Sleep Medicine 127 (2025) 1–15

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Systematic review: REM sleep, dysphoric dreams and nightmares as transdiagnostic features of psychiatric disorders with emotion dysregulation - Clinical implications

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ARTICLE INFO

Keywords:
MDD
PTSD
REM sleep
Nightmares
Emotion regulation
Dreams

ABSTRACT

Background: Fragmented rapid eye movement (REM) sleep disrupts the overnight resolution of emotional distress, a process crucial for emotion regulation. Emotion dysregulation, which is common across psychiatric disorders, is often associated with sleep disturbances. This systematic review explores how REM sleep and nightmares affect emotion processing and regulation in individuals with psychiatric disorders where emotion dysregulation is a key concern, suggesting novel sleep-related treatment pathways.

Methods: We performed a PRISMA-compliant systematic search of the PUBMED, Web of Science, and EBSCO databases from January 1994–February 2023. This systematic review targeted studies on REM sleep, nightmares, and emotion regulation in a postpubescent clinical population with affective dysregulation. The quality of the studies was assessed via the Newcastle–Ottawa Scale (NOS), adapted for cross-sectional studies.

Results: From the 714 screened records, 28 articles met the inclusion criteria and focused on REM sleep, dreams, or nightmares in individuals with mood disorders (k = 8), anxiety disorders (k = 1), posttraumatic stress disorder (PTSD) (k = 16), non-suicidal self-injury (NSSI), personality disorders (k = 2), and autism (k = 1). Fifteen studies used objective sleep measures, seventeen used self-reported assessments, six included treatment components, eight investigated nightmares, and three examined dreams. NOS scores ranged from moderate to high.

Conclusions: REM sleep disturbances represent a transdiagnostic feature across psychiatric disorders and are crucial for emotion regulation. Nightmares are associated with suicidal behaviour and emotion dysregulation. Targeted sleep interventions may improve emotion regulation and mental health outcomes. Future research should explore the role of REM sleep in disorder prognosis to develop tailored interventions.

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https://doi.org/10.1016/j.sleep.2024.12.037
Received 22 October 2024; Received in revised form 27 December 2024; Accepted 28 December 2024
Available online 31 December 2024

Abbreviations

ADI-R	Autism Diagnostic Interview-Revised
ASD	Autism Spectrum Disorder
BDI	Beck Depression Inventory
BPD	Borderline personality disorder
CAPS	Clinician-Administered PTSD Scale
CAPS-5	Clinician-Administered PTSD Scale for DSM-5
DASS-21	Depression, Anxiety and Stress Scale
DERS	Difficulties in Emotion Regulation Scale
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders (APA, 1994)
DLQ	Dream-like Quality Scale
HR	Heart Rate
HDRS	Hamilton Depression Rating Scale
HAMA	Hamilton Anxiety Scale
HAM-D	Hamilton Depression Scale
IAPS	International Affective Picture System
ISI	Insomnia Severity Index
NSSI	Nonsuicidal Self-Injury
MDD	Major Depressive Disorder
mPFC	Medial Prefrontal Cortex
MINI	International Neuropsychiatric Interview
PANAS	Positive and Negative Affect Schedule
SUDS	Subjective Units of Distress Scale
PCL-5	PTSD Checklist for the DSM-5
PTSD	Posttraumatic Stress Disorder
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
TECs	Trauma-Exposed Controls
TRNS	Trauma-related Nightmare Survey
SCID-I	Structured Clinical Interview for DSM-IV Axis I Disorders
SCL	Skin Conductance Level
SUDS	Subjective Units of Distress Scale
REM	Rapid Eye Movement
VAS	Visual Analogue Scale
VLPFC	Ventrolateral Prefrontal Cortex

1. Introduction

1.1. Disturbed sleep as a transdiagnostic mechanism in psychiatric disorders

Sleep disturbances are common in individuals with psychiatric disorders, with nearly 70 % reporting difficulty sleeping and over 30 % meeting the criteria for insomnia disorder [1]. Insomnia is especially prevalent in affective disorders such as major depressive disorder (45 %) and anxiety disorders (33 %), followed by bipolar disorder (23.8 %) and schizophrenia spectrum disorders (25 %) [2]. While traditionally viewed as symptoms, sleep disturbances are now recognised as bidirectional factors influencing mental health [2,3]. For instance, insomnia predicts the onset and prognosis of anxiety (OR 3.23) and depression (OR 2.83) [4], as well as borderline personality disorder (BPD) 5–8. Strong associations exist between insomnia, depression and anxiety disorders [9,10]. Poor sleep also exacerbates emotion dysregulation and suicidal tendencies, particularly in depressive populations [11,12]. Furthermore, insomnia exacerbates emotion dysregulation and is more strongly associated with anxiety and depression than traditional sleep quality indices (such as sleep efficiency) [13].

Polysomnography (PSG) studies reveal pervasive disruptions in sleep continuity across psychiatric disorders, such as prolonged sleep onset latency, frequent awakenings, and reduced total sleep duration [14–17]. These continuity disturbances likely reflect arousal imbalances that hinder emotion regulation [16]. This raises a critical question: which specific aspects of disturbed sleep most negatively impact mental health? Emerging evidence highlights specific REM sleep alterations—such as high density of rapid eye movements, fragmentation, dysphoric dream content, and thought-like nocturnal mentation—that undermine emotional processing [18,19]. REM sleep fragmentation, indicated by interrupting arousals and stage shifts during REM, disrupts REM sleep restorative processes [18–21]. Disturbed dreaming includes a wide range of dysphoric dream content (distressing or emotionally negative dreams), spanning from those recalled only upon waking (bad dreams) to those severe enough to cause nocturnal awakenings (nightmares) [22]. Thought-like nocturnal mentation, characterised by the recall of thought-like (fragmented, logical, or reality-oriented) rather than dream-like nocturnal mental content during REM, may contribute to emotion dysregulation [23,24]. The current review addresses the emerging insight that altered REM sleep impacts vulnerability to develop psychopathology by interfering with optimal emotion regulation.

1.2. REM sleep function in emotion regulation

Emotion processing involves multiple stages, from emotion reactivity and arousal to emotion modulation and regulation [25]. REM sleep plays a vital role in these processes. REM sleep is essential for maintaining emotional balance, reactivity, consolidation of emotional information, and reconsolidation of memories, particularly negative ones [26,27]. The influence of sleep on next-day mood and emotion is believed to be especially associated with REM sleep, where hyperactivity in limbic regions and reduced dorsolateral prefrontal cortex (dlPFC) function occur alongside normal activity in the medial prefrontal areas (mPFC), offering an optimal environment for processing and reprocessing and thus regulating emotional experiences [27,28].

Moreover, a recent model proposed that during healthy REM sleep, the silencing of the locus coeruleus allows for a “time-out” from noradrenergic activity, fostering synaptic plasticity in limbic circuits [20,29]. This neuromodulatory environment may promote fear extinction and adaptation to emotional experiences, as evidenced by the role of normal dreaming in emotion regulation [30]. In contrast, fragmented REM sleep disrupts these processes, leading to impaired emotional distress resolution and prolonged emotional arousal [29,31]. This disruption is thought to result from elevated norepinephrine levels, which impair synaptic plasticity and emotional memory integration [20,32].

In individuals with insomnia, fragmented REM sleep worsens negative thinking and contributes to the subjective experience of non-restorative sleep [18]. Dysfunctional emotional brain networks, including those in limbic and paralimbic areas, may underlie these disturbances [16,33]. Moreover, dysphoric dream content, such as nightmares, hinders emotional recovery during sleep [23]. Thus, the link between sleep disturbances, particularly altered REM sleep, and emotion dysregulation is key to understanding the connection between depression, anxiety, and sleep complaints.

1.3. REM sleep in psychiatric disorders

Alterations in REM sleep are commonly observed in individuals with psychiatric disorders, including affective, anxiety, autism, and schizophrenia spectrum disorders [16,34–39]. These disturbances, such as shortened REM latency, prolonged REM periods, and increased REM density, are associated with emotional dysregulation and memory deficits, influencing clinical outcomes [26,40] [16,26,28,34–41]. In particular, fragmented REM sleep and nightmares are associated with

increased risk and persistence of PTSD [42–44].

