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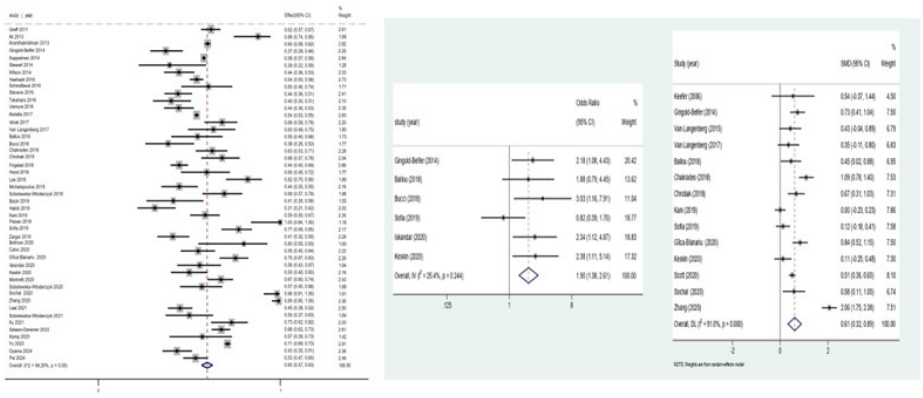

Poor sleep prevalence was 60% in IBD, 52% in UC, 56% in CD, and 68% in active IBD.

IBD patients had higher odds of poor sleep than controls (OR: 1.90, 95% CI: 1.38–2.61; SMD: 0.61, 95% CI: 0.32–0.89).

Active IBD patients showed elevated poor sleep risk versus those in remission (OR: 2.09, 95% CI: 1.50–2.90), driven by CD (OR: 2.40, 95% CI: 1.42–4.05)

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The sleep quality and its association with disease activity in patients with inflammatory bowel disease: a meta-analysis

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ABSTRACT

Background: Inflammatory bowel disease (IBD), including ulcerative colitis (UC) and Crohn's disease (CD), is linked to poor sleep quality, though evidence remains inconsistent. This meta-analysis aimed to evaluate sleep quality in IBD patients and its association with disease activity.

Methods: We systematically searched databases up to February 2025 for studies reporting sleep quality in IBD. Outcomes included pooled prevalence of poor sleep, odds ratios (ORs), and standardized mean differences (SMDs) comparing IBD patients to controls, UC vs CD, and active vs inactive disease. Heterogeneity was assessed using Cochran's Q and I^2 statistics.

Results: Fifty-five studies were included. Poor sleep prevalence was 60% in IBD, 52% in UC, 56% in CD, and 68% in active IBD. IBD patients had higher odds of poor sleep than controls (OR: 1.90, 95% CI: 1.38–2.61; SMD: 0.61, 95% CI: 0.32–0.89). No differences emerged between UC and CD (OR: 0.93; SMD: -0.04). Active IBD patients showed elevated poor sleep risk versus those in remission (OR: 2.09, 95% CI: 1.50–2.90), driven by CD (OR: 2.40, 95% CI: 1.42–4.05). SMDs for active vs inactive disease were 0.49 (IBD overall), 0.40 (UC), and 0.73 (CD).

Conclusion: Poor sleep is highly prevalent in IBD, particularly during active disease, with significantly higher rates than in healthy controls. Sleep quality did not differ between UC and CD. Addressing poor sleep in IBD management and exploring underlying mechanisms are crucial for improving patient outcomes.

Keywords: Sleep quality. Inflammatory bowel disease. Crohn's disease. Ulcerative colitis.

1. Introduction

Inflammatory bowel disease (IBD), which includes Crohn's disease (CD) and

ulcerative colitis (UC), is a chronic, progressive condition. It involves inflammation of the gastrointestinal tract and is linked to a rising number of cases and various complications. Recent studies have shown that the incidence and prevalence of IBD are increasing globally according to the Global Burden of Disease Study 2021[1]. Intestinal inflammation is a key feature of IBD. However, the condition also causes various symptoms outside the intestines, indicating that immune dysregulation in IBD are systemic[2]. The symptoms of IBD can range from mild, asymptomatic inflammation to widespread gastrointestinal issues. IBD often causes extraintestinal symptoms, such as erythema nodosum and peripheral arthritis, especially during active disease [3]. In addition to poor physical health, people with IBD often experience psychiatric and psychological issues such as depression, anxiety, pain, and poor sleep. Previous studies have shown that IBD patients are more likely to have sleep disturbances[4]. Factors affecting sleep quality in IBD patients include side effects of medication, nocturnal gastrointestinal symptoms, joint pain, depressed mood and disease activity[5-8]. Therefore, sleep management should be should be important part of IBD treatment.

Sleep is the foundation of good health, and a lack of sleep can bring negative health consequences. Not only are sleep disorders associated with hypertension, diabetes, and obesity, but depressive symptoms, physical illness, and fatigue are also reported to be linked to poor sleep quality and short sleep duration[9]. Clinical evidence shows that poor sleep is linked to increased histological activity and clinical relapse in IBD patients[10]. Poor sleep not only worsens symptoms but may also contribute to increased intestinal inflammation[11]. The mechanisms behind this connection involve inflammatory regulation. Sleep disorders trigger inflammatory responses and increase pro-inflammatory factors in immune cells[12]. Furthermore, sleep disorders may exacerbate the course of IBD through inflammatory regulation and gut-brain axis mechanisms[13].

In recent years, sleep disorders have gained increasing attention as a common comorbidity in patients with IBD. These disorders significantly impact patients' quality of life. However, there are important controversies in existing studies regarding the prevalence of sleep disorders in IBD patients and some related factors.

