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People with IBD evidence more microarousals during sleep architecture assessments

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ABSTRACT

Objective Poor sleep is common in inflammatory bowel disease (IBD) and may be associated with overall worse disease outcomes. While the sleep/IBD literature is growing, the data are often self-reported. Further, much of the research using objective measures of sleep architecture, or the overall pattern of sleep depth, rely on single-night assessments, which can be of questionable validity.

Design Participants with IBD and healthy controls were recruited from Dartmouth-Hitchcock Medical Center as part of a two-phase clinical trial. Sleep architecture was assessed using three nights of in-home electroencephalographic monitoring and scored according to the American Academy of Sleep Medicine guidelines.

Results Our sample included 15 participants with IBD and 8 healthy controls. Participants with IBD were more psychiatrically complex, with more self-reported insomnia, anxiety and depression. Participants with IBD evidenced greater microarousals than healthy controls. In participants with IBD, microarousals were associated with lower insomnia and greater depression scores. Within IBD, participants with clinically significant insomnia evidenced trend towards lower sleep efficiency, while self-reported disease activity did not significantly impact findings.

Conclusions The methodology of past research may have impacted findings, including the reliance on single-night assessments and limited generalisability. Future research that uses robust, multinight assessments of sleep architecture in large, diverse samples is clearly warranted, as is research exploring the impact of cognitive and behavioural factors on sleep architecture and arousal.

Trial registration number NCT04132024.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ People with inflammatory bowel disease report poor sleep. Past assessments of sleep architecture, the stages and cycles of sleep, have been limited.

WHAT THIS STUDY ADDS

⇒ Sleep architecture assessments were completed in home, over three nights, reducing the possible impact of adjustment to the device and night-to-night variations in sleep.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Better understanding of objective measures of poor sleep can inform prevention efforts and intervention development, improving disease course in IBD.

INTRODUCTION

Poor sleep is associated with greater disease activity and likelihood of hospitalisation in people with inflammatory bowel diseases (IBDs) including Crohn's disease (CD) and ulcerative colitis (UC).[1–4] People with both active and inactive IBD report difficulty falling asleep, sleeping pill use and poor overall sleep quality.[2] Objective data also indicates that people with IBD have a greater incidence of sleep fragmentation, characterised by brief electroencephalographic (EEG) arousals.[5 6]

Sleep disturbances impact sleep architecture—the stages and cycles of sleep that are comprised of rapid eye movement (REM) and non-REM sleep.[7 8] Emerging literature suggests that people with IBD spend a greater percentage of total sleep time in light sleep (stage 2 (N2)) and a smaller percentage in deep sleep (stage 3 (N3) or slow wave sleep) than healthy controls.[8–10] They also spend less time in REM sleep, more time in the N1 stage (a transitional stage between wake and sleep) and exhibit more EEG arousals.[8 10]

There are two key problems with the current literature. First, most research on sleep architecture in IBD has relied on a single night of laboratory data collection,[1 8 11 12] excepting a recent study by Zhang *et al*, which used two nights of in-lab polysomnography (PSG).[10] Single-night PSG can produce invalid data due to the subject's adjustment to the lab setting, testing device and/or procedures—termed the 'first night effect'.[13] Second, investigations into sleep architecture have typically excluded people with active IBD or sleep disorders, thereby limiting

generalisability.[1 8 11 12] Given the high prevalence of insomnia in IBD (~50%), it is likely important to include people with this sleep disorder for generalisability.[14]

Thus, to advance the current understanding of sleep architecture in IBD and produce more methodologically sound results, we used in-home, multinight wireless sleep-EEG testing to evaluate sleep architecture across three nights in people with and without IBD.

METHODS

Participants

Participants with physician-assessed mild-to-moderate CD or UC were referred from the Dartmouth-Hitchcock IBD Center as part of a two-phase pilot clinical trial, where phase I was an observational study. Detailed methods and results from this trial have been published elsewhere.[15] Healthy controls were recruited from advertisements posted around the medical centre and had no personal history of IBD, irritable bowel syndrome or coeliac disease, no current gastrointestinal symptoms or concerns and no family history of IBD or coeliac disease. All participants were recruited between October 2019 and January 2021, with a brief pause early in 2020 due to COVID-19. During this pause, the study methods were altered so as to be fully remote. Sample size was determined based on pilot study guidelines[16–18] and prior research on sleep architecture in IBD.[8 11 12]

For both groups, exclusion criteria were as follows: (1) Patient Health Questionnaire-9[19] Score>20, (2) Generalized Anxiety Disorder-7[20] Score>20, (3) current alcohol or substance abuse as assessed using the Alcohol Use Disorders Identification Test,[21] (4) current opioid use, (5) an unstable major psychiatric condition (via chart review), (6) probable or likely restless leg syndrome based on the Cambridge-Hopkins RLS Questionnaire[22] or (7) high risk for obstructive sleep apnoea based on the STOP-Bang Questionnaire[23] and current symptoms.

