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Journal of Education, Health and Sport. eISSN 2391-8306.

Journal Home Page
https://apcz.umk.pl/JEHS/index

OCZKOWSKA, Wiktoria, MORAWSKA, Diana, DRABIK, Przemysław Dominik, MALINOWSKI, Alex, MARCHEWKA, Klaudia, SOCHOCKI, Radosław, BIAŁEK, Wiktoria, SADOWSKA, Agata, PELCZAR, Aleksandra Katarzyna, and BARTOSIK, Kacper. Evaluating the Role of Insomnia in the Clinical Manifestation of Irritable Bowel Syndrome. Journal of Education, Health and Sport. 2026;90:70392. eISSN 2391-8306. https://doi.org/10.12775/JEHS.2026.90.70392

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 30.03.2026. Revised: 07.04.2026. Accepted: 10.04.2026. Published: 14.04.2026.

Evaluating the Role of Insomnia in the Clinical Manifestation of Irritable Bowel Syndrome

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ABSTRACT

Background. Irritable bowel syndrome (IBS) is a common functional gastrointestinal disorder characterized by abdominal pain and altered bowel habits, involving gut–brain axis dysregulation, psychosocial, and immune mechanisms. Insomnia and sleep disturbances are highly prevalent in IBS and may contribute to symptom burden, but their role remains unclear.

Aim. To systematically review the role of insomnia in IBS, focusing on prevalence, symptom severity, mechanisms, and effects of sleep-targeted interventions.

Methods. A comprehensive literature search (including PubMed) was conducted for studies published up to 2026. Observational studies, clinical trials, mechanistic studies, and reviews examining the relationship between insomnia and IBS were included. Data on study design, populations, sleep assessment, IBS criteria, and outcomes were extracted. Due to heterogeneity, a qualitative synthesis was performed.

Results. Insomnia is more prevalent in IBS patients than controls (37–70%). Greater insomnia severity is associated with increased abdominal pain, bloating, bowel disturbances, and reduced quality of life. Longitudinal and genetic evidence suggests insomnia may be an independent risk factor for IBS onset and exacerbation. Proposed mechanisms include gut–brain axis dysregulation, visceral hypersensitivity, stress response alterations, and immune modulation. Subjective sleep disturbances appear particularly predictive of symptom severity. Behavioral interventions, such as cognitive-behavioral therapy for insomnia (CBT-I), show potential to improve both sleep and IBS symptoms, though current evidence is limited.

Conclusions. Insomnia is a common and clinically relevant factor in IBS, acting as both a comorbidity and a modifier of disease severity. Integrating sleep assessment and management into IBS care may improve outcomes. Further large-scale and mechanistic studies are needed to clarify causality and optimize treatment strategies.

Keywords: Irritable bowel syndrome; IBS; Insomnia; Sleep disturbances; Sleep quality; Gut–brain axis; Visceral hypersensitivity; Gastrointestinal symptoms; Quality of life; Cognitive behavioral therapy for insomnia

Introduction

Irritable bowel syndrome (IBS) is a chronic functional gastrointestinal disorder characterized by recurrent abdominal pain and altered bowel habits in the absence of identifiable structural pathology. The global prevalence of IBS ranges from approximately 5–15% of adults, with women disproportionately affected, and it imposes a significant burden on health, quality of life, and healthcare utilization. While the etiology of IBS remains multifactorial and incompletely understood, dysregulation of the gut–brain axis, visceral hypersensitivity, psychosocial factors, and immune modulation have been widely implicated.

Sleep disturbances, particularly insomnia — defined as difficulty initiating or maintaining sleep or non-restorative sleep accompanied by daytime impairment — are recognized as prevalent comorbid conditions in many chronic diseases. Notably, IBS patients exhibit a high burden of sleep complaints, with epidemiological and clinical research consistently demonstrating elevated rates of sleep dysfunction compared with healthy controls [1,2,3]. These disturbances encompass difficulties falling asleep, frequent nocturnal awakenings, reduced sleep continuity, and poor sleep quality, phenomena that overlap with clinical insomnia syndromes and have been associated with worsened gastrointestinal symptoms.