Some studies reported that IBD patients have a much higher rate of poor sleep compared to healthy people, but the exact difference has not been fully measured. Others have suggested that disease activity and disease type (CD or UC) may significantly affect patients' sleep quality, but the results are inconsistent. Four meta-analyses on sleep quality in IBD patients have been published. However, these studies have several key limitations. For example, some new relevant studies are not included. These meta-analyses also lack comparisons between UC and CD in both active and remission phases. In addition, these meta-analyses do not report the prevalence of poor sleep separately for UC and CD patients. This makes it difficult to assess how each disease type affects sleep quality. These gaps highlight the need for more up-to-date and thorough analyses to better understand poor sleep in IBD patients.

To address these gaps in the research, this meta-analysis reviews existing studies on poor sleep in IBD patients. The goals of this study are: (i) to summarize the prevalence of poor sleep in IBD patients, (ii) to compare the risk of poor sleep between IBD patients and controls, (iii) to explore sleep differences between patients with CD and UC, and (iv) to assess sleep quality differences between patients in the active and inactive stages of IBD. This study aims to provide a scientific basis for better management of poor sleep in IBD patients and encourage future research on the complex relationship between IBD and sleep disorders.

2. Methods

2. 1. Search strategy

This systematic review and meta-analysis followed the PRISMA guidelines for preferred reporting items. We used a predetermined protocol and registered this meta-analysis (PROSPERO number: CRD42024624218). The search was conducted using PubMed, Ovid Embase, Medline, Cochrane CENTRAL, and conference abstracts such as European Crohn’s and Colitis Organization (ECCO), Digestive Disease Week (DDW), and United European Gastroenterology (UEG) week. The search covered studies from inception to February 2025. The search terms included: “sleep” OR “insomnia” OR “sleep quality” OR “sleep disorder” AND (“IBD” OR “inflammatory

bowel disease" OR "ulcerative colitis" OR "UC" OR "Crohn's disease" OR "CD" OR "colitis"). The results of the search were carefully reviewed. Any disagreements were resolved through discussion or by the whole team.

2. 2. Inclusion criteria and exclusion criteria

Each study that might meet the inclusion criteria was reviewed in full. Studies that clearly did not qualify were excluded. To be included, studies had to meet the following criteria: (1) be comparative in design (e.g., cross-sectional, case-control, cohort, or randomized controlled studies) that included at least two distinct groups; (2) assess sleep quality using a validated, patient-reported subjective sleep assessment questionnaire; (3) included patients with IBD using subjective measures such as the Crohn's Disease Activity Index (CDAI), Harvey-Blood-Shawin Index (HBI) or Mayo Partial Score (PMS), or objective measurements (such as endoscopy) to define inactive disease. The exclusion criteria were: (1) populations inappropriate for the study, such as children or adolescents; and (2) other types of papers, such as reviews, case reports, letters, editorials and non-English articles.

2. 3. Data extraction

Information about included studies was recorded in a standardized extraction form by two reviewers. This table was created to extract the following characteristics of the study: first author, country, study year, mean age, gender, sample size, sleep measures and IBD subtype. Additionally, measurement methods and main results were collected. Odds ratios (OR) and standardized mean differences (SMD) were also extracted.

2. 4. Quality appraisal

The Newcastle-Ottawa Scale (NOS) was used to assess the quality of the included studies. The selected studies were evaluated using the NOS based on three criteria: Selection, Comparability, and Exposure. The NOS awarded up to 2 stars in the Comparability domain and 1 star for each item in the Selection and Exposure domains. For this study, we used a shortened version of the scale. One item was

excluded because it measures the absence of disease in the control group, which could lead to misjudging results in studies comparing CD and UC. This change resulted in a maximum of eight stars for each study. Scores ranged from 0 to 8, with higher scores indicating better study quality. Any disagreements were resolved through consensus.

2. 5. Statistical analysis

In this study, data were analyzed using Stata SE 15.1 and Stata/MP 18.0. The primary outcomes were the overall incidence of poor sleep in IBD, UC, and CD patients. The secondary outcomes were the combined OR and SMD for poor sleep between IBD patients and controls, between UC and CD, and between active IBD and inactive IBD. Subgroup analyses were conducted based on disease duration and the type of sleep questionnaires used.

To assess heterogeneity, we used Cochran's Q test and the I^2 statistic. If there was significant heterogeneity ($I^2 > 50\%$), we applied a random-effects model. If heterogeneity was not significant ($I^2 \leq 50\%$), we used a fixed-effects model. Finally, publication bias was assessed using funnel plots and Egger-weighted regression tests.

3. Results

3.1 Study characteristics

The final number of included studies and the screening process were shown in the PRISMA flowchart (Figure 1). Table 1 showed the characteristics of the 55 studies included in this review[5, 6, 12, 14-65]. These studies were published between 2006 and 2024, and the studies were conducted in various countries and regions, including the United States, Japan, and Poland. The age of the study participants ranged from 25 to 52 years, and the average disease duration varied from 1.7 to 16.3 years. Although the studies used different indicators to measure disease activity, all of them employed standardized scores. Notably, all the included trials used standardized methods to assess sleep quality. The majority of studies used the Pittsburgh Sleep Quality Index (PSQI), another 8 studies used the PROMIS Sleep

Questionnaire, 2 studies used the Berlin Sleep Questionnaire (BNSQ), 1 study used the SLEEP-50 Sleep Disorders Assessment Scale, and 1 study used the Alpine Sleep Scale (AIS) (Table 1).

3.2 Prevalence of poor sleep in IBD and subgroup analyses

Forty-seven studies examined the incidence of poor sleep in patients with IBD. The overall combined rate of poor sleep in patients with IBD was 0.60 (95% CI 0.57-0.63; I^2= 94.20%) (Figure 2). Further analysis showed that the incidence of poor sleep was 0.52 (95% CI 0.45-0.59; I^2= 95.01%) in patients with UC and 0.56 (95% CI 0.50-0.62; I^2= 95.52%) in patients with CD. In patients with active IBD, the combined results from 6 studies showed an incidence of poor sleep of 0.69 (95% CI 0.60-0.77; I^2= 80.34%).