REM sleep-related nightmares, which are vivid and distressing, are prevalent across psychiatric conditions and have been associated with worsening mental health and increased suicide risk [30,45]. Dreaming may play an adaptive role in processing emotional experiences and resolving internal conflicts [46], but disrupted REM sleep undermines this capacity, contributing to prolonged emotional distress.

1.4. Objectives

As summarised above, REM sleep has been established as a key factor in emotion processing, emotion regulation and thus adaptation to distress [23,26]. However, no previous report has comprehensively reviewed the impact of REM sleep features (fragmentation, dysphoric dream content (e.g., nightmares) and thought-like nocturnal mentation) on emotion dysregulation as potential transdiagnostic markers in psychiatric disorders where emotion dysregulation is a primary concern. Bridging this gap may inspire novel sleep-targeted interventions that improve emotion regulation, overall emotion reactivity and well-being across individuals with affective psychiatric disorders.

This systematic review aims to address this relevant gap in the literature. We summarise all findings from relevant experimental and observational studies in adolescents and adults with psychiatric disorders in the following sections: (1) REM sleep and emotion processing; (2) dreams, nightmares and emotion processing; and (3) clinical intervention studies addressing REM sleep, dreams, nightmares and emotional processing.

2. Methods

2.1. Study design and search strategy

We conducted a PRISMA 2020-compliant systematic review of original studies investigating the relationship between processing and emotion regulation as a transdiagnostic feature in psychiatric disorders characterised by emotion dysregulation [47,48] with REM sleep features, including onset, duration, eye movement density, and (micro) arousals, as well as REM-associated subjective experiences such as dysphoric dream content (i.e., nightmares) and nocturnal mentation. The search included PUBMED, Web of Science, and EBSCOhost for papers published between January 1994 (corresponding to the release date of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, DSM-IV) and February 2023. The search strings applied combinations of the following search terms related to (1) emotion and emotion regulation; (2) REM sleep, nightmares, nocturnal mentation or sleep fragmentation; and (3) psychiatric disorders where emotion dysregulation is a primary concern: mood disorders, personality disorders, non-suicidal self-injury (NSSI), posttraumatic stress disorders, anxiety disorders, obsessive-compulsive disorders, neurodevelopmental disorders, eating disorders, and substance use disorders. The full search strings are available in the Supplement.

2.2. Inclusion and exclusion criteria

Study selection was completed by two independent authors (MMA and YB). The inclusion criteria were as follows: (1) peer-reviewed research excluding case studies, reviews, and meta-analyses, encompassing observational and experimental designs across various methodologies but excluding single case designs published in English, Dutch, Spanish, French, Italian, German or Catalan; and (2) adults and post-pubescent individuals with a confirmed diagnosis of a psychiatric disorder (as diagnosed via standardised systems such as the DSM or ICD).

The exclusion criteria were studies without original data, those published in languages not understood by the researchers, and those focused on psychosis spectrum disorders, subclinical populations or a sleep disorder diagnosis without psychiatric comorbidity.

2.3. Data collection

Authors MMA and JB independently screened titles and abstracts via Covidence software (average agreement percentage = 89 %; Kappa = .55). Studies meeting the predefined selection criteria were subjected to independent full-text review. The same authors completed full-text ratings (average interrater agreement 79 %; Kappa = .58). Discrepancies were discussed until a consensus was reached.

2.4. Risk of bias assessment

The Newcastle–Ottawa Scale (NOS) was used to evaluate the quality of the included studies, with higher scores indicating better methodological quality and a lower risk of bias. The NOS criteria were adjusted for applicability to cross-sectional study data (Supplementary Material) (Stang, 2010). Methodological quality ranged from moderate to high (Table 1).

2.5. Data extraction

Data extracted from each selected article included study design; sample characteristics (authors, publication date, primary diagnosis, setting, age, sample size, gender ratio); methodological and design aspects (tasks, stimuli, treatments, observational study details); and outcomes related to sleep, and emotion (using both objective and subjective measures; validated or open-ended instruments; and quantitative or qualitative data). The specified information was collected by both authors (MMA and YB) via a piloted data extraction template. A full overview of the extracted data can be found in Table 2.

3. Results

3.1. Description of studies

The study selection process is described in the PRISMA flowchart (Fig. 1). A total of 568 unique articles were screened to generate a final sample of 28 articles meeting the inclusion criteria, covering mood disorders (k = 8); anxiety disorders (k = 1), PTSD (k = 16); non-suicidal self-injury and personality disorders (k = 2); and autism (k = 1). We did not identify any additional articles that fulfilled the inclusion criteria through cross-referencing methods. A description of the studies included can be found in Table 2.

We summarise (1) all the observational (k = 6), experimental (k = 3), and quasiexperimental (k = 1) studies examining REM sleep and emotion processing; (2) all the observational (k = 9) and experimental (k = 1) studies on dreams, nightmares and emotion processing; and (3) all the clinical intervention studies (k = 8) addressing REM sleep, dreams, nightmares and emotional processing.

3.2. REM sleep and alterations in emotion processing

Two imaging studies using positron emission tomography (PET) revealed differences in brain metabolism during **REM sleep** between participants with MDD and PTSD patients. Participants with MDD presented (n = 13) hypermetabolic regions associated with affective processes (limbic, paralimbic, and basal ganglia) [49], not only during **REM sleep** but also during wakefulness, whereas participants with PTSD (n = 13) presented increased metabolism in these areas only during **REM sleep** [50]. Additionally, in patients with MDD (n = 13), **average REM density** correlated with hypofrontality (reduced activity in the prefrontal cortex) and reduced activity in brain areas (frontoparietal regions) implicated in regulating emotion-induced arousal in depression) [51].

A functional MRI study revealed that reduced **REM sleep** was associated with impaired extinction learning and heightened amygdala activation during fear conditioning, indicating that REM sleep integrity

Table 1
Assessment of the quality of the included cohort studies via the Newcastle–Ottawa Scale (NOS).

Study (first author)	Study design	Selection				Comparability	Outcome		TOTAL	EVALUATION
		Representativeness of the sample	Sample size	Non-respondents	Ascertainment of exposure	Based on design and analysis	Assessment of outcome	Statistical test		
Seo et al. (2022)	Cross-sectional	+	+	+	++	+	++	+	9/10	High
Tavakoli et al. (2022)	Cross-sectional	+			++	+	++	+	7/10	High
Borghese et al. (2022)	Longitudinal	+	+	+	++	+	++	+	9/10	High
Possemato et al. (2022)	Cross-sectional	+			+	+	++	+	6/10	Moderate
Paquet et al. (2022)	Longitudinal	+		+	++	+	++	+	8/10	High
Birchler-Pedross et al. (2022)	Cross-sectional	+			++	+	++	+	7/10	High
Ney et al (2021)	Cross-sectional	+	+	+	++	+	++	+	9/10	High
Sherrill et al. (2021)	Longitudinal	+	+		++	+	+	+	6/10	Moderate
Richards et al. (2021)	Cross-sectional	+	+	+	++	+	++	+	9/10	High
Lau et al. (2020)	Cross-sectional	+	+	+	++	+	++	+	9/10	High
Schäfer et al. (2019)	Cross-sectional	+		+	++	+	++	+	8/10	High
Lipinska et al. (2019)	Cross-sectional	+	+	+	++	+	++	+	9/10	High
Moraczewski et al. (2019)	Secondary analysis of data	+			++	+	++	+	7/10	High
Woodward et al. (2017)	Longitudinal	+	+		+	+	++	+	7/10	High
Ennis et al (2017)	Cross-sectional	+	+	+	++	+	+	+	8/10	High
Kobayashi et al. (2016)	Longitudinal	+		+	++	+	++	+	8/10	High
Littlewood et al. (2016)	Cross-sectional	+			+	+	++	+	6/10	Moderate
Short et al. (2015)	Cross-sectional	+	+		++	+	++	+	8/10	High
Cowdin et al. (2014)	Cross-sectional	+		+	++	+	++	+	8/10	High
Ebdlahad et al. (2013)	Cross-sectional	+			++	+	++	+	7/10	High
Selby et al. (2013)	Longitudinal	+		+	+	+	+	+	6/10	Moderate
Blanaru et al. (2012)	Longitudinal				++	+	++	+	6/10	Moderate
Rhudy et al. (2008)	Longitudinal	+		+	++	+	++	+	8/10	High
Daoust et al. (2008)	Cross-sectional	+			++	+	++	+	7/10	High
Cartwright et al. (2006)	Longitudinal	+			++	+	++	+	7/10	High
Germain et al. (2004)	Cross-sectional	+			++	+	++	+	7/10	High
Agargun et al. (2003)	Cross-sectional	+		+	++	+	++	+	8/10	High
Rao et al. (1999)	Longitudinal	+			++	+	++	+	7/10	High

