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Original Research

Respiratory function and sleep parameters in adults following recovery from acute COVID-19

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ABSTRACT

The impact of COVID-19 on lung function and sleep in otherwise healthy individuals has been subject to a limited number of studies. The aim of this study was to investigate the effect of COVID-19 on pulmonary function and sleep in adults. Participants, 50–85 years old, who had recovered from COVID-19 (COVID-19 group: n = 48) and those without history of COVID-19 (control group: n = 28) underwent pulmonary function assessment (Forced Vital Capacity, FVC, and Slow Vital Capacity, SVC) using spirometry. Sleep and circadian variables were measured objectively with wrist-worn actigraphy for seven days. Subjective sleep of participants was assessed using the Pittsburgh Sleep Quality Index (PSQI). There were no significant differences in age (60 ± 6 vs 62 ± 6 years), BMI (26.30 ± 4.25 vs 26.48 ± 3.60 kg/m^2), or pulmonary function (FVC, 4.02 ± 1.04 vs 3.80 ± 0.98 L, p = 0.36; and SVC, 3.82 ± 1.09 vs 3.89 ± 0.92 L, p = 0.76) between COVID-19 and control groups. The COVID-19 group had significantly reduced sleep efficiency (0.87 ± 0.04 vs 0.91 ± 0.04, p < 0.01), increased sleep disturbance (awakenings, 1.70 ± 1.02 vs 1.15 ± 1.15, p < 0.01; and wakefulness after sleep onset, 35:05 ± 25:37 vs 20:02 ± 12:48 min, p = 0.01) and PSQI score (5.19 ± 2.88 vs 3.93 ± 2.89, p = 0.01), compared to the control group. Individuals with history of COVID-19 demonstrate reduced sleep quality compared to a non-COVID-19 control group.

1. Introduction

Coronavirus disease 2019 (COVID-19) is a severe acute respiratory syndrome with common symptoms including cough, fever, fatigue, dyspnoea and myalgia [1,2]. Although the respiratory system is predominantly affected [1], neurological complications have been reported [3].

Many COVID-19 patients have experienced long-term altered sleep, with approximately one third diagnosed with a sleep disorder after hospital discharge [1,3–6]. Experiences vary, from excessive sleeping to chronic insomnia [3,7,8], with sleep disturbances, or night-time awakenings, commonly reported by COVID-19 survivors, of which 30–50 % experience them within the first year of infection onset [8]. Poor sleep, particularly altered sleep duration [9], sleep architecture [10], and sleep quality [11–13] has been reported up to 6 months post-infection particularly in cases of critical infection requiring hospitalisation or in those with post-COVID-19 condition (PCC; or long COVID). COVID-19 and sleep have a cyclic relationship: COVID-19 infection leads to poor sleep [6,14], and poor sleep leads to immune dysfunction [3], therefore the patient is more likely to be reinfected [15].

Both the respiratory and cardiovascular systems have a significant

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impact on sleep architecture and quality [10]. Pulmonary function and sleep are closely related, for example a lower respiratory rate (RR) is associated with a lower total sleep time (TST) and less deep sleep [10]. Obstructive sleep apnoea has an established link with more severe illness in COVID-19 patients [4,14,15]. COVID-19, as a primarily respiratory tract infection [5], may have impacted sleep quality indirectly through worsened respiratory health.

The aims of this study were to investigate the long-term effects of COVID-19 on pulmonary function and its relationship with sleep quality. We hypothesised that post-COVID-19 reduction in pulmonary function will be significantly associated with changes in objective (actigraphy-derived) and subjective (questionnaire-based) sleep parameters. We thus assessed pulmonary function through spirometry tests and sleep measures using actigraphy, sleep diaries and questionnaires in adults who had recovered from COVID-19 and those without a history of COVID-19. The combination of subjective and actigraphy-derived measures of sleep allowed us to gain insights into how participants assessed their own sleep which can be subject to age-, gender-, cultural- and recall-biases, compared to actigraphy which measures activity levels over several days permitting assessment of sleep (sleep onset latency, nocturnal arousals, awakenings, and total sleep time) as well as the rest-activity rhythm, non-invasively. Actigraphy has been validated against the best technique to measure sleep, i.e., polysomnography (PSG), in several populations [16–18]. While previous studies have primarily focused on the acute impact of critical COVID-19 on sleep, this study is among the first to examine the relationship between post-mild-to moderate-COVID-19 recovery pulmonary function and sleep health using both objective and subjective measurements. This study aims to bridge this gap by integrating both objective (actigraphy) and subjective (sleep questionnaires) measurements, providing a more comprehensive understanding of post-COVID-19 sleep disturbances.

2. Results

Participants without actigraphy recordings for a minimum of three weekday nights and one weekend night, were excluded from the analyses. Valid data were thus available for n = 48 COVID-19 and n = 28 Control participants.

2.1. Demographics

Demographic data for all participants are presented in Table 1. There was no significant difference in age, sex, BMI or MAP between the COVID-19 and control groups.

Table 1
Demographics of COVID-19 group, control group, and differences between groups. Data are expressed as mean ± standard deviation unless otherwise stated.

	Mean ± Standard Deviation		*p*-value
	COVID-19	Control	
Age (years)	59.5 ± 6.1	62.1 ± 6.0	0.08[a]
Sex, female (n, %)	28 (58.3 %)	18 (64.3 %)	0.61[c]
Height (cm)	169.7 ± 9.1	167.1 ± 8.9	0.21[b]
Weight (kg)	75.5 ± 14.5	74.3 ± 13.4	0.75[b]
BMI (kg/m^2)	26.3 ± 4.2	26.5 ± 3.6	0.88[b]
MAP (mmHg)	96.5 ± 12.4	100.4 ± 9.8	0.16[a]
Systolic pressure (mmHg)	134.6 ± 15.7	128.7 ± 16.5	0.22[b]
Diastolic pressure (mmHg)	83.0 ± 11.5	80.4 ± 8.3	0.12[b]
Time since infection (days)	216 ± 134	N/A	N/A

BMI, Body Mass Index; MAP, Mean Arterial Pressure.
[a] Welch's T-test.
[b] Mann-Whitney *U* test.
[c] Chi-square test.