Subgroup analyses were conducted to explore potential sources of heterogeneity. When stratified by geographic region, the pooled prevalence of poor sleep in patients with IBD was 0.61 (95% CI: 0.56–0.67; I^2 = 0.9%) in North America, 0.57 (95% CI: 0.38–0.75; I^2 = 1.0%) in Asia, 0.63 (95% CI: 0.51–0.73; I^2 = 0.8%) in Europe, 0.63 (95% CI: 0.49–0.75; I^2 = 0.0%) in Oceania, and 1.00 (95% CI: 0.00–1.00; I^2 = 0.0%) in South America. By age group, the pooled prevalence was 0.68 (95% CI: 0.51–0.81; I^2 = 93.1%) among participants under 40 years and 0.59 (95% CI: 0.54–0.64; I^2 = 85.8%) among those aged 40 years or older. In terms of sleep assessment tools, 39 studies using the PSQI reported a pooled prevalence of 0.61 (95% CI: 0.56–0.66; I^2 = 92.8%), while six studies using the PROMIS questionnaire reported 0.56 (95% CI: 0.52–0.59; I^2 = 92.2%). One study using the BNSQ reported a prevalence of 0.44 (95% CI: 0.40–0.49; I^2 = 0.0%), and one using the AIS reported 0.98 (95% CI: 0.91–1.00; I^2 = 0.0%).

3.3 Comparison of sleep quality between IBD patients and controls

Fourteen studies compared subjective sleep quality between IBD patients and controls. The results showed that IBD patients had significantly poorer sleep than controls. The combined OR was 1.90 (95% CI 1.38-2.61; I^2= 25.40%) and the SMD was 0.61 (95% CI 0.32-0. 89; I^2= 91.0%) (Figure 3). After removing one outlier study, the

SMD decreased to 0.48 (95% CI: 0.30–0.67) with I^2 reduced to 76.2%.

3.4 Comparison of sleep quality between UC and CD and subgroup analyses

Nineteen studies compared sleep quality between patients with UC and CD. The results showed that CD patients had poorer sleep than UC patients. However, the difference was small (OR = 0.93, 95% CI 0.76-1.12; I^2= 66.6%) (Figure 4) and not statistically significant.

Subgroup analyses showed mixed results using different sleep assessment tools. For the PSQI, the combined OR from 15 studies was 0.88 (95% CI 0.65-1.19; I^2= 65.8%). For the PROMIS, the combined OR from 3 studies was 0.87 (95% CI 0.70-1.08; I^2= 63.8%). For the BNSQ, the OR from 1 study was 0.93 (95% CI 0.76-1.12; I^2= 66.6%). We also performed a subgroup analysis on disease duration. The difference in sleep quality between patients with a disease duration of less than 8 years and those with more than 8 years was small. The OR for patients with less than 8 years of disease duration was 1.14 (95% CI 0.64-2.01; I^2= 64.4%). The OR for patients with more than 8 years of disease duration was 1.16 (95% CI 0.75-1.81; I^2= 62.4%).

3.5 Comparison of sleep quality between active IBD and inactive IBD and subgroup analyses

Twenty-one studies showed that patients with active IBD had significantly poorer sleep than those with inactive IBD. The combined OR was 2.09 (95% CI 1.50-2.90; I^2= 83.6%) and the SMD was 0.49 (95% CI 0.25-0.74; I^2= 84.9%) (Figure 5). In subgroup analyses, 18 studies using the PSQI showed an OR of 1.92 (95% CI 1.28-2.87; I^2= 81.8%) for patients with active IBD. Three studies using the PROMIS Sleep Questionnaire showed a combined OR of 2.09 (95% CI 1.50-2.90; I^2= 83.6%).

Subgroup analyses were conducted based on disease type. Two studies found no significant difference in sleep quality between patients with active and inactive UC. The combined OR was 1.78 (95% CI 0.49-6.46; I^2= 89.4%) and the SMD from 4 studies was 0.40 (95% CI -0.02 to 0.82; I^2= 85.9%). Eight studies found that patients with active CD had worse sleep quality than those with inactive CD. The combined

OR was 2.40 (95% CI 1.42-4.05; I^2= 70.3%) and the SMD for 4 studies was 0.73 (95% CI 0.31-1.16; I^2= 91.0%).

3.6 Quality assessment and publication bias

To assess the quality of the included studies, we used the revised NOS. This scale was widely used to evaluate the quality of observational studies. The total score ranged from 0 to 8, with higher scores indicating higher quality studies. In our analysis, most studies were of high quality. Sixteen studies scored 8, fourteen scored 7, nine scored 5, and one scored 3. The average score was 6.33, with most studies scoring between 6 and 8. Most of the included studies provided valid case definitions. This helped ensure that exposure variables, such as sleep quality, were assessed consistently. Studies also used clear inclusion criteria to distinguish between cases and controls. Additionally, the analyses were generally adjusted for known confounders, such as age, sex, disease activity, and treatment regimen. Another strength was the use of standardized tools to assess sleep quality, which improved the consistency and reliability of the results across studies. However, some limitations were present. Many studies used hospital-based case samples. This could limit the generalizability of the findings to all IBD patients. Some studies also did not report non-response rates in detail, which could introduce bias.

We also assessed potential publication bias. The funnel plots showed approximate symmetry, except for the six studies comparing IBD patients with controls. In this comparison, the funnel plot showed slight asymmetry, suggesting possible publication bias. However, Egger's test revealed that all studies had p-values greater than 0.05, meaning no significant bias was detected. Despite the observed asymmetry, the overall results were not affected by substantial publication bias. Thus, we could conclude that the studies included in this analysis were balanced in terms of publication and reporting, and the findings were not significantly influenced by systematic omissions.

