional human support may improve adherence.

The motivation for researching digital intervention delivery in BPD arises partly due to a significant gap in available care for the disorder (Iliakis et al., 2019b), which may stem from the resource-intensive nature of existing evidence-based psychotherapies for BPD (King et al., 2018). Sleep interventions are of particular interest in BPD, as sleep disturbance can be measured objectively, unlike most symptoms of the disorder. Sleep and rest-activity patterns are also more modifiable compared to other factors influencing BPD symptom severity, such

as genetic predispositions, adverse life experiences, or socioeconomic status. Moreover, sleep problems are transdiagnostic, meaning they cut across various disorders. This further motivates the development of sleep interventions, since they may benefit individuals with personality disorders broadly rather than exclusively targeting those with BPD. This broad applicability is becoming increasingly important as diagnostic criteria for personality disorders shift towards prioritising dimensional traits over specific disorder labels. Furthermore, transdiagnostic sleep interventions may appeal to patients who are either undiagnosed or opposed to a BPD diagnosis. Finally, if an RCT confirms the antidepressant effects of BLT and dCBT-I as reported in Chapter 4, this would mark a significant advancement in the treatment of BPD as current evidence supporting the antidepressant effects of psychotherapy in BPD is weak (Storebø et al., 2020).

The Chapter 2 analysis of a non-clinical group found no significant impact of personality-related factors on the uptake of digital Cognitive Behavioural Therapy for Insomnia (dCBT-I). Instead, worsened affective and insomnia symptoms were positive predictors of uptake. This aligns with previous research indicating that negative affect (or neuroticism) is the sole personality trait influencing attitudes toward treatment-seeking, as shown in both the PID-5 (Onyeukwu & Donahue, 2024) and the five-factor model of personality (FFM) (Jennings et al., 2017). Given that negative affect and insomnia are common features of BPD, these findings suggest that such experiences may encourage individuals to enrol in dCBT-I. However, further analysis in a clinical group is needed to confirm this.

Qualitative interview of the participants in the Sleep in BPD provided further insight into the acceptability of sleep and chronotherapeutic interventions among individuals with BPD.

Interviews indicated that participants were receptive to the idea of addressing sleep disturbances, and several shared positive reflections on the cognitive and behavioural changes they had made during the study. One factor cited in their decision to adhere or disengage from the interventions was perceived efficacy. In this context, the subjective improvements to sleep reported in Chapter 4 are further indicators of acceptability of dCBT-I and BLT for this group. Some participants noted rapid or immediate treatment responses during the Sleep in BPD study, which is common in both CBT-I (Morin et al., 2014) and BLT (Wirz-Justice & Terman, 2022). This rapid response contrasts with the more gradual improvements typically seen in psychotherapies indicated for BPD, which may be encouraging to participants and position sleep and chronotherapeutic as promising adjuncts to longer-term psychotherapy. Self-monitoring opportunities may capitalise on rapid treatment response: this was a key feature of the dCBT-I intervention assessed in the present research and is also supported by meta-analytic results in Chapter 6.

However, the dual-intervention approach also posed some challenges. Two participants withdrew due to the combined burden of treatment and stressful life events, highlighting the burden of the study. Importantly, the additional monitoring components, such as frequent check-ins and the use of both mobile apps and paper sleep diaries, likely amplified this burden as compared to the hypothetical deployment of dual CBT-I and BLT in a clinical setting. Nonetheless, participants highlighted numerous difficulties with both interventions and addressing these could significantly improve patient experience, adherence, and treatment effect of sleep and chronotherapeutic interventions in BPD.

Tailoring Digital Interventions for BPD

Human Support

Analysis of dCBT-I engagement from Chapter 2 suggests that providing university students with unmonitored, open-access digital interventions for insomnia may result in low adherence and rapid attrition. This raises questions about the amount of therapeutic benefit imparted on users of digital insomnia interventions in this context, which included most of the theorised risk factors for attrition (Eysenbach, 2005): recruitment was untargeted, and the unmonitored intervention was free to access with minimal management of participant expectation. Furthermore, there were no barriers to enrolment (*eg.* cost, travel) or attrition (*eg.* no need to notify the research team or return equipment). These factors may have increased casual enrolment and reduced feelings of accountability amongst participants. The Sleep in BPD study presented in Chapter 4 assessed the same dCBT-I intervention but altered many of these factors, namely assessing the intervention in a targeted group with more barriers to enrolment and attrition, increased management of participant expectation, and human support in the form of the research team, who were in contact with participants via email during the trial. The trial was also bookended by in-person study visits, possibly increasing feelings of observation and accountability amongst participants relative to the fully remote application of dCBT-I reported in Chapter 2. These factors may have contributed to the improved dCBT-I adherence during the Sleep in BPD study as compared to the non-clinical Flourish cohort from Chapter 2.

It is frequently posited that human support imparts increased treatment benefit on users of digital mental health interventions (Lakhtakia & Torous, 2022). However, in clinical trials the effects of human support on these interventions are inconsistent (Renfrew et al., 2021), as

evidenced by the non-significance of subgroup analysis in Chapter 6. This inconsistency may be due to between-study differences in the implementation of human support or in the participants themselves. Renfrew et al. (2021) propose that the degree of human support should be tailored according to user preference; this underscores the importance of determining use patterns for specific interventions.

Interviews with the Sleep in BPD study participants suggested that practical obstacles such as scheduling and travel may considerably reduce adherence to some components of the dual intervention, particularly SRT and consistent use of bright light therapy (BLT). Difficulty with adherence to CBT-I sleep restriction protocols and assigned wake times has been previously noted in patients with depression and attributed to hypersomnia and the tendency to use sleep as a means of avoiding negative emotions (Manber et al., 2011). Given the frequent occurrence of negative mood and comorbid depression in individuals with BPD, similar factors may also hinder their adherence to these protocols. Human support may be beneficial in resolving some of these difficulties. For example, clinicians or facilitators could assist with adjustments to wake times to maintain consistent BLT use despite changing occupational commitments or domiciles, which may be common in BPD. Clinician support may also be invaluable during the introduction to SRT, which is often challenging and unpleasant (Kyle et al., 2011) especially for those struggling with depressive symptoms or lack of motivation, which was frequently reported at interview. The importance of SRT in this cohort was indicated by the excess time in bed identified in Chapter 3.

Mohr et al. (2011) have suggested that the degree of human support should be inversely associated with the intrinsic motivation of users, such that more motivated users require less

support. Qualitative analysis of participant experience found that several participants in Stage II of the Sleep in BPD struggled to maintain motivation during the trial, implying that human support may be beneficial in digital sleep and circadian interventions for people with BPD. This is consistent with a trial of dCBT-I in a sub-clinical group which found that even small (mean = 14 min) increases in human support reduced risk of treatment failure (Forsell et al., 2019). The findings of this thesis, in conjunction with the extant literature, suggest that digital or remote-access sleep and circadian interventions for BPD may benefit from increased human support. Specifically, results from Chapters 4 and 5 imply that the introduction of BLT and SRT may form a critical period for the establishment of consistent adherence and treatment effect, during which time clinician or facilitator support be highly beneficial.

Digital Interfaces

Digital interfaces offer further opportunities for tailoring sleep and circadian interventions to BPD. Feedback from the Sleep in BPD study suggested that participants would benefit from more leniency in session access time, adjustable content playback speed, and alternative methods of progressing through familiar content, such as quizzes. These adaptations are relatively straightforward and centered on the need for increased flexibility, which could improve user experience by reducing frustration with the interventions. Increased interface flexibility would also allow tailoring to individuals not represented in the Sleep in BPD cohort, for example males or older adults who may have different interface preferences. Subgroup analyses reported in Chapter 6 highlight interface features that may promote effective treatment of BPD symptoms. Two of these features, reminders and self-monitoring tools, may assist with maintaining motivation over the treatment course, which was highlighted as a barrier to adherence during participant experience interviews in Chapter 5.

7.3 Future Research Directions

Further Phenotyping of Sleep and Circadian Rhythms in BPD

This thesis has identified several areas for future research to deepen the understanding of sleep and circadian rhythms in BPD. First, the discrepancy between previously reported circadian phase delay in BPD and the results of Chapter 3 warrants further investigation. An epidemiological analysis comparing actigraphy data from a BPD cohort and to a control population spanning from early to late adulthood would help determine whether circadian phase delay is a phenotypic feature of BPD and whether any phase delay relative to controls changes with age. If such a phase delay is identified, investigating possible underlying mechanisms, such as the length of the circadian period and light sensitivity, would provide a more nuanced understanding of the circadian phenotype of BPD. Alternatively, a longitudinal study could be employed to examine changes to circadian profile with relation to age and BPD symptom severity. This would also provide insight into the relationship between BPD remission and changes in sleep or circadian patterns, of interest since many young adults with BPD achieve remission (Gunderson et al., 2011; Zanarini et al., 2003). Finally, impulsivity is another symptom of BPD believed to be linked to circadian disruptions that warrants further investigation (Gillett et al., 2021; McGowan et al., 2020). Any associations between impulsivity and sleep or circadian disturbance would further motivate sleep and chronotherapeutic interventions in this group.

Considerations for Further Study of Sleep Interventions in BPD

Findings of the Chapter 4 feasibility study motivate a randomised controlled trial (RCT) of the combined dCBT-I and BLT intervention to confirm the treatment effects and determine effect size. Researchers should aim to overrecruit for this study given probable increased risk of attrition from research studies in personality disorder compared to other clinical groups (Iliakis et al., 2021), reflected by the high frequency of disruptive life events reported by participants in the Sleep in BPD study. A larger sample size would permit detection of changes to mood instability following sleep intervention.

Recruitment procedures in future studies should be aligned with the proposed dimensional criteria for personality disorder, which emphasise maladaptive personality traits (or dimensions) and severity of impairment instead of specific diagnoses such as borderline personality disorder. Though the consistency, reliability, and predictive power of dimensional personality measures has been demonstrated (Sharp & Miller, 2024), clinical research has yet to pivot towards this new approach and there is currently a deficit of RCTs employing appropriate screening and outcome measures (Zavlis et al., 2025). Most research on sleep in personality disorder focuses on BPD but sleep disturbance is likely to exacerbate transdiagnostic symptoms of personality disorder including emotion regulation, impulsivity, and behavioural inhibition (Van Veen et al., 2017), and thus future clinical studies should consider broadening inclusion criteria instead of relying on categorical diagnoses such as BPD. Furthermore, avoiding the BPD label may be preferrable to participants due to the stigma and reduced quality of physical healthcare associated with the diagnosis (Sanatinia et al., 2015; Stalker et al., 2005; Tedesco et al., 2024). For these reasons, future studies of sleep interventions in BPD community samples should consider recruiting based on self-reported

symptoms, measures of maladaptive personality traits, and screening tools for sleep problems rather than diagnostic status. Beyond recruitment, future studies of sleep or circadian rhythm in personality disorder should also use outcome measures that capture impairment due to generalised personality disorder severity as opposed to measures designed for categorical diagnoses, such as the ZAN-BPD. A dimensional measure of personality disorder severity reflective of Criterion A in the proposed DSM-5 dimensional model of personality disorder would assess impairment in contexts related to both self and social functioning relative to sleep and circadian rhythm disturbance (Krueger & Hobbs, 2020).

Chapter 4 reported significant improvements in subjective sleep experience despite a small sample size, indicating the possible influence of treatment expectation. Future trials of sleep interventions in this group should include active controls. Sham treatments delivered via mobile phone applications or websites, such as imagery visualisation, can be used to control for dCBT-I (Espie et al., 2012) and dim red light therapy can be assigned in place of BLT. While bright enough to be a plausible sham, dim red light does not stimulate intrinsically photosensitive retinal ganglion cells (ipRGCs) to the same extent as bright white light, and thus does not cause the photic entrainment required to shift circadian phase (Rahman et al., 2011). The antidepressant effect of red light is also significantly weaker compared to BLT (Glickman et al., 2006). Measurement of treatment expectations in both the treatment and control arms would also provide increased confidence in treatment effect (Boot et al., 2013).

Ideally, three important measures should also be added to future RCTs: the first is an endocrine measure of circadian phase, which would confirm successful circadian phase shift following BLT, rather than just an alteration of rest-activity patterns. The gold-standard

endocrine measure of circadian phase is dim-light melatonin onset time (DLMO), which is highly accurate but burdensome to participants as it requires remaining in dim lighting and provide multiple saliva or blood samples over the course of an evening and the following morning (Pandi-Perumal et al., 2007). Given the existing burden of BLT and dCBT-I, it may be worth allowing participants to opt-out of DLMO sampling and perhaps to offer additional compensation to those who complete this step. The second measure of interest is adherence to BLT. In the present study, this was estimated using sleep diary reports and light meters in the actigraphs, but a more reliable measure of adherence to BLT would be useful to establish a dose-response for phase shift and antidepressant effects. This would also contribute to a sparse literature on BLT and adherence, which thus far indicates that it is poor (Desan et al., 2004; Michalak et al., 2007b). An improved measure of adherence to BLT might be taken via light sensors worn on the upper body, for example in glasses (Wahl, 2019). However, this may be costly and again contributes to participant burden. A passive alternative would be the inclusion of elapsed time meters in the light therapy devices (Michalak et al., 2007b). This option does not directly measure light exposure and thus is less precise but would still represent an improvement over the protocol of the present study. Finally, a dimensional inventory of personality traits, such as maladaptive variants of the FFM, could be used to identify associations between personality features and specific sleep and circadian variables. This could deepen understanding of the relationship between personality, sleep, and circadian rhythm, and may provide insights to other personality disorders as well as sub-clinical groups.

7.4 Limitations

While specific limitations have been addressed in their respective chapters, several overarching limitations should be acknowledged in this thesis. One major limitation arises from the relatively limited body of research available on digital interventions in BPD. As a result, the quantitative analyses of adherence, efficacy, and interface features presented in Chapters 2 and 6 were based on non-clinical samples. This limits the direct applicability of these findings to individuals with BPD. However, these studies still offer valuable insights for multiple reasons. Firstly, broad patterns of digital intervention engagement, such as attrition, are observed consistently and unlikely to be drastically different in specific groups so long as the intervention and its implementation are held consistent. Furthermore, personality disorder is increasingly understood as an extreme variant of normal personality; therefore, research findings drawn from samples with subclinical symptoms of personality disorder may still provide useful foundations for further exploration in BPD populations.

In the chapters that did focus specifically on BPD samples (Chapters 3, 4, and 5), the sample sizes were often small, which restricted the statistical power of the analyses. This may have prevented the detection of certain associations. For example, daily mood is likely to have some effect on the following night's sleep, but this association was not detected in Chapter 3. The limited sample size presents a barrier to more robust findings and suggests the need for replication with larger cohorts in future research.

Chapters 3 and 4 relied on actigraphy data to draw conclusions surrounding sleep and circadian rhythms in BPD. However, actigraphy data can only directly measure rest-activity patterns which, although commonly used as a proxy, may not be indicative of sleep-wake

patterns. This is particularly true for insomnia populations, in which participants may be immobile but awake for extended periods as they attempt to sleep (Sadeh & Acebo, 2002). It is possible that this phenomenon led to the overestimation of sleep duration and efficiency, and overestimation of sleep latency in Chapters 3 and 4. This would explain the difference in objective and subjective measures of sleep reported in these chapters. In this case reductions in sleep latency after CBT-I or BLT also may not have been captured by actigraphy, which could have been the reason behind the marked improvement in subjective sleep experience reported by participants in Chapter 4.

