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ORIGINAL RESEARCH

Does COVID-19 Infection Continue to Affect Self-Reported and Objective Sleep? A Longitudinal Study of Good Sleepers

Jun Wu[1–3], Baixin Chen[1–3], Qingsong Qin[4], Yanyuan Dai[1–3], Le Chen[1–3], Dandan Zheng[1–3], Jiansheng Zhang[1–3], Yun Li[1–3]

[1]Department of Sleep Medicine, Mental Health Center of Shantou University, Shantou, Guangdong, People's Republic of China; [2]Sleep Medicine Center, Shantou University Medical College, Shantou, Guangdong, People's Republic of China; [3]Shantou University Medical College—Faculty of Medicine of University of Manitoba Joint Laboratory of Biological Psychiatry, Shantou, Guangdong, People's Republic of China; [4]Laboratory of Human Virology and Oncology, Shantou University Medical College, Shantou, People's Republic of China

Correspondence: Yun Li, Department of Sleep Medicine,Mental Health Center of Shantou University, North Taishan Road, Wanji District, Shantou, Guangdong, 515041, People's Republic of China, Email s_liyun@stu.edu.cn

Background: Whether COVID-19 infection continues to affect both self-reported and objective sleep is not clear. This longitudinal study aims to investigate the impact of COVID-19 infection on self-reported and objective sleep of good sleepers.

Methods: Fifteen good sleepers, with prior COVID infection, completed self-reported and objective sleep assessments at 3 time points: pre-COVID-19 infection, short-term post-COVID-19 infection for 1 month and long-term post-COVID-19 infection for 6 months. Self-reported sleep quality was assessed with the Pittsburgh Sleep Quality Index (PSQI). Self-reported sleep onset latency (s-SOL) and sleep efficiency (s-SE) were extracted from the PSQI. Objective sleep was assessed by overnight polysomnography. Nighttime electroencephalogram (EEG) relative power at central EEG derivations during sleep was calculated.

Results: Total scores of the PSQI (P=0.003), s-SOL (P=0.017) and s-SE (P=0.040) changed across the 3 time points. Specifically, total PSQI scores (P=0.002) and s-SOL (P=0.011) increased, while s-SE decreased (P=0.019) from the pre-COVID-19 infected period to the short-term post-COVID-19 infected period. However, there were no significant differences regarding PSQI scores, s-SOL or s-SE between short-term and long-term post-COVID-19 infected periods, or between pre-COVID-19 and long-term post-COVID-19 infected periods (all P>0.999). The changes in objective sleep were not significant across different periods except shorter o-SOL at the long-term post-COVID-19 infected period compared to the pre-COVID-19 (P=0.028) and short-term post-COVID-19 infected periods (P=0.010). Similarly, the changes in EEG relative power were not significant across different periods except the relative alpha EEG power during REM sleep (P=0.007).

Conclusion: COVID-19 infection has temporary adverse effect on self-reported sleep but no effect on objective sleep of good sleepers.

Keywords: COVID-19 infection, self-reported sleep, objective sleep

Introduction

The outbreak of a highly contagious, 2019 novel coronavirus (2019-nCoV/SARS-CoV-2), causing respiratory infections and pneumonia in humans, was observed in China in December 2019. Since then, the coronavirus disease 2019 (COVID-19) rapidly spread throughout China and worldwide. A meta-analysis revealed that poor sleep quality is associated with an elevated risk of COVID-19 infection and a greater likelihood of severe disease outcomes.[1] This suggests that maintaining good sleep may hold significant preventive value and practical implications for mitigating the impact of the COVID-19 pandemic and population susceptibility.[2] A large number of studies have found that the impact of the COVID-19 pandemic or COVID-19 infection on sleep is widespread.[3] The study found that for hospitalized patients 6 months after acute infection, COVID-19 survivors mainly presented with fatigue or muscle weakness, sleep difficulty, anxiety or depression.[4] Moreover, special populations such as medical staff, patients with COVID-19, patients with chronic diseases and patients with mental disorders have a higher prevalence of insomnia symptoms during the COVID-

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19 pandemic.[3] However, most of the previous reports were cross-sectional studies that based on only self-reported sleep questionnaires,[5] while objective sleep (ie, polysomnography [PSG] or actigraphy) for COVID-19 was not common.

From an electrophysiological perspective, in a study of sleep electroencephalogram (EEG) of COVID-19 survivors, abnormal brain waves were very common in COVID-19 survivors in the short term after infection, which could have similar effects as depression and insomnia.[6] The study by Sun et al found an increase in low-frequency EEG activity during wakeful EEG after SARS-CoV-2 infection.[7] These observations may help further explore changes in sleep after COVID-19 infection. Furthermore, longitudinal studies are needed to determine whether these changes are short-term or long-term. Evidence from longitudinal studies using objective sleep measurement is lacking. Whether COVID-19 infection continues to affect both self-reported and objective sleep is not clear.