2.2. Pulmonary function

All spirometry measurements except ERV exceeded predicted values for both cohorts (Table 2). The COVID-19 group had had a significantly lower SVC% than the control group (p = 0.01), but there were no other significant differences (Table 2).

2.3. Fatigue and mental health

There was no significant difference between control and COVID-19 results for the CFQ or any DASS-21 variables (depression, anxiety, stress) (Table 2). While anxiety was greater in male COVID-19 participants (Female 0.07 ± 0.38; Male 0.50 ± 1.00, p = 0.01) there was no difference in the dimension of anxiety (i.e. both were in the 'normal' range) between males and females. No correlations with demographics

Table 2
Sleep, circadian rhythm, questionnaire, and pulmonary function outcomes for COVID-19 and control groups, and differences between groups. Data are expressed as mean ± standard deviation unless otherwise stated. Significant results are in bold.

	Mean ± Standard Deviation		*p*-value
	COVID-19	Control	
Pulmonary Function			
FVC (L)	4.0 ± 1.0	3.8 ± 1.0	0.36[a]
FVC (%)	107.2 ± 17.8	112.1 ± 12.1	0.16[a]
FEV_1 (L)	3.1 ± 0.8	2.9 ± 0.7	0.29[a]
FEV_1 (%)	104.9 ± 16.5	107.1 ± 12.9	0.51[a]
FEV_1/FVC	77.5 ± 6.0	75.9 ± 7.7	0.36[a]
SVC (L)	3.8 ± 1.1	3.9 ± 0.9	0.76[a]
SVC (%)	103.5 ± 19.5	138.6 ± 13.0	**0.02**[b]
IC (L)	3.0 ± 0.9	3.0 ± 0.7	0.55[b]
IC (%)	114.0 ± 23.2	118.9 ± 19.8	0.71[a]
ERV (L)	0.8 ± 0.5	0.9 ± 0.5	0.52[b]
ERV (%)	62.7 ± 35.7	73.4 ± 34.7	0.14[b]
Questionnaires			
CFQ	12.9 ± 3.9	12.1 ± 2.6	0.49[b]
Depression	0.2 ± 0.7	0.2 ± 0.6	0.69[b]
Anxiety	0.3 ± 0.7	0.3 ± 0.8	0.67[b]
Stress	0.1 ± 0.5	0.1 ± 0.3	0.60[b]
Global PSQI	5.2 ± 2.9	3.9 ± 2.9	**0.01**[b]
Actigraphy-derived Outcomes			
Sleep variables			
Bedtime (BT)	22:55 ± 01:31	22:51 ± 00:51	0.61[b]
Wake-up time (WU)	07:25 ± 01:00	07:20 ± 01:05	0.75[a]
Time in bed (TIB)	08:30 ± 01:20	08:29 ± 01:05	0.97[b]
Total sleep time (TST)	07:22 ± 01:08	07:38 ± 00:52	0.20[b]
Sleep onset latency (SOL, mins)	16:14 ± 09:52	15:06 ± 10:23	0.20[b]
Sleep efficiency (%, SE)	86.86 ± 4.39	90.51 ± 3.88	**<0.01**[b]
Awakenings frequency	1.70 ± 1.02	1.15 ± 1.15	**<0.01**[a]
Wake after sleep onset (WASO, min)	35:05 ± 25:37	20:02 ± 12:48	**0.01**[b]
Cosinor functions			
Cosinor amplitude	2572.8 ± 950.9	2447.0 ± 608.8	0.49[a]
Acrophase	14:05 ± 01:24	13:40 ± 01:18	0.41[b]
Non-parametric functions			
Interdaily stability (IS)	0.5 ± 0.1	0.6 ± 0.1	0.61[a]
M10	4897.2 ± 1591.7	4729.0 ± 1617.4	0.93[b]
M10 onset	08:38 ± 02:01	08:37 ± 00:56	0.96[b]
L5	220.2 ± 191.5	245.8 ± 159.5	0.31[b]
L5 onset	01:56 ± 01:19	02:11 ± 01:08	0.34[b]
Relative amplitude (RA)	0.91 ± 0.07	0.93 ± 0.02	0.08[a]

Abbreviations: BT, bed-time; CQI, Chalder Fatigue Scale; ERV, expiratory reserve volume; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; IC, inspiratory capacity; IS, intradaily stability; IV, intradaily variability; L5, least active hours; M10; ten most active hours; PSQI, Pittsburgh sleep quality index; SE, sleep efficiency; SOL, sleep onset latency; SVC, slow vital capacity; TIB, time in bed; TST, total sleep time; WASO, wake after sleep onset; WU, wake-up time.
[a] Welch's T-test.
[b] Mann-Whitney *U* test.

were observed for the control cohort.

2.4. Sleep: actigraphy-derived and subjective measures

Actigraphy derived bedtime (BT) and wake-up time (WU) are reported as time on a 24hr clock (hh:mm, Table 2). There was no significant difference in sleep schedule between the COVID-19 and control groups as their main sleep periods fell at similar times and lasted similar durations. Sleep efficiency (SE) was significantly lower in the COVID-19 than the control group ($p < 0.01$), with the control cohort having significantly less disturbed sleep, with fewer awakenings ($p = 0.01$; Fig. 1b; Table 2) and a smaller WASO ($p = 0.01$; Fig. 1c; Table 2). There was no significant difference found in SOL between control and COVID-19 ($p = 0.20$, Fig. 1c; Table 2).