strongly influences brain responses to fear and extinction in individuals with more severe PTSD symptoms [52]. During early fear conditioning, subcortical and cortical regulatory (mPFC) and salience (dACC) network regions were activated in both PTSD (n = 63) and trauma-exposed control (TEC) individuals (n = 63). By the end of conditioning, TEC showed decreased activation, while PTSD participants maintained elevated neural activation, indicating prolonged emotional reactivity. During extinction learning, TEC engaged the mPFC and superior prefrontal cortex (SFC) for emotion regulation, while PTSD patients showed limited SFC activation. At extinction recall, PTSD patients activated the regulatory rACC region, unlike TEC. The only significant group difference was greater hippocampal activation in TEC during extinction recall [52].

3.2.1. Disturbed REM sleep and hyperarousal symptoms in emotion processing

Richards and colleagues (2022) found that consolidated **REM sleep** facilitated better threat-safety discrimination and reduced hyperarousal symptoms in trauma-exposed individuals with PTSD [53]. Using a nap protocol, this study revealed that signal safety learning was associated

Table 2
Characteristics of the included studies.

Study (first author)	Sample Size, diagnosis & recruitment setting (inpatient or outpatient)	Mean/median age and number of females and males (%)	Study Design	Evaluation of Sleep	Emotional Processing (i.e., affect, Mood) Evaluation	Clinical outcomes
Seo et al. (2022)	N = 126, PTSD (N = 63 PTSD and N = 63 trauma-exposed controls), not in treatment.	86 F/40 M, mean age: 24,17 years (aged 18–40)	Experimental; Prospective; Cross-sectional study	PSG, Actigraphy, sleep diary, Evening−Morning Sleep Questionnaire (EMSQ),	CAPS, PTSD Checklist for DSM-5 (PCL-5), Skin conductance level (SCL)	SCID I, CAPS-5, Peritraumatic Distress Scale (PDI)
Tavakoli et al. (2022)	N = 10, MDD, suicidal, post suicide attempt, inpatient.	8 F/2 M, mean age: 15.1 years - (aged: 12–17, SD = 1.6 years)	Experimental; Prospective; Cross sectional study	PSG	Emotional Go/NoGo task (facial expressions showing happy, sad, and neutral emotions)	Children's Depression Inventory - Second edition (CDI-2), Suicidal Ideation Questionnaire-JR (SIQ-JR), Suicidal Behaviors Questionnaire-Revised (SBQ-R)
Borghese et al. (2022)	N = 48, Social anxiety disorder, not in treatment	32 F/16 M, mean age TMR group: 24.7 ± 5.78 years vs control group: 24.12 ± 3.97	Experimental; Prospective; Randomised controlled trial; Longitudinal study	Insomnia Severity Index (ISI), Pittsburgh Sleep Quality Index (PSQI), dream diary, sleep agenda, PSG	heart rate, skin conductance, and SUDS	MINI module on social anxiety disorder, Liebowitz Social Anxiety Scale (LSAS), Beck Anxiety Inventory (BAI), BDI-II
Possemato et al. (2022)	N = 76, PTSD (at least subthreshold) and at-risk drinkers, army veterans at primary care clinics	8 F/68 M, mean age: 31 years, (SD: 8)	Observational; Retrospective; Cross-sectional study; Other: Secondary analysis of data	Five dream characteristics (realism, replication, symbolism, threat and recall)	Intensity and type of emotion (i.e., fear, shame, anger, sadness)	CAPS, PTSD intensity and severity, nightmare characteristics
Paquet et al. (2022)	N = 55, treatment-seeking individuals with a history of criterion A trauma and weekly nightmares, outpatient	43 F/11 M/ 1Transgender, mean age = 39.49 years (SD = 14.87)	Prospective; Nonrandomised experimental study; Longitudinal study	Trauma-related Nightmare Survey (TRNS)	Cognitive processing words (CPW) and emotional tone (ET) measured in PTNM	CAPS- 4 and 5, Posttraumatic Stress Diagnostic Scale (PDS), PCL-5, TRNS, BDI-II, Center for Epidemiological Studies for Depression Revised (CESD-R)
Birchler-Pedross et al. (2022)	N = 9 healthy controls and N = 8 unmedicated MDD	MDD: 24,3 years (age range 20–31) – Healthy controls: 25 years (age range 20–31)	Observational; Prospective; Cross sectional study	PSG, actigraphy, sleep logs, and Sleep Mentation Questionnaire (SQM),	MADRS, BDI, VAS	Salivary melatonin, Hamilton Depression Rating Scale with Atypical Depression Supplement (SIGH-ADS), BDI, Clinical interview DSM-IV
Ney et al. (2021)	N = 120, PTSD (n = 34), trauma-exposed (n = 52), nontrauma exposed controls (n = 42), outpatient	70 F/54 M, mean age: 26,4 years	Observational; Prospective; Cross-sectional study	PSQI, sleep diary, REM Sleep Behaviour Disorder Screening Questionnaire (RBDSQ)	Depression, Anxiety and Stress Scale (DASS-21), IAPS images recall	PCL-C, Traumatic Experiences Questionnaire (TEQ), DASS-21
Sherrill et al. (2021)	N = 80, PTSD, outpatient	29,9%F/70,1%M, mean age: 40.43 years (SD: 8.88)	Experimental; Prospective; Nonrandomised experimental study; Longitudinal study	ESS, nightmare and insomnia items on PCL-5	PCL-5	ESS-scores, PCL-5
Richards et al (2021)	N = 46, PTSD (PTSD, (n = 15), PTSD in remission (n = 18) or TEC (n = 13) with no PTSD, outpatient	22 F/24 M, mean age: 33,2 years	Experimental; Cross-sectional study; Other: secondary analysis of data	PSG	CAPS	CAPS
Lau et al (2020)	N = 112, MDD vs healthy controls, majority untreated	64,00–86,67 % Female, - Age ranging 16–60 years	Experimental; Prospective; Cross-sectional study	PSG recording, actigraph, PSQI, Sleep Conditioner Indicator (SCI)	PANAS, Emotional Intensity on Visual Analogue Scale	HAM-D, Beck Depression Inventory-II (BDI II), SCID
Schäfer et al (2019)	N = 54, PTSD, inpatient.	46 F/8 M, mean age: 41.45 years (SD = 9.06, range 20–59)	Experimental; Prospective; Randomised controlled trial; Cross-sectional study;	Actigraphy, sleep questionnaire A (SF-A), Mannheim Dream Questionnaire (MADRE)	MADRE: dream intensity and emotional tone of dreams	SCID-I (DSM-IV), BDI-II, PCL-5

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Table 2 (*continued*)

