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The effects of RBD medications on dream content: A critical review of evidence

Giorgia Bontempi [a], Elena Capriglia [b,c], Giulio Bernardi [a], Valentina Elce [a,*]

[a] Sleep, Plasticity, and Conscious Experience Group, IMT School for Advanced Studies Lucca, Lucca, Italy
[b] Unit of Sleep Medicine and Epilepsy, IRCCS Mondino Foundation, Pavia, Italy
[c] Department of Brain and Behavioral Sciences, University of Pavia, Pavia, Italy

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ABSTRACT

REM sleep behavior disorder, often an early marker of α-synucleinopathies, is characterized by the loss of REM-related muscle atonia and the occurrence of dream enactment behaviors. Dream reports in REM sleep behavior disorder are frequently described as more vivid, violent, and negative than typical dreams. However, evidence for this remains inconsistent, likely due to methodological variability and unaddressed confounds – most notably, the influence of pharmacological treatments. The extent to which medications affect REM sleep behavior disorder dream content remains poorly understood.

This systematic review critically evaluates the literature on the effects of medications commonly used in REM sleep behavior disorder and Parkinson's disease – including clonazepam, other benzodiazepines, melatonin, melatonin receptor agonists, levodopa, pramipexole, and other dopamine agonists - on dream content. We identified 27 relevant studies employing qualitative and/or quantitative methods.

Our analysis revealed major methodological limitations across studies, including a lack of standardized protocols and definitions, frequent reliance on retrospective or anecdotal reports, and predominantly qualitative or semi-quantitative assessments. These limitations constrain the interpretability and comparability of findings. Available evidence offers only limited support for clonazepam, melatonin, and pramipexole in reducing disturbing dream content. More consistent, but still preliminary, evidence links levodopa to increases in vivid or disturbing dreams, including nightmares.

This review highlights the need for rigorously controlled studies using standardized and reproducible methods to clarify the role of pharmacological treatments in shaping dream content in REM sleep behavior disorder. Future research should also integrate retrospective and prospective data-collection designs to account for potential recall biases.

1. Introduction

REM sleep is physiologically defined by a unique triad: wake-like cortical activation, rapid eye movements, and near-complete muscle atonia [1]. This muscular inhibition is believed to serve a protective function, preventing individuals from physically acting out dreams, which are especially vivid and emotionally intense during REM sleep [2].

REM sleep behavior disorder (RBD) is a parasomnia characterized by the partial or complete loss of muscular atonia, often leading to dream enactment behaviors. These can vary in complexity but are typically described as abnormal or violent — such as talking, yelling, kicking, or punching — and may result in injuries to the patient or their bed partner. These episodes often reflect vivid, aggressive, or threatening dream content, with a striking coherence between the movements and the dream reports provided upon awakening [3].

RBD is thought to reflect a pathological intrusion of wake-like motor activity into REM sleep and has been closely linked to neurodegenerative diseases, particularly α-synucleinopathies like Parkinson's disease (PD), dementia with Lewy bodies, and multiple system atrophy [4]. A distinction is made between isolated RBD (iRBD), diagnosed in the absence of known neurological disorders, and secondary RBD (sRBD),

Abbreviations: REM, Rapid Eye Movement; nREM, Non-Rapid Eye Movement; RBD, REM Sleep Behavior Disorder.

* Corresponding author.Piazza S. Ponziano, 6, 55100, Lucca, Italy.
E-mail addresses: giorgia.bontempi@imtlucca.it (G. Bontempi), elena.capriglia01@universitadipavia.it (E. Capriglia), giulio.bernardi@imtlucca.it (G. Bernardi), valentina.elce@imtlucca.it (V. Elce).

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associated with established neurodegenerative conditions. Importantly, iRBD is often a prodromal marker, with roughly 73 % of individuals developing a defined α-synucleinopathy within 12 years of diagnosis [5].

Since its initial description in 1986 [6], RBD has been associated with distinctly altered dream content. Reports have emphasized an increased prevalence of violent, negative, and action-filled dreams, often involving attacks by strangers or animals [7]. Moreover, patients often describe these unpleasant experiences as either distressing dreams or even nightmares – i.e., vivid, intensely frightening dreams that result in full awakening and detailed recall, often involving themes of threat [8,9]. These findings led to speculation that changes in dream content could serve as a diagnostic marker for RBD or even help predict neurodegenerative progression [7–9].

However, more recent studies have partially challenged this view, reporting a broader spectrum of dream contents and enactment behaviors, with evidence of nonviolent episodes [10–13] and of similar levels of aggressive or unpleasant dream content in RBD patients compared to healthy controls [14,15]. Research comparing patients with PD alone to those with both PD and RBD has also yielded mixed results: some studies reported more aggressive dreams in the PD-RBD group [16], while others found no differences [17,18]. Such inconsistencies in the literature may be explained by several non-mutually exclusive factors, including methodological differences related to how information about oneiric content is collected and analyzed, and the possible confounding effects of pharmacological treatments [19–21].

Indeed, study protocols can vary widely in their methodology, involving the use of structured questionnaires, the collection of verbal dream reports, or both. Dream data may be gathered retrospectively (asking participants to recall one or more dreams experienced over a given period, ranging from a week to their entire lifetime), prospectively (instructing participants to record their dreams immediately upon awakening on a daily basis), or through laboratory protocols. Each of these methods can systematically shape the type and quality of dream content reported, depending on factors such as memory demands, timing of recall, and contextual cues [9,22–25].

While each of these approaches has its own general strengths and weaknesses, their specific methodological differences become particularly critical in RBD research. Because violent dream enactment behaviors often cause awakening, and emotionally intense dreams – in line with the saliency hypothesis by Cohen and colleagues [26] – are more likely to be recalled, it is possible that aggressive dream content may be overrepresented [27]. Some authors have even suggested RBD might function as a "recall-bias syndrome", in which frequent awakenings and movements selectively enhance the recall of disturbing dreams. This effect is further confounded by the frequent use of retrospective methods in early studies [28].

