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THE CORRELATION BETWEEN SLEEP DISORDERS AND ULCERATIVE COLITIS AND OTHER AUTOIMMUNE DISEASES: A SCIENTIFIC REVIEW

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ABSTRACT

Ulcerative colitis (UC), a chronic inflammatory bowel disease (IBD), is an autoimmune disease and is increasingly recognised for its bidirectional relationship with sleep disorders. Emerging evidence suggests that sleep disturbances not only exacerbate UC symptoms but may also influence disease activity and relapse rates. Sleep deprivation is increasingly recognized as a significant factor influencing the pathogenesis and progression of autoimmune diseases. This review also synthesizes current evidence on the bidirectional relationship between sleep disturbances and autoimmune conditions, focusing on underlying mechanisms, clinical implications, and potential therapeutic strategies.

KEYWORDS

IBD, Ulcerative Colitis, Sleep Disorders, Autoimmune Diseases

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Introduction: Autoimmune diseases, with the expansion of medical knowledge, are diagnosed more frequently and are still a therapeutic challenge. Even though WHO doesn't have any specific statement, a cohort study from 2023 containing 22 million individuals, suggests that approximately 10% of the world's population may be affected by autoimmune diseases. [1]

Prevalence of Sleep Disorders in UC Patients

Studies indicate that sleep disorders are prevalent among IBD patients, including those with UC. [2][3] A cross-sectional study involving 208 IBD patients (58 with UC) and 199 healthy controls found that 59.6% of IBD patients experienced sleep disorders, a significantly higher rate than the 37.7% observed in healthy controls. A total of 208 patients with inflammatory bowel disease (IBD)—including 150 with Crohn's disease (CD) and 58 with ulcerative colitis (UC)—along with 199 healthy controls were studied. Sleep disorders were identified in 59.6% of the IBD group, with a slightly higher rate in females (63.5%) compared to males (56.9%), though this difference was not statistically significant ($P = 0.476$). Sleep disorders were significantly more common in IBD patients than in the healthy control group, where the prevalence was 37.7% (all $P < 0.01$). Among IBD subtypes, sleep disturbances were found in 58% of CD patients and 63.8% of those with UC ($P = 0.291$). Multivariate analysis indicated that older age (OR = 1.070; 95% CI: 1.035–1.105; $P = 0.000$), smoking (OR = 2.698; 95% CI: 1.089–6.685; $P = 0.032$), and depression (OR = 4.779; 95% CI: 1.915–11.928; $P = 0.001$) were significant risk factors for sleep disturbances in IBD patients. Conversely, a higher body mass index appeared to be protective (OR = 0.879; 95% CI: 0.790–0.977; $P = 0.017$).This prevalence underscores the need for routine screening and management of sleep disturbances in UC patients. [4]

The findings from 2021 meta analysis indicated that individuals with IBD experienced worse subjective sleep quality compared to healthy controls, with a moderate effect size (g =.49, 95% CI [.32 –.66], p <.001). No significant differences were observed between IBD subtypes (g = –.07, 95% CI [–.17 –.05], p =.208). Participants with active IBD reported significantly poorer sleep quality than those in remission, showing a large effect size (g =.66, 95% CI [.35 –.98], p <.001). In contrast, results from objective sleep measurements were inconsistent, offering no strong evidence of sleep disturbances in individuals with IBD. [5]

A total of 519 studies were reviewed and 36 met the criteria for inclusion in the meta-analysis from 2022, encompassing 24, 209 individuals with IBD. The combined prevalence of poor sleep among those with IBD was 56% (95% CI: 51–61%), with notable heterogeneity across studies. The prevalence remained consistent regardless of how poor sleep was defined. Meta-regression analysis showed that poor sleep

prevalence increased with age and with higher levels of objectively measured IBD activity, but it was not significantly associated with subjective IBD activity, depression, or the duration of the disease. [6]

An article from 2020 examined disease activity according to established clinical guidelines. There was a significant link between disease activity and subjective sleep quality, with a pooled odds ratio of 3.52 (95% CI: 1.82–6.83, $p < 0.001$). Additionally, a significant relationship was found between disease activity and sleep efficiency, with a pooled odds ratio of 4.55 (95% CI: 1.92–10.75, $p = 0.001$). [7]