This longitudinal study investigated the impact of post-COVID-19 infection on self-reported and objective sleep in good sleepers. We examined whether post-COVID-19 infection had adverse effects on self-reported and objective sleep of good sleepers throughout the short- and long-term post-COVID-19 infected status.

Materials and Methods

Participants

To minimize confounding effects of pre-existing sleep disorders and isolate the specific impact of COVID-19 infection on sleep, we recruited fifteen good sleepers from a good sleep control group, established before the COVID-19 pandemic, in an ongoing study comprised of adults (age≥18 years) without sleep complaints. The exclusion criteria for good sleepers included: (1) Chronic medical conditions known to disrupt sleep continuity (eg, chronic pain disorders, cardiopulmonary diseases); (2) Current major psychiatric condition, including but not limited to major depressive disorder and anxiety disorders; (3) Current or recent (within 1 month) treatment for insomnia, including pharmacotherapy (sleep medications or herbal medicines) or cognitive-behavioral therapy; (4) Sleep-disordered breathing (apnea-hypopnea index [AHI] ≥5 events/hour) confirmed by PSG or clinically diagnosed comorbid sleep disorders (eg, restless legs syndrome, circadian rhythm disorders). All the participants were subsequently infected with 2019-nCoV/SARS-CoV-2 between December 2022 and May 2023, and who had agreed to receive self-reported and objective sleep assessments during short- and long-term post-COVID-19 infection. Participants were evaluated through a standardized interview conducted by the same attending physician, and none of the participants were hospitalized for COVID-19. In this study, short-term post-COVID-19 infection follow-up was conducted around 1 month after their COVID-19 antibody testing became negative. Long-term post-COVID-19 infection follow-up was conducted 6 months after their COVID-19 antibody testing became negative. Both tests were conducted between December 2022 to May 2023. COVID-19 infection was confirmed based on the results of the individual's COVID-19 antigen-based lateral flow test from a nasopharyngeal swab.[8] We included adults who had COVID-19 infection between December 2022 and May 2023 and completed sleep assessments at their pre-COVID-19, short-term and long-term post-COVID-19 infection periods. We excluded subjects who (1) had a major mental condition (ie major depression) or sleep disorder (ie, insomnia and sleep apnea) pre-COVID-19; (2) had a central system infection or other major physical disease during the follow-up; (3) were pregnant or nursing; (4) refused to participate in the study or could not complete the follow-up surveys. Ultimately, 15 good sleepers were included. Figure 1 depicts the flow of this study. This study was approved by the Research Ethics Board of the Mental Health Center of Shantou University and informed consent was obtained from each participant. This study complied with the Declaration of Helsinki.

Measures

Clinical History and Physical Examination

All subjects completed a medical history and physical examination at their pre-COVID-19 infection period and at each post-COVID-19 infection follow-up in the sleep laboratory using a semi-structured questionnaire and a battery of clinical tests. Body mass index (BMI) was calculated based on weight (kg)/height^2 (m^2).