The substantial heterogeneity in methodological approaches inevitably interacts with another critical source of bias: pharmacological treatments. Indeed, several studies have suggested that pharmacological agents can influence dream phenomenology even in healthy individuals, through mechanisms involving both neurochemical modulation of dreaming and changes in arousal or alertness levels during sleep, further entangling the interpretation of findings in clinical populations [29–31]. Notably, while most individuals with RBD are treated with medications, especially *clonazepam*, studies often lack adequate control for their potential effects. Few include drug-naïve participants or incorporate washout periods, making it difficult to unravel the influence of the disorder from that of pharmacological treatments. The impact of medications on dream recall and content remains poorly understood, and their effects may mask, mimic, or modify core features of RBD-related dreaming.

In this systematic review, we critically examine the literature on the impact of commonly used medications for RBD and PD on dream content. Our primary aim is to clarify the extent to which pharmacological treatment may influence findings reported in RBD studies. By focusing on drug effects, regardless of the population under study, we seek to disentangle dream characteristics inherent to RBD from those potentially modified or obscured by pharmacological treatment. We also address key methodological challenges in dream research that must be considered when interpreting current evidence or planning further investigations.

2. Methods

We conducted a literature search on PubMed, considering all the papers published up to January 2024. Additional relevant studies were identified by screening the references of the retrieved papers.

Given the relatively limited number of studies specifically involving individuals with RBD, we broadened our inclusion criteria to encompass studies investigating the effects of medications commonly used to treat RBD in non-RBD populations (the diagnostic characteristics of each study sample are specified in Tables 1–3).

Search terms included reference to dream experiences (dream*, dream experience*, dream report*, dream recall, dream content*, dream content analysis*, nightmare*, sleep conscious experience*, sleep mentation*), medication (pharmacological treatment*, pharmaceutical treatment*, medication*, treatment*, drug*), and specific drugs or drug classes usually prescribed for the treatment of RBD or PD-RBD (clonazepam, rivotril, benzodiazepine*, melatonin, ramelteon, agomelatine, melatonin agonist*, pramipexole, levodopa, dopamine agonist*, dopaminergic drug*).

To maximize inclusivity of possible studies involving multiple medications, we also inspected works that contained the names of additional drugs or drug classes less specific for RBD treatment in our search terms (e.g., antidepressant*, gabapentinoid*, acetylcholinesterase inhibitor*).

Titles, abstracts, and keywords were screened based on the following inclusion criteria: (1) written in English; and (2) included a quantitative (QUAN), semi-quantitative (Semi-QUAN), or qualitative (QUAL) assessment of the effects of medication specifically on dream content.

Books, abstracts, comments, reviews, meta-analyses, and pre-prints were excluded. Eligible articles were selected following a multi-step process (title reading, abstract, and full-text assessment).

3. Results

3.1. Literature search and selection

Our literature search yielded 5969 results. After a multi-step screening process of titles, abstracts, and full texts, 27 papers published between 1970 and 2022 (inclusive) met our inclusion criteria and were selected for this review.

Of these, 8 studies examined the effects of *clonazepam* on dream content [7,17,20,32–36], including one also investigating the impact of *melatonin* [34], while 4 investigated the impact of other benzodiazepines [37–40].

Another 8 studies addressed the influence of *melatonin* (n = 5) [34, 41–44], including one also investigating the impact of *clonazepam* [34], or melatonin receptor agonists (n = 3) [45–47] on dream phenomenology.

Finally, 8 studies explored the role of dopaminergic drugs, including *levodopa* (n = 4) [48–51], *pramipexole* (n = 2) [52,53], and other dopamine agonists (n = 2) [54,55].

3.2. Clonazepam and other benzodiazepines

3.2.1. Mechanism of action

Benzodiazepines exert their effects by acting as positive allosteric modulators of the GABA-A receptors, binding specifically to their benzodiazepine site. Through this mechanism, they enhance the inhibitory action of GABA, the primary inhibitory neurotransmitter in the central nervous system. As a result, benzodiazepines produce a broad spectrum of effects, including sedation, hypnosis, skeletal muscle relaxation, anticonvulsant activity, and anxiolytic action [56,57]. Importantly, benzodiazepines differ in their pharmacokinetic and pharmacodynamic profiles, leading to variations in potency, selectivity, and therapeutic indications [58,59].

3.2.2. Use for RBD treatment

Among benzodiazepines, *clonazepam* is by far the most commonly used medication for the treatment of RBD symptoms, having become the first-line therapy following the seminal report by Schenck and colleagues in 1986 [6] and a subsequent series of large cohort studies [7,60,61], with other benzodiazepines being only rarely employed.

One critical and one systematic review and meta-analysis [62,63], while confirming the potential efficacy of *clonazepam* in the treatment of RBD symptoms, highlighted significant limitations in the available evidence, primarily due to the observational design of most studies and the frequent lack of objective measures assessing changes in dream enactment behaviors frequency or REM motor tone.

The mechanism by which *clonazepam* improves abnormal motor activity remains unclear. Studies conducted on both animal models [64] and humans [60,64–67] reported that *clonazepam* suppresses phasic EMG activity during REM sleep, effectively controlling motor behavior. These findings have been interpreted as suggesting that *clonazepam* may exert its effects on the locomotor system without fully restoring REM sleep atonia.

Evidence of the efficacy of benzodiazepines other than *clonazepam* for RBD treatment are very scarce, mainly based on case reports including only one or two participants [63,68]. Our literature search identified four studies [37–40] that used other benzodiazepines, but only three reported which specific medications were employed, namely *flunitrazepam* [38], *nitrazepam* [39], and *lormetazepam* [40].

To the best of our knowledge, no studies have specifically investigated the effects of *lormetazepam* and *flunitrazepam* on RBD symptoms. Instead, the limited available data on the efficacy of other benzodiazepines (including *nitrazepam*) for RBD treatment were summarized in a recent review [63], which reported that 86.4 % of a total of 22 patients across studies experienced no benefit.