In terms of self-reported outcomes, the global PSQI score was significantly higher for the COVID-19 group compared to control ($p = 0.01$), indicating that the COVID-19 participants ranked their sleep as significantly worse (Table 2). The COVID-19 group reported an average TIB of 08:05, TST of 06:49 and SE of 85 %. On average, the control group had a PSQI TIB of 08:22, TST of 07:21 and SE of 89 %. Between groups, there were no differences in TIB calculated from PSQI responses ($p = 0.35$) or SE ($p = 0.18$), but the COVID-19 group perceived a significantly shorter TST ($p = 0.04$) than the control. Further, within group analysis revealed that COVID-19 participants self-reported significantly longer TIB ($p = 0.05$) and TST ($p = 0.01$) than was recorded via actigraphy. The COVID-19 participants did not perceive their less efficient sleep that was observed using actigraphy, but believed they slept less than the control group (Fig. 2). There was no significant difference between actigraphy measured and perceived sleep statistics for the control cohort (TIB $p = 0.52$; TST $p = 0.14$; SE $p = 0.50$).

2.5. Non-parametric circadian rhythm analysis

Non-parametric circadian rhythm measurements (Table 2) similarly showed no significant differences between the groups. (Fig. 1a; Table 2). Relative amplitude (RA) calculated values of M10 and L5 was also not different between groups.

2.6. Date of assessment

The time of the year may have influenced the sleep of participants. The average date of visit for all participants was September 03, 2022 (SD = 76 days). Control participants mean visit date was significantly earlier than COVID-19 participants' (July 11, 2022, SD = 63 days *vs.* October 04, 2022, SD = 66 days $p < 0.01$). Overall, a later visit date was associated with more disturbed sleep for the COVID-19 participants, and less disturbed sleep for the control participants (Fig. 1a and b Relationships between sleep outcomes and visit date). WASO and awakenings were negatively correlated with time of year of participation in the control cohort (WASO $p < 0.01$; awakenings $p < 0.01$), but positively in the COVID-19 cohort (WASO $p < 0.01$; awakenings $p < 0.01$) (Fig. 3a and b). To assess whether time of year was responsible for the previously described differences in sleep quality between the groups, a range from the median of the control group (June 30, 2022) to the median of the COVID-19 group (October 19, 2022) was calculated (Fig. 3c), and only data from participants who visited between those dates were examined (COVID-19 n = 19 and Control n = 13). Within this range, visit date was not significantly different between the control and COVID-19 groups ($p = 0.16$). Within this subset both WASO and awakenings were significantly reduced in the control group compared to the COVID-19 group ($p = 0.01$ and $p < 0.01$, respectively) (Fig. 3d and e).

2.7. Correlations

A greater SE was associated with shorter SOL in the control group ($p < 0.01$) but not the COVID-19 group ($p = 0.52$) (Table 3).

Table 3 indicates sleep variable correlations. COVID-19 WU times were correlated with TIB ($p < 0.01$), TST ($p < 0.01$), M10 ($p = 0.01$) and cosinor amplitude ($p = 0.01$). COVID-19 BT was correlated with TIB ($p = 0.05$). Control WU time was positively correlated with TIB ($p < 0.01$), TST ($p < 0.01$), WASO ($p < 0.01$) and awakening frequency ($p = 0.03$), but negatively correlated with SE ($p = 0.04$). Control BT was correlated with M10 amplitude ($p = 0.03$). Awakenings positively correlated with BMI in control cohort ($p < 0.01$) but not COVID-19 ($p = 0.69$).

There were no significant correlations of spirometry (e.g. FVC, FVC %, FEV_1, FEV_1 %, FEV_1/FVC, SVC, SVC %, IC, IC %, ER, ERV %) with sleep statistics (e.g. BT, WU, TIB, TST, SOL, SE, WASO, Awake time) (Table 4).

3. Discussion

The present study demonstrated through use of actigraphy and subjective self-report measures, that a history of COVID-19 infection is associated with more interrupted and less efficient sleep but not reduced duration of sleep. Although the COVID-19 group did not take longer to fall asleep (SOL), and the two cohorts were in bed and asleep for similar durations (TIB and TST), the COVID-19 sleep efficiency (SE) was significantly lower than the control group. This was likely caused by the participants awakening more frequently and for longer (WASO), which potentially, contributed to the significantly worse global PSQI scores. That the COVID-19 participants believed they slept longer than their actigraphy-derived data indicated suggests a potential alteration in sleep perception. This has been noted in a study that examined sleep in non-hospitalised and hospitalised patients with post-COVID condition [19]. They noticed that TST were similar according to actigraphy and PSQI,

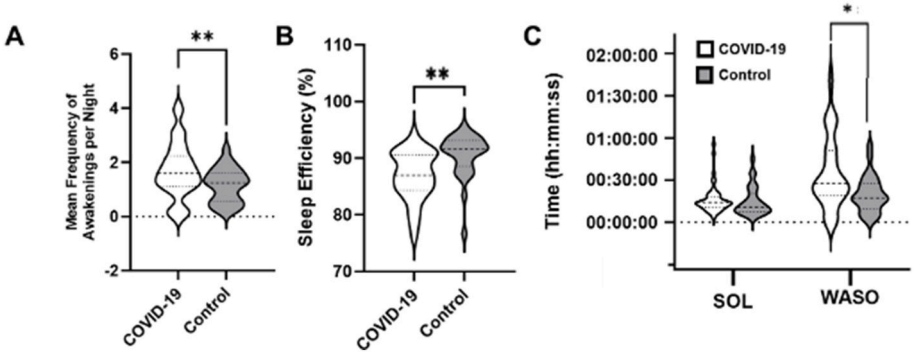

Fig. 1. Sleep quality mean values for each cohort. **A:** Night-time awakenings shown as mean frequency of awakenings in a night. **B: Sleep efficiency (SE) was calculated as the percentage of TIB spent asleep (TST/TIB x 100). C:** Sleep onset latency (SOL), and wake after sleep onset (WASO), measured in duration (hh:mm). *significant at the 0.05 level, **significant at the 0.01 level.