Study (first author)	Sample Size, diagnosis & recruitment setting (inpatient or outpatient)	Mean/median age and number of females and males (%)	Study Design	Evaluation of Sleep	Emotional Processing (i.e., affect, Mood) Evaluation	Clinical outcomes
Lipinska et al. (2019)	N = 60, PTSD and trauma-exposed non-PTSD, outpatient, and healthy controls	60 F, mean age: 25.10 years, SD = 4.45	Other: patient blind, placebo control Prospective; Cross-sectional study; Other: Quasiexperimental	PSG, PSQI	CAPS, BDI, International Affective Picture System (IAPS) scores, electrocardiogram (ECG), physical arousal parameters: SCL, heart rate (HR), preejection period (PEP) using the interval from left ventricular depolarisation to the opening of the aortic valve) and left ventricular ejection time (LVET).	The MINI International Neuropsychiatric Interview (MINI), Wechsler Abbreviated Scale of Intelligence (WASI), CAPS, BDI-II, physical arousal parameters (HR, PEP, LVET, SCL) and noradrenaline metabolites
Moraczewski et al. (2019)	N = 20, PTSD, outpatient	17 F/3 M, mean age: 39,8 years	Observational; Retrospective; Other: Secondary analysis of data	Disturbing Dreams and Nightmares Severity Index (DDNSI), ISI	CAPS, HRSD, Visual analogue scale (VAS) measurements	SCID, HRSD, CGI-S, MMSE, CAPS, PCL, Scale for Suicide Ideation (SSI), Columbia-Suicide Severity Rating Scale (C-SSRS),
Woodward et al. (2017)	N = 121, PTSD, outpatient	71 F/51 M Mean age: 38,95 years (aged 18–65)	Randomised controlled trial; Longitudinal study; Other: Secondary analysis of data	Sleep duration (sleep diary); Sleep quality (self-reported, 0–100); scale) Insomnia symptoms (CAPS item); Nightmares (CAPS and PDS item)	PDS and CAPS items: hyperarousal and prebed anxiety	PTSD Symptoms (CAPS and PDS) – sleep duration – sleep quality – insomnia symptoms – arousal, nightmares and prebed anxiety
Ennis et al. (2017)	Study 1: N = 313, self-injuring patients, outpatient. Study 2: N = 133, psychology students with NSSI or not, not in treatment	Study 1: 62.3%F/ 35.8%M/1.9%U, mean age: 26.08 years (aged 18–65) Study 2: 79.7%F/ 18.5%M,.8%U, Mean age: 19.64 (aged 18–38)	Observational; Prospective; Cross sectional study	ISI, Disturbing dreams and nightmare severity Index (DDNSI)	Depression and Anxiety Stress Scale DASS, BDI-II, DERS	DASS, BDI-II, NSSI screening and depression prevalence, prevalence of nightmares and insomnia symptoms
Kobayashi et al. (2016)	N = 29, full or subthreshold PTSD, not in treatment	15 F/14 M, mean age 28.07 years (SD = 10.99)	Experimental; Prospective; Randomised controlled trial; Longitudinal study	PSG	Subjective Units of Distress Scale (SUDS)	CAPS Scores - PTSD symptom evolution
Littlewood et al.(2016)	N = 91, PTSD	66 F/24 M/1 not specified, mean age: 28.87 years (SD:10.64)	Observational Cross-sectional study	CAPS items on falling asleep and maintaining sleep - insomnia/ nightmares	CAPS, Defeat scale, Entrapment scale, Beck Hopelessness scale	Posttraumatic Diagnostic Scale, Suicidal Behaviors Questionnaire-Revised (SBQ-R), Comorbid depression based on history of depression
Short et al. (2015)	N = 255, outpatient	65.1%F/34.9 %, mean age: 26.38 (SD = 10.34) years	Observational; Prospective; Cross sectional study	ISI, Disturbing Dreams and Nightmares Severity Index (DDNSI)	Ruminative response scale (RRS)	SCID-I, Clinical Global Impression (CGI), self-injurious behaviours (SCID-II screener for Axis II disorders), ruminative response scale (RRS)
Cowdin et al (2014)	Trauma-exposed/ resilient N = 22 - Trauma-exposed/ PTSD N = 28 current active symptoms - not in treatment	8 F/5 M and mean age: 20.77 years (SD = 2.68) resilient group 11F/6 M and mean age: 20.77 years (SD = 2.68) PTSD group	Observational; Prospective; Cross-sectional study	PSG, EEG	CAPS	SCID-I (DSM-IV), CAPS
Ebdlahad et al. (2013)	N = 25, 13 PTSD-veteran patients, 12 MDD-civilian patients, untreated	PTSD-patients: 3 F/10 M, mean age: 29.5 years (aged 22–45) MDD-patients: 9 F/3 M, mean age 34.0 years (aged 25–46)	Observational; Prospective; Cross-sectional study	PSQI (Pittsburgh Sleep Quality Index) PSG recordings on five nights	FDG-PET scan	Clinician-Administered PTSD Scale (CAPS), Structured Clinical Interview for DSM-IV Axis I disorders (SCID I) HRDS, Beck

(*continued on next page*)

Table 2 (*continued*)

Study (first author)	Sample Size, diagnosis & recruitment setting (inpatient or outpatient)	Mean/median age and number of females and males (%)	Study Design	Evaluation of Sleep	Emotional Processing (i.e., affect, Mood) Evaluation	Clinical outcomes
						Depression Inventory (BDI)
Selby et al. (2013)	N = 47, BPD (N = 16 BPD and N = 41 dysregulated behaviours), not in treatment	31 F/16 M, age not mentioned.	Observational; Prospective; Longitudinal study	Self-report questions on nightmares and sleep	Cognitive Emotion Regulation Questionnaire (CERQ), Daily Negative Emotion Assessment	Beck Scale for Suicide Ideation (BSS), MINI, SCID-II module for BPD, Daily Rumination Assessment
Blanaru et al. (2012)	N = 13 PTSD outpatients	5 F/8 M, mean age 45.7 years, SD = 11.4	Experimental; Prospective; Randomised controlled trial; Longitudinal study	Actigraph, Mini Sleep Questionnaire (MSQ), Technion Sleep Questionnaire	BDI, the State-Trait Anxiety Inventory (STAI) and the Hamilton Anxiety Scale (HAMA)	Beck Depression Inventory (BDI), the State-Trait Anxiety Inventory (STAXI) and HAMA
Rhudy et al. (2008)	N = 35, PTSD or trauma-exposed without PTSD, not yet in treatment.	25 F/10 M, mean age: 39 years, (SD = 12.02)	Experimental; Prospective; Randomised controlled trial; Longitudinal study	PSQI, PSQI, Trauma-related Nightmare Survey, Nightmare Content Interview (NCI)	Physiological arousal parameters (HR, skin conductance, muscle movement by EMG), ratings of emotional valence (unpleasantness–pleasantness) and arousal. Self-Assessment Manikin (SAM)	CAPS, BDI-II, PSQI, PSQI, Modified PTSD Symptom Scale–Self-Report (MPSS–SR), Trauma-Related Nightmare Survey, Dissociative Experiences Scale (DES), Pennebaker Inventory of Limbic Languidness (PILL).
Daoust et al (2008)	28 ASD, not in treatment, and 79 typically developed controls, not in treatment	ASD: 25 M/3 F, mean age = 22.7 years, range: 12–53) and healthy controls: 31 F/48 M, mean = 22.8 years, range: 15–57)	Observational; Prospective; Cross sectional study	Dream Habit Questionnaire, Dream content description, PSG 3 nights, Night 1 adaptation, Night 2 sleep architecture study, Night 3 dream reports following REM sleep awakening	Emotions and their frequency are reported in dreams.	ADI-R cut-off for autism and ASD diagnosis confirmed through DSM-IV criteria
Cartwright et al. (2006)	N = 30, depressive disorder vs control, no treatment	13 F/7 M in a depressed population	Observational; Prospective; Longitudinal study	-PSG -Foulkes Dream-like Fantasy Scale	-Profile of Mood States (POMS) - Current Concerns test	-SCID-III-R -BDI -HRSD
Germain et al. (2004)	N = 13, MDD	7 F/6 M, mean age: 39.2 years, S. D. = 9.2; range: 24.9–58.8	Observational, Cross-sectional study	PSG (Polysomnography)	HRSD (The Hamilton Depression Rating Scale), positron emission tomography (FDG-PET) scans	Sleep latency and duration, arousal index, sleep efficiency, REM latency, REM counts, regional cerebral metabolic rate of glucose (rCMRglc), Hamilton Rating Scale for Depression (HRSD)
Agargun et al. (2003)	N = 26, Depressive disorder, outpatient	16 F/10 M	Observational; Prospective; Cross sectional study	-PSG for three consecutive nights (Night 1 clinical control, Night 2 sleep architecture & Night 3 experimental), dream-like quality scale (DLQ) 5-point scale where low scores represent no-recall and higher scores represent a well-developed dream narrative	Affect Scores each half of the night by affect type (negative, neutral, positive, or unemotional) and strength (mildly emotional or strongly emotional)	BDI and HDRS Suicidality scores, REM sleep variables, DLQ
Rao et al. (1999)	N = 59, unipolar depressive disorder (N = 26, 92,9 % from baseline sample) and control group (N = 33, 94,3 % from baseline sample), in- and outpatient.	Depressed group: 17 F/9 M, mean age: 22.4 years (SD = 1.5) Control group: 20 F/13 M, mean age: 21.9 years (SD = 1.7)	Observational; Prospective; Longitudinal study; Prevalence study	PSG with EEG for three nights	Schedule for Affective Disorders and Schizophrenia-Lifetime version, HDRS	Parameters of depression, HDRS, DSM-III-R (suicidal ideation, anxiety disorder), GAF score, MPQ, REM latency, density, % 24 h cortisol near sleep onset.