Despite their shared mechanism of action, the reasons why *clonazepam* may be more effective for RBD treatment than other benzodiazepines is unclear. Pharmacokinetic differences may act as possible contributing factors.

3.2.3. Documented effects on dream content

Clonazepam. We identified eight studies investigating the impact of *clonazepam* on dream content (see Table 1). Six of these (75 %) [7,20,32–35] reported some form of influence of the drug on dreaming, while two (25 %) [17,36] found no effect. However, the nature and direction of these findings appeared to be contingent upon the methodological approach adopted.

Most studies relied on retrospective assessments, typically using standardized tools such as the REM sleep behavior disorder questionnaire (RBDQ) [33,69,70] or clinical interviews, and were more likely to report positive effects [7,20,32–35]. For instance, the two studies [32,33] evaluating RBD dream symptomatology in RBD [32] and iRBD participants [33] via the RBDQ consistently observed clear symptom alleviation under *clonazepam*, particularly when looking at the RBDQ composite 'Factor 1' score ($p = 0.023$; $p < 0.001$), capturing emotional tone and frequency of unpleasant dream experiences, and the specific RBDQ item related to the frequency of violent and aggressive dreams ($p = 0.027$; $p < 0.01$). Yet, this same tool failed to detect consistent changes in emotional dream content ($p = 0.221$; $p > 0.05$), and the two studies report inconsistent findings regarding the effect of *clonazepam* on frightening dreams ($p = 0.233$; $p < 0.01$) and nightmare frequency ($p = 0.098$; $p < 0.01$). Additional retrospective studies using broader clinical interviews [7,20,34] or PTSD-related questionnaires [35] also described reductions in nightmare frequency or disturbing dream features in RBD [7,20,33] or PTSD [34] participants.

In contrast, the two studies adopting prospective designs, either through a daily sleep diary consisting of self-administered questions [36] or content analysis of collected dream reports [17], did not find any significant changes in dream content under *clonazepam* ($p > 0.05$) in iRBD [17] or PTSD [36] participants. Notably, Cavallotti and colleagues [17] found no modulation in aggressiveness ($p = 0.254$) or threatening dream content ($p = 0.732$) by employing the Hall and Van de Castle coding system [71] and the Threat Simulation Scale [72].

Altogether, these findings suggest a methodological divide: *clonazepam*'s apparent effects on dreaming emerge more readily when assessed retrospectively, particularly via standardized self-report questionnaires, while prospective, report-based studies tend to reveal null results. This divergence raises important questions about how the timing and format of data collection may shape what is perceived or remembered as pharmacologically induced change in dreaming.

Other benzodiazepines. We identified four prospective studies investigating the effects of benzodiazepines on dream content (see Table 1). Despite their shared prospective design, these studies differed markedly in experimental setup and in how dream content was defined and measured.

Two studies [38,39] relied on semi-quantitative analyses of verbal dream reports rated through predefined scales [109–111]. In a double-blind, crossover design, Morgan and colleagues (1970) [39] collected verbal dream reports from healthy participants by telephone upon awakening and evaluated them for emotional content using specific categories. This assessment led to the identification of fewer hostile dreams after *nitrazepam* administration compared to placebo. Instead, Gaillard & Phelippeau (1976) [38] adopted a laboratory-based serial awakening protocol in which dream reports were collected from healthy participants from both REM and N2 sleep, during *flunitrazepam* and placebo nights. Although results were presented descriptively, REM sleep dreams under *flunitrazepam* appeared more unpleasant, aggressive, and sexual compared to placebo, suggesting a possible qualitative shift in dream content. The authors observed that N2 dream reports were generally low in emotional intensity and rarely featured unpleasant content in both drug and placebo conditions. Consequently, their further considerations focused primarily on REM-related dream reports. Notably, this was the only study among those reviewed to use a serial awakening paradigm, explicitly clarifying the sleep stage from which dream reports were obtained.

The other two studies used structured questionnaires. Schredl and colleagues (1998) [37] asked patients with primary insomnia to rate the emotional tone of their dreams each morning upon awakening [73]: patients treated with benzodiazepines reported more positively toned dream emotions than drug-free controls ($p = 0.0338$). Finally, Joseph and Hossain (1985) [40] administered *lormetazepam* over seven consecutive nights to participants with insomnia, who completed a daily morning sleep diary including a question about the "occurrence and type of dreams". While the authors reported no drug-related effects on dream content (p-value not specified), the absence of clearly defined content categories limited the interpretability of the findings.

Overall, the heterogeneity of these studies was reflected in the variability of their findings. While two studies reported positive changes in dream content, such as a reduction in hostility or an increase in positive emotional tone, one study associated benzodiazepines with more unpleasant and aggressive dreams, and another found no clear effects. Notably, the two studies reporting positive effects did so in relation to different dimensions or categories of dream content, raising doubts about the consistency and comparability of these findings.

Table 1
Description of the studies investigating the impact of clonazepam and other benzodiazepines on dream content.