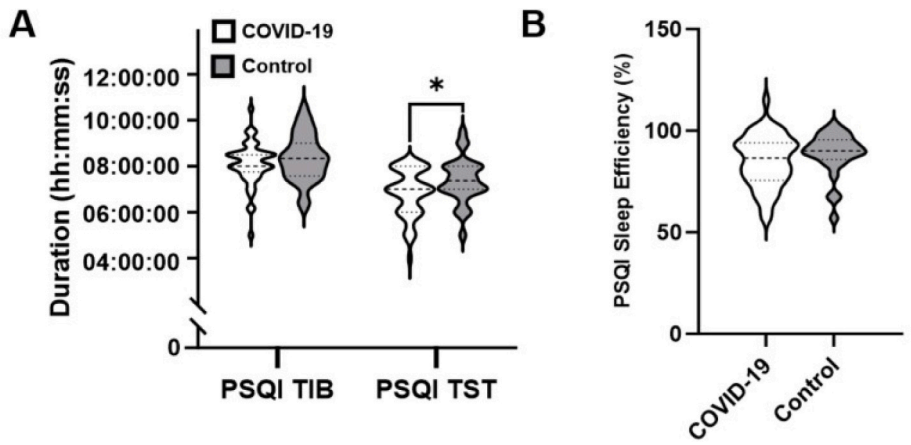

Fig. 2. Perceived sleep duration and efficiency in both the control and COVID-19 cohorts according to the Pittsburgh Sleep Quality Index (PSQI). **A:** Perceived TIB and TST in hh:mm according to PSQI. **B:** Perceived SE calculated from PSQI questions relating to number of hours slept and the number of hours in bed. *significant at the 0.05 level, **significant at the 0.01 level.

but did not find correlations between objective and subjective parameters of latency and sleep efficiency. Future research should explore the mechanisms underlying this discrepancy that exists in post-COVID, with and without PCC.

COVID-19 infection has been previously shown to negatively impact sleep [4,9,10]. Factors linked with sleep, such as cardiovascular health, pulmonary function and mental health, are also affected by COVID-19 [1,3]. However, no significant differences in mental health or pulmonary function were observed in this study. The COVID-19 group had a significantly lower SVC% than the control participants; however, both were within normal range [20], and the COVID-19 group performed better than predicted on average (Table 2). Although no significant respiratory function differences were observed between groups, even mild alterations in pulmonary mechanics may influence sleep quality. Future research could incorporate polysomnography to examine more subtle changes in sleep architecture related to respiratory health. To better understand the relationship between pulmonary function and sleep, additional respiratory measures such as nighttime oxygen saturation and respiratory rate should be considered in future studies.

Prior research has found that COVID-19 infection results in insomnia [7,14,15]. However, in this study we found that TST was unaffected, and that the COVID-19 participants did not struggle to fall asleep more than the control group. Rather, difficulty remaining asleep was observed, as the COVID-19 group demonstrated highly disturbed sleep and lower SE. Insomnia is defined as difficulty falling (onset insomnia) or staying (maintenance insomnia) asleep, despite sufficient opportunity to do so [21]. This study demonstrated that history of COVID-19 is associated with maintenance insomnia, rather than issues with sleep onset.

Sleep quality is affected by the time of year [22], and the control group visited significantly earlier, in the summer, than the COVID-19 autumn visits. Sleep disturbance measures (WASO and awakenings frequency) were significantly different between COVID-19 and control and correlated with visit date positively and negatively respectively (Figs. 1 and 2). However, when equalised to the median date range (Fig. 3), both WASO and awakenings were still significantly better in the control group than the COVID-19 (Fig. 3d and e). Therefore, the sleep disturbances were unlikely to have been caused by the later recording date. There were no significant differences in the timings of the main sleep periods, or most/least active times of day. Sleep schedule is an insufficient explanation for the altered sleep efficiency and more disturbed sleep. The recruitment and testing for this study took place during 2021 when SARS-CoV2 was circulating, and as such did not account for the time of year. Future studies should consider balancing participant recruitment across seasons to minimize potential confounding effects. To better understand the effect of seasonal variation on sleep quality, additional factors such as light exposure duration, ambient temperature, and melatonin levels, should also be considered in future studies.

In this cohort, it is impossible to identify whether COVID-19 or poor sleep came first. Future prospective studies are needed to establish a clear causal relationship. Poor sleep can both contribute to impaired immune function [15] and be caused by it, such as in COVID-19, where higher levels of proinflammatory cytokines can alter the sleep centres in the brain [3,6]. The increased stress during illness can lead to activation of the hypothalamic-pituitary-adrenal (HPA) axis, which contributes to insomnia [6]. Indeed, good sleep health (assessed by chronotype, sleep duration, insomnia, snoring, and daytime dysfunction) prior to SARS-CoV2 infection has been indicated as protective against PCC [23].

The blood brain barrier (BBB) is disrupted in cases of sleep loss and fragmentation, with risk increasing with age [24,25]. As the BBB becomes more permeable, pro-inflammatory molecules and COVID-19 can more easily gain entry to the brain, contributing to neuroinflammation, worse sleep, and COVID-19 reinfection risk [7,8,25].

