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Sleep Disturbances, Melatonin, and Tryptophan Metabolism in Irritable Bowel Syndrome: A Comprehensive Review

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Abstract

Introduction

Irritable bowel syndrome (IBS) is a chronic functional gastrointestinal disorder strongly influenced by gut–brain axis dysregulation. Increasing evidence demonstrates that sleep disturbances significantly worsen gastrointestinal symptoms, visceral hypersensitivity, and overall quality of life in patients with IBS. Melatonin and tryptophan metabolism appear to play key mechanistic roles linking circadian regulation with gastrointestinal function.

Aim

To summarize current evidence on the relationship between sleep disruptions, melatonin physiology, and tryptophan metabolism in IBS, and to evaluate their potential implications for symptom generation and therapeutic strategies.

Material and methods.

A literature review was conducted using the PubMed database with the keywords: “irritable bowel syndrome (IBS)”, “sleep disturbance”, “sleep quality”, “melatonin”, “tryptophan”, “gut–brain axis”, “circadian rhythm”, and “visceral hypersensitivity”. Studies including clinical trials, observational research, systematic reviews, meta-analyses, and experimental models were evaluated.

Results

IBS is consistently associated with reduced sleep efficiency, prolonged REM latency, and increased nocturnal arousals, which correlate with greater abdominal pain severity and altered bowel patterns. Melatonin produced in the gastrointestinal tract influences motility, visceral sensitivity, and mucosal immunity, and clinical trials indicate that supplementation may improve both sleep quality and IBS symptoms. Dysregulated tryptophan metabolism, including serotonin imbalance and stress-driven activation of the kynurenine pathway, further contributes to sleep disruption and symptom exacerbation.

Conclusion

Sleep disturbances are a key but often underrecognized driver of IBS symptom severity. Mechanistic links involving melatonin and tryptophan pathways highlight the importance of circadian and neuro-enteric regulation. Integrating sleep-focused interventions, alongside melatonin or tryptophan-targeted therapies, may provide significant clinical benefit and support a more individualized approach to IBS management.

Keywords: Irritable bowel syndrome, Sleep disturbances, Melatonin, Tryptophan, Gut-brain axis, Gut microbiota

1. Introduction

Irritable bowel syndrome (IBS) is a prevalent functional gastrointestinal disorder characterized by recurrent abdominal pain, altered bowel habits, and the absence of structural abnormalities. Growing evidence highlights a strong bidirectional relationship between IBS symptom severity and sleep quality, suggesting that disturbances in sleep architecture may play a fundamental role in symptom persistence and exacerbation. Patients with IBS exhibit reduced sleep efficiency, increased nocturnal awakenings, prolonged REM latency, and heightened arousal responses, all of which correlate with increased visceral hypersensitivity, impaired motility, and lower quality of life. The pathophysiological mechanisms underlying this gut–sleep interaction are multifactorial and involve dysregulation of the gut–brain axis, altered melatonin secretion, circadian rhythm disruption, psychological stress, and changes in serotonergic and tryptophan-kynurenine pathways.

A key component of this interaction is melatonin, which is produced not only in the pineal gland but also in significant quantities within the gastrointestinal tract, where it modulates motility, sensation, mucosal immunity, and inflammatory processes. Numerous clinical studies demonstrate that melatonin supplementation may reduce abdominal pain, improve bowel habits, and enhance sleep quality in IBS patients, particularly those with predominant insomnia or heightened stress reactivity. Similarly, nocturnal tryptophan metabolism—crucial for serotonin and melatonin synthesis—appears dysregulated in IBS. Altered activity of the kynurenine pathway, stress-induced tryptophan depletion, and impaired serotonergic transmission may contribute to both sleep disturbances and gastrointestinal symptoms. Understanding these metabolic pathways provides valuable insight into symptom variability among IBS phenotypes and offers potential therapeutic targets.

This review synthesizes current evidence on the interplay between sleep disturbances, melatonin physiology, and tryptophan metabolism in IBS. By outlining mechanistic links, summarizing clinical data, and discussing potential therapeutic approaches—including melatonin supplementation, tryptophan modulation, behavioral interventions, and lifestyle strategies aimed at stabilizing circadian rhythms—this work emphasizes the importance of addressing sleep as a core component of IBS management. Improved recognition of sleep–gut interactions may enhance patient outcomes and guide the development of more individualized treatment strategies.