Reference	Effect direction	Effect	Method	Diagnosis	Statistical design	Design (P/R)	Assessment
CLONAZEPAM							
[20] Schenck & Mahowald, 1990	⇓	Vivid, unpleasant, action filled, violent dreams, reported as severe nightmares	Clinical interview	RBD	Pre- vs Post-treatment	R	Semi-QUAN
[34] Lloyd et al., 2012	⇓	Nightmares	Clinical interview	RBD	Pre- vs Post-treatment	R	Semi-QUAN
[32] Ozone et al., 2020	⇓	Violent/aggressive dreams ($p = 0.027$) RBD dream symptoms (factor 1; $p = 0.023$)	Questionnaire (RBDQ [69])	RBD	Pre- vs Post-treatment	R	QUAN
	⇔	Emotional/sorrowful dreams Frightening dreams Nightmares					
[33] Li et al., 2016	⇓	Violent/aggressive dreams RBD dream symptoms (factor 1) Frightening dreams Nightmares ($p < 0.01$)	Questionnaire (RBDQ [69])	iRBD	Pre- vs Post-treatment	R	QUAN
	⇔	Emotional/sorrowful dreams ($p > 0.05$)					
[17] Cavallotti et al., 2022	⇔	Violent/aggressive dreams ($p = 0.254$) Threatening dreams ($p = 0.732$)	Report analysis with scales [71,72]	iRBD	Medicated vs Drug-free vs Healthy	P	QUAN
[7] Schenck et al., 1993	⇓	Vivid, unpleasant, action filled, violent dreams, reported as severe nightmares	Clinical interview	iRBD; sRBD	Pre- vs Post-treatment	R	Semi-QUAN
[35] Loewenstein et al., 1988	⇓	Nightmares	Questionnaire [35]	PTSD	Pre- vs Post-treatment	R	QUAL
[36] Cates et al., 2004	⇔	Nightmares frequency Nightmares intensity ($p > 0.05$)	Questionnaire in sleep diary [36]	PTSD	Drug vs Placebo	P	QUAN
OTHER BENZODIAZEPINES							
[40] Joseph & Hossain, 1985	⇔	Incidence and nature of dreams (p = N/A)	Questionnaire in sleep diary [40]	Insomnia	Nights vs Nights (always under Lormetazepam)	P	QUAN
[37] Schredl et al., 1998	⇑	Positive emotions in dreams ($p = 0.0338$)	Questionnaire (Sf-A [73])	Insomnia	Benzodiazepines vs Drug-free	P	QUAN
[39] Morgan et al., 1970	⇓	Hostility content	Report analysis with scales [109] Questionnaire [39]	Healthy	Nitrazepam vs Placebo	P	Semi-QUAN
[38] Gaillard & Phelippeau, 1976	⇑	Unpleasant dreams Violent/aggressive dreams Sexual dreams	Report analysis with scales [110,111]	Healthy	Flunitrazepam vs Placebo	P	Semi-QUAN
	⇔	Quantity and quality of dreams; unpleasant contents	Report analysis	Healthy	Flunitrazepam vs Placebo	P	QUAL

⇑, Increase; ⇓, Decrease; ⇔, No change; [purple], Retrospective; [green], Prospective.
Abbreviations: N/A, not available; RBDQ, REM sleep behavior disorder questionnaire [69]; Sf-A, Schlaffragebogen A; RBD, REM sleep behavior disorder; iRBD, Isolated RBD; sRBD, Secondary RBD; PTSD, Post-traumatic stress disorder; R, Retrospective; P, Prospective; QUAN, Quantitative; QUAL, Qualitative; Semi-QUAN, Semi-quantitative.
Scales for the analysis of reports included Hall and Van de Castle scale [71], Threat Simulation Scale [72], Hauri et al. (1967) scale [110], Gaillard & Phelippeau (1977) scale [111], Gottschalk et al. (1960) scale [109].

3.3. Melatonin and melatonin receptor agonists

3.3.1. Mechanism of action

Melatonin is a hormone secreted by the pineal gland in response to signals received from the hypothalamic suprachiasmatic nucleus, the so-called "circadian pacemaker". *Melatonin* secretion, which peaks during darkness, serves as a biological marker of night-time and plays a critical role in regulating circadian rhythms, including the sleep–wake cycle [74].

Exogenous *melatonin* and its analogues, known as melatonin receptor agonists, act on the G protein-coupled melatonin receptor 1 (MT1) and melatonin receptor 2 (MT2), which are distributed across various tissues. By targeting these receptors, they promote sleep by regulating circadian rhythms, rather than through a sedative effect [75,76].

Our search identified three melatonin receptor agonists studied in relation to dream modulation: *prolonged-release melatonin* (*PRM*), *ramelteon*, and *agomelatine*. *PRM*, designed for older adults with low *melatonin* levels, mimics the behavior of endogenous *melatonin* by ensuring a gradual release over 8–10 h [77]. *Ramelteon* offers higher circulating levels and a longer half-life than *melatonin* [76]. *Agomelatine*, an MT1/MT2 agonist and weak 5-HT2C antagonist, combines circadian regulation with antidepressant and anxiolytic properties [78–80].

3.3.2. Use for RBD treatment

Beneficial effects of *melatonin* at doses ranging from 3 mg to 12 mg on RBD symptomatology have been observed by several studies [41,81,82], with outcomes comparable to those obtained with *clonazepam* treatment [83]. In a recent review, Gilat and colleagues [63] reported that *melatonin* was effective in treating 59.9 % out of a total of 137 individuals with RBD across studies. However, most observations came from case reports and retrospective medical histories, with few available randomized placebo-controlled trials and prospective observational studies.

Moreover, findings from the few randomized placebo-controlled studies are inconsistent: two studies [43,45] reported no improvement in RBD symptoms following *PRM* treatment, while another [42] found only partial symptom improvement with *melatonin* compared to placebo. Although this evidence suggests the need for further adequately powered double-blinded assessments to confirm melatonin's efficacy, its higher tolerability compared to *clonazepam* makes it a safer first-line treatment for RBD, especially in the elderly patients and those with concomitant neurologic disorders [63,68].

Evidence for the efficacy of melatonin receptor agonists in RBD treatment is even scarcer, with preliminary data suggesting *ramelteon* may help in secondary RBD but not isolated cases [84–87], and only one case report supporting *agomelatine*'s potential benefit [47].

The exact mechanism by which *melatonin* improves RBD symptomatology is unclear. Some authors [41,88] have suggested that *melatonin*'s efficacy may depend on its role in reestablishing circadian rhythm synchronization. Additionally, studies employing polysomnographic monitoring showed that *melatonin*, unlike *clonazepam*, may restore REM sleep muscle atonia, as decreased percentage of REM sleep without atonia [42] and decreased tonic muscle activity during REM sleep [81] were observed after *melatonin* treatment.

3.3.3. Documented effects on dream content

We identified eight studies examining the impact of *melatonin* [34, 41–44] or melatonin receptor agonists [45–47] on dream content (see Table 2). Notably, the studies differed markedly in the methods used to assess dream content.