4. Discussion

Our analyses suggested that patients with IBD have a higher prevalence of

significantly poor sleep quality compared to controls. This finding aligned with the results of a cross-sectional study by Caloz et al. [66], which showed that sleep quality was significantly impaired in IBD patients. The PSQI revealed that 61. 3% of patients with IBD experienced sleep problems. In our meta-analysis, 39 studies using the PSQI showed a comorbid prevalence of poor sleep in IBD patients of 0.61. These results were nearly identical to those reported by Caloz et al. Thus, our study confirmed the high prevalence of poor sleep in IBD patients and underscores the importance of sleep quality in managing IBD. Subgroup analyses further suggested that geographic region may be a more important source of heterogeneity than age or sleep assessment tool. Recent studies have highlighted a strong link between poor sleep in IBD patients and inflammatory markers, particularly C-reactive protein (CRP). Elevated CRP was not only associated with the development of IBD but may also predicted the risk of sleep disorders like obstructive sleep apnea (OSA) and restless legs syndrome (RLS)[67]. This correlation was evident whether IBD was in an active stage or in remission. These findings suggested that inflammatory markers, such as CRP, could be useful in screening for sleep disorders in IBD patients. Future research should explore how elevated CRP may contribute to IBD-related poor sleep. In clinical practice, IBD patients with high CRP levels or extensive lesions should be regularly screened for OSA and RLS. Early detection and intervention could improve their overall quality of life. Additionally, molecular biology studies suggested that dysregulated circadian rhythm genes (e. g., BMAL1, CLOCK, NPAS2, and NR1D1) may contribute to sleep disorders in IBD patients[68]. This dysregulation affected the biological clock and may also worsen IBD by interfering with immune response and inflammation control.

Our study offered a more accurate assessment of sleep quality in patients with UC and CD, and provided a deeper comparative analysis between the two groups. We found no significant differences in sleep quality between UC and CD patients. The prevalence of poor sleep was similar in both groups. This finding aligned with a study by Calvo et al. [47], which reported no significant difference in sleep quality between UC and CD patients (UC: 22 cases, CD: 24 cases, p=0.842). In our study, 51.8% of UC patients and 48.2% of CD patients had poor sleep quality, showing only

a negligible difference. Our analysis further supported this conclusion, based on a larger study population, suggesting that disease type has little effect on sleep quality. Additionally, we performed subgroup analyses based on disease stage and sleep assessment tools. This approach helped us understand the potential impact of disease stage on sleep quality and also highlighted differences in results based on the assessment tool used. These findings suggested that sleep problems are common in IBD patients. Therefore, managing sleep quality in both UC and CD should focus on the individual patient's condition, rather than solely considering disease type. This insight was crucial for clinical management and emphasizes the importance of addressing sleep problems in IBD treatment plans.

The analysis showed that patients with active IBD had significantly poorer sleep quality compared to those with inactive IBD. This finding aligned with a recent meta-analysis of five studies, which also demonstrated a strong link between disease activity and sleep quality in IBD patients[69]. Subgroup analyses further confirmed this result, particularly when using tools like the PSQI and the PROMIS. For both instruments, patients with active IBD had worse sleep quality (PSQI OR = 1.92, 95% CI 1.28-2.87; PROMIS OR = 2.09, 95% CI 1.50-2.90). This relationship may be linked to increased levels of pro-inflammatory cytokines (e. g., TNF-α, IL-6), greater nighttime pain, and heightened psychological distress during active disease phases. Notably, gene expression of CLOCK and NR1D1 was significantly reduced in patients with active UC[68], suggesting that circadian rhythm disruption could play a crucial role in linking disease activity to sleep disturbances. Circadian disruption not only interfered with the normal sleep-wake cycle but may also exacerbated immune dysregulation, creating a vicious cycle. Pro-inflammatory cytokines, like TNF-α and IL-6, were significantly elevated during active disease. These cytokines could alter neuronal and glial cell function, affecting the central nervous system. They also negatively impacted the brain’s sleep regulatory mechanisms by activating neuroinflammatory pathways, leading to poorer sleep quality[70]. Although differences in sleep quality were observed between patients with UC and CD during the active phase, statistical analysis showed these differences were small and not statistically significant. This suggested that while active disease state affected sleep quality, the underlying

mechanisms in UC and CD during active phases may be similar. Larger sample sizes and further research are needed to explore the potential differences in sleep quality and their biological foundations in different types of IBD.

The main strength of this study lay in its use of a variety of high-quality statistical methods, such as ORs, SMDs, and subgroup analyses based on different sleep assessment tools and disease duration. These methods enhanced the reliability of the results and offered a more comprehensive analytical approach. Additionally, the review included 55 studies that accurately assessed the prevalence of sleep disorders in IBD patients worldwide, which improved both the comprehensiveness and accuracy of the data. This approach not only provided quantitative data on poor sleep in IBD patients but also highlighted potential differences in sleep quality between patients with UC and CD. This allowed for a clearer understanding of poor sleep across different IBD subtypes.

Subgroup analyses based on sleep assessment tools revealed consistent associations across the two primary instruments used-PSQI and PROMIS. Although only a few studies utilized alternative tools such as BNSQ, AIS, or SLEEP-50, their results were directionally aligned with the main findings. Due to the limited number of these studies, further pooled analyses were not feasible. Collectively, these observations suggest that variability in sleep assessment instruments had a minimal impact on the overall conclusions of the meta-analysis.