As poor sleep appears to act as both a risk factor and a symptom of COVID-19, sleep intervention such as improved sleep hygiene, stimulus control therapy or medication [21,26] would be a useful treatment, to ameliorate other sequelae, and to limit the risk of reinfection.

A limitation of this study is that it focussed on adults over 50 years, as they are at higher risk of experiencing sleep problems as a consequence of COVID-19 [7,27], as well as a general increase of sleep disruption with age [28]. The findings may thus be unique to this age group. Another limitation is the imbalance in the sample sizes that arose due insufficient actigraphy recordings (a minimum of three weekday nights and one weekend night, each with 24h of uninterrupted recording) from participants in each group. Although this criterion led to the exclusion of participants and a reduced and imbalanced sample ($n = 48$ from the COVID-19 group and $n = 28$ for the control group), the data analysed were robust and reliable. The sample size imbalance could have affected the study's statistical power, limiting the ability to detect smaller differences. Future studies should aim for more balanced group sizes or apply statistical corrections to mitigate this issue.

This observational study provides insight into the effect of prior COVID-19 infection on sleep but does not identify the cause. Some have theorised that neuro-inflammation, potentially due to BBB leakage, may be responsible [7,8,25]; this could be investigated further by comparing cytokine and inflammation marker levels. Another possible underlying cause may be the stress of infection leading to activation of the HPA axis, causing insomnia [6]. Biomarkers of HPA activation, including adrenocorticotropic hormone, arginine vasopressin and cortisol [29], may be useful to future studies of COVID-19 related sleep disturbance.

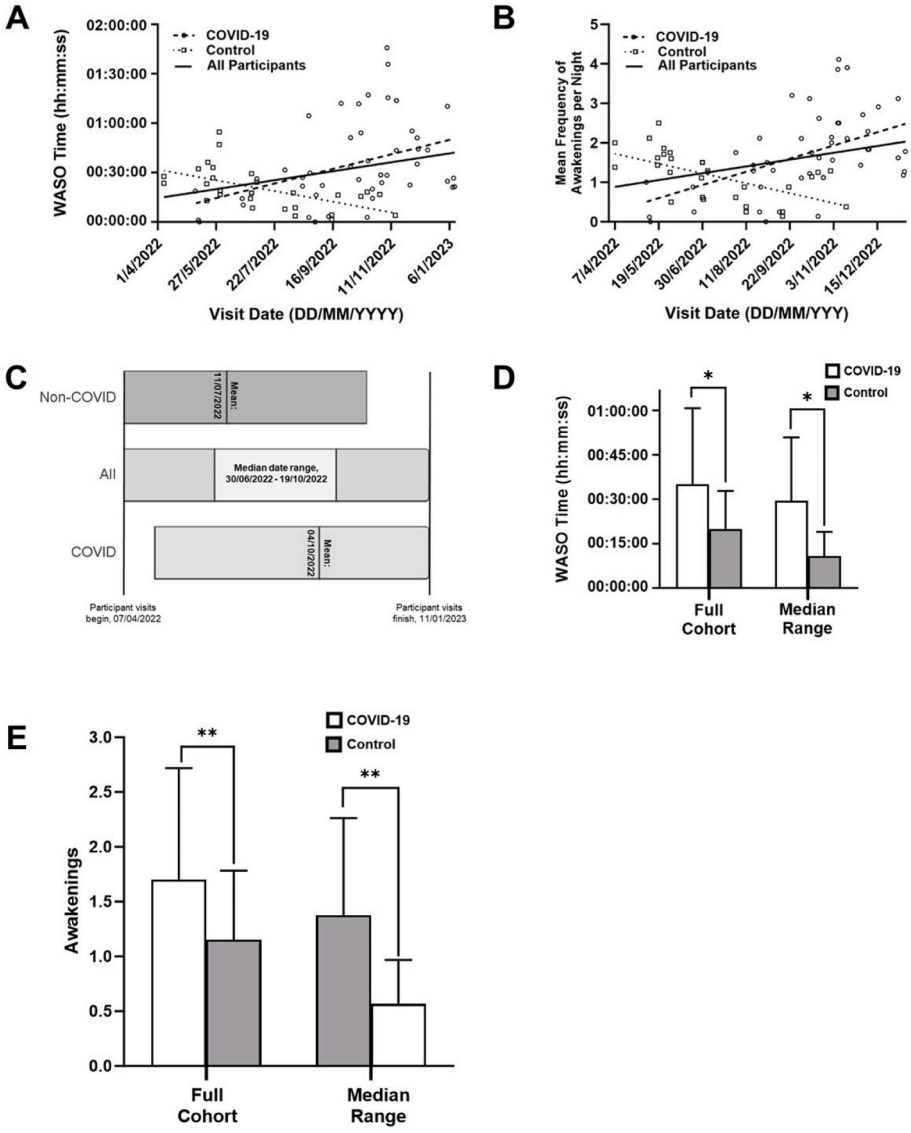

Fig. 3. Relationships between sleep outcomes and visit date A: Correlation between wake after sleep onset (WASO) and date. **B:** Correlations between mean awakenings per night and date. **C:** Timeline of visit dates and determination of the median date range **D:** Sleep disturbance measurements for the full cohort and the date-controlled range for WASO (wake after sleep onset). **E**: Sleep disturbance measurements for the full cohort and the date-controlled range for awakenings. *significant at the 0.05 level, **significant at the 0.01 level.

This study did not differentiate between COVID-19 patients who experienced severe, moderate or minor illness due to the inability to obtain meaningful subjective data on this outcome. Participants were not hospitalised due to acute COVID-19, indicating either mild or moderate acute infection and at the time of testing, all participants reported that they were able to do their pre-COVID-19 usual daily activities, and were therefore considered to have clinically recovered from COVID-19. Prior research has found a link between sleep quality and disease severity [3,4,14,15]; therefore, it would be useful to investigate this further. Furthermore, we did not have a sufficiently large sample size to carry out analyses of COVID-19 participants according to the period since recovery. This may have provided insights into the duration of the impact on sleep of COVID-19 infection.