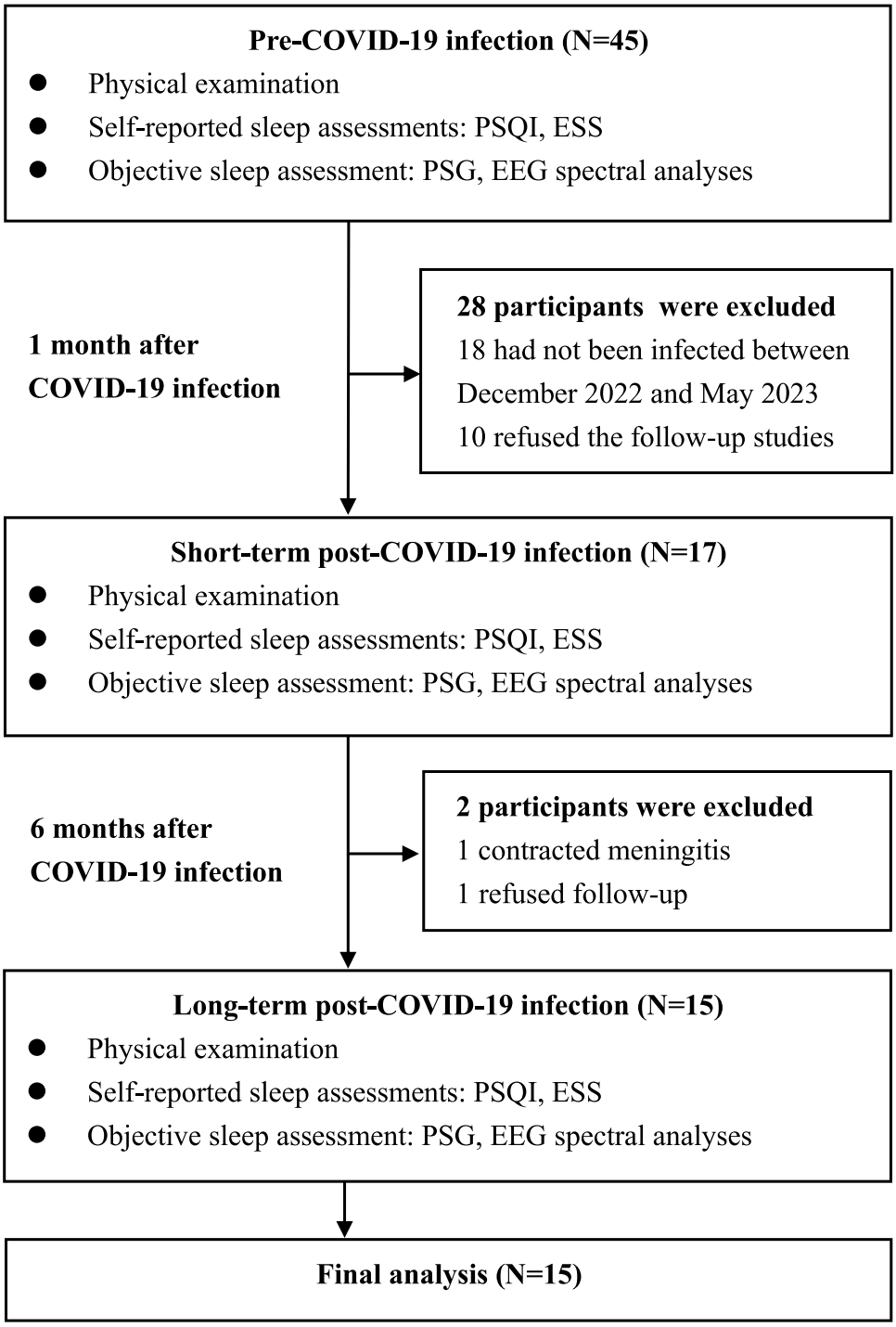

Figure 1 Study procedure.
Abbreviations: EEG, electroencephalogram; ESS, Epworth Sleepiness Scale; PSG, polysomnography; PSQI, Pittsburgh Sleep Quality Index.

Self-Reported Sleep Assessments

The Pittsburgh Sleep Quality Index (PSQI) was used to assess self-reported sleep (https://www.sleep.pitt.edu/psqi). Higher PSQI scores indicate worse self-reported sleep quality.[9] The self-reported sleep onset latency (s-SOL), total sleep time (s-TST) and sleep efficiency (s-SE) were calculated based on PSQI questions 1. During the past month, what time have you usually gone to bed at night? 2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night? 3. During the past month, what time have you usually gotten up in the morning? 4. During the past

month, how many hours of actual sleep did you get each night?[10] The Epworth Sleepiness Scale (ESS) was used to assess daytime sleepiness and the license was obtained. Higher ESS scores indicate more severe daytime sleepiness.[11] We used simplified Chinese version of the PSQI and ESS scale, which has been widely used and has achieved good internal consistency in our previous cross-sectional study with large sample (PSQI: Cronbach's α=0.74, ESS: Cronbach's α=0.82).[12]

Polysomnography

All subjects underwent overnight PSG monitorings in the sleep laboratory to assess their objective sleep at their pre-COVID-19 infection period and at each post-COVID-19 infection follow-up. Each subject could sleep according to their habitual sleep time, with the recording time ranging from 22:00–23:00 to 6:00–7:00. Subjects were continuously monitored with 16-channel polygraphs, including EEG, electrooculogram, electromyogram, and electrocardiogram. Respiration was monitored by use of thermocouples and a nasal pressure transducer at the nose and mouth and thoracic strain gauges throughout the night. Peripheral oxygen saturation was obtained with an oximeter attached to the finger. The sleep records were scored by a senior technician who was blind to the study, as according to the American Academy of Sleep Medicine Manual for the Scoring of Sleep and Associated Events version 2.3.[13]