While this meta-analysis provided valuable insights into the epidemiology of poor sleep in patients with IBD, several limitations suggested areas for improvement in future research. First, the observed heterogeneity in the studies was noteworthy. Sources of heterogeneity included differences in study design, sample characteristics, disease type and activity, sleep assessment tools, statistical methods, and selection bias. To address these factors, we performed subgroup analyses based on sleep assessment tools and disease duration, which helped to identify and partially explain the heterogeneity. Second, the analysis did not account for interactions with comorbidities, such as obesity, metabolic syndrome, and cardiovascular disease. These comorbidities likely affected sleep quality and disease progression, and their impact may have been underestimated. Future studies should

explore the synergistic effects of these factors and develop integrated treatment strategies to improve long-term outcomes and quality of life for IBD patients. The third important limitation was the reliance on patient self-reports of sleep quality. While this method was convenient, it lacked validation against objective measures, such as polysomnography or activity trackers. This reliance on subjective data could introduce bias and reduce the reliability of findings. To enhance the credibility of future studies, both subjective and objective measures of sleep quality should be used to provide a more comprehensive and accurate assessment.

Cognitive Behavioral Therapy for Insomnia (CBT-I) is a well-established, non-pharmacological intervention proven to improve sleep quality. Emerging evidence suggests that CBT-I may also mitigate common comorbidities in inflammatory bowel disease, including chronic pain and depression, while potentially reducing systemic inflammatory biomarkers[71]. Despite its demonstrated efficacy, CBT-I remains underutilized in IBD treatment protocols, highlighting a critical gap in clinical practice that warrants greater attention. Our findings indicate that sleep disturbances are highly prevalent among patients with IBD and are closely associated with disease activity. These results highlight the need to treat sleep problems as an integral component of IBD management. Given the bidirectional relationship between poor sleep and IBD disease activity, the incorporation of standardized sleep assessments-such as the PSQI-into routine clinical evaluations could facilitate earlier identification of high-risk patients. Such an approach may enable timely, targeted interventions to optimize sleep quality, thereby improving patient outcomes and enhancing overall disease management.

5. Conclusion

In this meta-analysis, we found that patients with IBD have a higher prevalence of poor sleep quality. Sleep quality was significantly worse in IBD patients compared to controls. It was also worse in patients with active IBD compared to those with inactive IBD. However, we did not observe significant differences in sleep quality between patients with UC and CD. Further subgroup analyses, especially regarding disease duration, also showed no significant differences. Future research should

focus on the mechanisms underlying poor sleep quality in IBD. Studies should also explore effective therapeutic strategies and medications to improve sleep quality in these patients. Additionally, the relationship between sleep quality and disease activity warrants further investigation.

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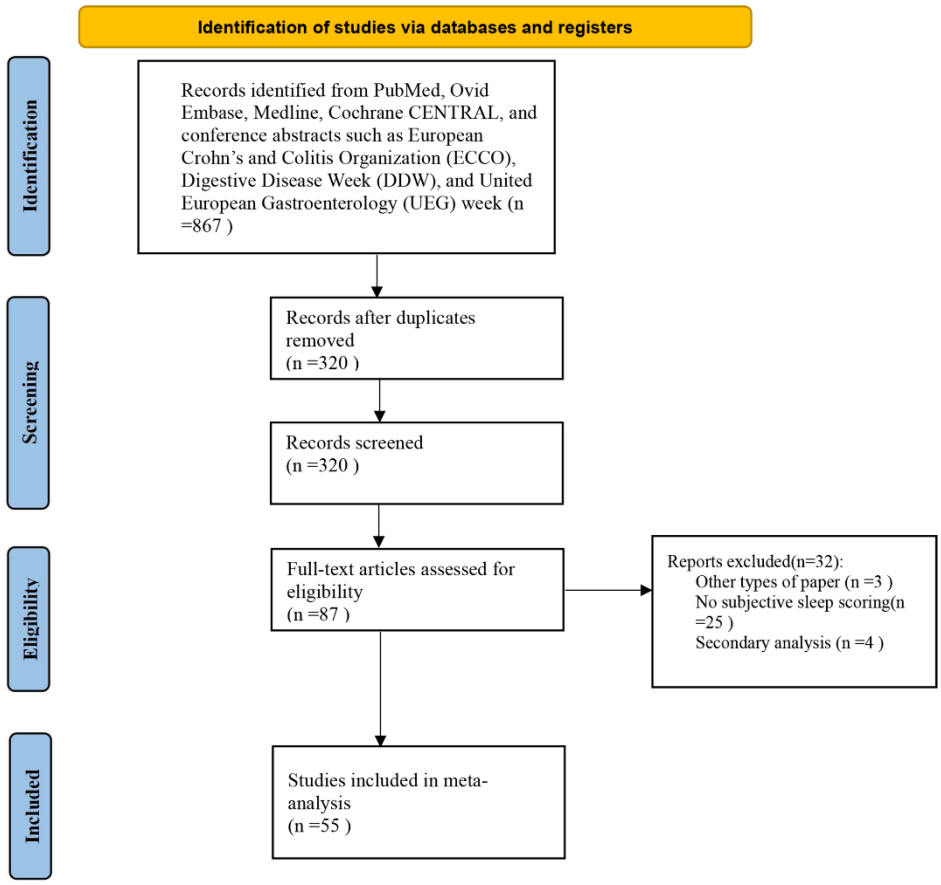

Figure 1: PRISMA flow diagram.