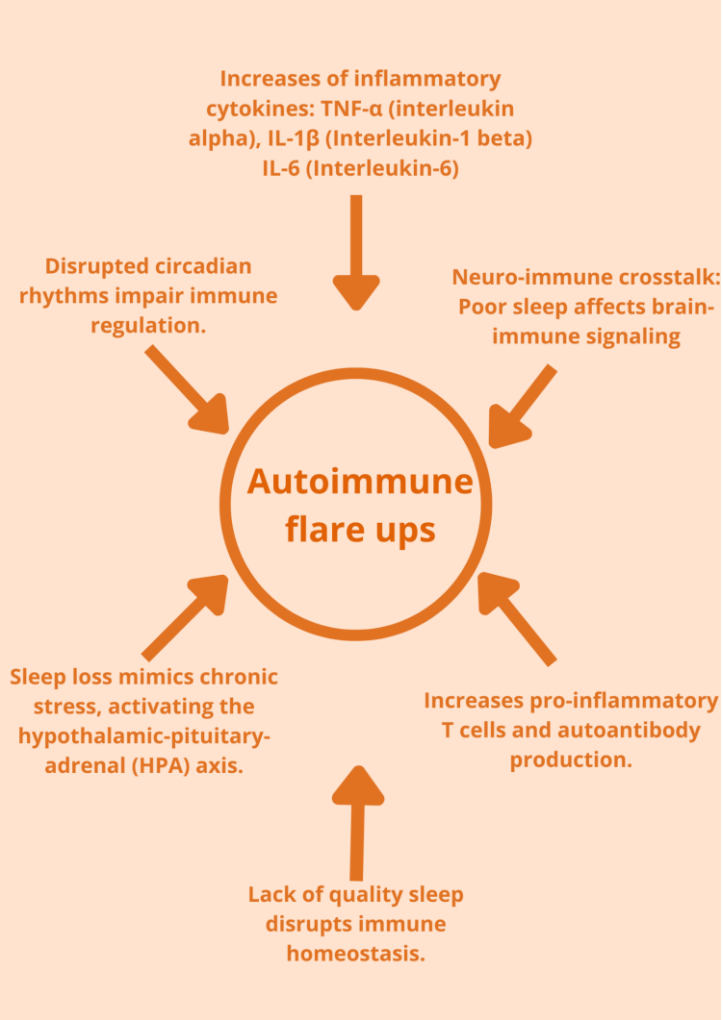

***Fig. 1**. An example of an infographic showing correlation between disruptions in sleep and autoimmune flare ups.*

Impact of Sleep Disorders on Disease Activity

The relationship between sleep disorders and UC extends beyond mere association; poor sleep quality has been linked to increased disease activity and relapse rates. [7, 8]

A prospective observational study in Japan which included 139 patients with inflammatory bowel disease (including 60 with chronic poor sleep), reported that patients with chronic poor sleep had a significantly higher relapse rate of UC (34.5%) compared to those with non-poor sleep (10.3%). Multivariate analysis identified chronic poor sleep as a clinical factor influencing UC relapse, with an odds ratio of 8.89. The rate of disease relapse was notably higher in individuals with poor sleep compared to those without sleep issues (28.3% vs. 8.9%; $p = 0.0033$). Specifically, patients with ulcerative colitis who experienced poor sleep had a significantly higher relapse rate than those with better sleep quality (34.5% vs. 10.3%; $p = 0.031$). Further analysis revealed that chronic poor sleep was a significant clinical factor associated with increased risk of relapse in inflammatory bowel disease (odds ratio [OR] = 6.69, 95% confidence interval [CI]: 2.23–20.0; $p = 0.0007$) and in ulcerative colitis (OR = 8.89, 95% CI: 1.57–50.2; $p = 0.014$). Additionally, Kaplan-Meier analysis demonstrated that patients with poor sleep had significantly lower cumulative treatment retention rates compared to those without