EEG Spectral Analyses

We also used EEG spectral analyses to assess objective sleep. Consistent with our previous studies, spectral analyses of EEG power were performed on central EEG derivations (C3-M2 and C4-M1) during non-rapid eye movement (NREM) and rapid eye movement (REM) sleep from the nighttime PSG.[14,15] After automatically eliminating epochs encompassing movements, arousals and poor electrode contact, we used artifact-free epochs of NREM and REM sleep for spectral analysis. In the spectral analysis, NREM sleep was defined as any period of NREM sleep persisting for at least 15 minutes, and REM sleep was characterized as any period of REM sleep lasting for at least 5 minutes.[16] We utilized sleepFFT software (Biosoft Studio), the fast Fourier transform (FFT) algorithm, to calculate relative spectral EEG power. The spectral resolution was set at 0.50 hz. After applying the Hann window, the power spectrum was calculated for each FFT window within the data. The averaged C3-M2 and C4-M1 relative EEG power of each frequency band (delta [0.5–4.0 hz], theta [4.5–8.0 hz], alpha [8.5–11.0 hz], sigma [11.5–15.0 hz], beta [15.5–30.0 hz]) and gamma [30.5–-49.5 hz] were used for analyses in this study.

Statistical Analysis

Data are presented as the mean ± standard deviation for continuous variables. One-way repeated measures analysis of variance was used to compare the changes across pre-COVID-19 infected periods, and the short- and long-term post-COVID-19 infection periods. Bonferroni correction was used to correct for multiple post hoc comparisons. These models were not adjusted for covariables such as sex because their interaction with time-effect were not significant (all $P \geq 0.221$) and our sample size was small. All analyses were performed using SPSS 26.0 (IBM Corp., Armonk, NY, USA). The G*Power 3.1.9.2 program was used for power calculation.[17] Among the detections for significant change in self-reported and objective sleep indicators, we obtained a power of at least 81.83% using "Post hoc: Compute achieved power - given α, sample size, and effect size" with an α of 0.05.

Results

Among 45 subjects from a good sleeper control group, 17 subjects were initially enrolled and completed the short-term post-COVID-19 infected follow-up. Among these 17 subjects, 2 subjects were excluded because one refused to do the long-term follow-up visit, and the other had a viral meningitis infection, that was not associated with 2019-nCoV/SARS-CoV-2, between the short- and long-term post-COVID-19 infection periods. Ultimately, our study included 15 subjects with a mean age of 25.33 ± 3.06 years and a mean BMI of 20.45 ± 2.91 kg/m^{2,} and 66.7% were females (Supplementary Table A.1).

Table 1 Self-Reported Sleep Across Different COVID-19 Infection Periods

Item	Pre-COVID-19 Infection	Short-Term Post-COVID-19 Infection	Long-Term Post-COVID-19 Infection	*F*	*Time effect P*	$\eta 2_p$	$P_{0\text{-}1}$ [a]	$P_{0\text{-}2}$ [a]	$P_{1\text{-}2}$ [a]
PSQI scores	3.87±1.46	5.20±1.42	4.33±1.84	9.738	**0.003**	0.600	**0.002**	>0.999	0.397
s-SOL (min)	20.33±10.08	27.67±12.23	23.40±14.61	5.688	**0.017**	0.467	**0.011**	>0.999	>0.999
s-TST (min)	398.00±44.59	378.00±50.46	404.00±59.86	1.989	0.176	0.234	0.196	>0.999	0.866
s-SE (%)	86.22±8.23	80.77±6.05	85.85±8.04	3.891	**0.040**	0.218	**0.019**	>0.999	0.162
ESS scores	5.93±2.87	5.27±4.27	4.20±2.62	1.448	0.253	0.094	>0.999	0.597	>0.999

Notes: Data are presented as mean ± standard deviation. *F*-values, *P*-values and $\eta 2_p$ were derived from univariate repeated-measures analysis. [a]. *P*-values were adjusted for multiple tests with use of the Bonferroni correction. $P_{0\text{-}1}$, *p*-value for pre-COVID-19 infection vs short-term post-COVID-19 infection; $P_{0\text{-}2}$, *p*-value for pre-COVID-19 infection vs long-term post-COVID-19 infection; $P_{1\text{-}2}$, *p*-value for short-term post-COVID-19 infection vs long-term post-COVID-19 infection. All $P \leq 0.05$ are indicated in bold.
Abbreviations: ESS, Epworth Sleepiness Scale; PSQI, Pittsburgh Sleep Quality Index; s-SE, self-reported sleep efficiency; s-SOL, self-reported sleep onset latency; s-TST, self-reported total sleep time.

Changes in Self-Reported Sleep

As shown in Table 1, the total PSQI scores (P=0.003), s-SOL (P=0.017), and s-SE (P=0.040) changed across the pre-COVID-19, short- and long-term post-COVID-19 infection periods. Specifically, the total PSQI scores (Figure 2A, P=0.002) and s-SOL increased (Figure 2B, P=0.011), and s-SE decreased (Figure 2C, P=0.019) for the short-term post-COVID-19 infection period compared to the pre-COVID-19 infection period. However, no significant differences were observed between pre-COVID-19 infection and long-term post-COVID-19 infection periods (all $P \geq 0.597$), or short- and long-term post-COVID-19 infection periods (all $P \geq 0.162$). We did not find any significant changes in s-TST across the different periods (P=0.176).