Measures

Sleep architecture

Sleep architecture was measured over the course of three nights with the 'sleep profiler PSG2' (Advanced Brain Monitoring, Carlsbad, California) device, a wireless ambulatory sleep-EEG monitor that measures sleep architecture via three channels (AF7, AF8, FPZ) of frontal EEG and a sensor for head movement. The sleep profiler has an autoscoring programme that has been validated against PSG.[24] Sleep architecture was evaluated over three nights to account for night-to-night variation in sleep patterns and for the possibility that night 1 may need to be excluded in the case of a first night effect.

In addition to per cent time in stage 1, stage 2, stage 3/slow wave sleep and REM sleep, we examined the following sleep continuity and sleep architecture variables:

- Sleep onset latency: the time it takes to fall asleep.
- Wake after sleep onset: time spent awake during the night.
- Total sleep time: total hours of sleep during the night.
- Sleep efficiency: per cent time spent in bed asleep (total sleep time/total time in bed×100).
- REM latency: the number of minutes from sleep onset to REM sleep.
- Microarousals: the number of brief (3–14 s) awakenings in which there is an abrupt change in EEG frequency.
- Awakenings: number of >30 s events where there is a transition from sleep → awake → back to sleep.

Patient-Reported Outcomes-3

Participants reported their average stool frequency, abdominal pain, rectal bleeding and general well-being for the past 7 days, with greater scores indicating more severe symptoms.[25] To keep measurement consistent between CD and UC, we classified participants' disease as active or inactive. CD activity was based on the severity of reported symptoms and is specified in our primary trial paper.[15] UC disease activity was based on a previously established scoring algorithm.[26]

Psychosocial questionnaires

Generalized Anxiety Disorder-7

This 7-item questionnaire assesses symptoms of generalised anxiety within the past 2 weeks, with greater scores indicating greater severity of symptoms.

Insomnia Severity Index (ISI)

This 7-item questionnaire assesses severity of insomnia disorder symptoms within the past 2 weeks, with higher scores indicating more severe insomnia.[27] Scores of 10 or greater are indicative of symptoms consistent with an insomnia disorder diagnosis.[28]

Pain Catastrophizing Scale

This 13-item scale assesses beliefs about pain across three dimensions—rumination, magnification and helplessness. Greater scores indicate more catastrophic thoughts about pain.[29]

Patient Health Questionnaire-9

This 9-item questionnaire assesses symptoms of depression within the past 2 weeks, with greater scores indicating greater severity of symptoms.

Pittsburgh Sleep Quality Index

This 10-item questionnaire assesses sleep habits and sleep disturbance in the past month, with greater scores indicating worse overall sleep.[30]

Insomnia-Related Behaviors Questionnaire

This 13-item questionnaire was designed by the first and senior author to assess frequency of behaviours that drive insomnia. Items are based on the Sleep Hygiene Index,[31] Sleep-Pain Behaviors Survey[32] and direct targets of behavioural insomnia treatment. For each item, a higher score indicates the behaviour occurs more often.

Patient and public involvement

The design of this research was discussed with clinicians, researchers who were and were not familiar with this disease population and trainees in medicine and psychology prior to recruitment. The data presented herein were collected as part of a pilot feasibility trial designed to investigate cognitive behavioural therapy for insomnia in people with IBD.[15] Participants who completed both the initial assessment and intervention were interviewed about their trial experiences and barriers and facilitators to participation. Interview data on participants' experiences were then used to inform subsequent research initiatives.

Data analytic strategy

Data were cleaned and evaluated for missingness and outliers. Sleep profiler data were manually scored in 30 s epochs to exclude invalid epochs and override misclassified sleep–wake states based on American Academy of Sleep Medicine criteria.[6] We adjusted lights out and wake times based on a standardised procedure manual developed by our lab.[33] One participant was excluded due to an intentional biphasic sleep pattern and two were excluded due to poor signal quality. Sleep profiler data were managed using the Advanced Brain Monitoring online portal.

Questionnaires were scored according to published guidelines and included no missing data. Comparisons between groups were made using independent samples t-tests and post hoc analyses included linear regressions. All analyses were completed using SPSS V.28.

RESULTS

In total, 15 participants with IBD and 8 healthy controls were included. Participants ranged in age from 23 to 67 years (M=43.65, SD=15.52) and 60.9% were women. Of the 15 participants with IBD, 9 had a diagnosis of CD and 6 had a diagnosis of UC. Participants with IBD received their diagnosis at a mean age of 31.53 years (SD=13.88). Demographics are presented in table 1.