A large meta-analysis showed that the prevalence of sleep disorders among IBS patients was approximately 37.6%, with an odds ratio significantly higher than that of non-IBS populations [2]. This epidemiological association suggests a robust link between sleep dysregulation and IBS beyond chance. Importantly, insomnia and sleep quality indices have been correlated with

IBS symptom severity and frequency in large cohort and online survey studies [4,5]. These findings underscore the need to understand insomnia not only as an associated comorbidity but also as a potential modifier of IBS clinical presentation.

The biological possibility of an IBS–sleep relationship is supported by several interlinked pathways involving the gut–brain axis, autonomic nervous system dysregulation, hypothalamic–pituitary–adrenal (HPA) axis activation, and immune modulation. Sleep disturbances can alter pain perception, amplify visceral hypersensitivity, change inflammatory profiles, and disrupt circadian rhythms that regulate gastrointestinal function. Moreover, subjective sleep disruption — even in the absence of objective sleep architecture abnormalities — has been shown to potentiate next-day abdominal pain and GI symptoms in IBS patients, suggesting that perceptual sleep disruption plays a central role in symptom amplification [5,19].

Despite evidence of association, causality remains an open question. A Mendelian randomization study demonstrated that genetic liability to insomnia significantly increased the risk of developing IBS, and this association persisted after adjusting for chronotype and short sleep duration, suggesting that insomnia may be an independent risk factor in the pathogenesis of IBS [3,20]. Conversely, IBS itself did not predict insomnia risk in reverse analysis in the same study, implying a uni-directional influence of sleep disturbance on IBS development rather than vice versa.

Nevertheless, the interplay between psychological factors such as depression and anxiety — both common in IBS — complicates the causal landscape. A population-based cohort in South Korea reported that the combination of insomnia and depressive symptoms was associated with a higher likelihood of having IBS than either factor alone, indicating interactive effects of mood and sleep disturbances on GI symptom expression [6].

Clinical Importance of Insomnia in IBS

Patient-reported outcomes consistently highlight that poor sleep quality and insomnia are associated with increased symptom severity, reduced quality of life, and greater healthcare use in IBS. In clinical actigraphy studies, a large proportion of IBS subjects screened positive for sleep disturbances — most commonly insomnia — and poor subjective sleep quality predicted next-day abdominal pain and GI symptoms in cross-lagged models, pointing toward directional effects of sleep on clinical manifestations [5,18]. Furthermore, self-reported sleep disturbances correlate with greater disease burden, and higher insomnia complaints are associated with

elevated subjective immune dysfunction and poorer perceived general health in IBS populations [4].

Behavioral intervention studies provide preliminary evidence that improving sleep may ameliorate IBS symptom burden. A randomized pilot study administering brief behavioral therapy for insomnia showed improved sleep outcomes and trends toward reduced IBS severity and abdominal pain compared with controls, laying groundwork for more definitive trials [7]. Similarly, cognitive-behavioral therapy for insomnia (CBT-I) has shown efficacy in reducing insomnia and IBS symptom severity in controlled trials among comorbid IBS populations, with reported improvements in sleep onset latency, sleep efficiency, and quality of life [14].

Moreover, the interplay between sleep dysregulation and central pain processing mechanisms — including heightened sympathetic activity, altered stress response, and cognitive arousal — suggests that insomnia may intensify visceral sensitivity in IBS, aggravating pain perception and symptom frequency. These neurophysiological and psychosocial dimensions highlight insomnia not just as a consequence but as a contributor to symptom maintenance and exacerbation in IBS.

Research Objective

The primary objective of this study is to systematically review and synthesize the existing literature on the role of insomnia in the clinical manifestations of irritable bowel syndrome (IBS). This review aims to assess the prevalence and severity of insomnia among IBS patients across diverse populations and clinical settings, examine the association between insomnia and IBS symptom severity—including abdominal pain, bowel habit alterations, and overall gastrointestinal symptom burden—and explore potential mechanisms linking insomnia to IBS, such as dysregulation of the gut–brain axis, stress response, immune modulation, and psychosocial factors. Additionally, it evaluates evidence on interventions targeting insomnia and their potential impact on IBS symptoms, quality of life, and clinical outcomes. Through this narrative synthesis, the study seeks to identify knowledge gaps, highlight areas of consistent evidence, and inform future research on the interplay between sleep disturbances and IBS.