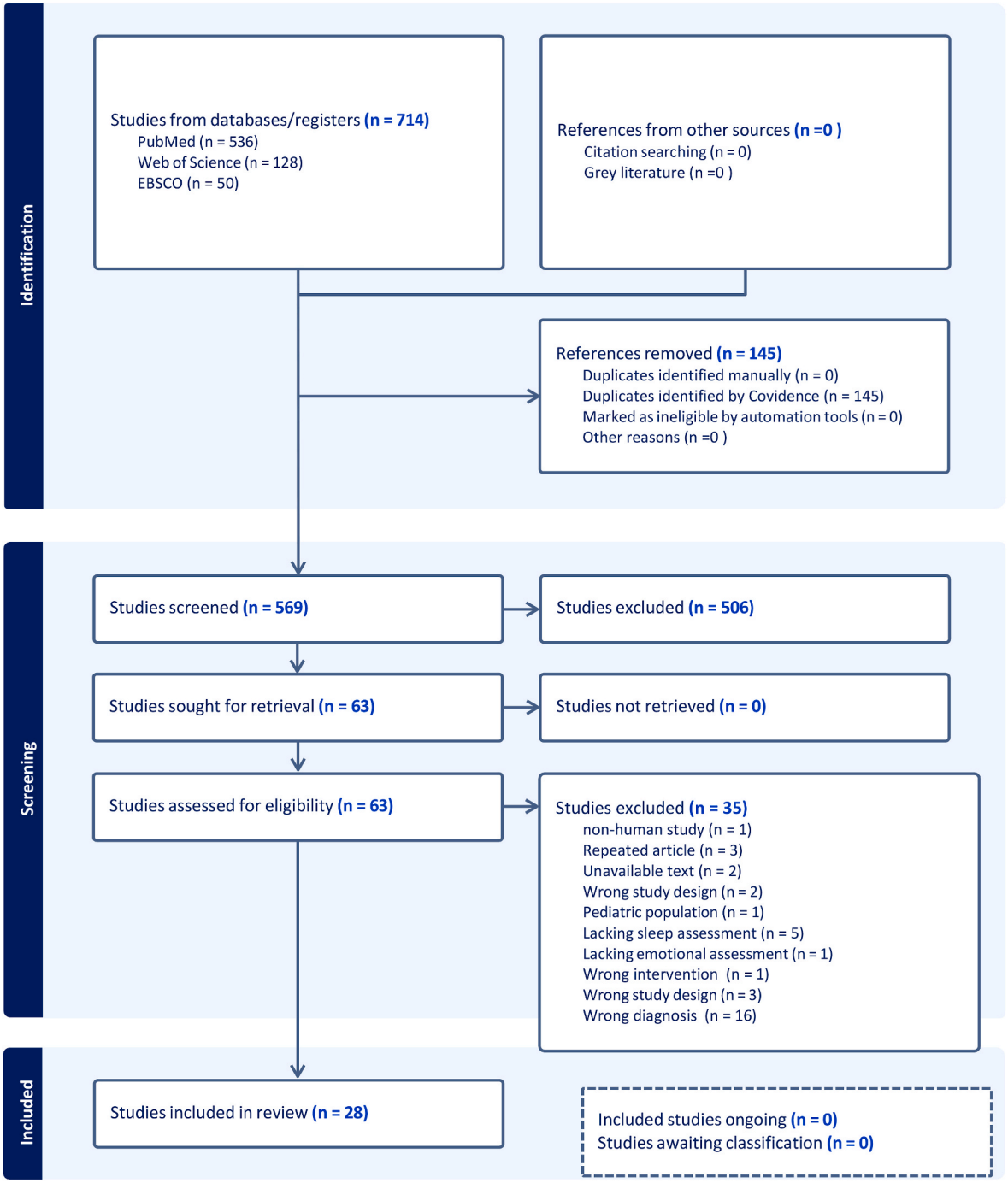

Fig. 1. PRISMA 2020 flow diagram for new systematic reviews, which included searches of databases.

with more consolidated **REM sleep**, which in turn improved the ability to discriminate between threat and safety signals the following day. Furthermore, **REM sleep** enhanced postsleep reactivity to conditioned stimuli and helped regulate responses in safe contexts. This bidirectional relationship underscores **REM sleep's** role in regulating conditioned responses. In addition, greater hyperarousal symptoms predicted poorer retention of safety signal learning, while higher PSG sleep quality facilitated a robust and rapid reduction in conditioned responses in safe contexts in the extinction session [53]. Furthermore, napping improves mood in both depressed (n = 46) and non-depressed individuals (n = 66) but increases the perception of angry faces in depressed individuals [54].

Lipinska and Thomas (2019) exposed their participants to emotional and neutral images before an 8-h overnight sleep delay and a subsequent recognition task. Higher **REM fragmentation** and nocturnal sympathetic activation were associated with lower recognition accuracy for emotional stimuli across PTSD (n = 21), non-PTSD (n = 19), and healthy control (n = 20) groups [55]. Furthermore, across the whole sample, higher activation and lower **REM sleep fragmentation** enhanced the recognition of **neutra**l images [55].

In a cross-sectional study, participants with PTSD had poorer subjective sleep quality, self-reported shorter total sleep duration, more **nightmares**, and greater levels of atypical REM behaviours symptoms than both trauma-exposed and non-trauma-exposed controls. Interestingly, these self-reported symptoms of **REM-related disturbances**, including **nightmares** and atypical REM behaviours, mediated the link

between PTSD status and intrusive memories. Indicating that REM symptoms (RBD symptomatology) may serve as a clinical marker for (impaired) trauma memory consolidation in trauma-exposed individuals, leading to PTSD [56].

PSG results from Cowdin and colleagues indicate that greater right prefrontal theta power (4–8 Hz) was present during the **first and last REM periods** in trauma-exposed resilient individuals (n = 13) than in participants with PTSD (n = 17). The authors suggest that right hemisphere prefrontal theta power during **REM sleep** is a potential biomarker of adaptive emotion memory processing/consolidation in trauma-exposed individuals who do not develop PTSD than in those who do [57].

3.3. Subjective REM indices: nightmares in emotion processing

Negative emotions during **dreaming** are amplified in individuals with MDD and extend across wakefulness and dreaming, highlighting the value of assessing dream recall and emotional load for understanding emotion regulation and early detection of MDD [58]. Compared with healthy controls (n = 9), women with MDD (n = 8) had twice the negative **dream** emotional load, regardless of sleep stage. A higher negative dream emotional load correlated with impaired mood levels during a 40-h multiple nap protocol in both groups [58]. Cartwright and colleagues (2006) reported a link between waking emotional states and **dream** experiences in (n = 20) untreated individuals diagnosed with major depression after marital separation or divorce [59]. The results suggest that non-remitted depressed individuals fail to incorporate concerns into their **dreams**, resulting in ineffective emotional processing during sleep and contributing to low mood in the morning and a lack of mood improvement during the day [59]. In the depressed individuals compared with the controls (n = 10), the degree of waking concern correlated with the number of **dreams containing worries** about some material, suggesting a role for **dreaming** in emotional regulation. Compared with those who remained depressed, the remitted participants had a greater percentage of well-developed dreams at follow-up [59].