Another limitation of this research is that neither BLT nor CBT-I were developed for treatment of personality disorder. Although several treatment targets of CBT-I are insomnia-perpetuating behaviours highly relevant to BPD (excess time in bed, irregular rest-activity patterns, anxiety and rumination), the therapy was originally developed for primary insomnia (Morin, 2004). Once efficacy of CBT-I had been established, dCBT-I was developed to increase intervention accessibility and scalability (Ritterband et al., 2009). There are other benefits to digital implementation of CBT-I, such as standardisation of treatment quality and automation of advice requiring calculation, like the recommended sleep window (Espie et al., 2012). However, standardisation and automation inherently reduce the flexibility of the intervention, which emerged as a major theme reducing adherence during participant debrief interviews in the Sleep in BPD study (Chapter 5). Pervasive instability is arguably the primary feature of BPD, as exhibited by EMA mood data (Chapter 3) and changes in domicile and occupation reported at interview by several participants. Treatment rigidity combined with personal instability may have contributed to the feelings of overwhelm reported by participants and ultimately reduced intervention adherence. For these reasons, a

face-to-face sleep intervention might be more appropriate for individuals with BPD than the self-guided programme deployed in Chapter 4, or the blended-care version proposed in Chapter 5. Importantly, to date no studies of face-to-face CBT-I in a BPD group have been published, and this may be an important step in identifying changes necessary to tailor CBT-I and BLT to individuals with BPD.

Finally, the samples used across the studies were all skewed towards individuals assigned female at birth, primarily cisgender women. This is common in BPD research, as in clinical settings the disorder is diagnosed more frequently in women (Silberschmidt et al., 2015). This imbalance limits the generalizability of the findings in this thesis to individuals assigned female at birth. It also reflects a broader issue in psychological research, where gender representation is often unbalanced, leading to a gap in the understanding of psychiatric disorders in males. Future research should prioritize the recruitment of male participants with BPD to determine whether sleep or circadian rhythm is distinctly different from those of women with BPD, as well as evaluate feasibility of sleep interventions in the group.

7.5 Conclusions

This thesis contributes to the growing body of literature pointing towards sleep disruption as a phenotype of BPD and exacerbating factor for mood instability in the disorder, although no evidence was found for circadian rhythm dysfunction or phase delay. These findings also suggest that digital cognitive behavioural therapy for insomnia and bright light therapy are feasible and acceptable to individuals with BPD and may reduce symptoms of insomnia and depression, though a randomised controlled trial is necessary to confirm this. This research motivates clinical assessment and treatment of sleep and rest-activity patterns in people with

BPD. Digital interventions with appropriate treatment targets and interface design may be effective as adjunctive treatments for symptoms of BPD. Future research should continue to refine these interventions, incorporating user feedback and addressing the unique challenges posed by this complex disorder.

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Appendix

Section II: Longitudinal associations between personality-related factors, insomnia symptoms & treatment

Table 2A.1. below shows the timing of questionnaire measures at the beginning and conclusion of the academic year. For measures take at entry, survey access opened three weeks after the beginning of the academic year and stayed open for three weeks. For measures taken at the end of the academic year, the questionnaire opened three weeks prior to end-of-year exams and stayed open until the exam period began. Table 2A.2. reports linear regression results from a model identical to the one reported in Chapter 2, except that the full PHQ-9 was used, including item referencing sleep problems ("Over the last two weeks, how often have you been bothered by trouble falling or staying asleep, or sleeping too much?"). The R^2 value for the model suggests that 52% of the variation in SCI score was explained by this model. As in the model reported in Chapter 2, baseline SCI (B = 0.67, SE = 0.03, $p <$ .001) and self-critical perfectionism (B = -0.22, SE = 0.09, p = .01) were significant predictors of end-of-year SCI score. With the inclusion of the sleep item, the PHQ-9 also became a significant predictor (B = -0.09, SE = 0.04, p = .03).

Table 2A.1. Timing of measures in the U-Flourish survey.

Measure	Entry (September)	End of Year (March)
Gender identity	x	
Self-reported sleep disorder diagnosis	x	
Sleep Condition Indicator (SCI)	x	x
Patient Health Questionnaire (PHQ-9)	x	x
Generalised Anxiety Screener (GAD-7)	x	x
Rosenberg's Self-Esteem (RSE)	x	
Clinical Perfectionism Questionnaire (CPQ)	x	

Table 2A.2. Linear regression predicting end-of-year SCI score from baseline symptoms and personality-related factors.

	ΔR^2	*B*	*SE B*	*t*	*p*
Baseline Model	0.52				
Intercept		5.93	1.85	3.21	.001
Female		0.23	0.36	0.64	.52
Lifetime sleep disorder [a]		0.83	0.84	0.98	.33
Baseline SCI		0.68	0.03	24.86	< .001
Baseline PHQ-9 [b]		-0.13	0.04	-3.12	.001
Baseline GAD-7		-0.08	0.04	-1.92	.055
Final Model	0.00				
Intercept		7.61	2.41	3.15	< .001
Female		0.31	0.35	0.86	.39
Lifetime sleep disorder [a]		0.72	0.84	0.85	.39
Baseline SCI		0.67	0.03	24.65	< .001
Baseline PHQ-9 [b]		-0.09	0.04	-2.14	.03

Baseline GAD-7	-0.06	0.04	-1.39	.16
Perfectionism Subscales				
Evaluative Concerns [c]	-0.22	0.09	-2.44	.01
Personal Standards [c]	0.01	0.05	0.24	.81
Self Esteem [d]	0.01	0.04	0.25	.80
Adj. R^2 for final model:	0.52			

[a] Self-reported diagnoses at entry to university. [b] PHQ-9 at entry to university.
[c] Subscale of the CPQ [d] Rosenberg Self-Esteem Scale

Section III: Phenotyping Sleep, Circadian Rhythm, and Mood Instability in Borderline Personality Disorder

Tables 3A.1 & 3A.2 compare mean mood ratings between the BPD and control groups for the LIRP and HIRP periods, respectively, without imputation. The BPD group reported significantly more intense anxiety, sadness, anger and irritability than the control group and less intense elation and energy (all $p < .001$). Tables 3A.3 & 3A.4 present comparisons of instability in each mood between the BPD and control groups for the LIRP and HIRP periods, respectively, without imputation.

Table 3A.1. Mean MoodZoom scores during the LIRP period (3 weeks). No imputation.

	Anxiety	Elation	Sadness	Anger	Irritability	Energy
BPD	2.17 (1.62)	1.34 (1.54)	1.96 (1.86)	0.88 (1.32)	2.00 (1.73)	1.68 (1.53)
HC	0.92 (1.25)	2.15 (1.87)	0.61 (1.23)	0.30 (0.89)	0.82 (1.27)	2.69 (1.66)
Statistic	W = 371370	W = 877483	W = 370711	W = 500909	W = 405470	W = 950477
p	< .001	< .001	< .001	< .001	< .001	< .001

Table 3A.2. Mean MoodZoom scores during the HIRP period (1 week). No imputation.

	Anxiety	Elation	Sadness	Anger	Irritability	Energy
BPD	2.16 (1.54)	1.51 (1.57)	1.64 (1.69)	0.84 (1.34)	2.02 (1.68)	2.03 (1.50)
HC	0.86 (1.15)	2.36 (1.88)	0.50 (1.01)	0.20 (0.71)	0.58 (1.01)	3.04 (1.49)
Statistic	W = 556789	W = 1430031	W = 637195	W = 803141	W = 537089	W = 1558578
p	<.001	<.001	<.001	<.001	<.001	<.001

Table 3A.3. RMSSD of MoodZoom scores during the full LIRP period, without imputation.

	Anxiety RMSSD	Elation RMSSD	Sadness RMSSD	Anger RMSSD	Irritability RMSSD	Energy RMSSD
BPD	1.75 (0.58)	1.64 (0.65)	1.96 (0.63)	1.36 (0.65)	1.80 (0.45)	1.87 (0.74)
HC	1.17 (0.58)	1.29 (0.50)	1.00 (0.65)	0.70 (0.61)	1.18 (0.63)	1.65 (0.51)
Statistic	T =-3.93	T =-2.37	T =-5.99	W = 221	W = 224	W = 407.5
p	< .001	.02	< .001	< .001	< .001	.23

Table 3A.4. RMSSD of MoodZoom scores during the full HIRP period, without imputation.

	Anxiety RMSSD	Elation RMSSD	Sadness RMSSD	Anger RMSSD	Irritability RMSSD	Energy RMSSD
BPD	1.41 (0.44)	1.31 (0.57)	1.24 (0.46)	1.09 (0.61)	1.48 (0.54)	1.38 (0.50)
HC	0.81 (0.47)	0.90 (0.49)	0.61 (0.54)	0.45 (0.44)	0.84 (0.53)	1.05 (0.38)
Statistic	t =-5.22	t =-3.00	W = 171.5	W = 197.5	W = 175	W = 273
p	< .001	< .01	< .001	< .001	< .001	<.01

Linear Models of Concurrent Sleep & Mood Instability

Tables 3A.5 – 3A.7 show associations between mean values of actigraphy-derived sleep variables and mean RMSSD of negative, positive, and irritable moods, respectively. All data arises from the week-long HIRP period. None of the covariates were significant predictors of

mood instability in either group. Tables 3A.8 – 3A.10 show associations between mean values of actigraphy-derived NPCRA variables and instability of negative, positive, and irritable moods, respectively. None of the covariates were significant predictors of mood instability in either group.

Table 3A.5. Linear models predicting RMSSD of the negative MoodZoom factor based on age, employment, and sleep variables. All data arises from the HIRP period. Model parameters are highlighted in blue for the BPD group and in green for the controls.

Term	BPD Estimate	BPD SE	BPD Test Statistic	BPD *p*	HC Estimate	HC SE	HC Test Statistic	HC *p*
Intercept	1.38	1.10	1.26	.22	0.89	1.22	0.73	.47
Age	0.01	0.02	0.38	.70	0.02	0.03	0.58	.57
Employed	-0.11	0.16	-0.71	.48	-0.19	0.33	-0.58	.57
TST	-1.72 e-06	2.88e-05	-0.06	.95	1.91e-05	5.15e-05	0.37	.71
SE	-0.0034	0.01	-0.29	.77	-0.01	0.02	-0.50	.62
SOL	-5.54 e-05	9.97e-05	-0.56	.58	-8.48 e-05	2.49e-04	-0.34	.74

BPD: borderline personality disorder, HC: healthy controls, TST: total sleep time, SE: sleep efficiency, SOL: sleep onset latency.

Table 3A.6. Linear models predicting RMSSD of the positive MoodZoom factor based on age, employment, and sleep variables. All data arises from the HIRP period. Model parameters are highlighted in blue for the BPD group and in green for the controls.

Term	BPD Estimate	BPD SE	BPD Test Statistic	BPD p	HC Estimate	HC SE	HC Test Statistic	HC p
Intercept	2.57	1.45	1.77	.09	1.71	1.16	1.48	.15

Age	-0.02	0.02	-1.09	.29	-0.02	0.02	-0.63	.54
Employed	-0.02	0.21	-0.07	.94	-0.16	0.31	-0.53	.60
TST	-1.24e-05	3.78e-05	-0.33	.75	-5.92e-05	4.89e-05	-1.21	.24
SE	-6.54e-03	0.02	-0.42	.68	0.01	0.02	0.73	.47
SOL	-8.66e-05	1.31e-04	-0.66	.52	-1.2e-04	2.37e-04	-0.49	.63

BPD: borderline personality disorder, HC: healthy controls, TST: total sleep time, SE: sleep efficiency, SOL: sleep onset latency.

Table 3A.7. Linear models predicting RMSSD of the irritable MoodZoom factor based on sleep variables. All data arises from the HIRP period. Model parameters are highlighted in blue for the BPD group and in green for the controls.

Term	BPD Estimate	BPD SE	BPD Test Statistic	BPD *p*	HC Estimate	HC SE	HC Test Statistic	HC *p*
Intercept	1.29	1.45	0.89	.38	1.67	1.11	1.51	.15
Age	4.65e-03	0.02	0.23	.82	-1.51e-03	0.02	-0.07	.95
Employed	0.03	0.21	0.13	.90	-0.22	0.30	-0.75	.46
TST	-1.03e-05	3.78e-05	-0.27	.79	3.15e-05	4.69e-05	0.67	.51
SE	-1.37e-03	0.02	-0.09	.93	-0.02	0.02	-0.99	.33
SOL	1.88e-05	1.31e-04	0.14	.89	-1.4e-04	2.27e-04	-0.60	.55

BPD: borderline personality disorder, HC: healthy controls, TST: total sleep time, SE: sleep efficiency, SOL: sleep onset latency.

Table 3A.8. Linear models predicting RMSSD of the negative MoodZoom factor based on NPCRA variables. All data arises from the HIRP period. Model parameters are highlighted in blue for the BPD group and in green for the controls.

Term	BPD Estimate	BPD SE	BPD Test Statistic	BPD *p*	HC Estimate	HC SE	HC Test Statistic	HC *p*
Intercept	0.38	1.55	0.24	.81	-1.70	2.30	-0.74	.47
Age	0.02	0.02	1.20	.26	0.04	0.05	0.78	.45
Employed	0.03	0.23	0.14	.89	0.13	0.40	0.32	.75
IS	-2.10	1.16	-1.82	.10	1.98	1.58	1.26	.23
IV	0.28	0.59	0.48	.64	-0.28	0.64	-0.44	.67
L5 Activity	3.11e-03	0.01	0.22	.83	-4.92e-03	0.02	-0.30	.77
M10 Activity	1.36e-03	1.15e-03	0.92	.38	-1.33e-03	1.90e-03	-0.70	.49
L5 Start	-0.05	0.09	-0.50	.63	0.08	0.15	0.53	.61
M10 Start	0.08	0.06	1.30	.22	0.06	0.06	1.01	.33

IS: interdaily stability. IV: intradaily variability. L5: least active five hours. M10: most active ten hours.

Table 3A.9. Linear models predicting RMSSD of the positive MoodZoom factor based on NPCRA variables. All data arises from the HIRP period. Model parameters are highlighted in blue for the BPD group and in green for the controls.

Term	BPD Estimate	BPD SE	BPD Test Statistic	BPD *p*	HC Estimate	HC SE	HC Test Statistic	HC *p*
Intercept	0.12	2.23	0.05	.96	-0.78	2.23	-0.35	.73

Age	0.01	0.03	0.44	.67	-4.34e-03	0.05	-0.09	.93
Employed	-0.09	0.32	-0.28	.78	0.15	0.38	0.40	.70
Interdaily Stability	-2.62	1.66	-1.58	.14	2.62	1.53	1.71	.11
Intradaily Variability	0.20	0.84	0.24	.81	-0.09	0.62	-0.15	.88
L5 Activity	-0.02	0.02	-1.02	.33	2.28e-03	0.02	0.14	.89
M10 Activity	3.53e-03	0.00	1.67	.12	-1.64e-03	1.84e-03	-0.89	.39
L5 Start	0.10	0.13	0.74	.48	0.18	0.15	1.23	.24
M10 Start	0.05	0.09	0.57	.58	-0.03	0.06	-0.55	.59

IS: interdaily stability. IV: intradaily variability. L5: least active five hours. M10: most active ten hours.

Table 3A.10. Linear models predicting RMSSD of the irritable MoodZoom factor based on NPCRA variables. All data arises from the HIRP period. Model parameters are highlighted in blue for the BPD group and in green for the controls.

Term	BPD Estimate	BPD SE	BPD Test Statistic	BPD *p*	HC Estimate	HC SE	HC Test Statistic	HC *p*
(Intercept)	1.94	2.26	0.86	.41	-0.31	2.26	-0.14	.89
Age	-0.01	0.03	-0.34	.74	0.04	0.05	0.75	.46
Employed	-0.10	0.33	-0.30	.77	-0.08	0.39	-0.22	.83

Interdaily Stability	-1.54	1.68	-0.92	.38	0.18	1.55	0.12	.91
Intradaily Variability	0.01	0.86	0.01	.99	-0.36	0.62	-0.58	.57
L5 Activity	0.01	0.02	0.63	.54	-0.01	0.02	-0.40	.70
M10 Activity	2.01e-03	2.15e-03	0.93	.37	-8.8e-04	1.86e-03	-0.47	.64
L5 Start	-0.03	0.14	-0.20	.84	0.01	0.15	0.07	.95
M10 Start	-0.02	0.09	-0.28	.79	0.07	0.06	1.15	.27

IS: interdaily stability. IV: intradaily variability. L5: least active five hours. M10: most active ten hours.