Changes in Objective Sleep

As shown in Table 2, significant change was observed in objective SOL (o-SOL, P=0.010) from pre-COVID-19 infection to long-term post-COVID-19 infection. Specifically, o-SOL was decreased at the long-term post-COVID-19 infection period compared to the pre-COVID-19 infection (P=0.028) and short-term post-COVID-19 infection periods (P=0.010). No differences were observed regarding other objective sleep parameters across the different periods (all $P \geq 0.155$).

Changes in EEG Power

As shown in Table 3, the relative alpha EEG power during REM sleep changed (P=0.007) across the pre-COVID-19 infection through long-term post-COVID-19 infection periods. However, in the post hoc analyses, no significant difference was observed between each period (all $P \geq 0.167$). No significant changes were observed in other EEG relative power during NREM or REM sleep across all three infection periods (all $P \geq 0.112$).

Discussion

This is the first study to investigate the changes in self-reported and objective sleep across different post-COVID-19 infected periods. Our results suggest that COVID-19 infection has adverse effects on self-reported sleep, but not objective sleep during the short-term post-COVID-19 infection period. In the long term, COVID-19 infection appears to have no effect on either self-reported or objective sleep for good sleepers.

A few studies have reported impaired self-reported sleep during the COVID-19 pandemic,[18] especially for short-term post-COVID-19 infection.[19] In the current study, we found that self-reported sleep quality and s-SE were decreased, and s-SOL was increased during the short-term post-COVID-19 infection period, which is consistent with most previous studies.[19,20] Several factors may contribute to this association. First, physiological stress and immune response associated with viral infections can disrupt sleep homeostasis and lead to self-reported sleep impairment.[21,22] COVID-19 infection may trigger excessive activation of the immune system and induce cytokines within the body, leading to an amplified immune response in brain tissue.[23,24] These consequent events can present as neuropsychiatric symptoms and sleep impairment.[25] Second, the psychological burden of the illness may exacerbate sleep complaints.[26–28] Interestingly, we found that self-reported sleep quality at 6 months post-COVID-19 infection improved to pre-COVID-19 levels,