The only two prospective studies adopted a quantitative approach based on structured questionnaires [43,44]. Gilat and colleagues [43] employed a parallel-group design to assess the frequency of vivid dreams in patients with PD-RBD using item 5 of the weekly CIRUS-RBDQ, which asks whether vivid dreams occurred and, if so, how many and how intense they were. The authors found no significant difference between *melatonin* and placebo groups ($p = 0.49$). In contrast, Kahan and colleagues [44] employed a within-subject design to examine the effects of *melatonin* on dream bizarreness in healthy participants, reporting no overall effect ($p > 0.05$). However, a significant interaction with sex emerged for two specific subcomponents of bizarreness. In both cases, *melatonin* increased the frequency of transformations, defined as the physical change of one element into another, in females, while decreasing it in males. This pattern was observed for transformations involving objects ($p = 0.02$) as well as for more general transformations affecting settings and dream characters ($p = 0.05$). Importantly, this was the only study to assess potential sleep differences in the analyses.

The study by Jun and colleagues (2019) [45] was the only retrospective study to use a quantitative approach and a standardized questionnaire with pre-post treatment assessment in a randomized controlled trial. Participants with iRBD received *PRM* at two different doses (2 mg/day or 6 mg/day) or placebo over four weeks, and changes in dream-related symptoms were evaluated using the 'Factor 1' composite score of the Korean version of the RBDQ [89]. Strikingly, this was also the only retrospective study to report no significant effect of treatment on dream content ($p = 0.782$), in line with the other two quantitative, albeit prospective, studies [43,44], which similarly did not detect significant effects of melatonin on dream experiences.

In contrast, the remaining five studies relied on clinical interviews, without the use of validated tools for dream analysis. Among these, four studies reported a marked reduction in disturbing dreams (variably described as frightening, vivid, or violent) following treatment with melatonin in RBD participants [34,41,42] or *agomelatine* in iRBD participants [47]. In all cases, disturbing dreams were present at baseline and appeared to remit completely after treatment durations ranging from four weeks [42] to six months [47]. However, these findings emerged from uncontrolled designs, and one of the studies [47] was a case report, thus considerably limiting the strength and generalizability of the conclusions.

The only study to report a negative effect of treatment on dream content was Shah and Kablinger (2015) [46], who identified nightmares as a potential adverse reaction to *ramelteon* in one patient with ADHD. Once again, the reliance on a single case in this study strongly limits the generalizability of the finding.

Taken together, the results appear to diverge depending on the methodological approach: while the most quantitatively robust study failed to detect any effect, regardless of whether the design was prospective or retrospective, findings from retrospective and observational designs pointed to a positive impact of melatonergic compounds on disturbing dream experiences.

3.4. Levodopa, pramipexole, and other dopamine agonists

3.4.1. Mechanism of action

PD is characterized by the progressive neurodegeneration of the nigrostriatal dopaminergic pathways, leading to the hallmark motor symptoms of the disease [90]. Pharmacological strategies aim to address this dopamine deficiency either by providing dopamine precursors or by directly stimulating dopamine receptors [91].

Dopamine itself cannot serve as pharmacological treatment of PD as it does not cross the blood-brain barrier. On the contrary, *levodopa*, an immediate precursor for catecholamines, readily penetrates into the brain where it is decarboxylated into dopamine, working as an effective

Table 2
Description of the studies investigating the impact of melatonin and melatonin receptor agonists on dream content.

Reference	Effect direction	Effect	Method	Diagnosis	Statistical design	Design (P/R)	Assessment
MELATONIN							
[41] Kunz & Bes, 1999	⇓	Frightening dreams	Clinical interview	RBD	Pre- vs Post-Treatment	R	QUAL
[34] Lloyd et al., 2012	⇓	Nightmares	Clinical interview	RBD	Pre- vs Post-Treatment	R	Semi-QUAN
[42] Kunz & Mahlberg, 2010	⇓	Frightening dreams	Clinical interview	iRBD; sRBD	Melatonin vs Placebo vs Baseline	R	QUAL
[43] Gilat et al., 2020	⇔	Vivid dreams	Questionnaire (wCIRUS-RBDQ [43])	PD-RBD	Melatonin vs Placebo	P	QUAN
[44] Kahan et al., 2000	⇔	Bizarreness ($p > 0.05$)	Bizarreness Questionnaire [44]	Healthy	Melatonin vs Placebo	P	QUAN
	⇓ M; ⇑ F	Transformations of objects ($p = 0.02$) Transformations in general ($p = 0.05$)					
MELATONIN RECEPTOR AGONISTS							
[47] Bonakis et al., 2012	⇓	Vivid, violent dreams	Clinical interview	iRBD	Agomelatine Pre- vs Post-Treatment	Case Report	QUAL
[45] Jun et al., 2019	⇔	RBD dream symptoms (factor 1; $p = 0.782$)	Questionnaire (RBDQ [69])	iRBD	Prolonged-release melatonin Pre- vs Post-treatment (inter- and intra-group)	R	QUAN
[46] Shah & Kablinger, 2015	⇑	Vivid nightmares	Clinical interview	ADHD and insomnia	Ramelteon Pre- vs Post-Treatment	Case Report	QUAL

⇑, Increase; ⇓, Decrease; ⇔, No change; (purple), Retrospective; (tan), Case report.
Abbreviations: M, males; F, females; wCIRUS-RBDQ, weekly CIRUS-RBD questionnaire; RBDQ, REM sleep behavior disorder questionnaire [69]; RBD, REM sleep behavior disorder; iRBD, Isolated RBD; sRBD, Secondary RBD; ADHD, Attention deficit hyperactivity disorder; R, Retrospective; P, Prospective; QUAN, Quantitative; QUAL, Qualitative; Semi-QUAN, Semi-quantitative.

dopamine precursor [92]. To enhance central availability and minimize peripheral side effects, *levodopa* is usually co-administered with a peripheral decarboxylase inhibitor, such as carbidopa [93]. Nevertheless, long-term *levodopa* therapy is frequently complicated by motor fluctuations and dyskinesias, likely due to pulsatile receptor stimulation and the progressive decline of remaining nigrostriatal neurons [94].