sleep issues (all patients: p = 0.0061; ulcerative colitis: p = 0.025).[9] Health-related QoL in IBD is influenced by aspects of sleep quality irrespective of IBD activity and mental health conditions. A total of 553 responses were analyzed, with 62.2% of participants diagnosed with Crohn's disease and over half (53.1%) receiving biologic therapy. Poor sleep and clinically significant insomnia were linked to reduced quality of life, as reflected in lower EQ-5D-5L scores (EQVAS and utility scores, all $p < 0.001$). Sleep quality was significantly correlated with the EQ-5D-5L domains of"pain" ($\rho = 0.35$, $p < 0.001$), "usual activities" ($\rho = 0.32$, $p < 0.001$), and"depression-anxiety" ($\rho = 0.37$, $p < 0.001$). Even after controlling for demographic factors, IBD activity, and mental health symptoms in multivariate regression, the"pain" domain remained significantly associated with PSQI components:"sleep quality, ""sleep disturbance, " and"sleep duration" (all $p < 0.001$). Clinically significant insomnia was independently linked to decreased quality of life (EQVAS, utility score), with an impact comparable to that of active IBD, regardless of disease activity ($p < 0.001$).[10, 11]

Mechanisms Linking Sleep and UC

Sleep disturbances have been linked to a higher risk of serious health issues, financial impacts, and notably, increased overall mortality. Research increasingly supports a connection between sleep, immune system function, and inflammation. However, the interplay between disrupted sleep and inflammatory conditions remains complex and not fully understood. Lack of sleep can raise levels of inflammatory markers such as interleukin (IL)-1β, IL-6, tumor necrosis factor-α, and C-reactive protein, which may further stimulate the body's inflammatory response. Interest has grown around the role of sleep in inflammatory bowel disease (IBD)—a chronic inflammatory condition of the digestive tract driven by immune dysfunction. Studies have found that individuals with both active and inactive IBD commonly report sleep disturbances. [12, 13] Recently, growing attention has been directed toward the potential influence of sleep, circadian rhythms, and melatonin in regulating inflammation within the gastrointestinal (GI) tract. Disruption of circadian rhythms has been shown to significantly worsen colitis in animal studies, and early research in humans suggests that individuals with IBD are more likely to experience disturbed sleep patterns. For example melatonin is present throughout various parts of the body, including the entire gastrointestinal tract (GIT). Although the pineal gland is the primary source of nighttime melatonin, the levels found in the GIT are approximately 400 times higher than those in the pineal gland. Melatonin functions as a potent antioxidant and free radical scavenger, and it has also been shown to reduce levels of tumor necrosis factor-alpha (TNF-α), both of which are pathways known to play significant roles in the development and progression of IBD. [14]

Risk Factors for Sleep Disorders in UC

Several factors contribute to the high prevalence of sleep disorders among UC patients. A study identified older age, smoking, and depression as significant risk factors, while a higher body mass index was associated with a lower risk of sleep disturbances. Addressing these modifiable risk factors may offer avenues for improving sleep quality and, by extension, disease outcomes in UC patients. [4, 15]

A 2025 systematic review compiled studies involving adults (aged 18 and over) diagnosed with inflammatory bowel disease—including Crohn's disease, ulcerative colitis, or indeterminate colitis—where fatigue and sleep were assessed using either validated self-report questionnaires or objective measurement tools. A total of 22 studies met the inclusion criteria, encompassing 16, 927 participants—58.7% (n = 9, 937) of whom had Crohn's disease, and 66.3% (n = 11, 226) were female. Among the participants, 56.1% reported experiencing fatigue, while 66.3% reported poor sleep. Fatigue was consistently linked to poor sleep quality and insomnia. Individuals with initially poor sleep were more likely to experience ongoing or worsening fatigue over a period of 12 to 24 months. Greater fatigue severity was also associated with clinically assessed (but not objectively measured) disease activity, as well as anxiety, depression, female sex/gender, and younger age. [16]

The impact on autoimmune diseases of sleep deprivation

Sleep deprivation is increasingly recognized as a critical environmental factor influencing the onset and progression of autoimmune diseases, which involve the immune system attacking the body's own tissues. [17] Genetic predisposition remains important; however, sleep disturbances have been shown to alter immune responses significantly. Sleep loss promotes a proinflammatory state by elevating cytokines such as IL-1β, IL-6, TNF-α, and CRP, thereby disrupting immune homeostasis and exacerbating autoimmune pathogenesis. [18, 19, 20] Additionally, disruption of the circadian rhythm, often caused by irregular sleep patterns, impairs immune regulation and increases susceptibility to autoimmune diseases. [7, 21, 22, 23, 24, 25] Sleep deprivation also affects the hypothalamic-pituitary-adrenal (HPA) axis, resulting in elevated cortisol levels,

which can both suppress immune defenses and promote chronic inflammation, further contributing to autoimmune disease development. [26] Experimental studies have even demonstrated that prolonged sleep deprivation can induce a cytokine-storm-like syndrome in mammals, underlining the profound immunological impact of insufficient sleep. [20]