With respect to autism spectrum disorder (ASD) (n = 28), fewer **dream** recollections, bad dreams, and emotions were reported (in particular, fewer reports of confusion, shyness, and sexual arousal and a lower intensity of anger and confusion) in the ASD group than in the control group [60]. In a sleep laboratory subgroup analysis, 17 subjects with ASD presented shorter dream narratives upon awakening from REM sleep than did the control group [60].

Autonomic responses in trauma-exposed individuals with **chronic nightmares** (n = 35) were associated with greater sleep disturbance and reported health symptoms, even when controlling for nightmare frequency [61]. Heart rate is associated with increased global sleep problems, fewer hours of sleep per night, panic upon waking, and health symptoms [61].

Nightmares containing realistic, easily recalled, and threatening material were associated with greater PTSD severity in combat veterans with PTSD and risky alcohol use (n = 76), reflecting inadequate emotional trauma processing [62]. Furthermore, anger in dreams emerged as a significant indicator of overall worsening PTSD symptom severity, particularly more severe traumatic themes, repetitiveness, and awakenings [63].

Individuals with BPD (n = 16) experienced more frequent **nightmares** than those without BPD did. Using generalised hierarchical linear modelling (GHLM), the study revealed that a baseline BPD diagnosis, when interacting with high trait rumination, prospectively predicted the highest number of nightmares during two weeks, even after accounting for depression, sleep quality, and PTSD [64]. Additionally, the high daily intensity of emotional cascades (negative emotion and rumination) in BPD patients predicted the greatest number of subsequent **nightmares**, even after controlling for covariates [64]. Another three-way interaction indicated that BPD diagnosis interacted with the **number of nightmares** to predict even higher levels of next-day negative emotional intensity, although not rumination [64].

3.3.1. REM sleep alterations, dysphoric dreaming and suicidal/non-suicidal self-injurious behaviour

Shorter **REM latency** and a higher **REM percentage** were correlated with suicidality in their observational cross-sectional study of depressed patients (n = 26) [65]. In the total sample, depressed individuals (n = 13) presented more **dream-negative** affect than non-suicidal depressed individuals did (n = 13), indicating a potential failure during sleep to regulate their mood and incorporate emotions into long-term memory networks.

Among depressed teens (n = 10), nocturnal awakenings and altered REM parameters were correlated with failures in processing emotional stimuli during suicidal crises. Notably, shorter **latency to REM** sleep onset and longer **REM duration** were significantly correlated with increased failure to inhibit inappropriate responses to positive emotional stimuli [66]. Furthermore, more frequent nocturnal awakenings were associated with failure to inhibit appropriate responses to negative emotional stimuli [66]. This highlights the importance of addressing sleep disturbances during suicidal crises, as they may lead to inhibition failures, delays or mistakes in the processing of emotional information. Furthermore, a shorter latency to **REM sleep onset**, increased **REM density**, and higher cortisol levels around sleep onset were reported in a subgroup of depressed teens with suicidal tendencies (n = 26) than in non-suicidal depressed teens and controls (n = 33) [67].

Littlewood et al. reported that **nightmares** predicted suicidal behaviour independently of insomnia and depression in PTSD patients (n = 91)n [68]. Suicidal behaviour was more common among those who reported experiencing nightmares [68]. These findings suggest that **nightmares** may hamper the adaptive consolidation of traumatic memories, resulting in continuous PTSD symptoms, associated suffering (perceptions of defeat and hopelessness) and then suicidal behaviour, increasing the risk of suicidal behaviour in PTSD [68].

In a clinical sample of outpatients (n = 255) with diverse diagnoses, including PTSD, depression, or comorbid diagnoses of both, Short and colleagues reported that **nightmare severity** mediated the relationship between PTSD and self-injurious behaviour after controlling for depression. Although insomnia was associated with self-injurious behaviour, it was not a significant mediator in the relationship between PTSD and self-injurious behaviour when **nightmares** were included in the model [69]. Another study revealed significant associations between nightmares, emotional dysregulation, and non-suicidal self-injury (NSSI), with emotion dysregulation fully explaining the link between nightmares and NSSI, which was consistent across both clinical (n = 313) and university samples (n = 133) [70]. This study revealed that **nightmares,** not insomnia symptoms, were associated with NSSI, even when accounting for depressive symptoms [70]. Finally, nightmare severity was shown to be related to the language narrative used, independent of PTSD and depression severity [71]. Paquet and colleagues reported that language use in **rescripted nightmares** during CBT was associated with lower PTSD and depression severity, highlighting its role in understanding PTSD patients' cognitive and emotional processes [71].

3.4. Role of sleep in treatment and targeted memory techniques

Sherrill et al. reported that daytime sleepiness, insomnia, or **nightmares** in individuals with PTSD did not interfere with the effectiveness of prolonged exposure therapy for PTSD [72]. In contrast, Woodward et al. demonstrated that psychotherapy improves sleep, with trauma-focused therapy (e.g., cognitive therapy for PTSD, CT-PTSD) yielding greater enhancements in self-reported sleep quality, **nightmares**, and insomnia symptoms compared to non-trauma-focused therapies in patients with PTSD (N = 121) [73]. Improvements were linked to memory-updating processes inherent in CT-PTSD.

Kobayashi et al. examined whether the timing of written narrative exposure (WNE) sessions relative to sleep affected PTSD symptom improvement (n = 21). Although proximity to sleep did not influence treatment outcomes, increased **REM density** and reduced N3 sleep predicted less symptom reduction. Conversely, longer sleep duration was associated with better emotional processing of trauma memories [74].

A randomised controlled trial suggested that music relaxation at bedtime improved subjective and objective sleep quality in PTSD patients (n = 13), highlighting its potential as an adjunctive treatment for sleep disturbances [75]. Similarly, olfactory interventions using pleasant odours were associated with a reduced negative emotional tone in **dreams** and improved dream intensity in PTSD patients [76].

Finally, targeted memory reactivation (TMR) strategies during sleep have shown promise in enhancing emotional extinction and reducing depressive and anxiety symptoms in clinical populations with social anxiety (n = 48) [77]. These findings suggest a potential role for interventions targeting **REM-related processes** in emotional regulation and memory consolidation.

4. Discussion

Our review underscores the critical role of REM sleep in emotion processing and regulation, particularly in affective psychiatric conditions such as MDD and PTSD. The findings in this review confirm that alterations in REM physiology are implicated in emotion dysregulation and memory processing deficits in psychiatric disorders [26,28,34–36,39–41]. In this discussion, we address three critical items: 1) the presence of REM sleep disturbances in psychiatric disorders and their relationship to emotion dysregulation in psychiatric disorders, 2) the mechanisms by which REM disturbances contribute to emotion dysregulation, and 3) the sleep-based treatment strategies and techniques that can be derived from these findings.

4.1. Is there evidence of REM disturbances in psychiatric disorders resulting in emotion dysregulation?

Evidence from various studies points to a consistent link between REM sleep disturbances and emotion dysregulation in psychiatric conditions such as MDD, PTSD, and suicidal behaviour. Our findings reveal underlying REM sleep and wakefulness disturbances in individuals with MDD and PTSD, which are associated with distinct neurobiological processes in brain regions associated with emotion regulation [49]. In MDD, alterations in REM sleep, such as reduced REM latency and, most importantly, increased REM density, have been shown not only to be characteristic of MDD but also to be associated with clinical outcomes [16,38,39,51,78]. This review further revealed a link between impairments in emotional regulation and REM density as a potential marker for emotion-induced arousal regulation in MDD [51]. Higher REM density was correlated with reduced metabolic activity in the PFC during the processing of emotional stimuli (specifically in the mPFC and VLPFC) [50,51]. Abnormal REM activity, including a greater negative dream emotional load, also resulted in difficulties with emotion processing, leading to worse emotion regulation and mood during the waking state [58]. In addition, the results of this review indicated that non-remitted individuals with MDD failed to integrate their waking concerns into dreams, negatively affecting emotion processing during sleep and resulting in poorer mood the following day; in contrast, remitted patients presented better emotion expression and memory integration in their dreams [58,59].