Day-lagged Models: Preliminary Reporting of Fixed Effects

Tables 3A.11 – 3A.16 report fixed-effects models predicting mood instability from previous nights' sleep variables.

Table 3A.11. Fixed effects-only model predicting RMSSD of negative mood based on the previous night's sleep in the BPD group.

Term	b	SE b	95% CI of b	p
Intercept	1.43	0.39	0.66, 2.21	<.001
Age	0.01	0.01	-4.32e-03, 0.03	.17
Employed	-0.18	0.08	-0.33,-0.03	.02
Weekend Day	-0.11	0.07	-0.25, 0.03	.14
TST	7.3e-06	8.3e-06	-9.14e-06, 2.37e-05	.38
SE	-8.67e-03	4.07e-03	-0.02,-6.47e-04	.03
SOL	-4.63e-05	2.70e-05	-9.95e-05, 6.92e-06	.09

Table 3A.12. Fixed effects-only model predicting RMSSD of negative mood based on the previous night's sleep in the control group.

Term	b	SE b	95% CI of b	p
Intercept	0.76	0.50	-0.24, 1.75	.13

Age	0.01	0.01	-0.01, 0.04	.28
Employed	-0.31	0.15	-0.61,-0.02	.04
Weekend Day	-0.06	0.08	-0.22, 0.10	.48
TST	5.6e-06	1.09e-05	-1.59e-05, 2.72e-05	.61
SE	-4.69e-03	5.76e-03	-0.02, 0.04	.42
SOL	-1.47e-05	5.25e-05	-1.18e-04, 8.89e-05	.78

Table 3A.13. Fixed effects-only model predicting RMSSD of positive mood based on the previous night's sleep in the BPD group.

Term	b	SE b	95% CI of b	p
Intercept	1.72	0.32	1.09, 2.36	<.001
Employed	-0.03	0.09	-0.21, 0.15	.77
Weekend Day	-0.06	0.08	-0.23, 0.10	.46
TST	1.9e-06	9.4e-06	-1.66e-05, 2.04e-05	.84
SE	-0.01	4.70e-03	-0.02, 3.70e-04	.06
SOL	-6.58e-05	3.13e-05	-1.28e-04,-3.98e-06	.04

Table 3A.14. Fixed effects-only model predicting RMSSD of positive mood based on the previous night's sleep in the control group.

Term	b	SE b	95% CI of b	p
Intercept	1.48	0.38	0.73, 2.23	<.001
Employed	-0.06	0.13	-0.32, 0.20	.65
Weekend Day	0.01	0.07	-0.14, 0.15	.93
TST	-2.35e-05	1.00e-05	-4.33e-05,-3.77e-06	.02
SE	-3.81e-04	5.35e-03	-0.01, 0.01	.94
SOL	-6.07e-05	4.88e-05	-1.57e-04, 3.55e-05	.21

3A.15. Fixed effects-only model predicting RMSSD of irritable mood based on the previous night's sleep in the BPD group.

Term	b	SE b	95% CI of b	p
Intercept	1.50	0.36	0.79, 2.21	<.0001
Employed	0.04	0.10	-0.16, 0.24	.70
Weekend Day	0.10	0.09	-0.09, 0.28	.30
TST	-6.09e-06	1.05e-05	-2.68e-05, 1.46e-05	.56
SE	-0.01	0.01	-0.02, 4.70e-03	.28

SOL	-2.20e-05	3.50e-05	-9.11e-05, 4.70e-05	.53

Table 3A.16. Fixed effects-only model predicting RMSSD of irritable mood based on the previous night's sleep in the control group.

Term	b	SE b	95% CI of b	p
Intercept	1.74	0.38	0.99, 2.50	<.0001
Employed	-0.31	0.13	-0.57,-0.05	.02
Weekend Day	-0.02	0.07	-0.17, 0.12	.74
TST	7.62e-06	1.00e-5	-1.21e-05, 2.74e-05	.45
SE	-0.01	0.01	-0.03,-4.01e-03	.01
SOL	-2.47e-05	4.88e-05	-1.21e-04, 7.17e-05	.61

Tables 3A.17 – 3A.19 report fixed-effects models predicting total sleep time in minutes from previous nights' sleep variables.

Table 3A.17. Fixed effects-only model predicting TST (in minutes) based on the previous day's mood instability in the BPD group.

Term	b	SE b	95% CI of b	p
Intercept	27109.72	1318.04	24507.89, 29711.56	<.0001
Employed	-2207.27	887.28	-3958.76, 455.77	.01
Weekend Day	204.51	828.96	-1431.88, 1840.90	.81s
Instability in Negative Mood	-1450.41	1038.90	-3501.21, 600.39	.16
Instability in Positive Mood	1122.75	770.22	-397.68, 2643.19	.15
Instability in Irritable Mood	-48.29	775.09	-1578.32, 1481.74	.95

Table 3A.18. Fixed effects-only model predicting TST (in minutes) based on the previous day's mood instability in the BPD group.

Term	b	SE b	95% CI of b	p
Intercept	27109.72	1318.04	24507.89, 29711.56	<.0001
Employed	-2207.27	887.28	-3958.76, 455.77	.01
Weekend Day	204.51	828.96	-1431.88, 1840.90	.81s

Instability in Negative Mood	-1450.41	1038.90	-3501.21, 600.39	.16
Instability in Positive Mood	1122.75	770.22	-397.68, 2643.19	.15
Instability in Irritable Mood	-48.29	775.09	-1578.32, 1481.74	.95

Table 3A.19. Fixed effects-only model predicting TST (in minutes) based on the previous day's mood instability in the control group.

Term	b	SE b	95% CI of b	p
Intercept	24316.07	1376.46	21598.09, 27034.06	<.0001
Employed	1056.36	1246.99	-1405.98, 3518.69	.40
Weekend Day	1117.49	647.62	-161.32, 2396.31	.09
Instability in Negative Mood	2073.71	837.98	419.02, 3728.40	.01
Instability in Positive Mood	-2189.54	774.44	-3718.17, 299.08	.01
Instability in Irritable Mood	-1311.54	815.66	-2922.17, 299.08	.11

Section IV: Feasibility study of bright light therapy & digital cognitive behavioural therapy for insomnia in borderline personality disorder

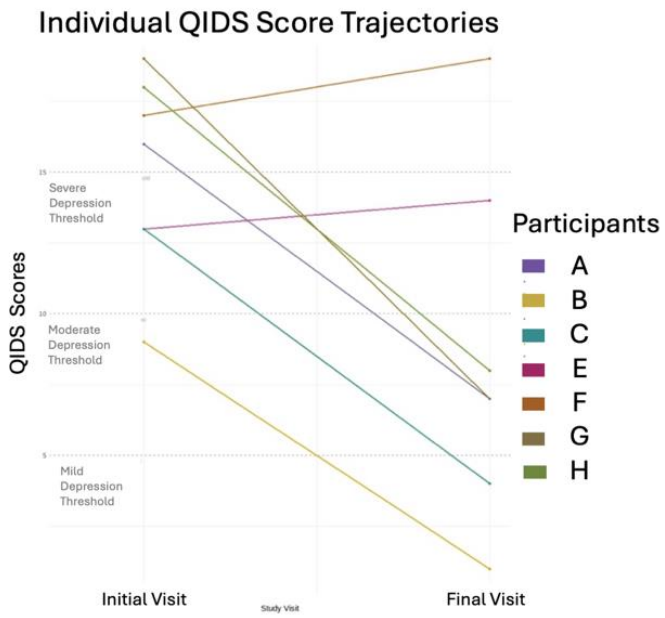


Figure 4A.1 (above). Change in QIDS scores between the first and final study visits. Lower QIDS scores indicate less severe depression symptoms. Score thresholds are indicated by dotted lines. Scores > 5 indicate mild depression, scores >10 indicate moderate depression and scores >15 indicate severe depression.

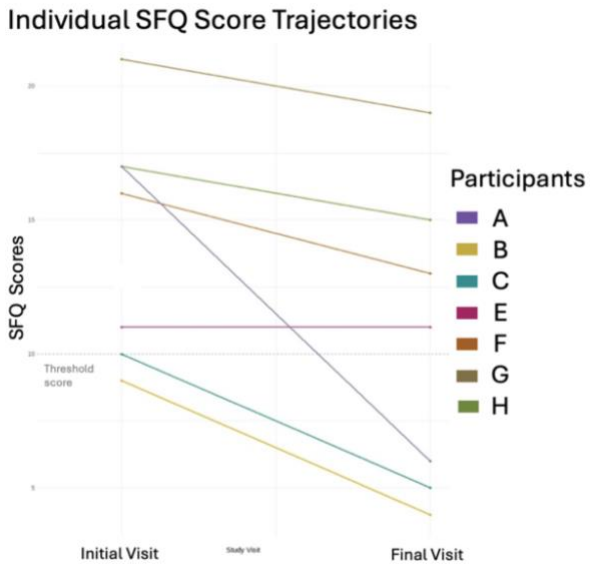
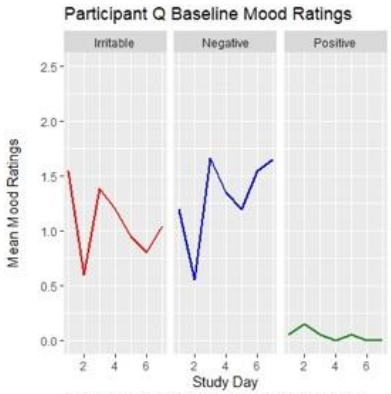


Figure 4A.2 (above) Change in SFQ scores between first and final study visits. Lower SFQ scores indicate improved social functioning.

4A.3. Individual Baseline vs. Post-intervention Mood Ratings & tRMSSD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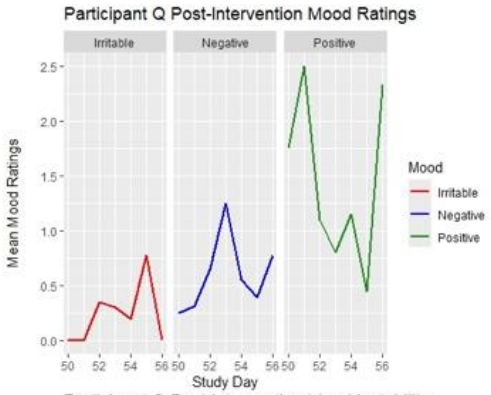
Irritable
Negative
Positive
Mean Mood Ratings
Study Day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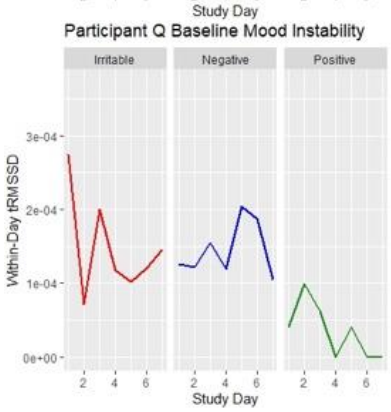
Irritable
Negative
Positive
Mean Mood Ratings
Study Day
Mood
Irritable
Negative
Positive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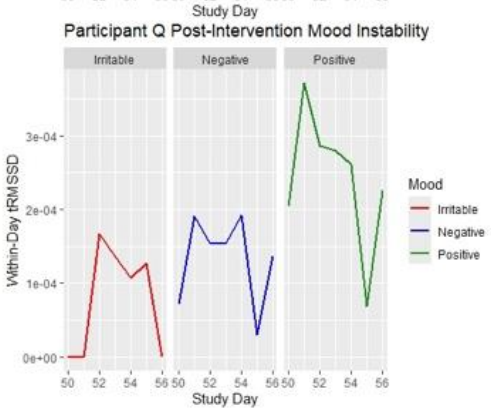
Irritable
Negative
Positive
Within-Day tRMSSD
Study Day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Irritable
Negative
Positive
Within-Day tRMSSD
Study Day
Mood
Irritable
Negative
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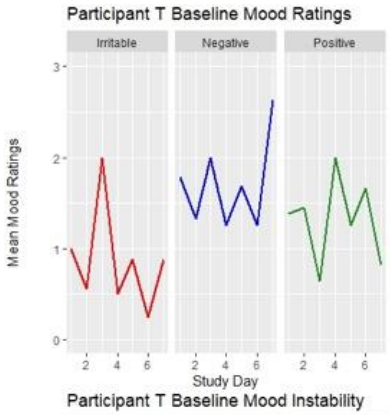
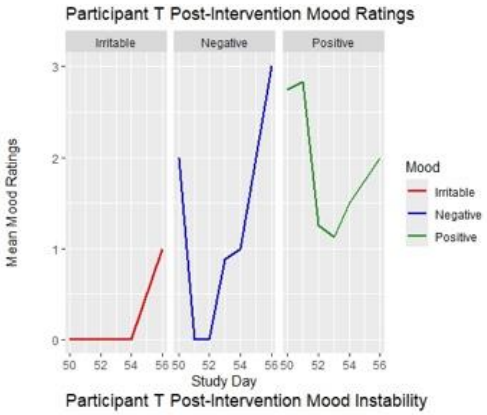
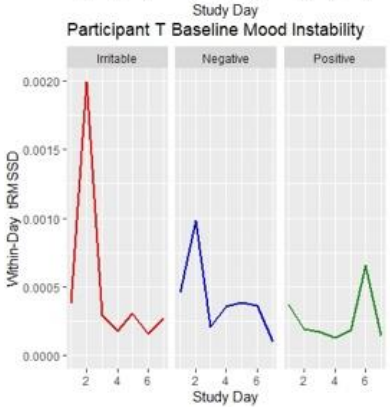
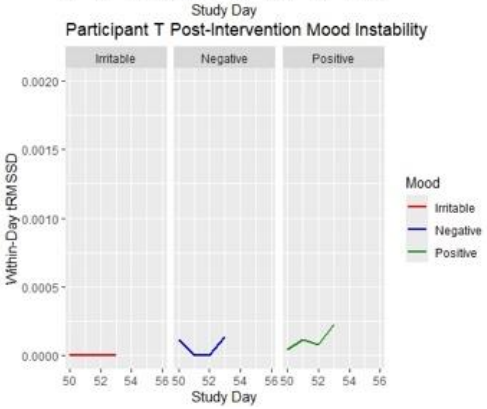

Participant T Baseline Mood Ratings
Irritable
Negative
Positive
Mean Mood Ratings
Study Day


Participant T Post-Intervention Mood Ratings
Irritable
Negative
Positive
Mean Mood Ratings
Study Day
Mood
Irritable
Negative
Positive