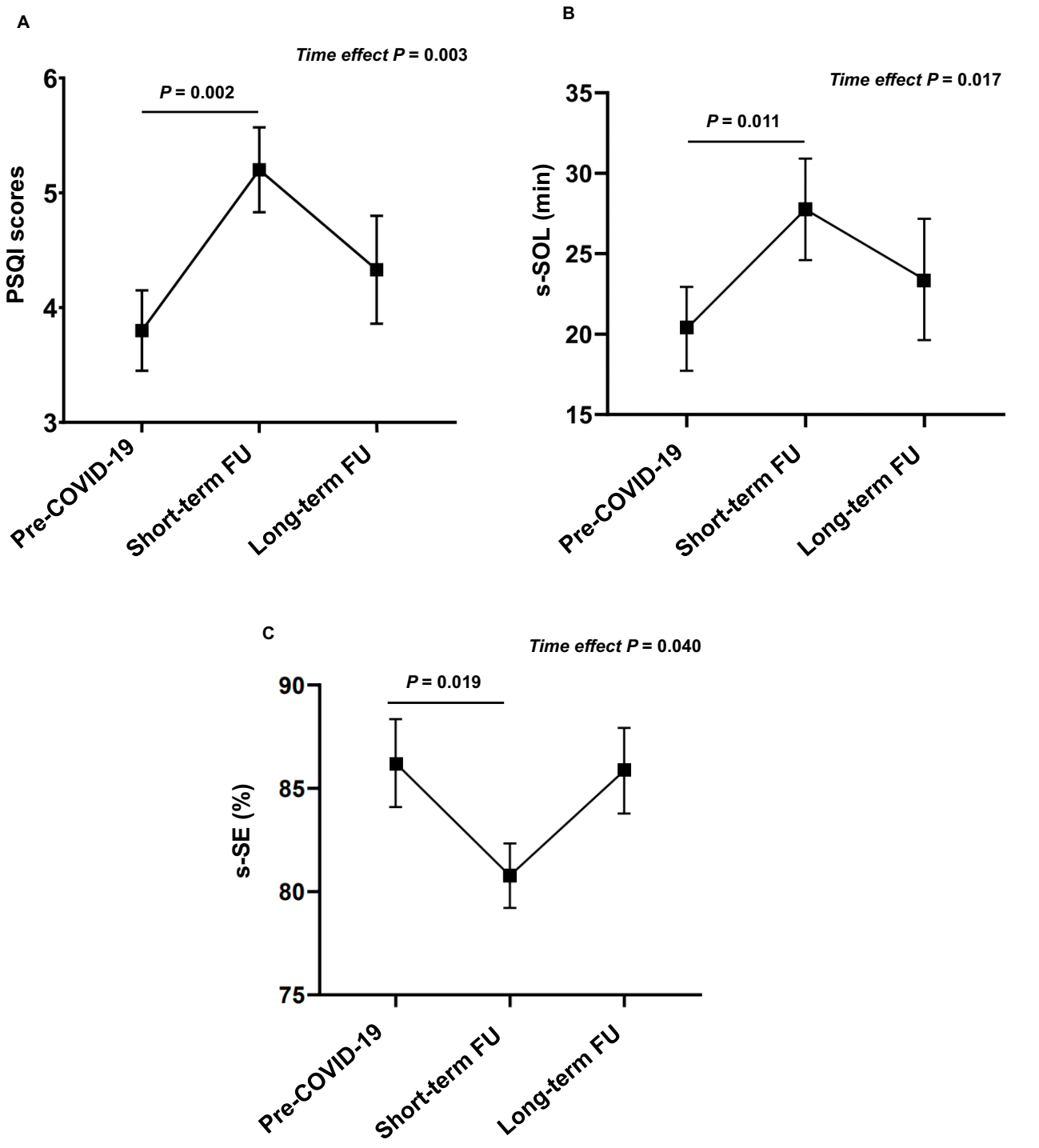

Figure 2 Changes in self-reported sleep. (**A**) Changes of PSQI scores across different COVID-19 infection periods. (**B**) Changes in s-SOL across different COVID-19 infection periods. (**C**) Changes in s-SE across different COVID-19 infection periods.
Notes: *Time effect p*-values are derived from univariate repeated-measures analysis with Bonferroni correction for multiple comparisons.
Abbreviations: FU, follow-up; PSQI, Pittsburgh Sleep Quality Index; s-SE, self-reported sleep efficiency; s-SOL, self-reported sleep onset latency.

suggesting that COVID-19 infection does not have long-term effects on self-reported sleep. Although there is no previous study to examine the long-term effect COVID-19 infection on sleep, our findings are consistent with previous studies in other viral infections, such as influenza.[29,30] The improvement in self-reported sleep may be associated with the resolution of acute physiological and psychological stressors as the body recovers from viral insult.[31] Furthermore, other potential factors may interpret the long-term recovery of sleep following COVID-19 infection, including the

Table 2 Polysomnography Parameters Across Different COVID-19 Infection Periods

Item	Pre-COVID -19 Infection	Short-Term Post-COVID-19 Infection	Long-Term post-COVID-19 Infection	*F*	*Time effect P*	η^2_p	$P_{0\text{-}1}$ [a]	$P_{0\text{-}2}$ [a]	$P_{1\text{-}2}$ [a]
o-SOL (min)	13.85±9.30	18.34±12.51	6.37±3.72	6.407	**0.010**	0.314	0.875	**0.028**	**0.010**
o-TST (min)	432.19±44.98	428.39±30.25	436.05±29.64	0.178	0.832	0.013	>0.999	>0.999	>0.999
o-SE (%)	89.59±7.72	90.49±4.45	93.50±3.60	2.068	0.155	0.129	>0.999	0.257	0.271
WASO (min)	30.23±31.18	26.85±15.81	23.97±17.41	0.256	0.729	0.018	>0.999	>0.999	>0.999
NREM sleep stage 1 (%)	8.44±2.97	8.67±4.36	11.61±7.49	1.933	0.176	0.121	>0.999	0.485	0.425
NREM sleep stage 2 (%)	48.99±9.03	52.15±7.42	48.13±5.41	1.785	0.196	0.113	0.628	>0.999	0.055
NREM sleep stage 3 (%)	21.18±8.17	18.52±5.88	19.87±6.18	0.724	0.481	0.049	0.711	>0.999	>0.999
REM sleep (%)	21.27±4.09	20.61±4.72	20.39±3.57	0.247	0.781	0.017	>0.999	>0.999	>0.999
AHI (events/hour)	2.01±2.02	1.77±1.32	2.14±1.79	0.192	0.814	0.014	>0.999	>0.999	>0.999
PLMI (events/hour)	1.42±1.99	1.27±2.11	2.71±4.43	0.657	0.535	0.092	>0.999	0.897	0.766

Notes: Data are presented as mean ± standard deviation. *F*-values, *P*-values and $\eta 2_p$ were derived from univariate repeated-measures analysis. [a]. *P*-values were adjusted for multiple tests with use of the Bonferroni correction. $P_{0\text{-}1}$, *P* for pre-COVID-19 infection vs short-term post-COVID-19 infection; $P_{0\text{-}2}$, *P* for pre-COVID-19 infection vs long-term post-COVID-19 infection; $P_{1\text{-}2}$, *P* for short-term post-COVID-19 infection vs long-term post-COVID-19 infection. All $P \leq 0.05$ are indicated in bold.
Abbreviations: AHI, apnea-hypopnea index; NREM, non-rapid eye movement; PLMI, periodic limb movement index; o-SE, objective sleep efficiency; o-SOL, objective sleep onset latency; o-TST, objective total sleep time; REM, rapid eye movement; WASO, wake after sleep onset.