To mitigate these complications, dopamine agonists have gained popularity, either as monotherapy or as adjuncts to *levodopa*. Compared to *levodopa*, dopamine agonists such as *pramipexole* provide a more continuous dopaminergic stimulation and may delay the onset of *levodopa*-induced motor complications. For this reason, they are considered a preferable initial therapy in younger patients, though their potential neuroprotective effects remain unconfirmed [95]. In older patients, *carbidopa-levodopa* remains the first-line therapy, as dopamine agonists have been associated with increased psychiatric adverse effects in this age group [96].

3.4.2. *Use for RBD treatment*

Evidence concerning the effect of *levodopa* on RBD symptoms is very limited and, given the nature of the medication, has been mainly assessed in patients with sRBD.

One retrospective study [97] and one case report [98] showed that *levodopa* treatment led to amelioration of RBD symptoms in six individuals with PD or iRBD and three individuals with iRBD, respectively. Conversely, Fernandez-Arcos et al. (2016) [99] observed that *levodopa* was ineffective in treating dream-enacting behaviors in one patient.

Treatment of RBD with dopamine agonists has produced mixed results. *Pramipexole* has been shown to improve RBD symptoms in some studies [52,100,101], while others [53,99] found it to be ineffective in controlling RBD. Finally, another study [102] involving a larger sample of participants found that people with iRBD responded significantly less to *pramipexole* compared to *clonazepam* monotherapy or combined *clonazepam* and *pramipexole* treatment. However, polysomnographic data suggested that *pramipexole* was specifically effective in a subset of patients with mild disease severity.

3.4.3. Documented effects on dream content

We identified eight studies examining the effect of dopaminergic drugs on dream content, focusing on *levodopa* [48–51], *pramipexole* [52, 53], and unspecified dopamine agonists [54,55] (see Table 3).

All studies assessing the impact of *levodopa* or *pramipexole* relied on retrospective methods, such as clinical interviews or standardized questionnaires [49,51,112]. The four studies involving *levodopa* [48–51] consistently associated this medication with an increase in vivid and disturbing dreams ($p < 0.0001$ [48]) [50,51], including nightmares ($p < 0.025$ [49]) [50] and night terrors [50] in PD patients. Nausieda and

Table 3
Description of the studies investigating the impact of levodopa, pramipexole, and other dopamine agonists on dream content.

Reference	Effect direction	Effect	Method	Diagnosis	Statistical design	Design (P/R)	Assessment
LEVODOPA							
[50] Sharf et al., 1978	⇑	Nightmares Vivid dreams Night terrors	Clinical interview	PD	Levodopa Pre- vs Post-Treatment	R	Semi-QUAN
[51] Nausieda et al., 1982	⇑	Altered dreams: vivid dreams antedating nightmares, night terrors, dreams with morbid content	Questionnaire [51]	PD	Levodopa PD with vs w/ sleep disturbances	R	Semi-QUAN
[48] Growdon et al., 1998	⇑	Vivid dreams (p < 0.0001)	UPDRS Thought Disorder Scale [112]	PD	Levodopa Pre- vs Post-Treatment	R	QUAN
[49] Kumar et al., 2002	⇑	Nightmares (p < 0.025)	Questionnaire [49]	PD	Levodopa Correlation dream symptoms-drug use	R	QUAN
PRAMIPEXOLE AND OTHER DOPAMINE AGONISTS							
[52] Sasai et al., 2012	⇓	Nightmares	Clinical interview	iRBD	Pramipexole Pre- vs Post-Treatment	R	Semi-QUAN
[53] Kumru et al., 2008	⇔	Unpleasant dreams (p = N/A)	PSQI [103]	PD-RBD	Pramipexole Pre- vs Post-Treatment	R	QUAN
[54] De Gennaro et al., 2016	⇓	Dream emotional load (p = 0.04) Dream bizarreness (p = 0.05)	Reports analysis with scales [54]	PD	Dopamine agonists Correlation dream symptoms-drug use	P	QUAN
[55] D'Agostino et al., 2010	⇔	Dream bizarreness (p > 0.05)	Reports analysis with scales [104]	iPD	Dopamine agonists Correlation dream symptoms-drug use	P	QUAN

⇑, Increase; ⇓, Decrease; ⇔, No change; (purple), Retrospective; (green), Prospective.
Abbreviations: N/A, not available; UPDRS, Unified Parkinson's Disease Rating Scale [112]; PSQI, Pittsburgh Sleep Quality Index [103]; PD, Parkinson's disease; iPD, Isolated PD; iRBD, Isolated REM sleep behavior disorder; PD-RBD, PD with concomitant RBD; R, Retrospective; P, Prospective; QUAN, Quantitative; QUAL, Qualitative; Semi-QUAN, Semi-quantitative.
Scales for the analysis of reports included 6-point Likert rating scales [54] developed by the authors, Bizarreness scale [104].

colleagues (1982) [51] further noted a progression in dream alterations over time, with vivid dreams preceding the onset of more severe symptoms such as night terrors and dreams with morbid content.

In contrast, the two studies examining the effects of *pramipexole* yielded only partially converging results. Sasai et al. (2012) [52] reported an apparent reduction in the frequency of disturbing dreams based on a semi-quantitative evaluation derived from clinical interviews in iRBD participants. Kumru and colleagues (2008) [53], by contrast, employed a more structured and quantitative approach using the relevant item of the Pittsburgh Sleep Quality Index (PSQI) [103] to quantify unpleasant dream frequency in PD-RBD participants. Although they observed a similar trend toward reduced dream disturbance, the change did not reach statistical significance, ultimately failing to demonstrate a reliable treatment effect (p-value not specified).