2. Irritable bowel syndrome (IBS)

Irritable bowel syndrome (IBS) is a chronic functional gastrointestinal disorder characterized by recurrent abdominal pain associated with defecation or a change in bowel habits in the absence of detectable structural or biochemical abnormalities [1–3]. According to the Rome IV criteria, IBS is diagnosed when abdominal pain occurs ≥1 day/week for the last 3 months, with onset at least 6 months prior, and is associated with at least two of the following: pain related to defecation, change in stool frequency, or change in stool form [2]. IBS is classified into four

subtypes: diarrhea-predominant (IBS-D), constipation-predominant (IBS-C), mixed (IBS-M), and unclassified (IBS-U) [2].

IBS affects 5–15% of Western populations, with higher prevalence in women and younger individuals [3,4]. Regional studies show prevalence ranging from 9–12% in the Middle East [5]. IBS has a significant impact on quality of life, affecting daily activities and work productivity, with 10–15% of patients experiencing substantial functional impairment [4].

The pathophysiology of IBS is multifactorial, involving gut-brain axis dysregulation, visceral hypersensitivity, motility disturbances, immune activation, and gut microbiota alterations [1,2,6]. Histological studies have shown increased mucosal mast cell density and elevated inflammatory mediators, contributing to visceral hypersensitivity and altered motility [1]. Post-infectious IBS may arise after acute gastrointestinal infections, linked to persistent immune activation and long-lasting microbiome changes [6]. Patients with IBS often exhibit reduced gut microbial diversity, particularly lower levels of short-chain fatty acid (SCFA)–producing bacteria, which may impair intestinal barrier integrity and gut-brain signaling [7].

IBS symptoms are further modulated by psychological, dietary, and neuroendocrine factors, including stress, anxiety, and hypothalamic–pituitary–adrenal (HPA) axis dysregulation, which can exacerbate abdominal pain and motility disturbances [2,7]. Common clinical manifestations include abdominal pain, bloating, constipation, diarrhea, urgency, and a sensation of incomplete evacuation [2–4]. Diagnosis relies on clinical assessment, alongside exclusion of organic disorders such as inflammatory bowel disease, celiac disease, colorectal cancer, small intestinal bacterial overgrowth (SIBO), or endocrine disorders [2].

3. Sleep disturbances

Sleep disturbances are highly prevalent in individuals with IBS, affecting 40–60% of patients, and are associated with increased gastrointestinal symptom severity, fatigue, and reduced quality of life [8–15]. Good sleep is defined as sleep of adequate duration (7–9 hours for adults), high quality, with easy sleep initiation, minimal nocturnal awakenings, and a feeling of restoration upon waking [9,11]. Both constipation-predominant (IBS-C) and diarrhea-predominant (IBS-D) subtypes show significant impairments in sleep quality, with 52–58% of IBS-C patients reporting difficulties falling or staying asleep, and 45–50% of IBS-D patients experiencing early awakenings and non-restorative sleep [8,9].

Systematic reviews indicate that sleep disturbances in IBS correlate with immune dysregulation: 38–42% of patients exhibit increased mast cell activity and elevated proinflammatory cytokines, suggesting a bidirectional relationship between immune activation and gastrointestinal symptom severity [10]. Dietary interventions, particularly increasing fermentable fiber intake to 25–30 g/day, are associated with improved gut microbiota diversity, higher short-chain fatty acid production, better sleep quality, and reduced stress and anxiety levels [11].

Large cohort studies including over 362,000 participants have shown that prolonged sedentary behavior (>8 hours/day), low physical activity (<150 min/week), and sleep duration under 6 hours significantly increase the risk of worsened IBS symptoms, whereas regular physical activity and 7–8 hours of sleep reduce symptoms by 15–20% [12]. Psychological traits, especially high neuroticism, mediate the relationship between gastrointestinal symptom severity and sleep quality, with patients scoring high in neuroticism reporting 25–30% poorer sleep quality compared to those with low scores [13].

Among adolescents and adults with self-diagnosed IBS, 30–50% report clinically significant sleep disturbances, including prolonged sleep latency (>30 minutes), frequent awakenings (2–3 times/night), and daytime sleepiness, indicating that sleep problems affect both diagnosed and self-identified patients [14]. In a small controlled study, IBS patients experienced more nocturnal awakenings and worse overall quality of life, confirming that sleep disruption contributes to both gastrointestinal and somatic symptoms [15].