Research Problems and Gaps

Despite robust evidence of association between insomnia and IBS, several key problems remain unresolved. Most existing studies are cross-sectional, limiting causal inference. Longitudinal

and mechanistic studies are needed to disentangle directionality and underlying pathways. Also objective sleep measures (polysomnography) often fail to align with subjective sleep complaints in IBS, raising questions about the role of perceptual sleep disruption versus physiological sleep pathology [1,8]. The overlap of insomnia with depression, anxiety, and stress complicates the understanding of distinct pathways through which sleep disturbance affects IBS symptoms. While preliminary trials suggest that treating insomnia may improve IBS outcomes, larger, well-powered randomized controlled trials are needed to confirm efficacy and generalizability.[9,10]

Research Hypotheses

Based on current evidence, the following hypotheses will be tested:

1. **Hypothesis 1**: Clinical insomnia is significantly more prevalent in individuals with IBS than in matched controls without IBS.

2. **Hypothesis 2**: Higher insomnia severity is associated with increased IBS symptom severity, including abdominal pain, bowel irregularity, and reduced quality of life.

3. **Hypothesis 3**: Insomnia severity independently predicts future IBS symptom exacerbation when controlling for mood disorders and stress.

4. **Hypothesis 4**: Behavioral or cognitive interventions targeting insomnia will lead to significant improvements in IBS symptom burden compared with standard care.

Materials and Methods

A comprehensive literature review was performed to identify studies investigating the relationship between insomnia and the clinical manifestations of irritable bowel syndrome (IBS). Electronic databases, including PubMed, were systematically searched for articles published up to 2026.

The search strategy employed combinations of the following keywords: Irritable bowel syndrome; IBS; Insomnia; Sleep disturbances; Sleep quality; Gut–brain axis; Visceral hypersensitivity; Gastrointestinal symptoms; Quality of life; Cognitive behavioral therapy for insomnia.

Inclusion criteria were studies that examined the role of sleep disturbances, particularly insomnia, in patients with IBS. Eligible studies included human observational studies, clinical trials, experimental studies, mechanistic investigations, and relevant review articles published in peer-reviewed journals and written in English. Studies were excluded if they did not directly address the association between insomnia or sleep disturbances and IBS, or if only conference abstracts without full text were available.

Titles and abstracts were first screened for relevance, followed by a full-text evaluation of potentially eligible studies. Data extraction focused on study design, sample characteristics, measures of sleep quality and insomnia, IBS diagnostic criteria, and reported associations between sleep disturbances and IBS symptom severity.

Due to the heterogeneity in study designs, populations, and outcome measures, a qualitative narrative synthesis was conducted to summarize current evidence regarding the prevalence, severity, and potential mechanistic links between insomnia and IBS, as well as the impact of sleep interventions on IBS symptomatology.

Results

Prevalence of Insomnia in IBS Populations

Across multiple studies, insomnia was consistently reported at higher rates among individuals with IBS compared with control populations. Tu et al. (2016) [1] conducted a systematic review and reported that sleep disturbances, including insomnia, affect a significant proportion of IBS patients, with prevalence estimates ranging from 40% to 70%, depending on assessment methodology. Similarly, Wang et al. (2018) [2] conducted a meta-analysis including 25 studies and reported a pooled prevalence of approximately 37.6% for sleep disorders in IBS, with a significantly higher odds ratio compared to non-IBS cohorts. These findings suggest that insomnia represents a highly prevalent comorbidity in IBS and may contribute meaningfully to disease burden.

Notably, population-based studies incorporating self-reported sleep complaints and objective sleep measures indicate that women are disproportionately affected by sleep disturbances in IBS, consistent with the female predominance of IBS itself [2]. Actigraphy-based studies confirm that both objective sleep duration and efficiency are often reduced in IBS cohorts, although discrepancies between subjective and objective sleep quality are frequently observed

[8,1]. This misalignment emphasizes the clinical relevance of perceived sleep disruption, which may independently influence symptom perception.

Relationship Between Insomnia and IBS Symptom Severity

Evidence from cross-sectional and longitudinal studies consistently demonstrates that insomnia is associated with increased IBS symptom severity. Topan et al. (2023) [5] reported that poor subjective sleep quality predicted next-day abdominal pain and bowel symptom frequency in women with IBS. Similarly, Lee et al. (2017) [6] found that the combination of insomnia and depressive symptoms significantly increased the likelihood of IBS presence, suggesting additive or interactive effects between sleep and mood disturbances on gastrointestinal symptom expression.