Participant T Baseline Mood Instability
Irritable
Negative
Positive
Within-Day tRMSSD
Study Day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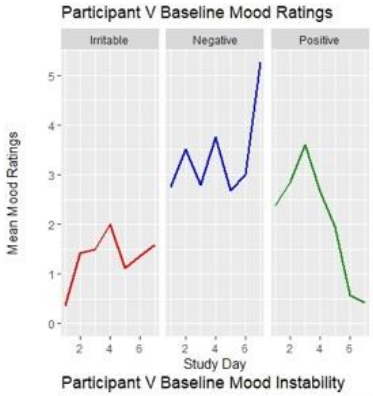
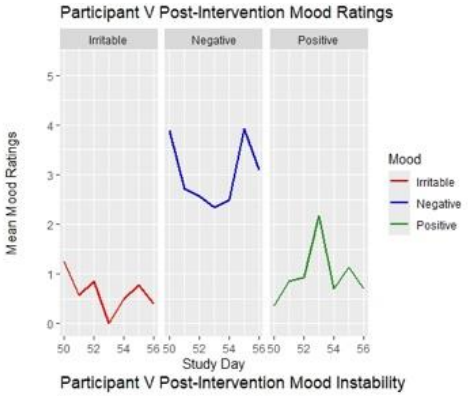
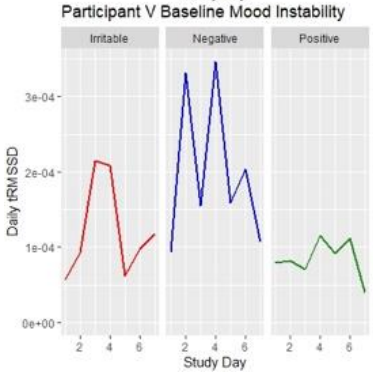
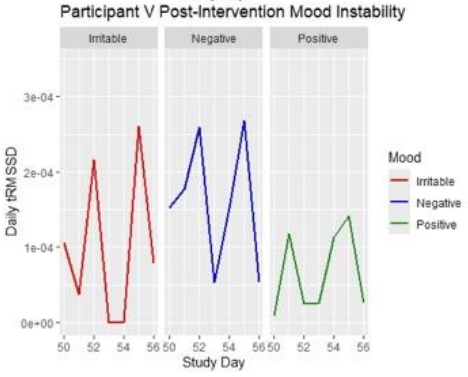
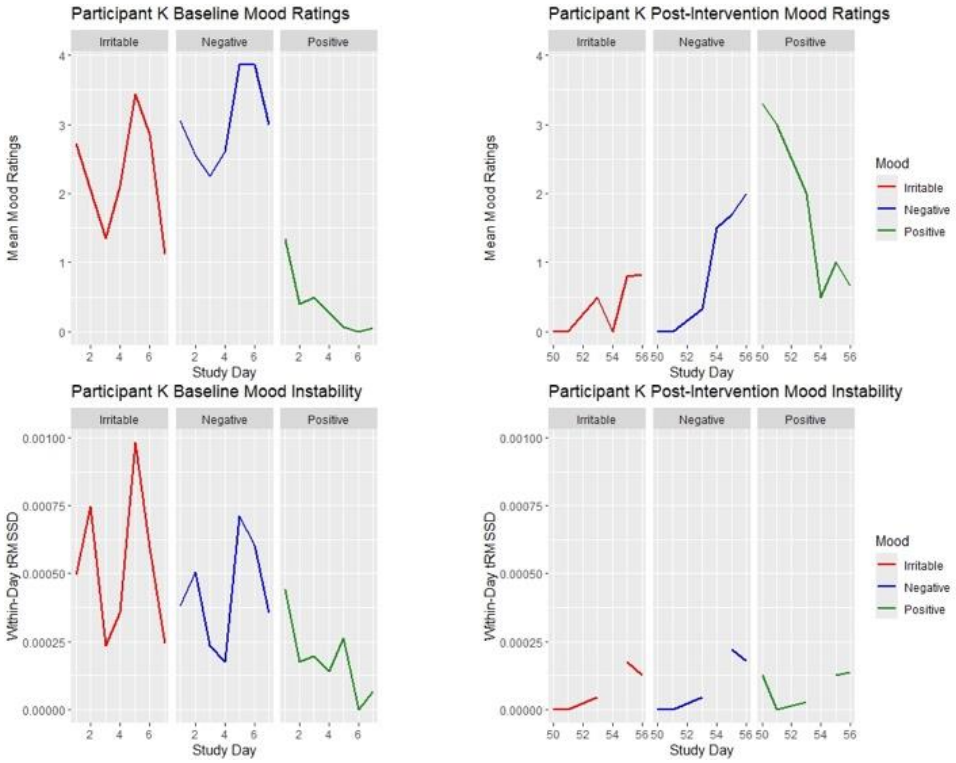
Irritable
Negative
Positive
Within-Day tRMSSD
Study Day
Mood
Irritable
Negative
Positive


Participant V Baseline Mood Ratings
Irritable
Negative
Positive
Mean Mood Ratings
Study Day


Participant V Post-Intervention Mood Ratings
Irritable
Negative
Positive
Mean Mood Ratings
Study Day
Mood
Irritable
Negative
Positive


Participant V Baseline Mood Instability
Irritable
Negative
Positive
Daily tRMSSD
Study Day


Participant V Post-Intervention Mood Instability
Irritable
Negative
Positive
Daily tRMSSD
Study Day
Mood
Irritable
Negative
Positive



Supplementary Actigraphy-Derived Results

Distribution of Sleep Variables

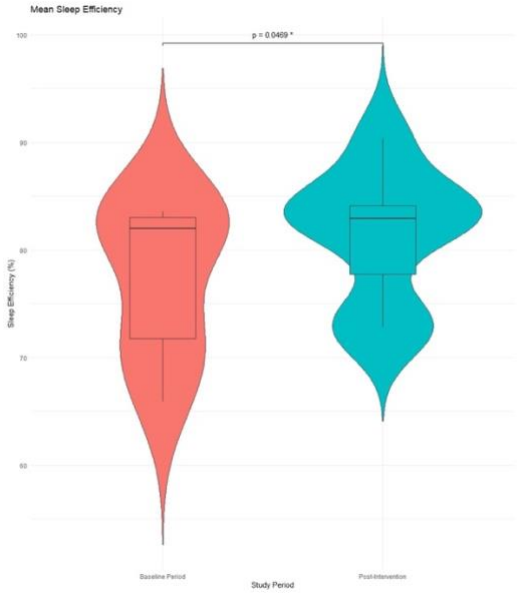


Figure 4A.4. (above) Sleep efficiency in baseline versus post-intervention weeks, difference significant at p = 0.0469.

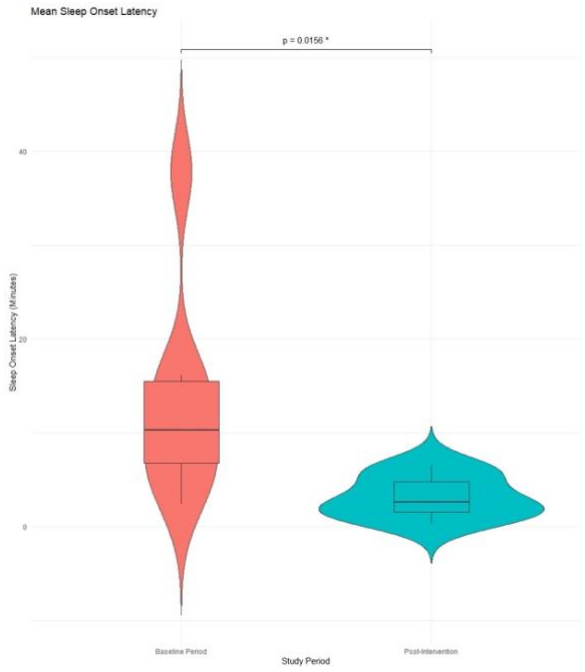


Figure 4A.5. (above) Sleep onset latency in baseline versus post-intervention weeks, difference significant at *p* = .02

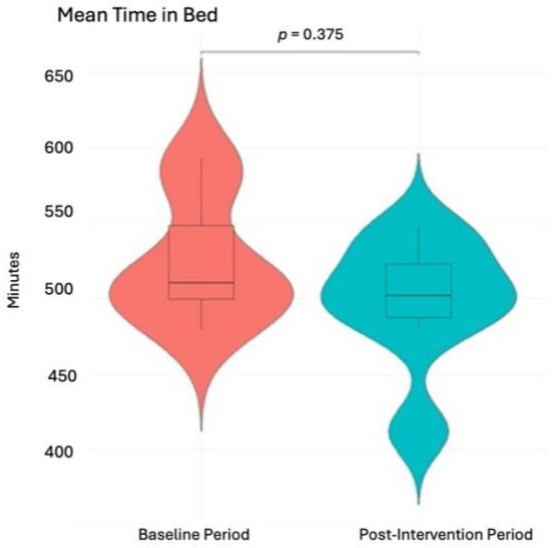


Figure 4A.6. (above) Time in bed in baseline versus post-intervention weeks, difference insignificant (p = .38).

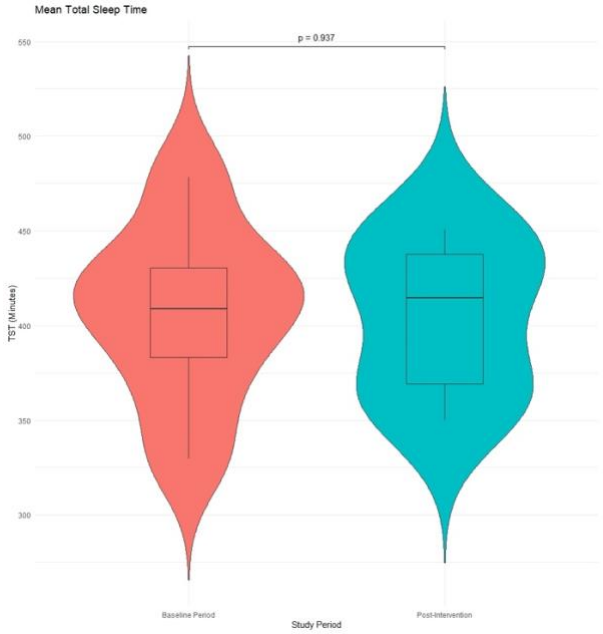


Figure 4A.7. (above) Total sleep time in baseline versus post-intervention weeks, difference insignificant (p = .94).

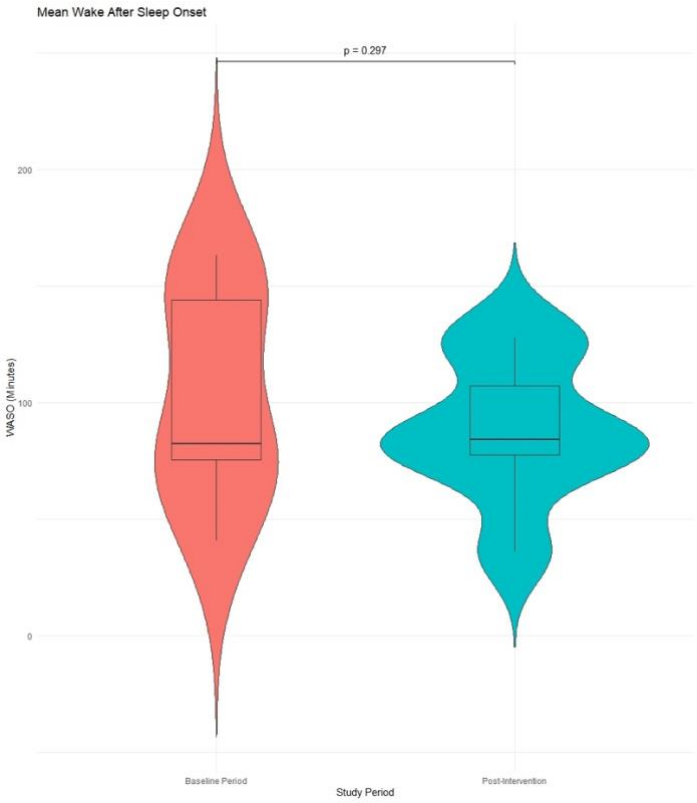


Figure 4A.8. (above) Wake after sleep onset (min) in baseline versus post-intervention weeks, difference insignificant p = 0.30.

Comparative Distributions of Circadian Rhythm Structure

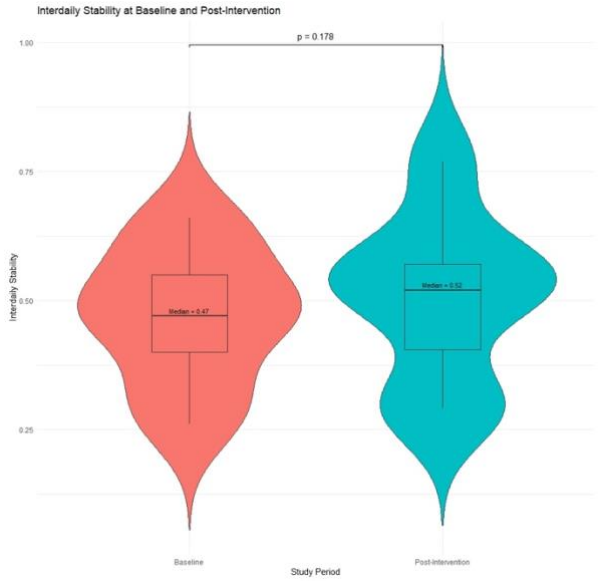


Figure 4A.9. (above) Interdaily stability in baseline versus post-intervention weeks, difference insignificant at p = .18.

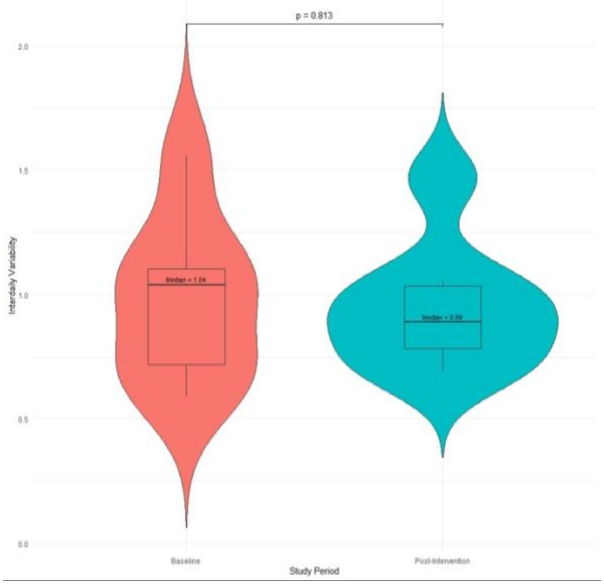


Figure 4A.10. (above) Intradaily Variability in baseline versus post-intervention weeks, difference insignificant at p = .81.