Sleep architecture

We investigated the presence of a first-night effect by comparing night 1 with an average of nights 2 and 3 separately for participants with and without IBD. Healthy controls evidenced significantly more microarousals on night 1 than nights 2 and 3 combined (8.43 vs 4.86, respectively, p=0.03), though participants with IBD did not (13.53 vs 13.87, respectively, p=0.88). Thus, we excluded night 1 data for all analyses.

Results of sleep architecture analyses are displayed in table 2. The only significant difference was that participants with IBD had significantly more microarousals than participants without IBD (13.87 vs 4.86, p=0.008). The effect size for this difference was large (Cohen's d=1.07, 95% CI: 0.10 to 2.01). We also investigated microarousals based on insomnia status, separating our IBD participants with insomnia (n=9) and without insomnia (n=6). A one-way analysis of variance (ANOVA) investigating the number of microarousals in healthy participants, participants with IBD and no insomnia and participants with IBD and insomnia trended towards significance ($F(2,19)=2.60$, p=0.10). Mean microarousals were lowest in healthy participants (M=4.86, SD=3.31) and greater in participants with IBD and no insomnia (M=14.08, SD=7.30) and in participants with IBD and insomnia (M=13.72, SD=11.65).

We further investigated the impact of insomnia on sleep architecture specifically in participants with IBD. There were no significant differences between the participants with and without insomnia and only sleep efficiency (76.66% vs 89.41%, respectively, p=0.08) trended towards significance (online supplemental table 1). Additionally, analyses indicated no significant differences in sleep architecture based on self-reported IBD disease activity, with all p values>0.13 (online supplemental table 2).

Post hoc arousal analyses

We performed exploratory post hoc analyses to better understand possible associations with microarousals in IBD and to distinguish it from insomnia. Specifically, we evaluated whether sleep quality, pain catastrophizing, anxiety and depression would be associated with microarousals above and beyond insomnia. In a linear regression, we controlled for insomnia symptoms (ISI scores) and then allowed psychosocial variables to be part of the model in a forward regression (p<0.05 for inclusion). While the initial model with insomnia predicting microarousals was not significant (p=0.99), the final model including both insomnia and depression was significant (F=5.03, p=0.03). Both insomnia (beta=−1.18, p=0.02) and depression (beta=1.37, p=0.009) contributed to the model significantly and independently.

Finally, we also investigated whether specific insomnia-related behaviours were associated with microarousals in IBD. Pearson correlations revealed that none of the insomnia-related behaviours reached the level of significance, though two items were of at least a moderate association. Specifically, microarousals were positively correlated with getting up at different times from 1 day to the next by at least 30 min (r=0.41, p=0.13) and negatively correlated with taking sleep medication (prescription or over the counter) in response to difficulty sleeping (r=−0.47, p=0.08). All other correlations were <0.28.

DISCUSSION

Overall, many of our analyses did not replicate prior research, with our only significant finding indicating that participants with IBD evidenced more microarousals than healthy controls.[5 8–10 34] It is possible that people with IBD are more influenced by a laboratory setting than healthy controls. For example, worry about bathroom access in an unfamiliar location could result in lighter sleep. In this case, our methodology would likely yield a more

Table 1 Participant demographics

	Full sample	IBD	Healthy control	Significance test
Age	43.65 years (SD=15.52)	43.80 years (SD=15.07)	43.38 years (SD=17.39)	p=0.95
Gender	60.9% women	60% women	62.5% women	p=1.0
Race	87% white	93.3% white	75% white	p=0.53
IBD type and location	–	9 Crohn's disease Location: 4 ileal only 3 ileocolonic 2 colonic only Behavior: 3 inflammatory 4 stricturing 2 penetrating Perianal Involvement: 4 yes 5 no 6 ulcerative colitis Extent: 5 left-sided disease 1 proctitis	–	–
Years since IBD diagnosis	–	M=31.53, SD=13.88	–	–
IBD medications	–	3 adalimumab 1 azathioprine 3 infliximab 3 mesalamine 1 methotrexate 1 sulfasalazine 2 ustekinumab 2 vedolizumab 2 prednisone (<10 mg)	–	–
Sleep and/or psychiatric medications*	–	3 antidepressants (citalopram, fluoxetine, bupropion) 1 anticonvulsant 1 atypical antipsychotic	–	–
Sleep quality (PSQI)	M=7.13, SD=4.37	M=8.93, SD=3.71	M=3.75, SD=3.54	**p=0.004**
Insomnia severity (ISI)	M=8.96, SD=7.70	M=12.13, SD=7.31	M=3.00, SD=4.14	**p=0.004**
Anxiety (GAD-7)	M=5.05, SD=6.22	M=7.79, SD=6.34	M=0.25, SD=0.46	**p=0.003**
Depression (PHQ-9)	M=4.64, SD=5.62	M=7.14, SD=5.67	M=0.25, SD=0.71	**p=0.003**
Pain catastrophizing (PCS)	M=9.0, SD=9.34	M=11.64, SD=10.34	M=4.38, SD=4.98	p=0.08