Polysomnography and actigraphy studies highlight that increased sleep onset latency, nighttime awakenings, and non-restorative sleep are associated with higher abdominal pain scores, bloating, and stool frequency irregularities [8,4]. Additionally, Bao et al. (2022) [3] provided Mendelian randomization evidence indicating that genetic predisposition to insomnia is causally linked with increased IBS risk, supporting the hypothesis that insomnia is not merely a comorbid condition but a potential modifier of disease manifestation.

Sleep disruption may amplify visceral hypersensitivity and alter central pain processing, including augmented sympathetic nervous system activity and dysregulated hypothalamic-pituitary-adrenal (HPA) axis responses [1,2,5]. These findings are supported by studies demonstrating that IBS patients with chronic sleep disturbances report greater pain severity and reduced tolerance thresholds in experimental visceral sensitivity paradigms [2,22].

Immune Function, Stress, and Insomnia in IBS

Insomnia in IBS is also associated with altered perceived immune functioning. Balikji et al. (2018) [4] reported that higher insomnia severity correlated with elevated subjective immune complaints and greater gastrointestinal symptom burden. Chronic stress appears to exacerbate these associations, as studies examining stress-related sleep disruption found that poor sleep quality magnifies the effect of stress on abdominal pain and bowel irregularity [12]. These findings indicate that insomnia may contribute to IBS pathophysiology via immunomodulatory and stress-related pathways, potentially influencing systemic inflammation and visceral sensitivity [22].

Moreover, anxiety and depression, frequent comorbidities in IBS, further compound the influence of insomnia. Meta-analytic evidence indicates that IBS patients with coexisting mood disorders and sleep disturbances report higher symptom severity, reduced quality of life, and increased healthcare utilization [11,6]. The bidirectional relationship between psychological factors, sleep disruption, and gastrointestinal symptoms underscores the complexity of IBS symptom modulation by insomnia.[21]

Impact of Insomnia-Targeted Interventions on IBS Outcomes

Intervention studies suggest that improving sleep may ameliorate IBS symptomatology. Ballou et al. (2019) [7] conducted a pilot study of brief behavioral therapy for insomnia in IBS patients, reporting significant improvements in sleep latency, sleep efficiency, and trends toward reduced abdominal pain and stool irregularities. Similarly, CBT-I interventions have demonstrated efficacy in both improving insomnia symptoms and reducing IBS symptom severity in controlled trials among student populations [14].

Mechanistically, these interventions may reduce central arousal, modulate stress responses, and enhance visceral pain thresholds, contributing to improvements in symptom perception and quality of life [13-16]. These findings support the concept that sleep-focused therapies are not only feasible but potentially clinically meaningful in IBS management, though larger trials with long-term follow-up are needed to confirm effectiveness.

Objective vs. Subjective Sleep Findings

Across studies, objective measures such as polysomnography often reveal relatively subtle alterations in sleep architecture among IBS patients compared to subjective reports of severe sleep disruption [8,1]. This discrepancy highlights the clinical importance of **perceived insomnia**, which may independently influence symptom severity and quality of life even in the absence of measurable sleep pathology. Topan et al. (2023) [5] found that perceived poor sleep predicted increased abdominal pain the following day, reinforcing the clinical relevance of self-reported sleep quality in IBS assessment.

Gender Differences and Epidemiological Patterns

Women with IBS consistently demonstrate higher rates of insomnia than men, aligning with the overall female predominance of IBS [2,22,23,24]. These gender differences extend to

symptom reporting, sleep disruption perception, and response to sleep interventions, suggesting potential sex-specific mechanisms in the insomnia–IBS relationship.

Table 1. Summary of Key Findings

Key Finding	Details / Evidence
High prevalence of insomnia in IBS	Pooled estimates show around 37–40% prevalence [1,2].
Association with symptom severity	Insomnia linked to worse IBS symptoms: abdominal pain, bloating, stool irregularities [5,4,6].
Potential causal role	Mendelian randomization studies suggest insomnia may contribute to IBS development [3,15].
Perceived sleep quality matters	Subjective sleep assessments predict symptom exacerbation as much as objective measures [1,5,8].
Effectiveness of sleep-focused interventions	Behavioral interventions, including CBT-I, can reduce insomnia and IBS symptom severity [7,14,16].
Interaction with psychological factors	Comorbidities and chronic stress amplify IBS symptoms when insomnia is present [6,11,12].
Sex differences	Female IBS patients show higher susceptibility to insomnia and symptom severity [2,22,23,24].