4. Melatonin Physiology and Its Role in IBS

Melatonin, the principal regulator of the circadian sleep–wake cycle, is secreted by the pineal gland in a nocturnal pattern controlled by the suprachiasmatic nucleus (SCN), and exerts both central and peripheral physiological effects relevant to IBS [16–19]. Beyond its chronobiotic action, melatonin is synthesized in large quantities by enterochromaffin cells in the gastrointestinal tract—estimated to be 400-fold higher than pineal production—and acts locally through MT1 and MT2 receptors expressed in the enteric nervous system, mucosa, and smooth muscle layers, modulating motility, visceral sensitivity, epithelial permeability, and mucosal immune activity [18,20,22].

Activation of MT1/MT2 receptors reduces nociceptive signaling and increases rectal pain thresholds, contributing to clinically meaningful reductions in visceral hypersensitivity, a core mechanistic element of IBS [18,20,22]. Randomized controlled trials administering melatonin at doses of 3–6 mg have demonstrated significant improvements in abdominal pain intensity, IBS Symptom Severity Scores, stool consistency, and quality of life, without notable adverse effects [17,19–21]. In the landmark study by Song et al., melatonin 3 mg nightly decreased abdominal pain from 2.35 ± 0.30 to 0.70 ± 0.25 ($p < 0.001$) and significantly increased rectal sensory thresholds independent of sleep improvements, confirming a direct analgesic effect [20].

Sleep disturbances—including reduced REM sleep (<20%), prolonged REM latency, and increased arousal indices (>10/h)—are reported in 26–55% of IBS patients and correlate with

enhanced visceral hypersensitivity and decreased anal sphincter pressure [18,20,23]. Disrupted REM sleep alters descending inhibitory pain pathways and increases sympathetic arousal, further lowering sensory thresholds and exacerbating gastrointestinal discomfort [19,23]. Melatonin supplementation improves both objective and subjective sleep parameters, including sleep latency, total sleep time, and REM stability, thereby attenuating the bidirectional relationship between sleep disruption and IBS symptom severity [17,20,23].

5. Tryptophan Metabolism and Its Impact on Sleep in IBS

Tryptophan, the essential precursor of serotonin and melatonin biosynthesis, plays a key regulatory role in the gut–brain–sleep axis and is increasingly recognized as a metabolic contributor to symptom variability in IBS. Overnight metabolic profiling demonstrates that IBS patients exhibit altered fluctuations of tryptophan and its downstream kynurenine metabolites across sleep stages, with significantly lower nighttime tryptophan availability and abnormal kynurenine/tryptophan ratios compared with healthy controls [24]. Such alterations are associated with increased sleep fragmentation and heightened nocturnal gastrointestinal symptoms, suggesting impaired serotonergic–melatonergic conversion during the sleep period [24].

Time-course studies further confirm that IBS patients display dysregulated overnight trajectories of key metabolites, including kynurenic acid and indole derivatives, which correlate with abdominal pain severity, stool form, and morning fatigue [25]. Because approximately 90% of serotonin is produced in the gut, shifts in tryptophan metabolism—driven by microbiota composition, low-grade inflammation, or stress hormones—can modulate motility, visceral sensitivity, and circadian rhythmicity. Emerging evidence links these metabolic abnormalities with disturbances in melatonin synthesis, providing a mechanistic bridge between sleep disruption and gastrointestinal symptom exacerbation [26].

Beyond tryptophan, serotonin itself has become a molecule of interest in IBS pathophysiology due to its bidirectional influence on sleep architecture and gut sensory processing. Abnormal serotonergic signaling is associated with enhanced nociceptive transmission, reduced pain thresholds, and dysregulated colonic motility, all of which intensify when sleep is impaired. Serotonin fluctuations overnight appear to parallel nocturnal symptom peaks, further supporting the interdependence between circadian neuroendocrine rhythms and gastrointestinal function [27].