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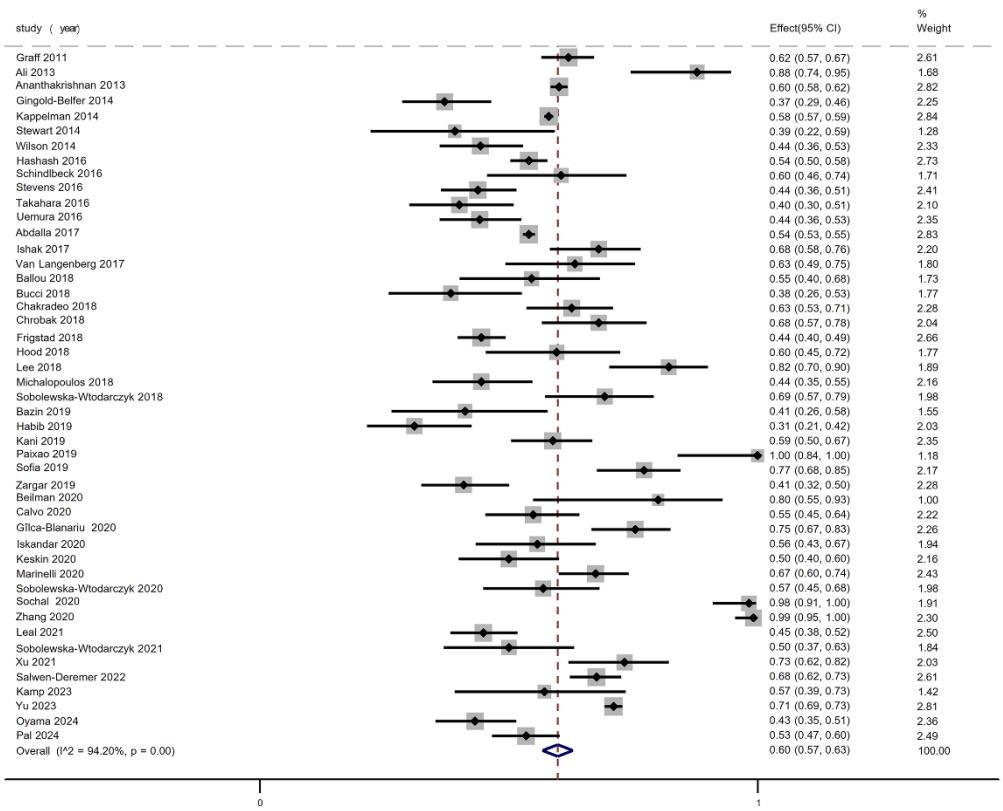

Figure 2: Forest plot depicting the pooled rates of poor sleep in IBD patients.

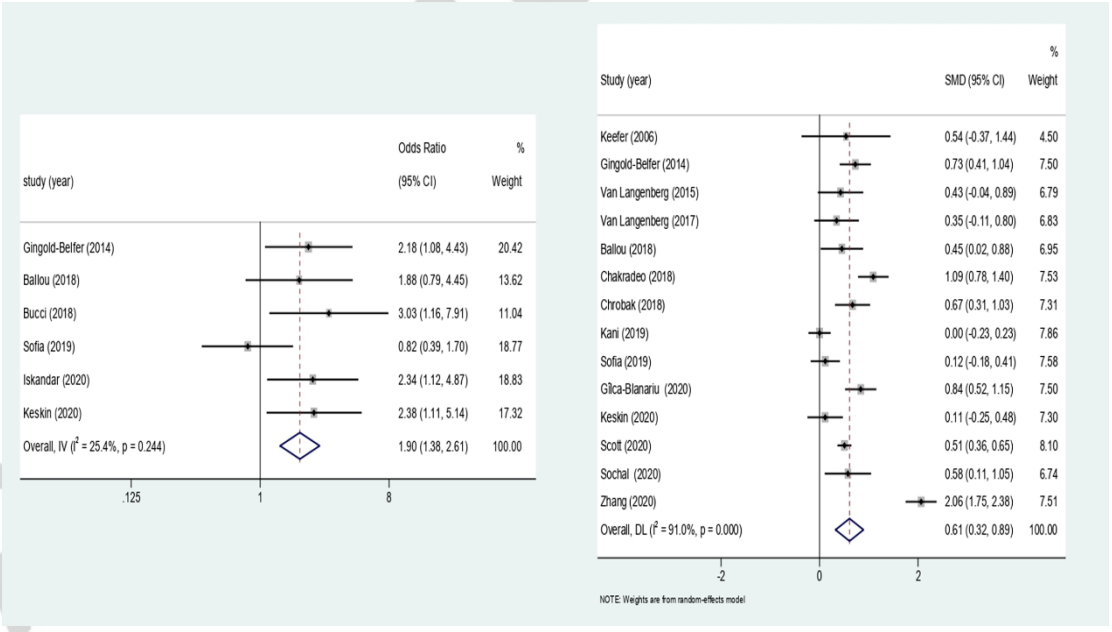

Figure 3: Forest plot for the sleep quality between IBD patients and controls.