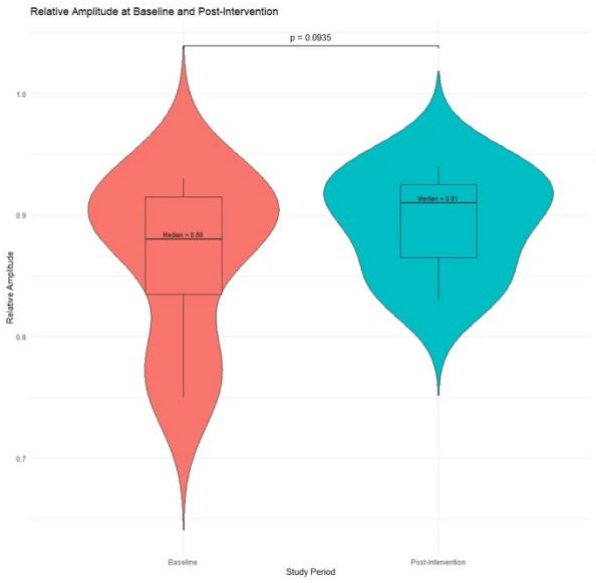
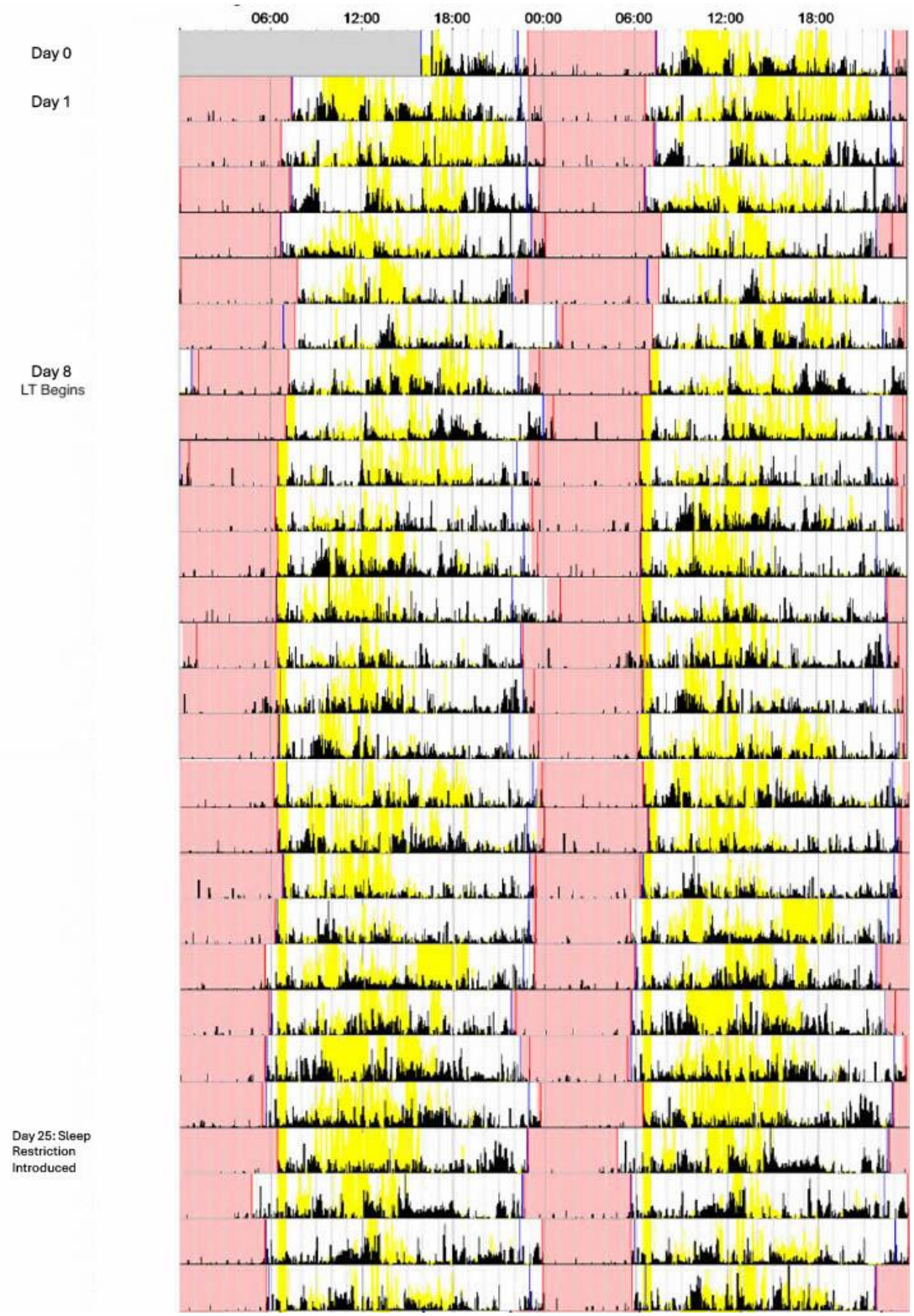


Figure 4A.11. (above) Mean relative amplitude in baseline versus post-intervention weeks, difference insignificant at p = .09.

Figure 4A.12. Participant C's actogram. Sleep attempt windows are demarcated in red and bright light exposure in yellow. Adherence to BLT is evident from Days 9 -50.



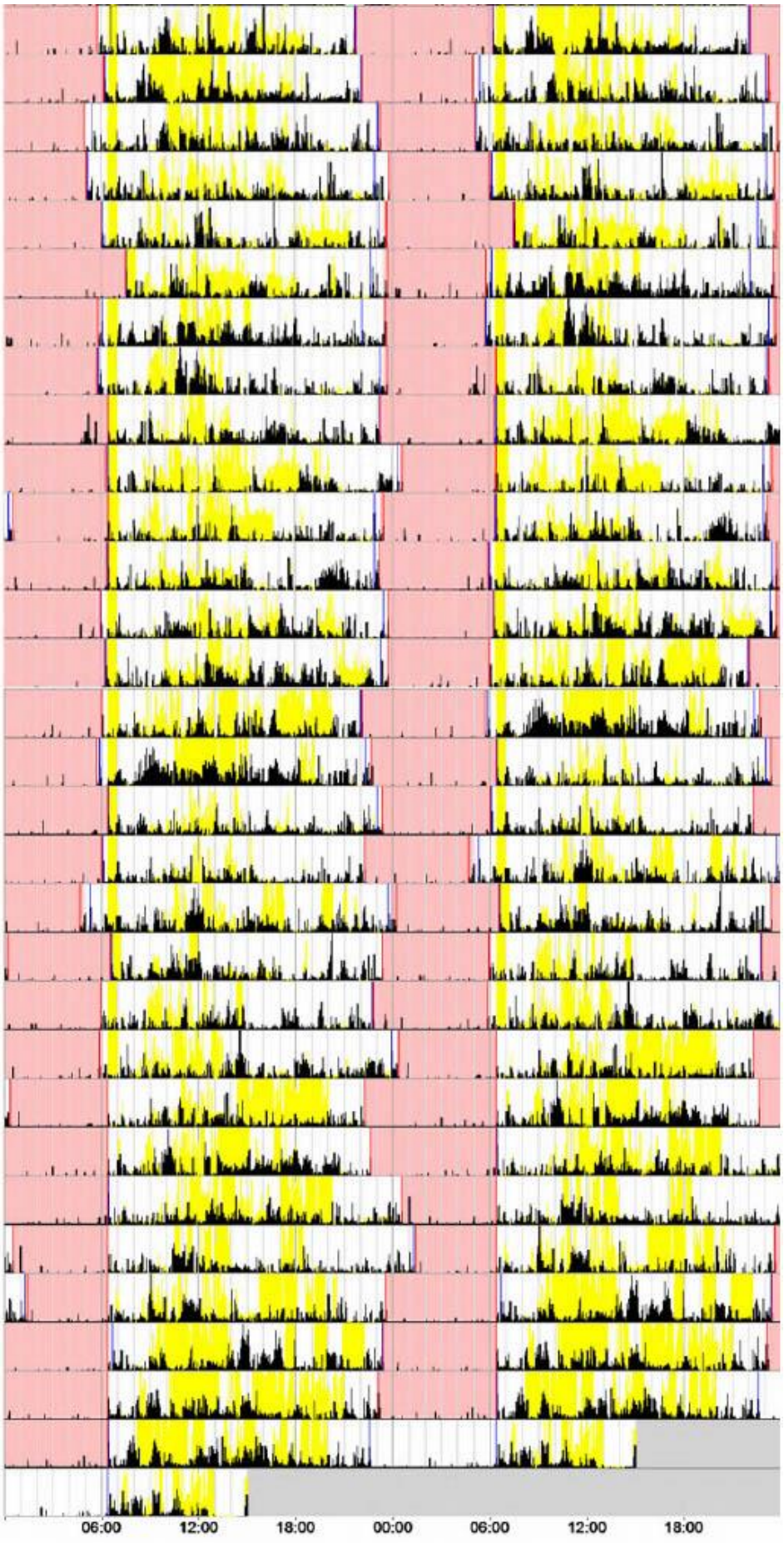
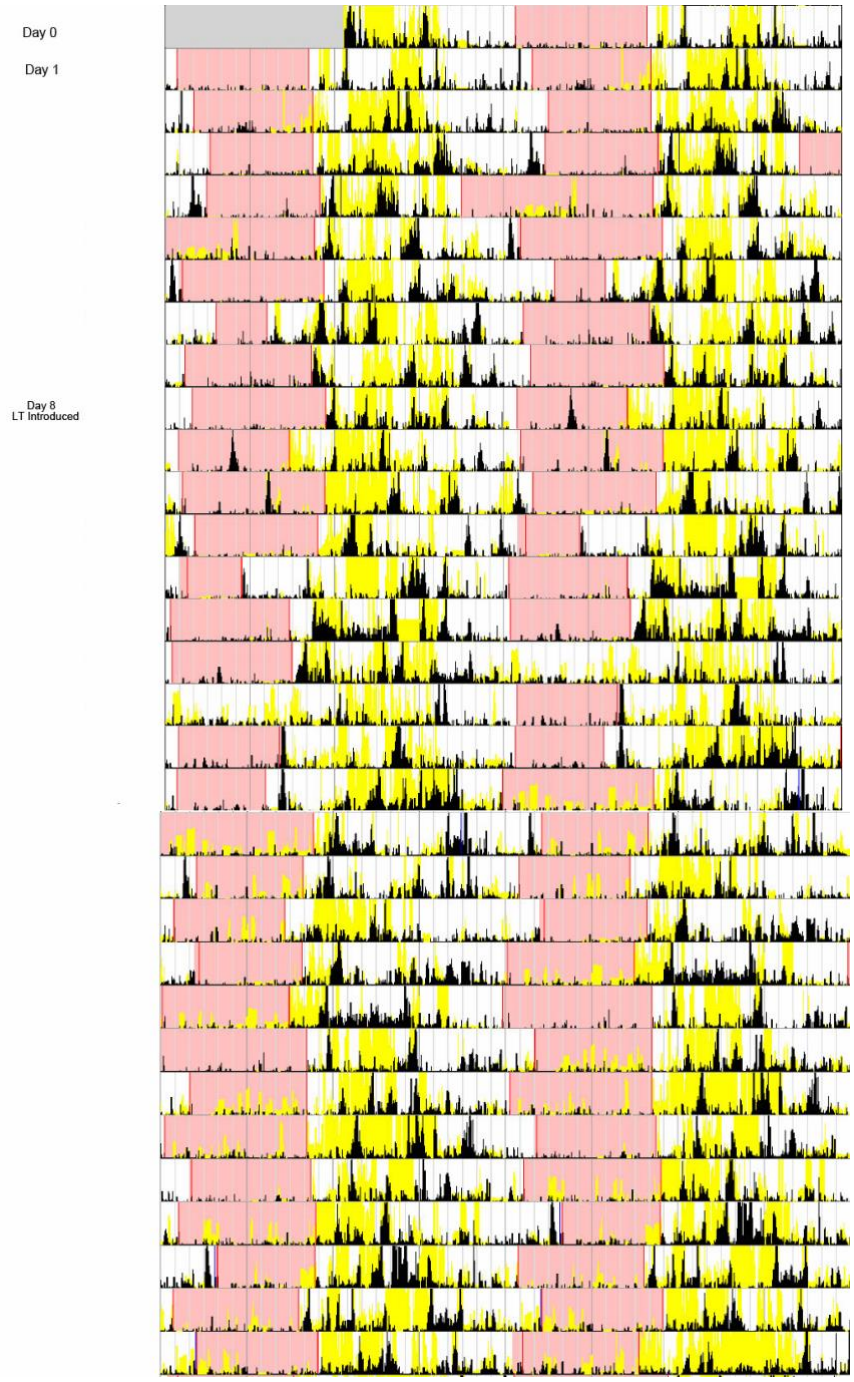
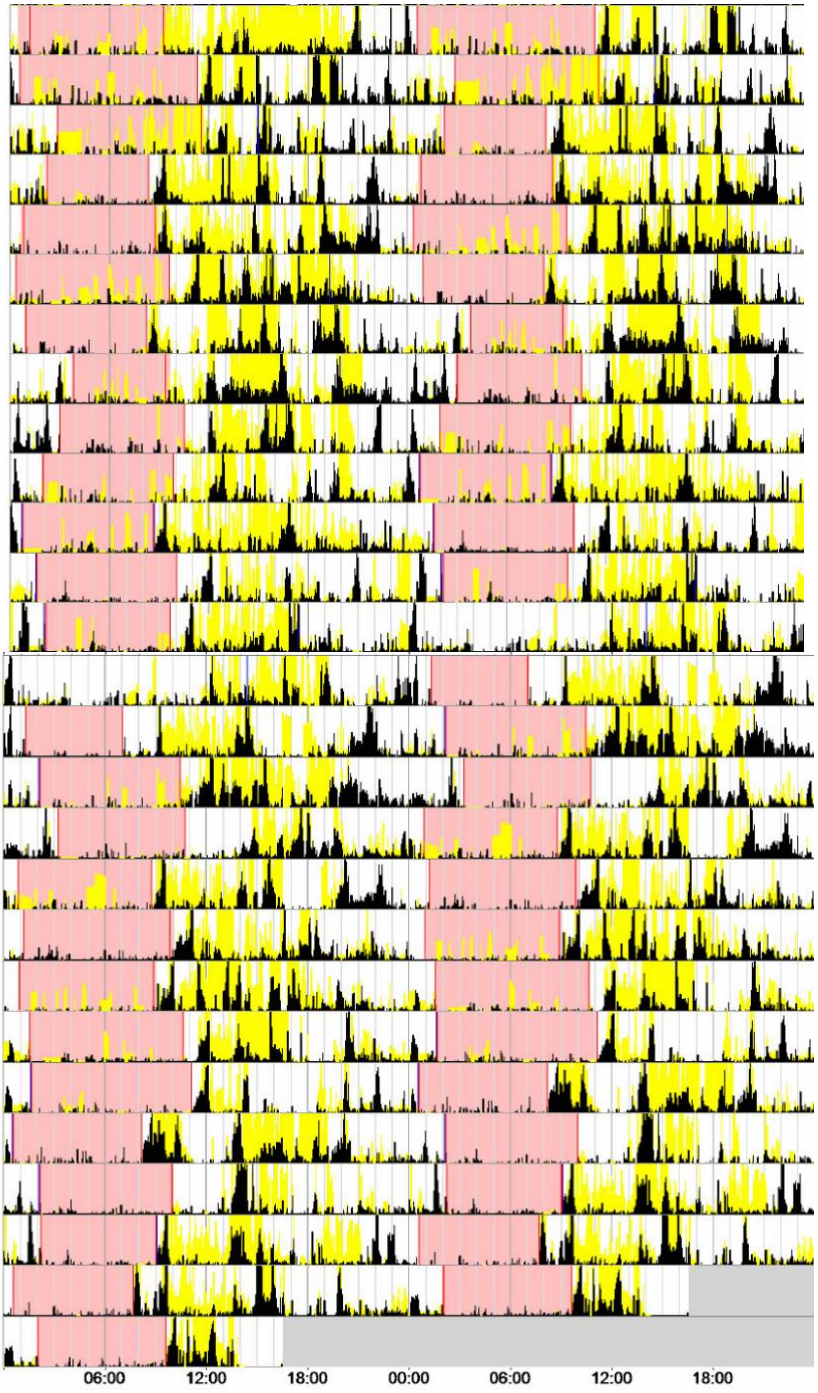

Day 50:
LT Ends
06:00
12:00
18:00
00:00
06:00
12:00
18:00

Figure 4A.13 Actogram from Participant B. Sleep attempt windows are demarcated in red and bright light exposure in yellow.




06:00
12:00
18:00
00:00
06:00
12:00
18:00

Section VI. Systematic Review & Meta-Analysis of Digital Interventions for Symptoms of BPD

6A.1 Search strategy

The search strategy, which was developed by JL and EH, is reproduced below in full using Ovid Embase syntax, with the number of results returned at the end of each line. Terms related to BPD and its symptoms are captured by lines 1-12 and 30-44. Lines 13-24 were derived from a validated filter developed by the National Institute for Health and Care Excellence with the objective of capturing the many possible descriptors used for application-based digital interventions (1). Lines 25, 26, and 45 were added to this filter to account for other delivery formats including web-based interventions, serious games, and interventions mislabeled as telehealth. Lines 27, 28, 50 and 52 capture terms related to symptoms and psychiatric illness. Lines 46-48 capture terms related to clinical trials and experimental settings. This final concept was added to the search to filter out observational studies. The search can be automatically executed by following the referenced link (*OVID Embase Search Strategy*, 2022). References were managed using EndNote 20 and Rayyan (www.rayyan.ai).