Epidemiological and clinical studies have established associations between sleep deprivation and various autoimmune disorders. Women with chronic short sleep duration have been found to possess nearly a threefold increased risk of developing systemic lupus erythematosus (SLE). [27] Rheumatoid arthritis (RA) patients commonly experience sleep disturbances, which correlate with heightened disease activity and pain. [17, 24] Similarly, poor sleep quality in multiple sclerosis (MS) patients is linked to increased fatigue and disease severity, suggesting that sleep loss impairs immune regulation and exacerbates disease progression. [27] Other autoimmune conditions, including Hashimoto's thyroiditis, Graves' disease, and alopecia areata, have also been associated with sleep disturbances influencing immune modulation. [24] Clinical management should incorporate assessment and treatment of sleep disorders through cognitive behavioral therapy for insomnia (CBT-I), pharmacological treatments, and lifestyle modifications, which have shown promise in improving disease outcomes and patient quality of life. [19, 28] Future research should focus on elucidating precise mechanisms linking sleep deprivation with autoimmunity, supported by longitudinal studies and clinical trials. Such efforts may pave the way for personalized medicine approaches targeting sleep and immune function to prevent or mitigate autoimmune disease progression. [20]

Implications for Clinical Practice

Given the significant impact of sleep disorders on UC disease activity and patient quality of life, healthcare providers should routinely assess sleep quality in UC patients. Implementing interventions such as cognitive behavioural therapy for insomnia (CBT-I), pharmacological treatments, and lifestyle modifications may help mitigate sleep disturbances and improve disease management. It's worth noting that]the findings indicate that individuals with IBD experience notable sleep disturbances even during periods of disease remission. These sleep issues may negatively impact quality of life, exacerbate gastrointestinal symptoms, impair coping mechanisms, and potentially influence disease severity or increase the likelihood of flare-ups. Whether sleep disturbances are a primary condition or secondary to IBD, they should be recognized and managed as part of routine clinical care for IBD patients. [29][30]

Conclusions

The bidirectional relationship between sleep disorders and ulcerative colitis (UC) underscores the necessity of adopting an integrated, multidisciplinary approach to patient care. Individuals with UC frequently experience sleep disturbances, which may include insomnia, poor sleep quality, fragmented sleep, and increased sleep latency. These disturbances not only diminish quality of life but also have the potential to exacerbate intestinal inflammation, contributing to disease flares and increased symptom severity. Conversely, active UC symptoms such as abdominal pain, diarrhea, and nocturnal urgency can significantly disrupt sleep patterns, creating a self-perpetuating cycle that may hinder disease management. Given this complex interplay, it is essential that clinicians consider sleep health as a core component of UC management. Addressing both gastrointestinal and sleep-related symptoms in tandem—rather than treating them in isolation—could enhance treatment efficacy and improve patient-reported outcomes. Integrating sleep assessments into routine clinical evaluations for individuals with UC may allow for earlier identification of sleep disorders and facilitate timely interventions, such as behavioral therapy, sleep hygiene education, or pharmacological support when appropriate. Furthermore, additional research is urgently needed to better characterize the specific types and patterns of sleep disturbances that affect patients with inflammatory bowel disease (IBD), including both UC and Crohn's disease. A more nuanced understanding of these disturbances could enable the development of targeted therapeutic strategies that simultaneously address inflammation and sleep dysfunction. Research should also investigate how various IBD treatments—such as biologics, immunosuppressants, and dietary interventions—impact sleep quality, and whether improving sleep can lead to measurable improvements in disease activity and long-term outcomes. Ultimately, recognizing and managing sleep disturbances as modifiable risk factors in UC could represent a valuable strategy for influencing the course of the disease. In doing so, healthcare providers can move toward a more holistic model of care—one that not only controls intestinal inflammation but also supports overall well-being and quality of life.

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

In preparing this work, the author(s) used ChatGPT for the purpose of improving the language and verifying the styles of citations. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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For full transparency, all submitted manuscripts must include an Author Contribution Statement stating the work of each author. For research articles with multiple authors, a short paragraph must be provided stating their individual contributions.

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