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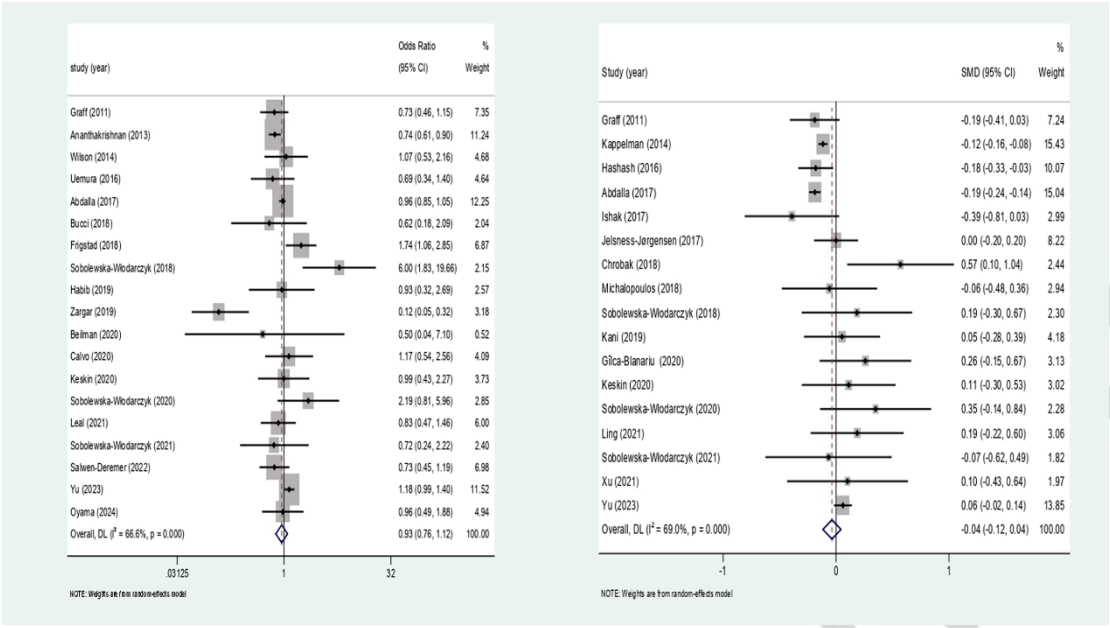

Figure 4: Forest plot for the sleep quality between UC and CD.

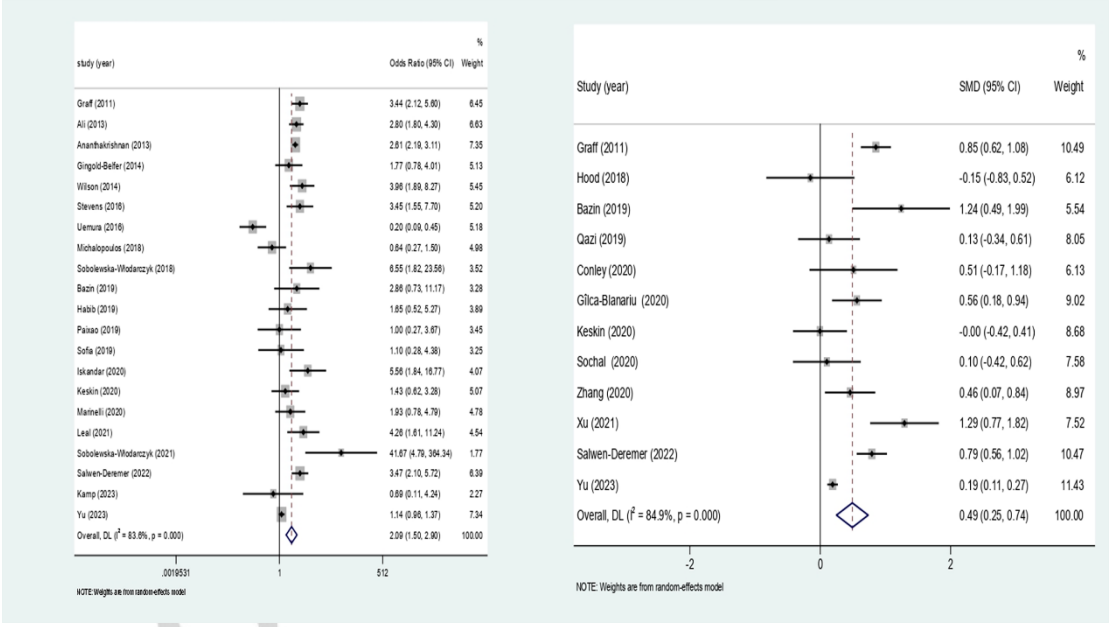

Figure 5: Forest plot for the sleep quality between active IBD and inactive IBD.

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Table 1: Characteristics of the included studies.

Author	Year	Country	Mean age	Female（%）	IBD sample size	UC	CD	Definition of poor sleep	Assessment of disease activity	Duration of IBD	Control sample size
Keefer	2006	USA	41.4	9（56）	16	8	8	PSQI-PSG	UCAI(UC);CDAI(CD)	9.86±10.17	7
Graff	2011	USA	43	103（32）	318	158	160	PSQI	Powell-Tuck(UC);HBI(CD)	6.4±2.1	-
Ali	2013	USA	37.5	27（66）	41	18	23	PSQI	MMS(UC);HBI(CD)	-	-
Ananthakrishnan	2013	USA	44	2316（73）	3173	1094	2079	PROMIS-PSQI	SCCAI(UC);SCDAI(CD)	-	-
Gingold-Belfer	2014	Israel	40.3	47（44）	108	-	108	PSQI	CDAI	10.22±8.6	66
Kappelman	2014	USA	44.1(UC), 44(CD)	7569（71）	10634	3945	6689	PROMIS	SCCAI(UC);SCDAI(CD)	12.5±11.01(UC); 16.3±12.09(CD)	-
Stewart	2014	USA	52	10（43）	23	-	-	PSQI	-	-	34
Wilson	2014	USA	25	72（55）	131	53	78	PROMIS	-	-	-
Van Langenberg	2014	Australia	44	29（60）	48	-	48	PSQI	HBI(CD)	14	30

Van Langenberg	2015	Australia	44	29（59）	49	-	49	PSQI	-	14	31
Hashash	2016	USA	44	363（53）	685	267	418	PSQI	UCAI(UC);HBI(CD)	-	-
Schindlbeck	2016	Germany	47	31（72）	43	13	30	PSQI	Partial Mayo Score(UC);HBI(CD)	-	-
Stevens	2016	USA	35	77（48）	160	66	94	PROMIS	-	-	-
Takahara	2016	Japan	42	34（43）	80	46	34	PSQI	-	-	-
Uemura	2016	Japan	42	60（44）	136	88	48	PSQI	Partial Mayo Score(UC);HBI(CD)	-	-
Abdalla	2017	USA	43.7	4479（71）	6309	2362	3947	PROMIS	SCCAI(UC);SCDAI(CD)	11.5±11(UC); 15.9±14(CD)	-
Ishak	2017	USA	42.4	46（47）	97	35	62	PROMIS	-	-	-
Jelsness-Jørgensen	2017	Norway	40	201（49）	410	180	230	BNSQ	SCCAI(UC);SCDAI(CD)	6(UC);11(CD)	-