Table 3 Indicators of EEG Power Across Different COVID-19 Infection Periods

EEG Power	Pre-COVID-19 Infection	Short-Term Post-COVID-19 Infection	Long-Term Post-COVID-19 Infection	*F*	*Time effect P*	η^2_p	$P_{0\text{-}1}$ [a]	$P_{0\text{-}2}$ [a]	$P_{1\text{-}2}$ [a]
NREM-Delta (%)	76.64±3.24	77.13±3.11	75.66±3.82	0.782	0.458	0.053	>0.999	>0.999	0.543
NREM-Theta (%)	11.34±1.79	11.29±1.35	12.15±1.62	1.333	0.278	0.087	>0.999	0.770	0.170
NREM-Alpha (%)	4.36±1.02	4.46±1.20	4.92±1.52	1.009	0.371	0.067	>0.999	0.741	0.966
NREM-Sigma (%)	5.70±1.90	4.99±1.62	5.10±1.79	0.642	0.502	0.044	0.966	>0.999	>0.999
NREM-Beta (%)	1.92±0.71	2.08±0.81	2.12±0.84	0.380	0.679	0.026	>0.999	>0.999	>0.999
NREM-Gamma (%)	0.05±0.03	0.05±0.02	0.05±0.02	0.210	0.786	0.015	>0.999	>0.999	>0.999
REM-Delta (%)	65.13±6.09	68.30±4.72	66.68±5.15	2.353	0.126	0.144	0.180	>0.999	0.452
REM-Theta (%)	18.78±4.27	17.14±3.50	17.64±3.33	0.843	0.422	0.057	0.641	>0.999	>0.999
REM-Alpha (%)	6.47±1.46	5.98±1.06	6.61±2.04	7.552	**0.007**	0.537	0.803	>0.999	0.167
REM-Sigma (%)	3.95±0.95	3.79±1.29	4.03±1.38	0.166	0.836	0.012	>0.999	>0.999	>0.999
REM-Beta (%)	5.51±2.05	4.67±1.88	4.91±2.23	1.413	0.260	0.092	0.433	0.799	>0.999
REM-Gamma (%)	0.17±0.10	0.12±0.07	0.13±0.09	2.488	0.112	0.151	0.207	0.552	>0.999

Notes: Data are presented as mean ± standard deviation. *F*-values, *P*-values and η^2_p were derived from univariate repeated-measures analysis. [a]. *P*-values were adjusted for multiple tests with use of the Bonferroni correction. $P_{0\text{-}1}$, *P* for pre-COVID-19 infection vs short-term post-COVID-19 infection; $P_{0\text{-}2}$, *P* for pre-COVID-19 infection vs long-term post-COVID-19 infection; $P_{1\text{-}2}$, *P* for short-term post-COVID-19 infection vs long-term post-COVID-19 infected period. Delta: 0.5–4.0 hz; theta: 4.5–8.0 hz; alpha: 8.5–11.0 hz; sigma: 11.5–15.0 hz; beta: 15.5–30.0 hz. All $P \leq 0.05$ are indicated in bold.
Abbreviations: EEG, electroencephalogram; NREM, non-rapid eye movement; REM, rapid eye movement.

rebound of sleep homeostasis and adaptation of emotional responses.[32,33] In a study on sleep vulnerability, it was observed that individuals whose sleep was susceptible to stress demonstrated a compensatory mechanism that prevented acute sleep disturbances from developing into chronic issues.[34] This finding contributes to the understanding of the stress-compensation process.[34] In our study, this process can be used to explain changes in self-reported sleep as a stressor in the context of COVID-19.

Regarding objective sleep, this is the first PSG-based longitudinal study to examine the short- and long-term changes after COVID-19 infection. In our study, despite the adverse short-term impact of COVID-19 infection on self-reported sleep, most indicators of PSG and EEG relative power during sleep were not affected, suggesting that good sleepers had good objective sleep stability in response to the COVID-19 infection. EEG power indicators have been noted to be biomarkers for blood-brain barrier (BBB) leakage.[35] As evidenced by our results showing no changes in EEG relative power after COVID-19 infection, the BBB may not be affected in good sleepers due to their stable objective sleep. Furthermore, shortened o-SOL was observed during the long-term post-COVID-19 infection period compared to pre-COVID-19. However, it could not be associated with excessive sleepiness, since their self-reported daytime sleepiness

did not change, as measured by ESS. In addition, we found that objective parameters for sleep-related breathing disorder (ie, AHI) or periodic limb movement disorder (ie, periodic limb movement index) did not change across different pre- and post-infection periods, suggesting that COVID-19 infection may not affect sleep-related breathing or periodic limb movement disorders.