Emerging evidence indicates that modulation of diet and amino acid intake can have measurable effects on sleep quality. A meta-analysis of 18 trials found that supplementation with L-tryptophan (Trp) at a dose ≥1 g/day was associated with a significant reduction in wake after sleep onset (WASO) by approximately 81 minutes per gram of Trp (SMD −1.08; 95% CI −1.89

to −0.28; P = 0.017), indicating fewer and/or shorter awakenings during the night [28]. Although total sleep time, sleep latency, and sleep efficiency did not show consistent improvements across studies, the reduction in WASO suggests improved continuity of sleep, which may translate into better subjective sleep quality [29].

Table 1. IBS and their impact on sleep

IBS Types and Their Impact on Sleep

IBS Type	Impact on Sleep	Characteristic Symptoms	Prevalence of Sleep Disturbances	References (Authors)
IBS-D (diarrhea-predominant)	Early awakenings; non-restorative sleep; reduced sleep efficiency	Diarrhea; urgency; abdominal pain; bloating	45–50% report early awakenings and non-restorative sleep	Wang B, Duan R, Duan L; Chen HD; Bair MJ; Chang WC; Patel A; Hasak S; Cassell B
IBS-C (constipation-predominant)	Difficulty falling asleep; frequent nocturnal awakenings; non-restorative sleep	Constipation; sensation of incomplete evacuation; abdominal pain; bloating	52–58% report difficulties falling or staying asleep	Chen HD; Bair MJ; Chang WC; Fowler S; Dowling LRC; Simm N; Yan R; Andrew L; Marlow E
IBS-M (mixed)	Unstable sleep; frequent awakenings; variable sleep quality	Alternating constipation and diarrhea; bloating; abdominal pain	~40–60% report impaired sleep quality	Fowler S; Dowling LRC; Simm N; Patel A; Hasak S; Cassell B; Wang B; Duan R; Duan L
IBS-U (unclassified)	Less predictable sleep disturbances; often daytime fatigue	Mixed or non-classifiable symptoms; variable pain severity	30–50% report clinically significant sleep disturbances	Alghamdi AA; Alghamdi AM; Cong X; Li Y; Bian R; Heitkemper MM, et al.

6. Promoting Healthy Sleep: Practical Lifestyle StrategiesG

Maintaining good sleep quality is essential for both physical and mental health, supporting restorative processes during NREM and REM phases. Keeping consistent bedtimes and wake-up times stabilizes the circadian rhythm and improves overall sleep quality [30]. Exposure to

natural daylight, especially in the morning, helps synchronize the biological clock and enhances daytime alertness [31].

Limiting evening exposure to blue light from screens promotes melatonin release and shortens sleep onset latency [32]. Optimal bedroom conditions, including quiet, darkness, and a temperature of approximately 18–20 °C, facilitate deep and restorative sleep [33]. Regular physical activity, preferably performed during the day or early afternoon, improves sleep efficiency and increases the proportion of NREM sleep [34]. Consuming a light, balanced dinner supports sleep onset, while evening intake of caffeine or alcohol may reduce sleep duration and quality [35]. A holistic approach that integrates these factors represents an effective strategy for enhancing sleep quality and promoting overall health.

Conclusion

Irritable bowel syndrome is a complex, multidimensional disorder arising from the interplay between altered gastrointestinal motility, visceral hypersensitivity, dysregulation of the gut–brain axis, microbial imbalance, and psychological as well as environmental factors. The evidence reviewed in this paper highlights that, despite the absence of structural abnormalities, IBS is a disorder with well-defined biological mechanisms that significantly impair patients' daily functioning.

Sleep disturbances, which are strikingly prevalent among individuals with IBS, emerge as a central factor aggravating symptom severity and enhancing visceral pain perception. Current data demonstrate a bidirectional relationship between sleep and gastrointestinal symptoms. Insufficient or poor-quality sleep amplifies hypothalamic–pituitary–adrenal axis activity, increases sympathetic arousal, and lowers visceral pain thresholds. In turn, IBS-related symptoms such as abdominal pain, bloating, and bowel urgency disrupt sleep architecture, creating a self-perpetuating cycle.