1 borderline state/ 15927

2 ((borderline or border-line) adj3 (personalit* or state*)).ti,ab,kw. 11882

3 ("Axis II" or "Cluster B" or flamboyant or "emotionally unstable personality" or EUPD or "F60.3" or "F60.30" or "F60.31").kw,tw. 4678

4 (idealization adj5 devaluation).kw,tw. 34

5 ((vulnerable or hyberbolic) adj3 temperament).kw,tw. 12

6 (((unstab* or instab* or poor or disturb* or fail* or weak or dysregulat*) adj3 (self* or impuls* or interperson* or identit* or relationship* or emotion* or affect*)) and (personality or character or PD)).kw,tw. 4918

7 (impulsiv* adj5 (behavio?r or character or personalit*)).kw,tw. 6309

8 (self adj3 (injur* or damag* or destruct* or harm* or hurt* or mutilat*)).kw,tw. 27728

9 (suicidal adj3 (behavior or behaviour)).kw,tw. 13793

10 (feel* adj3 (empt* or bored*)).kw,tw. 607

11 (anger adj5 control*).kw,tw. 1465

12 (risk-taking adj3 (behavior or behaviour)).kw,tw. 2785

13 exp mobile application/ 25719

14 exp mobile phone/ 47811

15 text messaging/ 7699

16 personal digital assistant/ 1833

17 computer assisted therapy/ 4861

18 (app or apps).ti,ab. 59462

19 ((online or web or internet or digital* or virtual) adj3 (based or application* or intervention* or program* or therap*)).ab. 119319

20 (phone* or telephone* or smartphone* or cellphone* or smartwatch*).ti. 32393

21 ((phone* or telephone* or smartphone* or cellphone* or smartwatch*) adj3 (based or application* or intervention* or program* or therap*)).ab. 22835

22 (mobile health or mhealth or m-health or ehealth or e-health or emental or e-mental).ti. 9450

23 ((mobile health or mhealth or m-health or ehealth or e-health or emental or e-mental) adj3 (based or application* or intervention* or program* or therap*)).ab. 6510

24 (mobile* adj3 (based or application* or intervention* or device* or technolog*)).ti,ab. 27130

25 exp telemedicine/ 71490

26 (telemedicine or tele-medicine or telehealth or tele-health or wearable*).ti,ab. 66756

27 symptom*.ti,ab,kw. 2128090

28 exp symptom/ 169701

29 27 or 28 2173329

30 borderline state/ 15927

31 "borderline psychosis".tw,kw. 16

32 abandon*.tw,kw. 34055

33 ("interpersonal effectiveness" or "interpersonal sensitivity" or "interpersonal hypersensitivity" or "rejection sensitivity" or "rejection hyper-sensitivity").tw,kw. 2145

34 ("identity disturbance" or "uncertain sense of self" or "shifting identity").tw,kw. 184

35 impulsiveness/ 27235

36 impulsiv*.tw,kw. 34929

37 exp suicidal behavior/ 127019

38 exp automutilation/ 24507

39 (self-injur* or selfinjur* or "self harm*" or "self-inflicted injur*" or selfinflict* or para-suicid* or parasuicid* or suicid*).tw,kw. 133863

40 ("emotion dysregulat*" or "emotional dysregulat*" or "emotion regulat*" or "emotional regulat*" or "affective instabil*" or "affective labil*" or "mood instabil*" or "mood labil*" or "aversive tension").kw,tw. 22796

41 anger management therapy/ or anger/ 22458

42 (anger or angry).tw,kw. 29497

43 paranoia/ 11981

44 (depersonaliz* or depersonalis* or derealiz* or derealis* or dissociat* or paranoi* or suspicio*).tw,kw. 368290

45 ("serious game*" or gamifi* or "applied game*").tw,kw.3106

46 exp clinical trial/ or "clinical trial (topic)"/ or clinical trial protocol/ 1954147

47 human experiment/ or therapeutic research/ 642337

48 (treatment* or task* or therap* or pre-therap* or post-therap* or interven* or train* or tool*).kw,tw. 12945540

49 46 or 47 or 48 13970931

50 (psychopathology or risk* or prevention or crisis or crises or psychiatr* or disorder* or "psychological distress" or "psychological state" or "mental health" or harm*).kw,tw. 6934344

51 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43 or 44 686992

52 symptom/ 164738

53 mental disease/ 277629

54 27 or 28 or 50 or 52 8263048

55 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 45 357217

56 49 and 51 and 54 and 55 3524

57 limit 56 to english language 3455

58 limit 57 to yr="2000 -Current" 3429

6A.2 Study Characteristics, presented by symptom

Tabulation of BPD-Specific Interventions

Four interventions were identified which listed their treatment targets as BPD broadly rather than a specific symptom (Table 6A.1). These studies, published from 2017 to 2021, include data from 376 participants.

Table 6A.1: Study Characteristics of BPD Interventions

Author, Year, Location, Type of study	***Intervention name*, therapeutic approach, duration, frequency & access method. Degree of Human Facilitation.**	**Population, Enrolment/Assignment, Loss to follow-up, Comparator**	**Outcome measure(s) & Summary**	**PSD Elements Employed**
Jacob, Hauer [4] 2018. Germany, Single arm pre-post pilot study. 2018.	*priovi,* schema therapy, web. Adjunctive to in-person treatment.	Outpatients with BPD diagnosis 14 enrolled, 2 (14%) lost to FU	Significant reductions in BPDSI & BPD-CL scores	Reduction Tunneling Tailoring Personalisation Reminders Suggestion Similarity Liking
Klein, Hauer-von Mauschwitz [5] 2021. Germany, RCT.	*priovi,* schema therapy, 1 year, twice weekly, web. Technical support only.	Outpatients with diagnosis or probable diagnosis of BPD Digital intervention arm: 103 assigned, 42 (41%) lost to FU Control (TAU) arm: 101 assigned, 26 (26%) lost to FU	BPD symptom frequency (BPDSI) had significantly greater decrease in *priovi* group	Reduction Tunneling Tailoring Personalisation Reminders Suggestion Similarity Liking Rewards
Laursen, Helweg-Jørgensen [6] 2021. Denmark, RCT & economic evaluation	*mDiary*, DBT, 40 weeks to 1 year depending on study site, daily, mobile application. Adjunctive to in-person treatment.	Outpatients with EUPD (F60.3) Digital intervention arm: 42 assigned Active control arm: 36 assigned Drop-out acknowledged but not reported.	Between-group differences in ZAN-BPD scores were insignificant	Reduction Self-monitoring Rehearsal Reminders Suggestion Liking

Zanarini et al. [7] 2017. USA, RCT	(none), DBT, 6 weeks, weekly, web. None.	Community sample of women meeting diagnostic criteria for BPD Digital intervention arm: 40 assigned, 1 (2.5%) lost to FU. Control arm: 40 assigned, 2 (5%) lost to FU.	ZAN-BPD: no significant difference in total score between groups. BEST: no significant difference between groups	Interface not described in text and no response from authors

Tabulation of Suicidal Ideation Interventions

Twenty studies targeting symptoms of suicidal ideation were identified (Table 6A.2), with publication years ranging from 2016 to 2023. These studies include data from 3,983 participants.

Table 6A.2: Study characteristics of suicidal ideation interventions

Author, Year, Location, Type of study	***Intervention name*, therapeutic approach, duration, frequency, access method, human facilitation**	**Population, Enrolment, Attrition & Adherence**	**Outcome measure(s) & Summary**	**PSD Elements Employed**
Batterham, Calear [8]. 2018. Australia, RCT (3 arms)	FitMindKit, multiple, 2 weeks, daily, website access. None.	Community sample of adults with moderate mood disorder symptoms. Digital intervention static version: 62 randomised, 41 (66%) lost to follow-up. Digital intervention tailored version: 66 randomised, 41 (62%) lost to follow-up. Control arm: 66 randomised, 37 (56%) lost to follow-up.	SIDAS: no significant decreases or time x condition effects in suicidal ideation.	Reduction Tunneling Rehearsal Reminders Suggestion Similarity Liking Social Role
Crosby and Witte [9] 2021. USA, Single arm	Sleep Scholar, CBT-I, 1 session + 14 sleep diaries, website access, None.	University students with insomnia & suicidal ideation 40 enrolled, 2 (5%) excluded due to technical difficulties, 5 (13%) lost to follow-up.	DSI-SS: No significant reduction in suicidal ideation	Reduction Tailoring Personalisation Self-monitoring Simulation Rewards

				Suggestion
De Jaegere, van Landschoot [10] 2019. Belgium, RCT	Think Life (adaptation of Living with Deadly Thoughts), CBT, 6 weeks, weekly, website access, None.	Adults with suicidal ideation. Digital intervention arm: 365 randomised, 270 (74%) lost to follow-up. Control arm: 359 randomised, 187 (52%) lost to follow-up.	BSS & SIDAS: Significantly greater reduction in both measures of suicidal ideation for the Think Life group.	Reduction Self-monitoring Rehearsal Reminders Suggestion
Depp, Parrish [11] 2023. USA, RCT (pilot)	mSTART, CBT, 3 months, daily use, mobile application, adjunctive to in-person treatment.	Outpatients with SMIs & suicidal ideation Digital intervention arm: 38 assigned, 11 (29%) lost to follow-up. In-person only: 40 assigned, 10 (25%) lost to follow-up.	BSSI: No significant effect of group at post-treatment	Reduction Tailoring Personalisation Self-monitoring Simulation Rehearsal Rewards Suggestion
Eylem, van Straten [12] 2021. Netherlands & UK, RCT	Living with Deadly Thoughts (Turkish version), 6 weeks, weekly, website access, regular content support.	Community sample of adults with suicidal ideation. Digital intervention arm: 10 randomised, 0 lost to follow-up. Control arm: 8 randomised, 2 (25%) lost to follow-up.	BSS: No significant time x condition effects.	Reduction Self-monitoring Rehearsal Reminders Suggestion
Frey, Osteen [13] 2023. USA, RCT	Man Therapy, stigma reduction, 3 months open dosage, website, none.	Community adults with suicidal ideation. Digital intervention arm: 279 assigned, 72 (26%) lost to follow-up. Sham control: 275 assigned, 33 (12%) lost to follow-up.	C-SSRS: no significant effect of group at post-treatment.	Reduction Tailoring Personalisation Simulation Suggestion Similarity Liking Social Role
Franklin, Fox [14] 2016. USA, RCT	TEC (Study 1 version), evaluative conditioning, 2 months, open dosage, mobile application, none.	Adults with recent self-cutting Active digital intervention arm: 55 randomised, 14 (25%) lost to one-month follow-up. Sham digital intervention arm: 59 randomised, 21 (36%) lost to one-month follow-up.	Significantly fewer general NSSI episodes, self-cutting episodes, and suicidal plans in the active group than the sham group.	Rewards
Franklin, Fox [14] 2016. USA, RCT	TEC (Study 2 version), evaluative conditioning, 2 months, open dosage, mobile application, none.	Adults with recent self-cutting Active digital intervention arm: 62 randomised, 18 (29%) lost to follow-up.	Significantly fewer self-cutting episodes in the active group as compared to the sham group.	Rewards

		Sham digital intervention arm: 69 randomised, 17 (25%) lost to follow-up.	No difference in self-cutting events (ie. Total number of cuts) or general NSSI episodes.	
Franklin, Fox [14] 2016. USA, RCT	TEC (Study 3 version), evaluative conditioning, 2 months, open dosage, mobile application, none.	Community sample of adults with suicidal ideation. Active digital intervention arm: 75 randomised, 39 (52%) lost to one-month follow-up. Control digital intervention arm: 84 randomised, 30 (36%) lost to one-month follow-up.	SITBI: Significant reduction in suicide plans for active group, no significant difference in ideation between groups	Rewards
Hooley, Fox [15] 2018. USA, RCT (3 arms)	Autobiographical Self-Enhancement Training (ASET), Expressive Writing (EW), journaling, 1 month, daily, website access, as-needed treatment support.	Adults with recent NSSI ASET: 49 randomised, 8 (16%) lost to one-month follow-up. EW: 49 randomised, 4 (8%) lost to one-month follow-up. Basic journaling control: 46 randomised, 9 (20%) lost to one-month follow-up.	SITBI – NSSI & suicidal ideation frequency: Basic journaling group had significantly fewer days of suicidal ideation than EW group. No other significant effects.	Reduction Self-monitoring Rehearsal Reminders Suggestion
Laursen, Helweg-Jørgensen [6] 2021. Denmark, RCT & economic evaluation	mDiary, DBT, 40-52 weeks depending on study site, daily, mobile application, adjunctive to in-person treatment.	Outpatients with EUPD (F60.3) Digital intervention arm: 42 assigned Paper diary control arm: 36 assigned Drop-out acknowledged but not reported.	Unadjusted between-group differences in suicidal behaviour (SBQ) were insignificant	Reduction Self-monitoring Rehearsal Reminders Suggestion Liking
Mühlmann, Madsen [16] 2021. Denmark, RCT	Living with Deadly Thoughts (Danish version), CBT, 6 weeks, daily, website access, as-needed content support	Community sample of adults with suicidal ideation. Digital intervention arm: 196 randomised, 15 (8%) lost to follow-up. Control arm: 206 randomised, 24 (12%) lost to follow-up.	BSS & SIDAS: Significant reduction in both measures of suicidal ideation for intervention arm	Reduction Self-monitoring Rehearsal Reminders Suggestion
O'Toole, Arendt [17] 2019. Denmark, RCT	Life App'tite, 8 weeks, daily, mobile application,	Outpatients with suicidal ideation.	SSF: Significant time x condition effect on suicide risk.	Reduction Self-monitoring Rehearsal Suggestion

	adjunctive to in-person treatment.	Digital intervention arm: 60 randomised, 34 (57%) lost to follow-up. Control (TAU) arm: 69 randomised, 30 (43%) lost to follow-up.		
Pauwels, Aerts [18] 2017. Belgium, Single arm pre-post	BackUp, crisis planning, 1 week, open dosage, mobile application, none.	Community sample of adults with suicidal ideation. 45 enrolled, 24 (53%) dropped out.	BSS, non-significant decrease in suicidal ideation	Reduction Personalisation Suggestion Liking
Rodante, Kaplan [19] 2022. Argentina, Cluster RCT	Calma, DBT, 1 month, open dosage, mobile application, adjunctive to in-person treatment	Outpatients with suicidal ideation Digital intervention + DBT: 11 randomised & allocated, 2 (18%) lost to follow-up. DBT only: 11 randomised & allocated, 1 (9%) lost to follow-up.	No significant differences between groups.	Reduction Tunneling Tailoring Self-monitoring Rehearsal Reminders Suggestion Liking
Tighe, Shand [20] 2017. Australia, RCT	iBobbly, ACT, 6 weeks, open dosage, mobile application, none.	Young indigenous adults with suicidal ideation and depression/distress. Digital intervention arm: 31 randomised, 2 (6%) lost to follow-up. Control arm: 30 randomised, 0 lost to follow-up.	DSI-SS: significant decrease in suicidal ideation for intervention arm, but no time x condition effect.	Reduction Tunneling Personalisation Self-monitoring Rehearsal Suggestion Similarity Liking
Torok, Han [21] 2022. Australia, RCT	Life Buoy, DBT, 6 weeks, open dosage, mobile application, none.	Community adults with suicidal ideation Digital intervention arm: 228 randomised, 66 (29%) lost to follow-up. Sham control arm: 227 randomised, 57 (25%) lost to follow-up.	SIDAS: Significant reduction in suicidal ideation scores & significant time x condition effect in intervention arm.	Reduction Tunneling Self-monitoring Simulation Rehearsal Reminders Suggestion Liking
van Spijker, van Straten [22] 2018. Netherlands, RCT	Living with Deadly Thoughts (Dutch version), CBT & DBT, 6 weeks, daily, website access, none.	Adults with mild to moderate suicidal ideation. Digital intervention arm: 116 randomised, 11 (9%) lost to follow-up Control arm: 120 randomised, 10 (8%) lost to follow-up.	BSS: Significant reduction in suicidal in intervention arm	Reduction Self-monitoring Rehearsal Reminders Suggestion

van Spijker, Werner-Seidler [23] 2018. Australia, RCT	Living with Deadly Thoughts (English version), CBT & DBT, 6 weeks, daily, website access, crisis support as needed.	Community adults with current suicidal ideation of any severity. Digital intervention arm: 207 randomised, 90 (43%) lost to follow-up. Sham control arm: 211 randomised, 102 (48%) lost to follow-up.	No group differences in suicidal ideation.	Reduction Self-monitoring Rehearsal Reminders Suggestion
Wilks, Lungu [24] 2018. USA, RCT	None, iDBT-ST, 8 weeks, weekly, website access, technical support as-needed.	University students with suicidal ideation, heavy drinking & emotion dysregulation. Digital intervention arm: 30 enrolled Control arm: 29 enrolled In total, 21 (36%) were lost to follow-up.	SSI & DERS: No significant time x condition effects on suicidal ideation or emotion regulation.	Reduction Tunneling Self-monitoring Rehearsal Rewards Suggestion

Subgroup Analysis of Population in Suicidal Ideation Interventions

Separating the studies by population (outpatient versus community-drawn samples) did not suggest significant differences in treatment effect between groups (Table 6.A3).