Collectively, these findings provide a coherent narrative that insomnia is not merely a comorbidity but a potential modifier and amplifier of IBS symptom expression, influencing pain perception, gastrointestinal function, and overall quality of life.

Discussion

This narrative synthesis highlights the significant role of insomnia in the clinical manifestation of irritable bowel syndrome (IBS), providing consistent evidence across epidemiological, mechanistic, and interventional studies. Across multiple investigations, insomnia was

consistently shown to be highly prevalent among IBS patients, with prevalence estimates ranging from 37% to over 60% in some cohorts [1,2,5]. This prevalence is markedly higher than in non-IBS populations, suggesting that sleep disturbances are not incidental but likely integral to IBS pathophysiology [2,1].

Mechanistic Insights: Gut–Brain Axis, Visceral Sensitivity, and Stress

Several studies provide mechanistic insights linking insomnia to exacerbated IBS symptoms. Disruption of the gut–brain axis appears central, with insomnia contributing to altered autonomic regulation, increased visceral sensitivity, and dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis [1,5]. Polysomnography and actigraphy studies demonstrate that fragmented sleep, prolonged sleep onset latency, and reduced sleep efficiency are associated with increased abdominal pain and stool irregularities, suggesting that objective sleep disruption interacts with perceptual symptom amplification [8,4,5].

Sleep disturbances are also closely intertwined with chronic stress and immune modulation. Higher insomnia severity correlates with subjective reports of impaired immune functioning and greater gastrointestinal symptom burden [4,12]. Stress and mood disorders, including depression and anxiety, further exacerbate IBS symptoms and frequently coexist with insomnia, creating a multifactorial interplay that amplifies disease burden [6,11]. Evidence from cohort studies demonstrates that insomnia combined with depression significantly increases the likelihood of IBS diagnosis compared to either factor alone [6].

Genetic studies provide further support for a potential causal role of insomnia in IBS. Mendelian randomization analyses indicate that genetic liability to insomnia increases IBS risk independently of chronotype and sleep duration, suggesting that insomnia itself may be a risk factor rather than merely a consequence of gastrointestinal dysfunction [3,15]. Conversely, IBS does not appear to predict insomnia in these analyses, reinforcing a uni-directional influence from sleep disruption to gastrointestinal symptoms.

Clinical Relevance of Subjective Sleep Disturbance

Subjective sleep quality, often assessed via patient self-report or validated instruments, appears to have equal or greater clinical relevance than objective measures such as polysomnography [1,5,8]. Studies consistently report that perceived poor sleep predicts next-day abdominal pain, bloating, and bowel habit irregularities [5]. This emphasizes the importance of assessing

patient-reported sleep complaints in routine IBS management, as objective sleep measures alone may underestimate the clinical impact of insomnia.

Epidemiological studies also indicate sex-specific differences, with women disproportionately affected by sleep disturbances in IBS [2,22,23,24]. Female patients report higher rates of insomnia, poorer sleep quality, and greater symptom severity, highlighting the need for gender-sensitive assessment and management strategies.

Interventional Evidence

Behavioral interventions targeting insomnia show promising effects on IBS symptomatology. Brief behavioral therapy for insomnia (BBTI) in IBS patients improved sleep efficiency and reduced abdominal pain and stool irregularities in pilot trials [7]. Cognitive-behavioral therapy for insomnia (CBT-I) has demonstrated robust improvements in both sleep outcomes and IBS symptom severity, suggesting that modulating sleep can positively influence gastrointestinal symptom expression [14,16]. Randomized controlled trials among college populations further support these findings, highlighting improvements in sleep latency, sleep efficiency, and quality of life following CBT-I [14].

The success of these interventions suggests that insomnia is not merely an epiphenomenon but a potential modifiable factor influencing IBS outcomes. Effective management of insomnia may therefore reduce symptom burden, improve quality of life, and potentially mitigate downstream complications associated with chronic stress and immune dysregulation [7,14,16].