Although MDD and PTSD often coexist, with up to 36 % of depressed patients also experiencing PTSD [79], PTSD is characterised by more profound REM sleep disturbances, particularly in the form of nightmares and increased REM fragmentation. Furthermore, disrupted REM sleep significantly contributes to the development and persistence of PTSD symptoms, including emotional disturbances [18,42,44]. Therefore, REM sleep fragmentation, nightmare severity, and increased sympathetic tone after trauma have been identified as predictors of PTSD symptomatology, indicating their role as clinical biomarkers of emotion dysregulation [42,80,81].

In suicidality, REM disturbances also play a pivotal role in emotion dysregulation. Suicidal individuals with depression display shorter REM latency and an increased REM percentage, which have been associated with a failure to inhibit emotional responses, especially negative ones [66]. Suicidal adolescents, in particular, show higher REM percentages and more negative emotional dream content, suggesting heightened emotional dysregulation due to REM sleep abnormalities [65].

4.2. What is the presumed mechanism by which REM disturbances lead to emotion dysregulation in patients with psychiatric disorders?

The underlying mechanisms connecting REM disturbances to emotion dysregulation involve disruptions in brain regions responsible for emotion regulation, memory consolidation, and arousal control. In MDD, REM sleep abnormalities, such as increased REM density, are associated with hypofrontality, specifically in areas such as the VLPFC and mPFC, which are crucial for emotion regulation [51,82]. These frontal areas are typically involved in downregulating negative emotional arousal [82–87]. Reduced activity in these regions during REM sleep may impair emotion processing and result in ineffective integration of emotional experiences from wakefulness into dreams. This deficiency can manifest as recurrent negative dreams, poor mood upon awakening, and persistent emotional dysregulation during the day [58,59]. Over time, chronic REM sleep disruptions (i.e., REM sleep fragmentation) can lead to a rebound effect marked by shortened REM latency, increased REM density, and heightened emotional reactivity to negative stimuli [39]. Chronic REM sleep fragmentation may lead to enhanced emotional sensitivity, which could contribute to the onset of MDD [39].

REM fragmentation has been suggested to play a key role in the associations among insomnia, PTSD and MDD [21], with inadequate noradrenergic suppression during fragmented REM sleep leading to potential persistent hyperarousal [29]. Our results seem to suggest, at least partially, the same mechanisms in PTSD as in people with insomnia, where overnight regulation of emotional distress is impaired by fragmented REM sleep as well as by subjective indices of altered REM sleep, such as nightmares [23]. Uninterrupted REM sleep is associated with improved recognition of emotional and neutral material, with emotional arousal differentially affecting memory [55]. It can be hypothesised that when REM sleep is uninterrupted, providing a "time-out" from noradrenergic activity, synaptic plasticity in limbic circuits may facilitate the overnight adaptation of emotional experiences [20,29]. Therefore, a night of unfragmented REM sleep may enable individuals to process information the following day more unbiasedly [28,41,55,88,89].

Nevertheless, REM sleep is fragmented in PTSD, where sympathetic arousal remains elevated, preventing effective emotional memory consolidation [42]. Hyperarousal, a key symptom of PTSD, exacerbates REM disturbances, further reducing the ability to consolidate emotional memories during REM sleep [53,90]. This review has shown that trauma processing during REM sleep, characterised by right prefrontal theta power and overnight reduction in sympathetic arousal, is essential for adaptive emotional memory consolidation in individuals exposed to trauma [55,57]. Specifically, decreased nocturnal arousal predicts better emotional memory accuracy, whereas increased arousal is associated with improved recall of neutral material [55]. These findings support the idea that REM sleep integrates memories into existing cortical networks, reducing negative emotional components [41] while enhancing neutral ones [55]. Ultimately, the emotional memory processing that occurs during REM sleep can serve an adaptive function that can be indexed by right frontal theta activity [91].

REM sleep disruptions such as microarousals, nightmares, and

altered dreaming significantly affect cognitive and emotional processes in trauma-related disorders by interacting with cognitive and emotional processes [35,42]. Another potential mechanism is the impairment of adaptive emotion processing during memory consolidation due to heightened sympathetic activity and amygdala hyperactivity. In PTSD, hyperactivation of limbic structures, particularly the amygdala, during REM sleep disrupts fear extinction and adaptive emotion processing, as the amygdala—which is crucial for fear conditioning—is typically suppressed during healthy REM sleep to facilitate the extinction of fear-related memories [52]. The results of this review indicate that trauma-exposed individuals exhibit greater sympathetic arousal and frequent REM sleep disruptions, leading to poor emotional memory consolidation, difficulty distinguishing between threat and safety signals, and increased PTSD symptom severity [42,53,55,62].

Nightmares in PTSD are strongly linked to impaired trauma processing and contribute significantly to emotional dysregulation during wakefulness [42]. Nightmares containing anger or fear often signal unresolved trauma and heightened emotion dysregulation in PTSD patients [63]. Trauma-related nightmares vary in severity, ranging from loosely trauma-associated themes to vivid replays of the traumatic event [92]. Neurocognitive models propose that dreaming typically facilitates fear memory extinction through a regulatory network involving the amygdala, medial prefrontal cortex (mPFC), hippocampus, and anterior cingulate cortex (ACC) [93]. However, under conditions of high stress or trauma, this network can become dysfunctional, resulting in dysphoric dreams that span from occasional bad dreams to repetitive nightmares [92,93].

Consistent with this framework, more realistic, easily recalled, and threatening nightmares are hypothesised to reflect inadequate trauma processing during the night, which is associated with greater PTSD severity [62]. Increased REM activity is associated with the persistence of nightmares, which function as repeated simulations of unresolved traumatic events, further reinforcing emotion dysregulation and hyperarousal [62,94].

The neurophysiological mechanisms underlying repetitive dreams, such as those in PTSD, appear to differ significantly from those involved in undefined or non-specific nightmares. Recurrent distressing nightmares, often aligned with the Intrusive Traumatic Re-Experiencing Domain (ITRED), involve trauma-specific reactivation in areas like the anterior cingulate cortex (ACC) and generalised sensory representations in the visual cortex, reflecting their vivid sensory and emotional specificity [95]. These nightmares function as intrusions, involuntary reactivations of trauma memories that maintain hyperactivity in sensory and emotional brain regions, such as the amygdala while showing reduced activity in conceptual/semantic areas like the superior temporal cortex [96]. This imbalance hinders the integration of trauma into broader memory networks, perpetuating the cycle of emotional dysregulation [96]. The failure to distinguish traumatic memories from perceptually similar cues further solidifies nightmares as a hallmark symptom of PTSD.

In contrast, undefined nightmares, lacking direct ties to traumatic memory, may involve less distinct reactivation of memory-related networks, instead relying more on perceptual processing systems [95–97]. Research identifies the posterior parieto-occipital region as a critical "hot zone" supporting perceptual aspects of dreaming independent of memory recall [97]. When dreams contain thought-like experiences (nocturnal mentation), increased high-frequency activity in the parieto-occipital region is accompanied by activation in portions of the prefrontal cortex during REM sleep [97]. This aligns with findings by Wassing et al. (2016) that associate nocturnal mentation with fragmented REM sleep, heightened eye movement density, and disrupted distress resolution, ultimately culminating in hyperarousal [23].

These results suggest that recurrent nightmares in PTSD reflect impaired integration of trauma-related memories into the brain's regulatory and semantic networks, while undefined nightmares may stem from broader disruptions in perceptual and regulatory functions. The vividness and sensory detail of recurrent nightmares likely stem from excessive sensory generalisation and trauma-specific neural reactivation, contrasting with the perceptual but less specific basis of undefined nightmares. The association of recurrent nightmares with intrusions, as outlined in ITRED, underscores their role in perpetuating emotional dysregulation and hyperarousal in PTSD [95–97].