We found that a history of COVID-19 infection is associated poor sleep quality but not reduced total sleep duration. External zeitgebers such as seasonal light differences or personal sleep schedules, or internal factors such as mental or respiratory health, were not significantly different between the two groups. Sleep disturbance is likely associated with COVID-19 infection in a cyclic relationship, where they each contribute to the occurrence of the other. Therefore, sleep intervention treatment may not only help the symptoms of more frequent awakenings but may reduce the risk of reinfection. It is important to consider sleep health both as a sequela and risk factor for COVID-19 and must be closely monitored and strategies must be developed for better sleep quality to avoid future sleep health related complications.

4. Methods

This study integrates both subjective (PSQI, DASS-21, Chalder Fatigue Scale) and objective (actigraphy, spirometry) measurements to assess the impact of COVID-19 on respiratory function and sleep. Previous research has typically focused on either self-reported surveys or physiological assessments alone. By combining these methodologies, this study offers a more comprehensive understanding of the long-term physiological and psychological effects of COVID-19.

Table 3
Sleep variable correlations for both COVID-19 (n=48) and control (n=28) groups. Significant results are in bold. All values from Spearman correlation tests within groups.

		BT	WU	TIB	TST	SOL	SE	WASO	Awake	M10
COVID-19										
WU	**r**	−0.09								
	p	0.56								
TIB	**r**	−0.29	0.41							
	p	**0.05**	**<0.01**							
TST	**r**	−0.19	0.46	0.91						
	p	0.19	**<0.01**	**<0.01**						
SOL	**r**	−0.11	0.19	0.32	0.27					
	p	0.48	0.21	**0.03**	0.07					
SE	**r**	0.24	0.09	−0.09	0.27	−0.10				
	p	0.10	0.55	0.53	0.06	0.52				
WASO	**r**	−0.21	0.08	0.38	0.09	−0.18	−0.75			
	p	0.15	0.58	**0.01**	0.56	0.22	**<0.01**			
Awake	**r**	−0.19	0.07	0.36	0.11	−0.22	−0.60	0.89		
	p	0.20	0.65	**0.01**	0.46	0.13	**<0.01**	**<0.01**		
M10	**r**	−0.10	0.37	0.01	0.05	0.09	0.10	−0.07	−0.01	
	p	0.51	**0.01**	0.96	0.76	0.56	0.50	0.64	0.95	
Cosinor amplitude	**r**	−0.04	0.36	−0.04	−0.01	−0.02	0.08	−0.08	−0.02	0.93
	p	0.79	**0.01**	0.80	0.98	0.89	0.59	0.61	0.89	**<0.01**
Control										
WU	**r**	0.18								
	p	0.36								
TIB	**r**	−0.17	0.64							
	p	0.38	**<0.01**							
TST	**r**	−0.24	0.55	0.91						
	p	0.22	**<0.01**	**<0.01**						
SOL	**r**	−0.07	0.18	0.45	0.23					
	p	0.73	0.36	**0.02**	0.24					
SE	**r**	−0.08	−0.40	−0.61	−0.35	−0.67				
	p	0.70	**0.04**	**<0.01**	0.07	**<0.01**				
WASO	**r**	−0.12	0.53	0.68	0.54	0.15	−0.62			
	p	0.53	**<0.01**	**<0.01**	**<0.01**	0.45	**<0.01**			
Awake	**r**	−0.07	0.42	0.47	0.46	−0.17	−0.33	0.83		
	p	0.72	**0.03**	**0.01**	**0.01**	0.39	0.09	**<0.01**		
M10	**r**	0.10	−0.48	−0.38	−0.51	0.12	−0.11	−0.20	−0.27	
	p	0.61	**0.01**	**0.05**	**0.01**	0.53	0.57	0.31	0.16	
Cosinor amplitude	**r**	0.09	−0.37	−0.26	−0.37	0.10	−0.06	−0.12	−0.16	0.88
	p	0.64	0.06	0.18	0.05	0.63	0.76	0.53	0.41	**<0.01**

Abbreviations: Awake, frequency of awakenings; BT, Bed-time; SE, sleep efficiency; SOL, sleep onset latency; TIB, time in bed; TST, total sleep time; WASO, wake after sleep onset; WU, wake-up time.

4.1. Study design and participants

This was a sub-study of a larger study to evaluate the effects of COVID-19 on cardiovascular structure and function [30,31]. Ethical approval for the study was granted by the Coventry University Research Ethics Committee (P125303) and the UK Health Research Authority National Health Service East Midlands – Leicester South Research Ethics Committee (22//0090), and all methods were performed in accordance with the relevant guidelines and regulations. Written, informed consent was obtained from all participants. Adults, aged 50–85 years old and fully vaccinated against COVID-19, were invited to participate in the study between April 2022 and January 2023. The age range of 50–85 y was chosen due to emerging data from Public Health England, primary care networks, and global datasets indicating that patients aged over 50 years were at the highest risk of severe COVID-19 and mortality, and this age group was amongst the first to be vaccinated in the UK [32]. By focusing on this demographic, our study aims to provide a more accurate assessment of long-term effects in a high-risk population. Those who self-reported as having no history of COVID-19 and when testing was not available, did not have symptoms of COVID-19 served as the control group, while those who had received a positive COVID-19 test (PCR or lateral flow) 1–18 months before their visit and had recovered, were assigned to the COVID-19 group. Other inclusion criteria included: aged 50–85 years; COVID-19 vaccinated (i.e. at least two doses of an NHS-approved vaccine). Exclusion criteria included known chronic respiratory or cardiovascular conditions (i.e. COPD, emphysema, pulmonary hypertension, coronary artery disease), severe hypertension (systolic blood pressure >180 mmHg, diastolic blood pressure >120 mmHg), acute or chronic neurological impairment or progressive neurological disease, use of medication known to directly affect cardiac function (i.e. beta-blockers), current smoker, body mass index >35 kg/m^2, previously ventilated during COVID-19 infection and exceeding current physical activity guidelines defined by the World Health Organisation [33]. Participants (COVID-19 group: n = 84; control group: n = 40) attended the lab on a single occasion for clinical assessments [30,31,34] completion of questionnaires and were given an actigraphy device to wear for at least seven days to objectively measure their sleep and activity. Incomplete data were excluded from analysis, and only participants with complete actigraphy data sets were included in the final evaluation. We thus report data from COVID-19 group: n = 48; control group: n = 28. However, no sensitivity analysis was conducted to assess the potential impact of missing data on the results, which may introduce a limitation regarding generalizability. Data from participants who did not return complete actigraphy data sets are not reported on here; Data from COVID-19 participants were those collected at their baseline visit only, prior to being assigned to groups for the randomised controlled trial [30].