Table 6A.3: Subgroup analysis comparing suicide ideation intervention studies recruiting from community versus outpatient samples

	n studies	SMD	95% CI	P	I^2	95% CI	$P_{subgroup}$
Population							.77
Community	13	-0.14	-0.28 to 0.00	.04	31.1%	50.1 – 83.8%	
Outpatient	5	-0.09	-0.51 to 0.32	.56	71.5%	0 – 73.6%	

Tabulation of Paranoia Interventions

Five studies of interventions targeting paranoia were identified, with publication years ranging from 2018 to 2021 and enrolling a total of 679 participants (Table 6A.4).

Table 6A.4: Study characteristics for interventions targeting paranoia

Author, Year, Location, Study Type.	*Intervention name*, modality, duration, frequency, access method, human facilitation	Population, Enrolment, Attrition & Adherence	Outcome measure(s) & Summary	PSD Elements Employed
Muneghina, Van Gordon [25] 2021, UK, RCT.	*Nature Based Intervention (NBI),* mindfulness, 5 days, daily, website access, none.	Community sample of healthy adults Digital intervention arm: 37 allocated, 0 lost to follow-up Control arm: 35 allocated, 0 lost to follow-up	Significant time x condition effect on Paranoia Scale scores	Rehearsal Suggestion Liking
Sood and Newman-Taylor [26], 2020, UK, RCT.	*No name,* attachment-based imagery, 1 session, website access, none.	Community & university sample with elevated but non-clinical paranoia Digital intervention arm: 61 allocated Active control arm: 56 allocated	Significant time x condition effect on Paranoia Scale score	Suggestion
Shore, Strauss [27], 2018, UK , RCT.	*Learning Meditation Online,* mindfulness, 2 weeks, open dosage, website access, as-needed technical support.	Community & university sample of healthy adults Digital intervention arm: 56 randomised, 27 (48%) lost to follow-up. Mean days of use: 11.83 (SD = 3.68) Active control arm: 54 randomised, 25 (46%) lost to follow-up	Significant time x condition effect on Paranoia Scale score	Reduction Rehearsal Suggestion
Newman-Taylor, Kemp [28], 2018, UK, RCT.	*No name,* attachment-based imagery, 1 session, website access, none.	University sample of healthy adults Digital intervention arm: 140 allocated Active control arm: 161 allocated	No significant effect on Paranoia Scale scores	Suggestion
Newman-Taylor, Sood [29], 2021, UK, RCT.	*None,* attachment-based imagery, 5 days, daily, website access, none.	Community & university sample with elevated but non-clinical paranoia Digital intervention arm: 42 allocated, 0 lost to follow-up Active control arm: 37 allocated, 0 lost to follow-up	Significant time x condition effect on Paranoia Scale score	Suggestion

Tabulation of Non-Suicidal Self-Injury

Six studies of interventions for NSSI were identified, with publication years ranging from 2016 to 2020 and enrolling a total of 633 participants (Table 6A.5). All studies were assessed at moderate risk of bias (19, 30-32).

Table 6A.5: Study characteristics of NSSI Interventions

Author, Year, Location, Type of Study	***Intervention name*, modality, duration, frequency & access method. Degree of Human Facilitation.**	**Population, Enrollment, Attrition & Adherence**	**Outcome measure(s) & Summary**	**PSD Elements Employed**
Franklin et al. [14] 2016, USA, RCT	*TEC (Study 1 version)*, evaluative conditioning, 2 months, open dosage, mobile application. None.	Adults with recent self-cutting Active *TEC* arm: 55 randomised, 14 (25%) lost to one-month follow-up. Sham *TEC* arm: 59 randomised, 21 (36%) lost to one-month follow-up.	Significantly fewer general NSSI episodes, self-cutting episodes, and suicide plans in the active group than the sham group.	Rewards
Franklin et al. [14] 2016, USA, RCT	*TEC (Study 2 version)*, evaluative conditioning, 2 months, open dosage, mobile application. None.	Adults with recent self-cutting Active *TEC* arm: 62 randomised, 18 (29%) lost to follow-up. Sham *TEC* arm: 69 randomised, 17 (25%) lost to follow-up.	Significantly fewer self-cutting episodes in the active group as compared to the sham group. No difference in self-cutting events (ie. Total number of cuts) or general NSSI episodes.	Rewards
Franklin, Fox [14] 2016, USA, RCT	*TEC (Study 3 version)*, evaluative conditioning, 2 months, open dosage, mobile application, none.	Community sample of adults with suicidal ideation. Active digital intervention arm*:* 75 randomised, 39 (52%) lost to one-month follow-up. Control digital intervention arm*:* 84 randomised, 30 (36%) lost to one-month follow-up.	SITBI: Significant reduction in suicide plans for active group, no significant difference in ideation between groups	Rewards
Hooley et al. [31] 2018, USA, RCT	*Autobiographical Self-Enhancement Training (ASET), Expressive Writing (EW),* journaling, 1	Adults with recent NSSI ASET: 49 randomised, 8 (16%) lost to one-month follow-up.	SITBI – NSSI & suicidal ideation frequency: Basic journaling group had significantly fewer days of suicidal ideation than EW	Self-monitoring, Rewards, Reminders

	month, daily, website access. Treatment support, as-needed.	EW: 49 randomised, 4 (8%) lost to one-month follow-up. Basic journaling control: 46 randomised, 9 (20%) lost to one-month follow-up.	group. No other significant effects.	
Drabu et al. [32] 2022, Singapore, RCT	None, self-compassion training, 1 week, daily, audio recordings accessible online. None	Adults with NSSI and self-criticism Intervention arm: 30 randomised, 0 discontinued Control arm: 33 randomised, 0 discontinued.	SITBI: Significant time x condition effect on inclination to self-injure	Self-monitoring, Rehearsal, Suggestion
Rodante [19] 2020, Argentina, Cluster RCT	Calma, DBT, 1 month, open dosage, mobile application, adjunctive to in-person treatment	Outpatients with suicidal ideation Digital intervention + DBT: 11 randomised & allocated, 2 (18%) lost to follow-up. DBT only: 11 randomised & allocated, 1 (9%) lost to follow-up.	No significant differences between groups.	Reduction Tunneling Tailoring Self-monitoring Rehearsal Reminders Suggestion Liking

Meta-Analysis of NSSI RCTs

A random-effects model fitted to the data found a small, insignificant reduction in number of NSSI episodes after digital intervention: $N_c = 5$, $g = -0.06$ with 95% CI: [-0.32; 0.19], $P = 0.52$. The between-study heterogeneity variance was estimated at $\tau^2 = 0.00$, 95% CI: [0.00; 0.46] and between-study heterogeneity at $I^2 = 6\%$ [0.0%; 80.5%] (Figure 6A.1). A funnel plot of studies of interventions for NSSI does not show evidence of publication bias (Figure 6A.2).

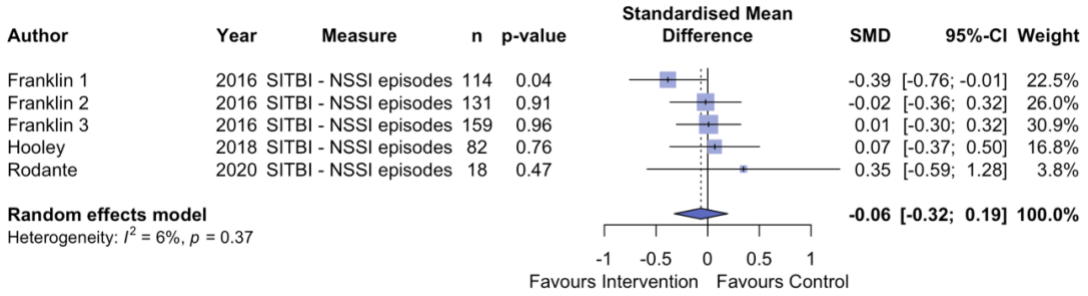


Figure 6A.1: Forest plot of SMD in NSSI episodes after digital intervention

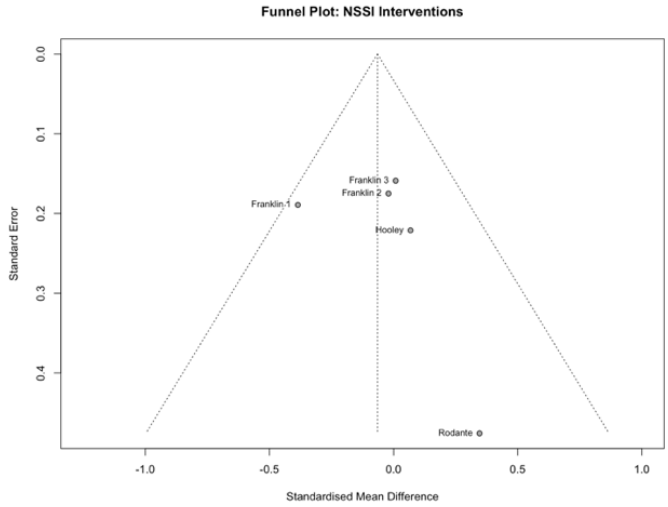


Figure 6A.2: Funnel plot of SMD of NSSI episodes does not show risk of publication bias.

Tabulation of Emotion Regulation Interventions

Six studies (4 RCTs) of interventions for emotion regulation were identified, with publication dates ranging from 2011-2021 and enrolling a total of 599 participants (Table 6A.6). Of these, one study was assessed to have low risk of bias (33), three at moderate risk of bias (34-36) and two at high risk of bias (37, 38).

Table 6A.6: Study Characteristics of emotion regulation interventions

Author, Year, Location,	*Intervention name*, modality,	Population, Enrollment, Attrition & Adherence	Outcome measure(s) & Summary	PSD Elements Employed

Type of study	duration, frequency & access method. Degree of Human Involvement in Treatment.			
Flujas-Contreras et al. [33] 2021, Spain, Single-arm pre-post	*Parenting Forest,* ACT, 6 weeks, weekly, website access. Reminders only.	Parents, either with inflexible parenting or with children with behavioural difficulties. 27 enrolled, 15 (56%) withdrawn/lost to follow-up. Remaining participants' time to completion was mean 64.2, SD = 20.5	DERS: non-significant decrease at post-treatment	Reduction, Tunneling, Tailoring, Personalisation, Self-monitoring, Reminders, Suggestion
Fonesca et al. [34] 2019, Portugal, RCT	*Be a Mom,* CBT, 5 weeks, weekly, website access. Technical support and reminders only as needed.	Early postpartum women with risk factors or symptoms of postpartum depression *Be a Mom* arm: 98 allocated, 33 (34%) lost to follow-up. Control arm: 96 allocated, 14 (14%) lost to follow-up.	DERS-SF: Significantly greater decrease in emotion regulation difficulties in intervention arm	Reduction, Tunneling, Tailoring, Personalisation, Self-monitoring, Rehearsal, Reminders, Suggestion, Similarity, Liking
Bernstein et al. [37] 2022, USA, Single-arm pre-post	*None,* CBT, 4-5 weeks, 6x daily, mobile application. None.	Outpatients (immediately post-discharge) with suicidal ideation or behaviour 25 enrolled, 6 (24%) lost to follow-up.	ERS: Significant decrease in ERS from baseline to post-assessment	Reduction, Tunneling, Personalisation, Self-monitoring, Rehearsal, Rewards, Suggestion, Similarity

Stappenbeck et al. [35] 2021, USA, RCT	*None*, DBT & Social Learning Theory, 2 weeks, daily, website access. Reminders only.	Female university students with lifetime self-reported sexual assault. Intervention arm: 100 allocated, 5 (5%) lost to follow-up. Mean completion 10.0/14.8 (SD=4.8) Control arm: 100 allocated, 10 (10%) lost to follow-up	DERS: significant time x condition effect with reduced emotion regulation difficulties in intervention arm	Tunneling, Tailoring, Personalisation, Rehearsal, Reminders, Suggestion,
Salamin et al. [36] 2019, Switzerland, open-label uncontrolled pilot	*e-motion*, DBT, 16 weeks, intended frequency not stated, website access. Treatment support as-needed (via online forum).	Close relatives of people with mental illness. Online intervention arm: 44 enrolled, 13 (30%) lost to follow-up In-person intervention arm: 60 enrolled, 22 (37%) lost to follow-up	DERS: significant time x condition medium effect size on difficulties with emotion regulation	Reduction, Tunneling, Self-monitoring, Simulation, Rehearsal, Praise, Suggestion, Social Role
Glück & Maercker [38] 2011, Germany & Austria, RCT	*None*, mindfulness, 2 weeks, 6 days per week, website access. Technical support as-needed and reminders.	Adults (no further inclusion criteria) Intervention arm: 28 allocated, 2 (7%) lost to follow-up, 10 (36%) completed <6 days of training Control arm: 21 allocated, 3 (14%) lost to follow-up.	SEK-27: no significant effect on emotion regulation for time, condition, or time x condition	Reduction, Tunneling, Rehearsal, Suggestion

Meta-Analysis of Emotion Regulation RCTs

A random-effects model fitted to the data found a small, insignificant effect of digital interventions for emotion regulation: $N_c = 4$, $g = -0.02$ with 95% CI: [-0.55; 0.51], $P = 0.90$. The between-study heterogeneity variance was estimated at $\tau^2 = 0.06$, 95% CI: [0.00; 1.69] and between-study heterogeneity at $I^2 = 56.8\%$ [0.0%; 85.7%] (Figure 6A.3). The funnel plot in Figure 6A.4 does not show evidence of publication bias.

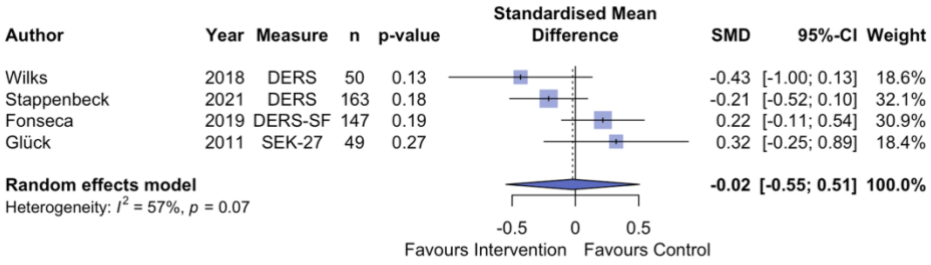


Figure 6A.3: Forest plot of SMD in difficulties with emotion regulation. Note: the SEK-27 measures emotion regulation competencies so its SMD was reversed for inclusion in this meta-analysis

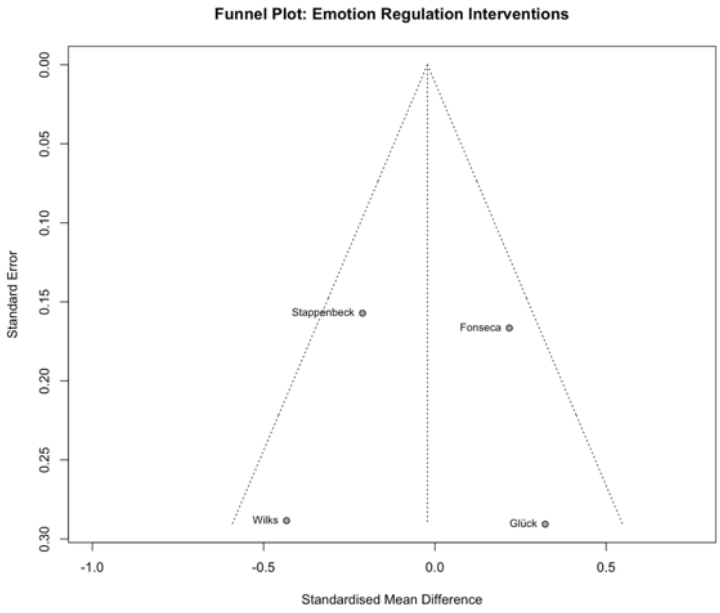


Figure 6A.4: Funnel plot of effect sizes of emotion regulation interventions does not show evidence of publication bias

Tabulation of Anger Interventions

Three studies, all RCTs, targeting symptoms of anger were identified, with publication years ranging from 2014 – 2020 (Table 6A.7). A total of 655 participants were enrolled. Two of the studies were deemed to have moderate risk of bias (39, 40) and one was at high risk of bias (41).