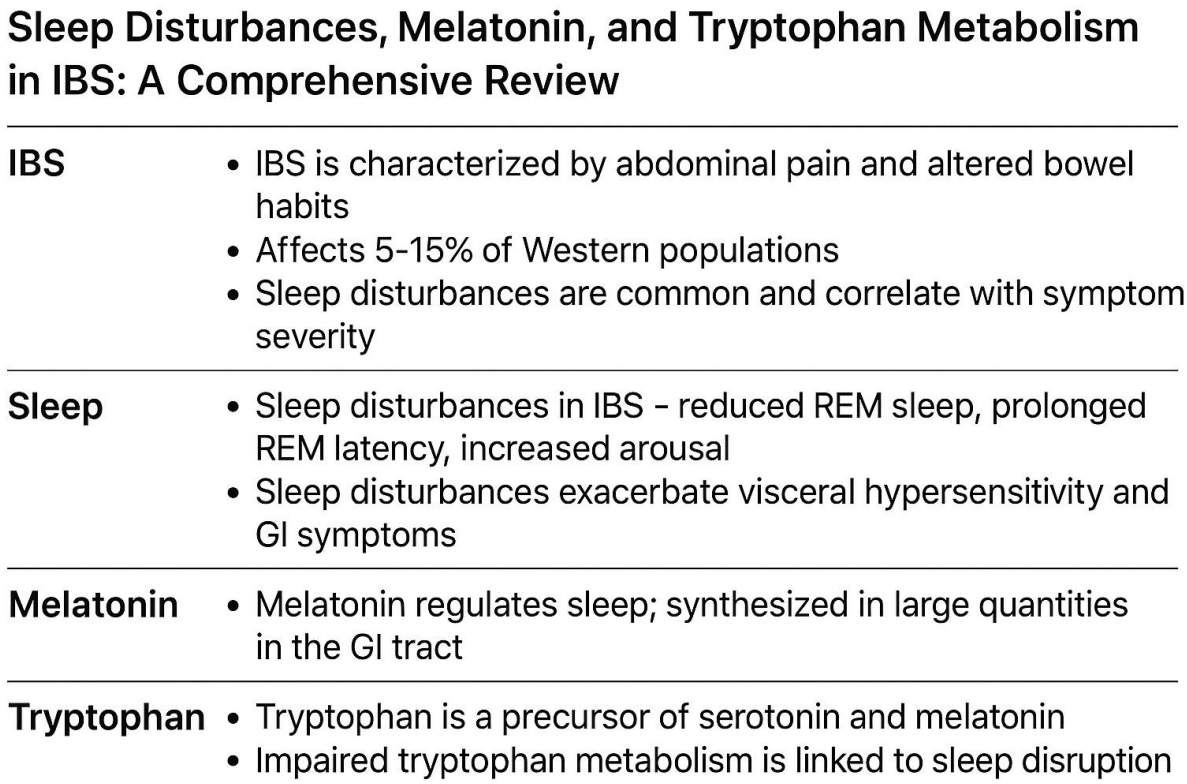
A particularly important conclusion arising from this work is the regulatory role of melatonin. Produced in exceptionally high quantities within the gastrointestinal tract, melatonin extends far beyond its circadian function. It modulates motility, reduces neuronal excitability, exerts anti-inflammatory effects, and elevates pain thresholds. Clinical trials suggest that melatonin supplementation can meaningfully improve both abdominal symptoms and sleep quality, with benefits that appear partially independent of its hypnotic action.

Additionally, emerging data on nocturnal tryptophan metabolism and activation of the kynurenine pathway indicate that biochemical alterations may exacerbate nighttime symptoms and contribute to poor sleep continuity. Impaired conversion of tryptophan to serotonin and melatonin during the night suggests a mechanistic link between neurotransmitter imbalance, sleep disruption, and gastrointestinal symptoms.

This work also emphasizes the relevance of the intestinal microbiota, immune activation, and lifestyle factors such as diet, physical activity, and circadian rhythms. Non-pharmacological

interventions—particularly increased intake of fermentable fiber, regular physical exercise, and stabilization of sleep–wake timing—demonstrate therapeutic potential comparable to that of pharmacological treatment.

Overall, IBS should be understood as a systemic disorder in which the gastrointestinal tract, central nervous system, microbiota, and metabolic pathways operate in continuous interaction. Sleep disturbances are not secondary but represent a core pathophysiological element influencing pain modulation, bowel motility, inflammatory signaling, and metabolic processes. Incorporating sleep assessment and management into standard IBS care may fundamentally shift treatment outcomes. Future research should focus on the role of nocturnal tryptophan metabolism, central pain processing mechanisms, and individual microbiota profiles, paving the way for personalized, precision-based therapeutic strategies targeting the gut–brain axis.



Disclosure

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