4.2. Clinical assessments

Participants undertook a physical examination at the beginning of their visit. Anthropometric measurements were taken, and body mass

Table 4
Spirometry correlations with sleep statistics for both COVID-19 (n=48) and control (n=28) groups. Significant results are in bold. All values from Spearman correlation tests within groups.

		FVC	FVC %	FEV_1	FEV_1 %	FEV_1/FVC	SVC	SVC%	IC	IC %	ERV	ERV %
COVID-19												
BT	**r**	0.01	−0.06	0.02	−0.12	−0.08	0.14	0.06	0.11	0.06	0.12	0.09
	p	0.94	0.71	0.87	0.43	0.58	0.33	0.70	0.46	0.68	0.43	0.53
WU	**r**	0.22	0.08	0.21	0.15	0.07	0.16	0.02	0.18	0.08	0.13	0.10
	p	0.13	0.59	0.15	0.30	0.65	0.26	0.88	0.22	0.60	0.40	0.49
TIB	**r**	−0.06	0.00	−0.04	0.10	0.26	−0.05	0.01	0.04	0.10	−0.04	−0.05
	p	0.67	0.99	0.81	0.49	0.07	0.72	0.96	0.81	0.49	0.80	0.74
TST	**r**	0.02	0.05	0.03	0.11	0.16	0.03	0.04	0.13	0.15	−0.01	−0.04
	p	0.89	0.72	0.87	0.45	0.27	0.85	0.79	0.37	0.32	0.93	0.77
SOL	**r**	0.03	0.07	0.04	0.13	0.17	0.00	0.02	−0.08	−0.03	0.14	0.14
	p	0.83	0.66	0.78	0.37	0.26	0.99	0.92	0.59	0.87	0.34	0.33
SE	**r**	0.15	0.12	0.07	−0.03	−0.27	0.10	0.02	0.11	0.00	0.13	0.09
	p	0.32	0.43	0.62	0.82	0.07	0.51	0.89	0.45	0.99	0.38	0.55
WASO	**r**	−0.27	−0.22	−0.25	−0.14	0.20	−0.17	−0.05	−0.13	0.00	−0.20	−0.13
	p	0.07	0.14	0.09	0.35	0.18	0.24	0.73	0.40	0.98	0.18	0.39
Awake	**r**	−0.23	−0.27	−0.24	−0.25	0.12	−0.20	−0.23	−0.21	−0.21	−0.16	−0.11
	p	0.12	0.06	0.11	0.09	0.42	0.17	0.12	0.16	0.15	0.27	0.44
Control												
BT	**r**	−0.18	−0.04	−0.23	−0.23	−0.22	−0.25	−0.15	−0.13	0.02	−0.16	−0.01
	p	0.36	0.84	0.23	0.25	0.26	0.20	0.44	0.51	0.90	0.43	0.95
WU	**r**	0.17	−0.14	0.16	0.04	−0.04	0.10	−0.20	0.05	0.07	−0.07	−0.09
	p	0.39	0.47	0.43	0.86	0.85	0.62	0.30	0.81	0.74	0.73	0.66
TIB	**r**	0.10	−0.02	0.07	0.11	0.01	0.10	−0.08	0.04	−0.02	0.10	0.03
	p	0.61	0.91	0.72	0.59	0.98	0.63	0.70	0.83	0.92	0.63	0.89
TST	**r**	0.12	−0.13	0.09	−0.04	−0.08	0.14	−0.05	0.13	0.03	0.05	−0.10
	p	0.56	0.51	0.67	0.83	0.68	0.49	0.80	0.50	0.88	0.78	0.62
SOL	**r**	0.12	0.35	0.15	0.28	0.11	0.05	0.16	0.10	0.08	0.13	0.12
	p	0.53	0.07	0.45	0.16	0.58	0.81	0.41	0.62	0.70	0.50	0.53
SE	**r**	−0.09	−0.09	−0.06	−0.21	−0.08	0.00	0.09	0.03	0.06	−0.12	−0.14
	p	0.64	0.67	0.75	0.29	0.69	0.99	0.64	0.87	0.78	0.53	0.47
WASO	**r**	0.12	−0.16	0.14	0.15	0.12	0.08	−0.27	−0.03	−0.11	0.06	0.04
	p	0.55	0.43	0.47	0.46	0.53	0.70	0.16	0.86	0.56	0.77	0.86
Awake	**r**	0.13	−0.22	0.17	−0.01	−0.11	0.15	−0.26	0.04	−0.02	0.10	0.06
	p	0.51	0.25	0.39	0.98	0.59	0.45	0.18	0.85	0.93	0.63	0.76

Abbreviations: Awake, frequency of awakenings; BT, Bed-time; ERV, expiratory reserve volume; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; IC, inspiratory capacity; SE, sleep efficiency; SOL, sleep onset latency; SVC, slow vital capacity; TIB, time in bed; TST, total sleep time; WASO, wake after sleep onset; WU, wake-up time.

index (BMI) determined. Resting blood pressure and 12-lead electrocardiogram (ECG) (CardioExpress Sl18A, Spacelabs Healthcare, Snoqualmie, WA, USA) was performed to ensure inclusion criteria were met.