Integrated Findings and Implications

This synthesis demonstrates a consistent pattern: insomnia in IBS is highly prevalent, associated with increased symptom severity, amplified by psychological comorbidities, and potentially modifiable through behavioral interventions. Sleep disruption impacts visceral sensitivity, gut–brain communication, stress responses, and immune perception, all of which contribute to exacerbated abdominal pain, bloating, and bowel irregularities [1-5,12].

Moreover, the alignment of evidence across epidemiological, genetic, and interventional studies reinforces a model in which insomnia acts both as a risk factor and a symptom amplifier in IBS. This has direct clinical implications: assessing and treating insomnia in IBS patients may not

only improve sleep but also reduce gastrointestinal symptom burden, enhance quality of life, and decrease healthcare utilization [1,2,5,7,14,16].

Limitations in the Current Literature

Despite consistent associations, several limitations exist. Many studies are cross-sectional, limiting causal inference and temporality assessment [1,2,4]. Discrepancies between objective and subjective sleep measurements underscore the need for comprehensive assessment approaches incorporating both modalities [8,1]. Small sample sizes in interventional trials restrict generalizability, and heterogeneity in outcome measures complicates direct comparisons across studies [7,14]. Additionally, comorbid depression, anxiety, and chronic stress frequently confound associations, making it challenging to disentangle the independent effect of insomnia [6,11,12,17].

Future Research Directions

Future research should prioritize longitudinal designs to clarify temporal relationships between insomnia and IBS symptom exacerbation. Large-scale, multicenter randomized trials examining the efficacy of sleep-focused interventions are needed to determine whether improvements in insomnia can consistently ameliorate IBS symptoms across diverse populations [14,7,16]. Objective and subjective sleep assessment should be integrated, with attention to sex differences and psychosocial moderators. Finally, mechanistic studies exploring gut–brain axis modulation, immune function, and stress physiology may elucidate pathways linking insomnia to gastrointestinal symptom exacerbation [1,3,5,12].

Conclusion

The synthesis of the current literature demonstrates that insomnia is a highly prevalent and clinically meaningful factor in patients with irritable bowel syndrome (IBS). Beyond being a common comorbidity, insomnia appears to actively influence the clinical manifestation of IBS, intensifying abdominal pain, bloating, and bowel habit irregularities, and contributing to a reduced quality of life. The interplay between disrupted sleep and the central and peripheral mechanisms of gastrointestinal function suggests that insomnia may serve both as a modifier of symptom severity and a potential trigger for symptom exacerbation.

Behavioral and cognitive interventions targeting sleep disturbances show promising results in improving both sleep quality and gastrointestinal symptoms, indicating that addressing insomnia could be an effective strategy to alleviate overall disease burden. Moreover, the relationship between sleep disruption and psychological factors such as stress, anxiety, and depression highlights the importance of considering a holistic, biopsychosocial approach to IBS management. Interventions that simultaneously address sleep, mental health, and gastrointestinal function may be particularly effective in mitigating the cyclical reinforcement of symptoms.

From a clinical perspective, the recognition of insomnia as an integral component of IBS underscores the need for routine screening of sleep disturbances in this patient population. Tailored sleep management strategies, alongside standard IBS therapies, could not only improve patient-reported outcomes but also reduce long-term complications associated with chronic stress, immune dysregulation, and impaired daily functioning.

Finally, this review highlights critical gaps in current research, including the need for large-scale, longitudinal studies to clarify causal relationships, and for integrated mechanistic studies to better understand how insomnia interacts with the gut–brain axis and visceral sensitivity. By bridging these gaps, future research can inform the development of precision-based interventions, enabling clinicians to offer more effective, individualized care. Overall, the evidence supports the conclusion that insomnia is not merely a secondary symptom in IBS but a significant contributor to disease pathophysiology, representing both a challenge and an opportunity for improving patient outcomes.

Disclosure

Supplementary Materials

There are no supplementary data connected with this article.

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All authors have read and agreed to the published version of the manuscript.

Funding

W. Oczkowska

Institutional Rview Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Data sharing is not applicable to this article.

Acknowledgements

Not applicable.

Conflicts of Interest

The authors declare no conflicts of interest.

Deceleration of the Use of Generative AI and AI-Assisted Technologies in the Writing.

Process

During the preparation of this work, the authors used Grammarly for the purpose of improving language and readability. After using this tool, the authors reviewed And edited the content as needed and took full responsibility for the substantive content of the publication.

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