Disrupted REM sleep may mechanistically contribute to intrusive memory formation by impairing emotional processing [98,99]. While REM sleep initially heightens emotional reactivity, it facilitates emotional processing over subsequent nights, reducing intrusion frequency and distress in healthy individuals [98–100]. Meta-analytic findings suggest sleep primarily influences intrusions by modulating explicit trauma memory rather than reprocessing emotional components of traumatic memories, though the evidence is mixed [99]. However, the precise relationship between intrusion frequency, distressing nightmares, and emotional distress in PTSD remains unresolved [98–100].

4.3. What sleep-based treatment strategies can be derived from these findings?

The recognition of REM sleep disturbances as a significant contributor to emotion dysregulation in individuals with psychiatric disorders provides a strong rationale for incorporating sleep-focused interventions into treatment. Our study highlights the need for targeted therapies to reduce REM sleep disturbances, which are crucial for managing emotion dysregulation and preventing suicide in PTSD patients [61,68–70].

Treatments for MDD that target REM sleep abnormalities and dream experiences could help reduce suicidality and improve emotion regulation [51,59,78,101]. Dream analysis may provide a therapeutic avenue, as examining the content of REM dreams could help clinicians address unresolved emotional conflicts or trauma in patients with depression [45,59]. Napping interventions have also been shown to improve mood and emotional regulation in both depressed and non-depressed individuals by facilitating the consolidation of positive emotional memories [54], whereas the number of nightmares predicts elevated negative emotion in BPD [64]. Novel approaches that could restore PFC activity and improve emotion regulation are a primary goal. Neurofeedback approaches using EEG and fMRI signals are novel techniques that may provide a new avenue for treating emotion dysregulation in patients with BPD [102]. Neurofeedback training has been shown to modulate not only amygdala activity but also frontal regions of the brain, particularly those involved in top-down emotion regulation, such as the dorsal anterior cingulate cortex (dACC) and left dorsolateral prefrontal cortex (dlPFC) [102]. Future research should explore their effects on sleep disturbance.

For PTSD, interventions such as narrative exposure therapy, which involves trauma reprocessing, have shown promising results in reducing emotion dysregulation and PTSD symptoms [72–74]. Massed delivery of prolonged exposure (daily sessions for two weeks) has been highlighted to enhance engagement and facilitate fear extinction, thereby reducing the impact of sleep disturbance on treatment outcomes [72]. Although the treatment of PTSD symptoms through trauma-focused psychotherapy may alleviate sleep problems such as nightmares and insomnia, a critical area for future research is whether the treatment of sleep disturbances can also alleviate PTSD symptoms. Possible approaches include exposure to positive emotional stimuli, such as olfactory stimulation during sleep and music relaxation at bedtime, which have been shown to improve both objective and subjective sleep quality and may play a role in emotion regulation, as music has been shown to alleviate the intensity and negative emotional tone of dreams [75,76]. Therefore, these approaches could help reduce nightmare frequency and promote trauma processing during REM sleep. Interestingly, recent research has proven the effectiveness of TMR using auditory stimuli linked to treatment memories during REM sleep to potentiate the effects of imagery rehearsal therapy for nightmare disorders [103]. In this study, TMR not only reduced the frequency of nightmares even after 3 months but also

increased the positive tone of dreams after 2 weeks [103]. Another recent study provided a proof of concept of TMR during SWS as an augmentation strategy after exposure-based treatment in PTSD patients [104]. Future studies should explore the potential complementary effects of combined SWS-REM-TMR in the emotional depotentiation of traumatic memories in psychiatric populations [105]. In particular, TMR may play a role in reducing physiological manifestations of anxiety and stress during REM sleep in people with social anxiety disorder, possibly by enhancing extinction and emotional depotentiation [77].

Emerging treatments addressing "trauma-associated sleep disorder" (TSD), which combines features of PTSD and REM sleep behaviour disorder, may offer new ways to target sleep disruptions in trauma-exposed individuals [56,106]. These parasomnias, characterised by nightmares and physical restlessness, could be treated with a combination of pharmacological and behavioural interventions aimed at regulating both sleep and emotion processing [107]. In addition, addressing nightmare-related autonomic arousal may improve sleep quality and health, reducing risks such as cardiovascular disease [61,108]. Future studies targeting sensory generalisation reduction, conceptual distinctiveness, and trauma-specific reactivation during sleep may provide novel therapeutic insights for managing PTSD-related nightmares and trauma intrusions [96].

Cognitive Behavioural Therapy for Insomnia (CBT-I) has been shown to be effective in the treatment of insomnia in individuals with psychiatric disorders, such as MDD and PTSD [1,109–112]. Reesen and colleagues (2024) recently highlighted the importance of addressing insomnia in individuals with affective and emotion dysregulation disorders and the urgent need for a transdiagnostic approach to evaluate the potential dual effectiveness of incorporating CBT-I for treating various psychiatric disorders characterised by emotion dysregulation [113], as currently, van Trigt is testing in BPD [114]. Nevertheless, to our knowledge, the impact of CBT-i on emotion regulation outcomes in clinical populations has not been reported. Therefore, larger studies are needed to confirm the benefits of sleep interventions in improving overall psychiatric outcomes and emotion dysregulation, as well as the beneficial effects of prioritising the treatment of insomnia, nightmares, and REM sleep disturbance before initiating standard psychological therapies, as quality sleep is vital for alleviating emotional distress 8, 115. The shift in the DSM-5 to a separate insomnia diagnosis recognises the comorbid nature of insomnia and advocates for treating insomnia itself in addition to any concurrent medical or psychiatric disorders [116].

4.4. Overall conclusion

REM sleep disturbances are a critical component of emotion dysregulation in psychiatric disorders, particularly MDD, PTSD, and suicidality. Evidence suggests that improving sleep through targeted interventions—such as narrative exposure therapy, music relaxation, and olfactory stimulation—can significantly improve emotion processing and treatment outcomes in PTSD patients. While sleep has not been a primary focus of past clinical interventions for psychiatric disorders, understanding its role in emotion consolidation and regulation during treatment is an essential area for future research. Understanding how insomnia and psychiatric conditions interact can inform more effective treatment strategies [117]. Future research should focus on uncovering the mechanisms behind REM sleep disturbances and their impact on emotion regulation, applying these insights to interventions for mental health disorders. Sleep research offers valuable translational opportunities in this field.

4.5. Limitations

Our evidence was mostly limited to studies on mood disorders (k = 8) and anxiety disorders (k = 17), which limits the transdiagnostic generalizability of our results to all psychiatric disorders with affective symptoms regardless of their core complaint (e.g., psychosis spectrum disorders). Dimensional and longitudinal approaches are needed to elucidate symptom interactions and changes over time to optimise treatment for sleep-related disorders in people with psychiatric disorders.

CRediT authorship contribution statement

Mariana Mendoza Alvarez: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Yannick Balthasar:** Writing – review & editing, Validation, Software, Methodology, Investigation, Formal analysis, Data curation. **Johan Verbraecken:** Writing – review & editing, Supervision. **Laurence Claes:** Writing – review & editing, Supervision. **Eus van Someren:** Writing – review & editing, Supervision. **Hein J.F. van Marle:** Writing – review & editing, Supervision. **Marie Vandekerckhove:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Livia De Picker:** Writing – review & editing, Supervision, Methodology, Conceptualization.

Data availability statement

The search strategies are presented in the supplementary material, and further inquiries can be directed to the corresponding author.

Declaration of AI and AI-assisted technologies in the writing process

During the preparation of this work, the author(s) used the ChatGPT AI language model from OpenAI to refine the language. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

Funding

The University Psychiatric Centre Duffel (UPCD), Belgium, provided funding for this study. The sponsor was not involved in the study design, data collection, analysis, interpretation, report writing, or decision to submit the paper for publication.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

We express our gratitude to the research teams at the University Psychiatric Centre Duffel (UPCD) for their assistance in this study.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2024.12.037.

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