Bolded values are significant at p<0.05.
*Sleep and psychiatric medications not reported by healthy controls.
GAD-7, Generalized Anxiety Disorder-7; IBD, inflammatory bowel disease; ISI, Insomnia Severity Index; PCS, Pain Catastrophizing Scale; PHQ-9, Patient Health Questionnaire-9; PSQI, Pittsburgh Sleep Quality Index.

accurate comparison of sleep architecture. Additionally, several prior studies excluded people with *diagnosed* sleep disorders, but did not comprehensively screen for sleep disorders. It is possible that they inadvertently included more participants with sleep disorders in the IBD group, impacting results. Herein, participants with IBD and insomnia evidenced worse sleep efficiency.

Post hoc analyses suggested that microarousals in participants with IBD were associated with decreased insomnia symptoms and increased depressive symptoms. These analyses preliminarily suggest that microarousals are not a product or cause of insomnia, but instead are related to more complex psychological factors. We suggest that disease-related distress or helplessness about

Table 2 Differences in sleep architecture in participants with and without IBD

	IBD	Healthy	Significance (t-test)
Sleep onset latency	18.97 min (SD=26.51)	20.93 min (SD=19.43)	p=0.86
Wake after sleep onset	50.77 min (SD=24.91 min)	40.86 min (SD=20.24)	p=0.37
Total sleep time	6.73 hours (SD=1.47)	6.09 hours (SD=0.66)	p=0.29
Sleep efficiency	81.76% (SD=13.86%)	85.78% (SD=7.49%)	p=0.48
REM latency	81.47 min (SD=31.86)	84.14 min (SD=50.84)	p=0.88
REM sleep, %	20.85% (SD=6.92%)	22.28% (SD=6.40%)	p=0.65
Stage 1 sleep (N1), %	5.11% (SD=2.76%)	5.66% (SD=4.07%)	p=0.71
Stage 2 sleep (N2), %	40.43% (SD=14.61%)	42.87% (SD=16.19%)	p=0.73
Stage 3 sleep (N3), %	30.28% (SD=13.02%)	29.19% (SD=17.64%)	p=0.87
Microarousals/hour	13.87 (SD=9.83)	4.86 (SD=3.31)	**p=0.03**
Awakenings	33.83 (SD=16.16)	28.50 (SD=13.28)	p=0.46

Bolded value is significant at p<0.05.
IBD, inflammatory bowel disease; REM, rapid eye movement.

IBD (eg, concern about night-time bowel movements, hypervigilance to internal cues that suggest a need to evacuate one's bowels) may create a state of hyperarousal that predisposes people with IBD to being more impacted by both internal and external stimuli. For patients who report night-time bowel movements but do not evidence objective markers of active disease, it is possible that this hypervigilance and hyperarousal contributes to the persistence of night-time bowel movements.

While this study adds to a growing literature, it does have limitations, including a small, homogeneous sample that limited our power and generalisability.[35] While our sample size is consistent with others investigating sleep architecture in IBD,[8 11 12] future research using larger, more diverse samples is clearly warranted. Additionally, our assessment of disease activity was not objective; future research including measures of inflammation such as C reactive protein are needed to better understand the relationships among sleep disturbances, inflammation and IBD symptoms. Further, while our findings on psychosocial and behavioural contributors to microarousals in IBD are novel, they are very preliminary and should be explored in samples adequately powered for these analyses. While depression was clearly related to microarousals in these analyses, we were unable to more thoroughly investigate this phenomenon due to our small sample. The relative contributions of psychosocial and behavioural factors should also be investigated in healthy controls; we were unable to investigate these relationships herein due to the sample size. Finally, antidepressants may impact sleep architecture and accounting for them in future research is warranted.[36] Herein, only three of our participants with IBD were using medication for sleep and/or psychiatric conditions, thus we were unable to investigate this phenomenon.

CONCLUSIONS

Participants with IBD evidence significantly more microarousals during sleep than healthy controls. In participants with IBD, only sleep efficiency differed based on insomnia symptoms. Post hoc analyses suggested microarousals may be related to insomnia and depression. Research investigating the impact of cognitive factors on night-time arousal is warranted.

Contributors JS-D designed the study with critical input from MTS and CS. JS-D and CS collected the data and JS-D and MR analysed the data. JS-D developed the initial draft of the paper with critical input from SJW and all authors critically revised the manuscript for important intellectual content. All authors approved the final version of this manuscript. JS-D is the guarantor for this article.

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Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by the Dartmouth-Hitchcock Health Human Research Protection Program (IRB #02000051). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement The data underlying this article cannot be shared publicly due to the privacy of individuals that participated in the study. The data will be shared on reasonable request to the corresponding author.

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