Van Langenberg	2017	Australia	44	28（57）	49	-	49	PSQI	HBI(CD)	14	31
Ballou	2018	USA	44.5	31（70）	44	-	-	PSQI	-	-	41
Bucci	2018	Italy	39.7(UC);38.5(CD)	22（47）	47	19	28	PSQI	-	6±6.5(UC);8.1±8.5(CD)	47
Chakradeo	2018	USA	41.4	71（62）	115	-	-	PSQI	Harvey-Bradshaw questionnaire(UC); CDAI(CD)	-	76
Chrobak	2018	Poland	42.6(UC);35.8(CD)	38（53）	72	38	34	PSQI	-	6.5(UC);8.5(CD)	57
Frigstad	2018	Norway	40	196（48）	405	178	227	BNSQ	SCCAI(UC);HBI(CD)	6(UC);11(CD)	-
Hood	2018	USA	42.6	26（53）	47	47	-	PSQI	UC-CDAI	-	-
Lee	2018	USA	45	37（66）	56	17	39	PSQI	LSS(UC);HBI(CD)	-	-
Michalopoulos	2018	Greece	45.4(UC);37.3(CD)	42（47）	90	36	54	PSQI	Mayo score(UC);CDAI(CD)	10.2 ±9.3(UC);6±5.4(CD)	-

Sobolewska-Włodarczyk	2018	Poland	42(UC);38.7(CD)	27（42）	65	35	30	PSQI	Mayo score(UC);CDAI(CD)	6.3±8.2(UC);5.3±4(CD)	-
Bazin	2019	France	40	15（44）	34	-	34	PSQI-Actigraphy	HBI(CD)	-	-
Habib	2019	Iran	38.2	43（61）	71	44	24	PSQI	UCAI(UC);CDAI(CD)	8.2±5.5	-
Kani	2019	Turkey	41.7(UC);37.1(CD)	58（43）	136	64	72	PSQI	Mayo score(UC);CDAI(CD)	6.2±5.08(UC);5.33±4.04(CD)	168
Paixao	2019	Brazil	46.4(UC);30.9(CD)	13（65）	20	11	9	PSQI-PSG	CDAI	-	-
Qazi	2019	USA	36.2	23（32）	72	-	72	PROMIS,Actigraphy	HBI;CRP	13(CD)	-
Sofia	2019	USA	43	57（62）	92	-	92	PSQI	HBI(CD)	-	82
Zargar	2019	Iran	38.6	57（50）	115	85	30	PSQI	-	-	-
Beilman	2020	Canada	35.3	11（73）	15	8	7	PSQI-PSG	-	-	-

Calvo	2020	Spain	45	44（102）	102	51	51	PSQI	Partial Mayo Score(UC);HBI(CD)	-	-
Conley	2020	USA	38	21（37）	37	19	18	PSQI-Actigraphy	-	12.3	-
Gîlca-Blanariu	2020	Romania	42.4	51（46）	110	76	34	PSQI	Mayo score(UC);CDAI(CD)	-	66
Iskandar	2020	USA	32	-	61	-	61	PSQI-Actigraphy	HBI(CD)	-	60
Keskin	2020	Turkey	36.8	59（66）	90	49	41	PSQI	-	-	44
Marinelli	2020	Italy	44	78（47）	166	79	87	PSQI	-	-	-
Scott	2020	UK	33.9	335（82）	409	155	254	SLEEP-50	SCCAI;Seo	9.12 ±8.52	377
Sobolewska-Włodarczyk	2020	Poland	40.7	31（48）	65	35	30	PSQI	Partial Mayo Score(UC);CDAI(CD)	6.3 ±8.2(UC);5.3 ±3.9(CD)	-
Sochal	2020	Poland	36.5	36（62）	58	-	58	AIS	HBI(CD)	-	26

Zhang	2020	China	36.1	59（49）	120	103	17	PSQI-PSG	MES(UC);HBI(CD)	-	120
Leal	2021	Portugal	32	100（49）	205	83	122	PSQI	Partial Mayo Score(UC);HBI(CD)	7	-
Ling	2021	China	30.5	34（32）	106	33	73	PSQI	Mayo score(UC);CDAI(CD)	-	165
Sobolewska-Włodarczyk	2021	Poland	41.5(UC);38.7(CD)	26（50）	52	20	32	PSQI	Partial Mayo Score(UC);CDAI(CD)	5.4±5.6(UC); 7.4±9.2(CD)	-
Xu	2021	China	-	26（37）	71	18	53	PSQI	Partial Mayo Score(UC,CD);CDAI(CD)	-	68
Salwen-Deremer	2022	USA	48.62	207（66）	312	122	188	PSQI	Patient-Reported Outcomes-3	-	-
Kamp	2023	USA	33.4	17（61）	28	7	21	PROMIS;PSQI	SCCAI,MES(UC); HBI,SESCD(CD)	11	-
Yu	2023	China	41.6(UC);33.4(CD)	931（38）	2478	1371	1107	PSQI	MMS(UC);CDAI(CD)	-	-

Oyama	2024	Japan	-	-	139	68	71	PSQI	UCAI(UC);CDAI(CD)	-	-
Pal	2024	Indian	31	69（34）	202	79	123	PSQI	SCCAI(UC);CDAI(CD)	1.7	-