Table 6A.7: Study Characteristics of Anger Interventions

Author, Year, Location	***Intervention name*, modality, duration, frequency & access method. Degree of Human Involvement in Treatment**	**Population, Enrollment, Attrition & Adherence**	**Outcome measure(s) & Summary**	**PSD Elements Employed**
Howie & Malouff [39] 2014, USA, RCT	*None,* CBT, 1 month, daily, website access. Treatment support, as-needed.	Adults with elevated trait anger CBT arm: 37 randomised, 10 (27%) lost to follow-up, 26 (70%) did not complete intervention Control arm: 38 randomised, 6 (16%) lost to follow-up	TAS: Completer analysis found significant time x condition effects on trait anger, ITT analysis was borderline significant (*P* = 0.06)	Reduction, Self-monitoring, Rehearsal, Reminders, Suggestion.
Johnson et al. [40] 2020, USA, RDICT (randomised delayed-intervention controlled trial)	*None,* relaxation therapy, 6 weeks, weekly, website access. None.	Adults with aggression and emotion-related impulsivity. 235 randomised in total, intervention arm: 127, waitlist: 108. 121 (57%) non-response. 4.31 (out of 6) sessions completed on average.	MOAS: Significant time x condition effects on overt aggression scale.	Reduction, Tunneling, Self-monitoring, Rehearsal, Suggestion
Osgood et al. [41] 2020, USA, RCT, *Study 2 only.*	*None,* hostile bias modification training (HBMT), 1 session. None.	Adults (no other specified characteristics). 345 were randomised but 116 (34%) were withdrawn after randomization for inadequate performance on HBMT. Active HBMT arm: 117 completed, 8 (7%) lost to follow-up. Sham HBMT arm: 112 completed, 4 (4%) lost to follow-up	TAS: No significant effect on trait anger	No PSD elements mentioned or apparent based on publication

Meta-analysis of Anger-Targeting RCTs

A random-effects model fitted to the data found a small, insignificant effect of digital interventions for anger: $N_c = 3$, $g = -0.17$ with 95% CI: [-1.03; 0.69], $P = 0.49$. The between-study heterogeneity variance was estimated at $\tau^2 = 0.10$, 95% CI: [0.01; 4.43] and between-study heterogeneity at $I^2 = 80.9\%$ [40.3%; 93.9%] (Figure 6A.5). The funnel plot in Figure 6A.6 shows possible evidence of publication bias, but there are too few studies to conduct Egger's test.

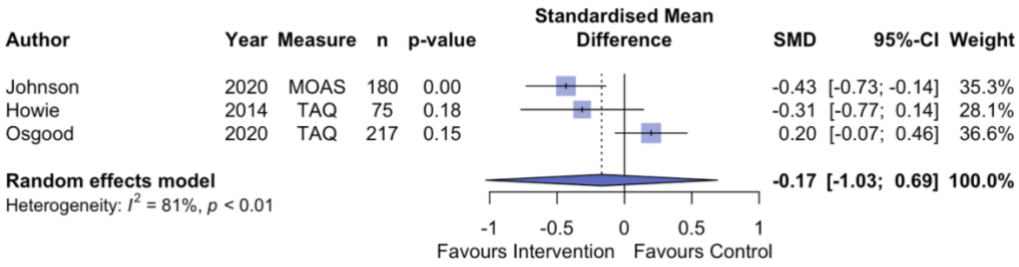


Figure 6A.5: Forest plot of SMD of digital anger interventions

Funnel Plot of Anger-Targeting RCTs

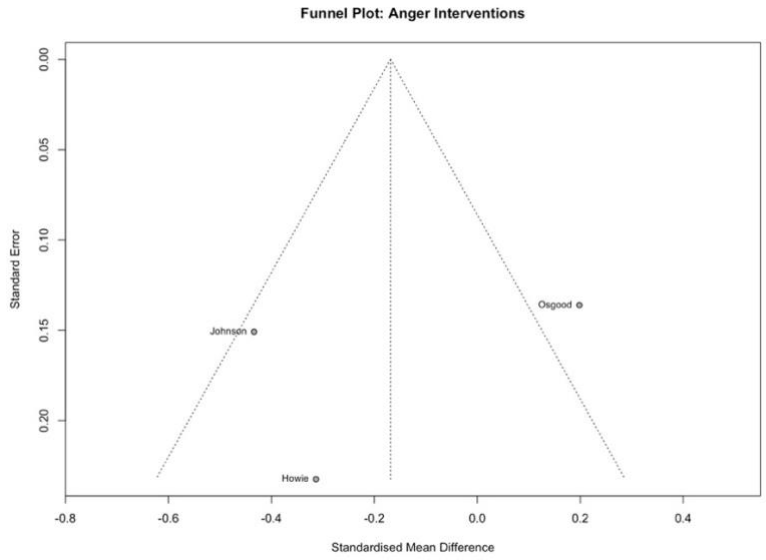


Figure 6A.6: Funnel plot of digital interventions for anger shows possible publication bias, but there are too few studies to conduct an Egger's test

Risk of Bias Results

i. Randomised Controlled Trials:

RCTs were assessed for risk of bias using Cochrane's ROB2 tool and visualized using the *robvis* tool (42). Figure 6A.7 (below) shows outcomes for each domain within the tool. There was widespread risk of bias, with only 3 (9%) of the RCTs judged to have overall low risk, 22 (69%) for which authors had some concerns, and 8 (25%) at high risk of bias. Most of this risk arose in Domain 5: Bias in selection of the reported result, in which 22 (69%) of studies were judged to have some concerns or high risk. There were some concerns about bias due to deviations from the intended interventions (10 studies, 31%) and missing data (11 studies, 34%).

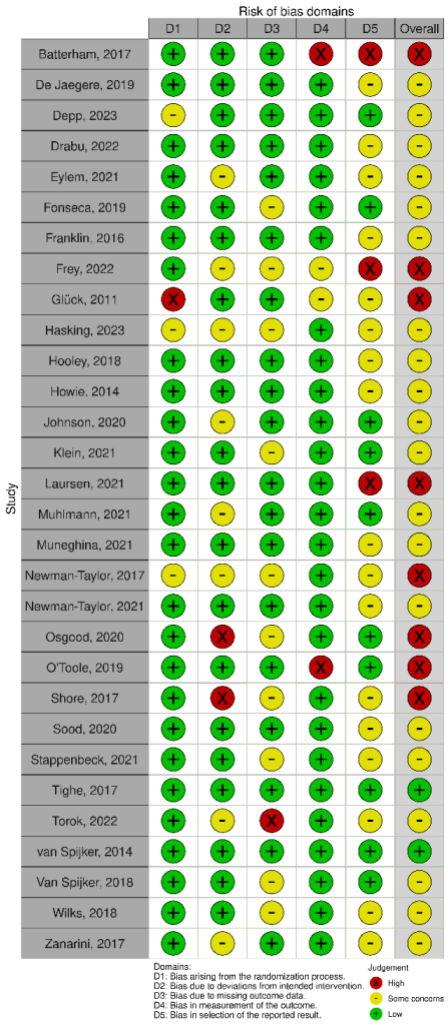


Figure 6A.7: Risk of Bias results for RCTs, presented by bias domain.

Figure 6A.8 shows a summary of results for each domain. Most of the risk came from Domain 5: selection of the reported result, which was due to trials not pre-registering their protocols and analysis plans.

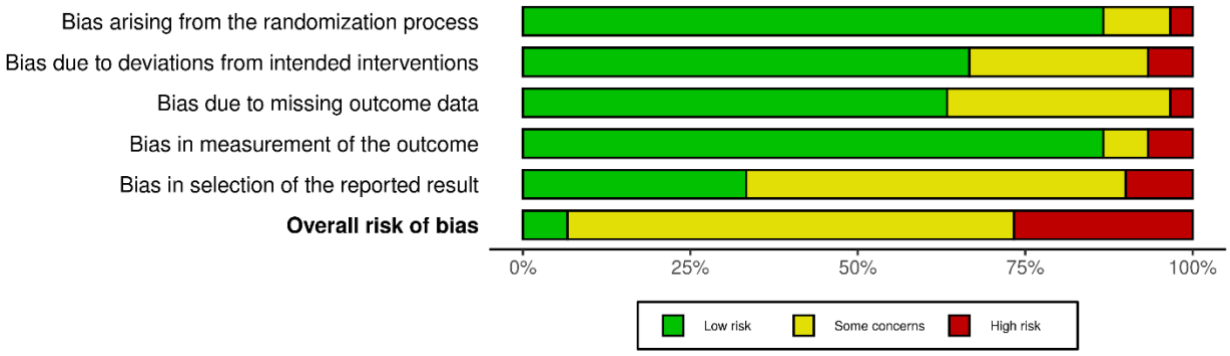


Figure 6A.8: Risk of bias summary for each domain

ii. Cluster trial:

There was one cluster trial included in the review, for which risk of bias was assessed using Cochrane's ROB2 for Cluster Trials (Figure 6A.9). We had some concerns about bias in the solitary cluster-randomised controlled trial, primarily due to the lack of a pre-registered analysis plan. Other concerns arose because it was not specified whether the randomization allocation sequence was concealed until allocation. Also, as with most studies of psychological interventions, some degree of risk arose due to participants self-reporting outcome measures without being blinded to their allocation.

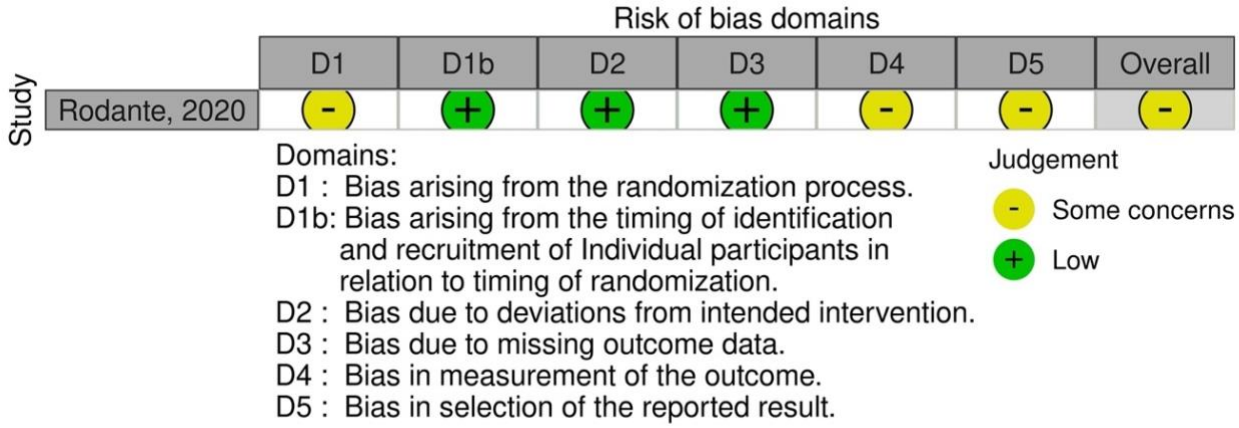


Figure 6A.9: Risk of bias domain results for cluster trial

iii. Single-Arm & Open-Label Studies:

Studies were assessed using the National Institute for Health's Study Quality Assessment tool. Figure 6A.10 below shows the outcomes for each study and domain, while Figure 6A.11 shows the summary breakdown by domain. These studies also carried a considerable risk of bias, with just 1 (11%) deemed "Good" using the NIH Quality Assessment tool. All the studies were at risk of bias from Domain 8: Assessor Blinding, which as discussed above is difficult to avoid with psychological interventions. Most studies (8 studies, 89%) also failed each of Domain 9: Loss to Follow-Up and Domain 11, which rewards studies that took multiple baseline and follow-up measures of the primary outcome variable. We deemed Domain 11 to be overly stringent for our assessment since taking multiple baseline measures is not common practice in studies of psychological interventions.

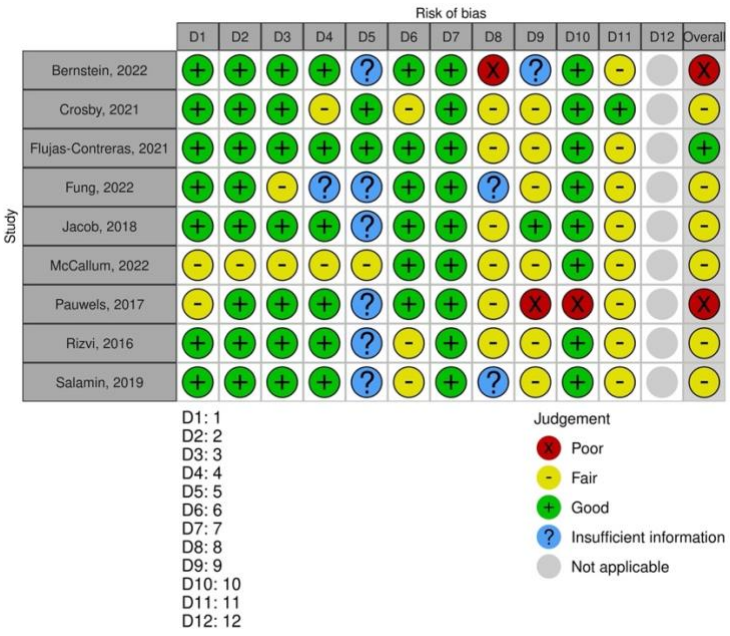


Figure 6A.10: Risk of bias domain results for single-arm and open-label studies, by domain

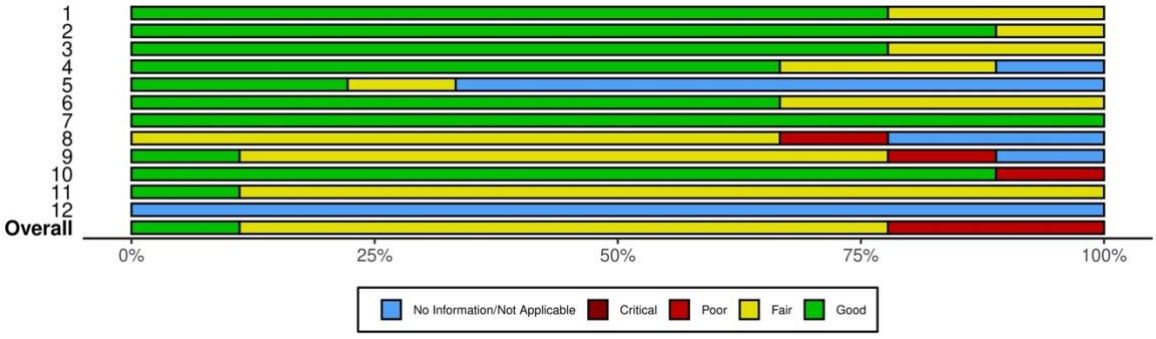


Figure 6A.11: Summary of quality assessment results for single-arm and open-label studies, by domain

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