Previous studies with objective sleep assessment to examine the impact of COVID-19 on sleep were cross-sectional studies. Rouen et al[36] found no significant difference in objective sleep between COVID-19 patients with insomnia and those with chronic insomnia without COVID-19 infection, but cross-sectional studies limited the causal inferences about the effect of COVID-19 infection on sleep. Although altered sleep architecture was found in long COVID-19 patients compared with non-COVID-19 patients in another study,[37] that study lacked EEG-related information and had no baseline data. Findings of these cross-sectional studies showed that patients during COVID-19 infection or with long COVID-19 had shorter TST[36] and deep sleep duration,[37] as well as increased awakenings.[38] The inconsistent findings with our study could be interpreted by inclusion of different study subjects (ie, subjects with COVID-19 infection vs post-COVID-19 infection status).

The discrepancy between self-reported and objective sleep has been observed in various clinical populations, particularly patients with insomnia.[39,40] Our discrepancy between self-reported and objective sleep may be interpreted as paradoxical insomnia during the short-term post-COVID-19 infection period, where the proportion of subjects with PSQI scores >5 was increased from 13.33% to 26.67%.

The current study has several strengths. This is the first study examining both self-reported and objective sleep across the pre-COVID-19 infection, and short- and long-term post-COVID-19 infection periods. The EEG relative power analysis in addition to PSG collectively suggest that COVID-19 infection does not affect objective sleep of good sleepers. Some limitations also need to be acknowledged. First, the sample size was relatively small. However, the power calculation showed good statistical significance with at least 81.83% power to detect changes in the 15 subjects. Second, due to the restricted access of COVID-19 positive patients to sleep laboratories during the pandemic and potential ethical issues, no data were obtained in the acute phase of COVID-19 infection. However, considering the long-term effects of COVID-19 infection, the results of this study provide a reference for future research. Third, our subjects were predominantly young female. Although the interaction effect of gender and time-effect was not significant, our results should be interpreted cautiously in those with older age or medical conditions. Future studies including larger populations with different medical conditions are needed.

Conclusions

In conclusion, COVID-19 infection may have adverse impacts on self-reported sleep but not objective sleep during the short-term period. Over an extended duration, no observable influence of COVID-19 infection was detected on either self-reported sleep or objective sleep.

Abbreviations

AHI, apnea-hypopnea index; BBB, blood-brain barrier; BMI, body mass index; EEG, electroencephalogram; ESS, Epworth Sleepiness Scale; FFT, fast Fourier transform; NREM, non-rapid eye movement; PSG, polysomnography; PSQI, Pittsburgh Sleep Quality Index; REM, rapid eye movement; SE, sleep efficiency; SOL, sleep onset latency; TST, total sleep time.

Data Sharing Statement

Data and protocols are available upon reasonable request from the corresponding author.

Ethics Approval and Informed Consent

This study was approved by the Research Ethics Board of the Mental Health Center of Shantou University (Approval No. 202303) and informed consent was obtained from each participant. This study complied with the Declaration of Helsinki.

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Author Contributions

Jun Wu: Methodology, Writing- Original draft preparation, Formal analysis, Visualization, Investigation; **Baixin Chen**: Formal analysis, Project administration, Writing - Review & Editing; **Qingsong Qin**: Visualization, Investigation, Writing - Review & Editing; **Yanyuan Dai**: Data Curation, Visualization, Writing - Review & Editing; **Le Chen**: Data Curation, Visualization, Writing - Review & Editing; **Dandan Zheng**: Data Curation, Visualization, Writing - Review & Editing; **Jiansheng Zhang**: Data Curation, Visualization, Writing - Review & Editing; **Yun Li**: Conceptualization, Writing - Review & Editing, Supervision. All authors contributed significantly to the work reported whether in conception, study design, execution, data acquisition, analysis and interpretation, or in all of these areas. All authors contributed to drafting or writing, or substantively revised or critically reviewed the article. All the authors have reached consensus on the article will be submitted to the journal. All authors agree to review all versions of the article prior to submission, during revision, acceptance of the final version for publication, and any significant changes introduced during the proofreading phase, and to be responsible for the content of the article.

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Disclosure

All authors report no potential conflicts of interest for this work.

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