A different methodological approach was adopted by the two studies that prospectively investigated the effects of unspecified dopamine agonists, both relying on the daily collection of dream reports upon awakening and their subsequent scoring using predefined scales. Despite the use of similar procedures, the studies yielded divergent results. De Gennaro and colleagues (2016) [54] reported a significant association between dopaminergic treatment and reduced dream bizarreness ($p = 0.05$) and emotional intensity ($p = 0.04$), as rated on a 6-point Likert scale in PD participants. In contrast, D'Agostino and colleagues (2010) [55], employing the scale developed by Hobson and colleagues (1987) [104], found no significant correlation between dopamine agonist intake and dream bizarreness ($p > 0.05$) in PD participants.

Taken together, retrospective studies involving *levodopa* appear to consistently link dopaminergic treatment with altered or intensified dream experiences, while less conclusive evidence are obtained by retrospective and prospective approaches investigating the impact of pramipexole and other dopamine agonists on nightmares, unpleasant dreams, or dream emotional intensity and bizarreness.

4. Discussion

This review assessed the potential effects of medications commonly used to treat iRBD or sRBD on dream content. We found that, although *clonazepam* and *melatonin* are commonly assumed to lessen nightmares and violent or frightening dreams, the evidence supporting this assumption is limited and based mainly on few retrospective studies employing qualitative or semi-quantitative methods. Evidence for the dopaminergic agent *pramipexole* was also mixed: one study reported a reduction in unpleasant dream content, whereas another found no significant changes in nightmare frequency under treatment. Similarly, no consensus emerged concerning the impact of other dopamine agonists on dream content, as one study observed a reduction in dream emotional load and bizarreness, while the other found no changes in dream bizarreness. More consistent evidence emerged for *levodopa*, with several retrospective studies indicating an increase in the frequency or intensity of vivid or disturbing dreams, including nightmares. Overall, though, a critical methodological appraisal of the available literature highlighted the need for great caution when summarizing and interpreting the available pieces of evidence.

Drawing robust conclusions about the potential impact of RBD medications on dream content is challenging because the literature is sparse and methodologically inconsistent, making studies difficult to compare. Moreover, recent reviews [62,63,68] highlight that most studies on RBD treatments are underpowered and rarely employ double-blind, placebo-controlled designs, increasing the risk of bias. These limitations are especially critical in the context of the present review as most of the included studies did not focus on dream content as a primary outcome, often assessing it only incidentally or anecdotally. A considerable number of the reviewed studies did not include statistical comparisons to support their observations, or failed to clearly report them. Most relied on pre-post treatment comparisons, and only a few included comparisons with placebo or drug-free groups. Critically, in many cases, data were not systematically collected through structured paradigms, but rather derived from clinical records, often lacking detail about how dream reports were elicited, recorded, and analyzed. As summarized in Supplementary Table S1, information regarding concomitant medications, dosages, timing of administration, and screening for additional sleep disorders was not reported systematically across studies. When available, the data revealed a highly heterogeneous picture: treatment regimens varied considerably in dosage and administration schedules, additional medications with potential effects on sleep and dreaming were not always controlled for, and screening for comorbid sleep disorders was either absent or applied inconsistently. This lack of systematic reporting further complicates the comparison of findings across studies and limits the strength of any conclusions regarding medication effects on dream content.

A particularly important source of variability and potential inconsistency across studies appeared to lie in the methods used to collect and analyze dream reports. In line with common practices in dream research, most of the reviewed studies relied on retrospective data collection, with only 10 of the 27 included articles (37 %) adopting instead a prospective approach [17,36–40,43,44,54,55]. Crucially, retrospective approaches are easier to implement in clinical settings but more susceptible to cognitive biases that can reshape memories through integration and reprocessing [23]. Therefore, while such methods can offer a broader and potentially more integrated picture of an individual's oneiric life over extended periods, their interpretive value must be considered with caution. Conversely, prospective approaches offer a narrower yet more temporally precise and ecologically valid snapshot of dream experiences, especially when based on the last dream upon awakening. The partly distinct focus of these methods and their susceptibility to different sources of bias may lead to divergent results in certain circumstances. In this respect, the case of *clonazepam* — one of the few medications for which both retrospective and prospective studies were available — seems particularly worthy of discussion. Indeed, positive effects of *clonazepam* on dream content were reported only in the retrospective quantitative studies [32,33] but not in prospective ones [17,36]. This discrepancy raises the question of whether and to which degree pharmacological effects on oneiric activity reported by some studies reflect genuine changes in dream phenomenology or recall biases related to the data collection method.

On the one hand, *clonazepam*'s anxiolytic properties may actually promote more positively toned dreams, pointing to a direct effect of the medication on dream generation mechanisms [17,19,56,57]. Yet, on the other hand, a reduction of overt dream enactment behaviors could lead to improved sleep quality and continuity, with a lower frequency of awakenings caused by violent movements or involuntary impacts against surrounding objects or the bed partner. Indeed, dreams that are not shortly followed by an awakening typically fail to be encoded in memory and are not recalled upon the final morning awakening [17,23]. Thus, the observed reduction in negative or aggressive dream content may stem from fewer enactment-related awakenings, limiting recall opportunities rather than altering dream generation. Since dream enactments often coincide with emotionally intense content and facilitate memory encoding, studies relying solely on retrospective recall may overrepresent such experiences [17,23].