4.3. Pulmonary function tests

Lung function was evaluated through spirometry tests (Breeze, Ultima Series, Medgraphics, UK) at rest as per American Thoracic Society and European Respiratory Society guidelines [20]. The maximum volume of air exhaled after either a single forced expiration (forced vital capacity, FVC) or a maximal inspiration without forced effort (slow vital capacity, SVC) were recorded. Forced expiratory volume in 1 s (FEV_1), inspiratory capacity (IC) and expiratory reserve volume (ERV) were measured. Each test was repeated three times, and the highest value was accepted. Results were expressed as percentages of the predicted maximum according to participant height, weight and age.

4.4. Questionnaires

Participants were asked to complete a set of questionnaires about their sleep habits, general health, levels of fatigue, and mental health during their visit. The Pittsburgh sleep quality index (PSQI) [35] was used to assess the sleep habits of participants prior to the study. The PSQI consists of 19 items, the scores of which are summed for a global score. A score of >5 is used to denote poor sleep quality [35]. PSQI sleep efficiency was determined from the answers to questions 1, 3, and 4 relating the number of hours slept and the number of hours in bed [35]. Self-reported bedtime, wake-up time, sleep onset latency and total sleep time (TST), and time-in-bed (TIB; calculated as the difference between reported bedtime and wake-up time) were determined from the PSQI. Levels of physical and mental fatigue in participants were assessed using Chalder Fatigue Scale (CFQ), where the Likert scoring system was used [36]. The Depression, Anxiety and Stress Scale (DASS)-21 measures aspects of mental health as felt over the past week [37]. There are 21 items in total with seven items related to each aspect, measured on a four-point scale, ranging from 0 = Did not apply to me at all, to 3 = Applied to me very much, or most of the time. Outcome variables were analysed as continuous variables for depression, anxiety and stress scores, where higher scores denoted greater severity.

4.5. Sleep and circadian rhythm assessment via actigraphy

Participants wore an ActTrust2 actigraphy device (Condor Instruments, Brazil) on their non-dominant wrist continuously for seven days. They were asked to keep a sleep diary recording their bedtime, when they fell asleep, and wake time, for the same period. For a dataset to be included, a minimum of three weekday nights and one weekend night, each with 24h of uninterrupted recording was required. This criterion meant that we are reporting on the data of only n = 48 from the COVID-19 group and n = 28 for the control group.

Data were collected in 60s epochs and included: ambient and skin temperature; environmental light (red, blue, green, infrared, UVA and UVB); and activity. Sleep scores were calculated in ActStudio using the Cole-Kripke algorithm [38]. Actigraphy data were manually reviewed and validated against sleep diaries. Data artefacts were identified and removed based on ambient and skin temperature fluctuations, as well as environmental light data. This process ensured accuracy in the determination of sleep parameters. Outcome variables were average (i.e.

seven-day) bedtime, wake-up time, time-in-bed (TiB, h; duration of actigraphy scored inactivity period), total sleep time (TST, h; duration of sleep between sleep onset and wake time), sleep onset latency (SOL, min; the time taken to fall asleep determined by a threshold of inactivity, comparing activity counts for an epoch and those surrounding it), sleep efficiency (SE, %; the percentage of the period of inactivity that a participant spent asleep), wake after sleep onset (WASO, min; the amount of time spent awake between the onset of sleep and wake up time). ActSudio (Version 1.0.24) was also used for the analyses of circadian rhythm parametric functions: cosinor amplitude (to assess the robustness of the circadian rhythm), and acrophase (the time point in the cycle of highest amplitude); and non-parametric functions: total activity in most active 10-h period (M10), onset of most active 10-h period (M10-onset), total activity in least active 5-h period (L5), onset of least active 5-h period (L5-onset), relative amplitude (M10 minus L5, divided by M10 plus L5) interdaily stability (IS) as a measure of entrainment of the circadian rhythm, intradaily variability (IV) as a measure of rhythm fragmentation [39].

4.6. Data analysis

All data were screened for univariate and multivariate outliers before analyses using IBM SPSS Statistics (Version 27) and excluded if not physiologically viable. Normality was evaluated using Kolmogorov-Smirnov tests. Control and COVID-19 datasets were compared using Welch's t-tests, Mann-Whitney U tests, and Chi-squared tests, as appropriate. Strength of relationship between actigraphy-derived and spirometry variables within groups was assessed by coefficient of correlation (Pearson's or Spearman's as appropriate). Results were deemed significant if $p < 0.05$.

CRediT authorship contribution statement

Olivia Hood: Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Data curation. **Sophie L. Russell:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Mushidur Rahman:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Data curation. **Nduka C. Okwose:** Writing – review & editing, Supervision, Methodology, Investigation, Data curation, Conceptualization. **Djordje G. Jakovljevic:** Writing – review & editing, Validation, Supervision, Methodology, Funding acquisition, Conceptualization. **Laura C. Roden:** Writing – review & editing, Validation, Supervision, Resources, Methodology, Formal analysis, Data curation, Conceptualization.

Data availability

The datasets generated during the current study are available from the corresponding author on reasonable request.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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