These considerations underscore the need for studies combining both retrospective and prospective methodologies. In particular, prospective approaches should aim to sample dream content independently of enactment episodes to reduce potential bias introduced by enactment-triggered arousals or by awakenings caused by the bed partner, who may be struck or alerted by the onset of enactment behaviors. This could be achieved through randomized serial awakenings or by focusing on the last experience upon physiological morning awakening. Notably, none of the reviewed studies attempted to dissociate these factors, leaving the relationship between dream content and motor behaviors largely unexplored. Yet, clarifying whether current pharmacological treatments act on dream generation or on dream recall has important implications that go beyond methodological concerns. From a clinical standpoint, it would deepen our understanding of the mechanisms underlying RBD, including the potential of dream alterations to serve as early markers of neurodegenerative diseases. Moreover, if certain dream features, such as emotional intensity or threat perception, contribute to the likelihood or severity of enactment behaviors, then dreams themselves may represent a meaningful therapeutic target. In this light, if current treatments mainly affect dream enactment rather than content, developing interventions targeting dream features could valuably complement existing therapies. Another key limitation of both retrospective and home-based prospective protocols is their inability to determine the sleep stage from which dream reports originate. REM and nREM dreams differ in intensity, complexity, and the presence of social content, likely reflecting distinct underlying mechanisms and neuromodulatory environments that may interact differently with medications. In contrast, prospective laboratory-based approaches, such as serial awakenings or structured morning collections, enable precise attribution of dream content to specific sleep stages. However, such methods remain underutilized. Among all the studies reviewed, only two [37,38] employed prospective, laboratory-based protocols, and only Gaillard & Phelippeau [38] used a serial awakening design with explicit sleep stage classification. Critically, the lack of control over the sleep stage may significantly reduce consistency across studies, potentially masking genuine pharmacological effects on dream content.

A further source of methodological variability and concern lies in the nature of the collected dream data. Of the 27 studies reviewed, 9 (33 %) relied solely on clinical observations or patient history [7,20,34,41,42,46,47,50,52], a choice that limits objectivity and reproducibility of their findings. Only 5 studies (19 %) collected extensive dream reports [17,38,39,54,55], with only one combining them with a predefined questionnaire [39]. The remaining 13 studies (48 %) relied exclusively on predefined questionnaires [5,32,33,35–37,40,43–45,48,49,51,53]. Among the latter, most included only one or two questions about dreams, except for those employing versions of the RBDQ [32,33,45]. Questionnaires can help target specific aspects of dream content, but they may also limit participants' responses. This can introduce bias and reduce descriptive richness, especially when only a few questions are included. By contrast, collecting and analyzing full dream reports is more time-consuming, but it provides extensive information about the narrative and logical structure of dreams, particularly relevant when evaluating pharmacological effects. Given the current lack of a coherent and comprehensive literature on this topic, formulating specific, hypothesis-driven research questions is often not feasible. In this context, relying solely on narrowly focused questions may further limit our understanding of how medications influence dream content.

Once collected, dream reports can be analyzed using predefined scoring systems (e.g., the Hall and Van de Castle method [71]) or (semi-) automated computational linguistic methods to extract information about dream content. All the studies included in this review that collected verbal reports relied on manual coding based on predefined scales for dream content analysis [17,38,39,54,55]. However, manual scoring introduces potential within- and across-studies variability due to inter-rater discrepancies. Furthermore, each study employed a different coding scheme with specific semantic categories, further hindering cross-study comparisons and reproducibility of results. Semi-automated or fully automated methods could offer a more objective and reproducible alternative, enabling consistent extraction of quantitative features from textual data [105].

Another critical limitation affecting the available literature is the lack of clear and consistent definitions for the phenomena under investigation. Most of the reviewed studies, indeed, failed to define key terms such as "frightening dreams", "sorrowful content", "vividness", or "bizarreness", for which no standardized operational definitions exist. This lack of specification undermines the interpretability and comparability of findings, as participants may have construed these terms differently, resulting in inconsistent data within and across studies. Establishing and reporting precise definitions is essential to ensure that participants provide responses that accurately reflect the aim of the study, as well as methodological reproducibility. The same holds true for the term "dream" itself. As discussed elsewhere [22,106,107] [22,107,108], the term "dream" may seem straightforward, and most people would easily answer a question like "Did you have a dream last night?". Yet, establishing a commonly accepted scientific definition has proven difficult. According to the chosen framework, the term can refer to a wide range of subjective experiences — from simple, static perceptions (isolated images, sounds, bodily sensations, or thoughts), to more complex and dynamic experiences — or exclusively to immersive, multisensory, and narrative-like experiences that are more common in REM sleep. The lack of clear definitions may increase variability in the obtained responses, as each participant may apply a slightly different idea of what should be considered and reported. Importantly, such a variability may be expected to be further amplified by interindividual differences in psychological and cognitive traits, which may shape how dreams are subjectively experienced, remembered, and reported [108]. This additional layer of variability introduces noise that may obscure the specific effects of experimental manipulations or pharmacological treatments on dream content. Providing participants with specific, detailed instructions on what aspects should be attended to and reported could help mitigate these biases, enhancing both the consistency of data collection and the reproducibility of findings.

5. Conclusion

In summary, while the influence of *clonazepam* and other pharmacological treatments on dream content cannot be definitely ruled out, it appears to be only weakly supported by the available literature. At a critical methodological assessment, the available literature showed crucial weaknesses, primarily stemming from methodological heterogeneity and the lack of objective, structured, and reproducible paradigms shared across studies.

Future research should prioritize well-controlled, adequately powered trials, ideally employing both retrospective and prospective designs, as well as systematic dream collection through both detailed reports and focused questionnaires. The use of objective and reproducible analytic tools, including computational linguistic methods, should be also prioritized. Equally important is the establishment of shared, operationalized definitions for key terms, to enhance comparability across studies and ensure internal coherence. Finally, future studies may also benefit from integrating electrophysiological and neuroimaging approaches, which could help clarify how pharmacological treatments influence specific brain activity patterns during sleep and, in turn, how these neural changes relate to alterations in dream content and features.

Addressing these methodological limitations is essential to advance our understanding of how medications influence dream content and to distinguish direct pharmacological effects on dream experiences from those mediated by changes in sleep architecture or behavior.

CRediT authorship contribution statement

Giorgia Bontempi: Writing – review & editing, Writing – original draft, Methodology, Investigation, Data curation, Conceptualization. **Elena Capriglia:** Writing – review & editing. **Giulio Bernardi:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Investigation, Conceptualization. **Valentina Elce:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Investigation, Conceptualization.

Data availability statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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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.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2025.106878.

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