

Article

Effects of Dynamic Increases in Indoor CO₂ Concentration on Daytime Sleepiness: An EEG-Based Study

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Abstract

Elevated indoor carbon dioxide (CO₂) concentrations are increasingly recognized as a critical determinant of occupant health and cognitive performance; however, the neuro-physiological mechanisms linking CO₂ exposure to daytime sleepiness remain a subject of debate. This study investigated the effects of dynamically rising CO₂ concentrations on subjective sleepiness and neural oscillation patterns in a controlled environment. A within-subject repeated-measures experiment was conducted with 18 healthy university students exposed to CO₂ levels gradually increasing from a baseline of ~800 ppm to ~2000 ppm over a 40-min period. Subjective sleepiness was assessed using the Karolinska Sleepiness Scale (KSS), while electroencephalogram (EEG), heart rate (HR), and heart rate variability (HRV) were monitored continuously. Results indicate a significant increase in subjective sleepiness scores concurrent with rising CO₂ levels ($p < 0.001$). Power spectral density (PSD) analysis revealed that elevated CO₂ exposure significantly enhanced power in low-frequency EEG bands (δ , θ , and α), indicative of a transition from alertness to drowsiness. Specifically, α -band power peaked between 20 and 30 min (increasing by 0.15 μV^2 relative to the 0–10 min baseline), while θ and δ bands peaked earlier (10–20 min) and sustained elevated levels through 30 min. Topographic mapping identified the central, parietal, and occipital regions as the primary loci of this low-frequency activity. Additionally, HR and HRV measures showed upward trends, suggesting autonomic modulation. It is noted that low-frequency EEG power declined during the final 30–40 min interval, potentially reflecting physiological saturation or acclimatization. These findings provide objective neurophysiological evidence that dynamic CO₂ accumulation accelerates drowsiness onset, underscoring the necessity of optimized ventilation strategies in educational settings. However, these findings should be interpreted with caution due to the small sample size and the absence of a control group with constant CO₂ concentration. Future studies with larger, diverse samples and stable-concentration control groups are recommended to validate these trends.



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1. Introduction

Carbon dioxide (CO₂) is an odorless, tasteless, and colorless gas that influences multiple aspects of human physiology [1,2]. While historically considered primarily a tracer for ventilation efficiency, a growing body of evidence emerging in the post-pandemic era

suggests that CO₂ acts as a significant environmental stressor contributing to human health risks and neurophysiological alterations [3–5]. The average indoor CO₂ concentration usually ranges from 600–1000 ppm, but can exceed 2500 ppm in cases of inadequate ventilation, particularly in densely occupied spaces such as classrooms, meeting rooms, and dormitories [6–8]. Consequently, updating indoor air quality (IAQ) standards has become a global priority, with organizations proposing stricter limits to safeguard occupant well-being. For instance, the General Administration of Quality Supervision, Inspection and Quarantine of China (AQSIQ) recommends an indoor standard of ≤ 1000 ppm (CS GB/T17094-2022) to mitigate potential adverse effects. Even at relatively low concentrations, exposure to CO₂ can lead to a variety of psychological and physiological changes in humans [9–11]. When the CO₂ concentration in the environment reaches 1000 ppm or more, individuals may begin to exhibit symptoms such as sleepiness, heart rate (HR) changes, headache symptoms, and cognitive decline [2,3,8,10,12]. At higher thresholds (e.g., around 3000 ppm), CO₂ exposure has been shown to further dysregulate the autonomic nervous system and exacerbate feelings of fatigue [8]. Elevated indoor CO₂ is increasingly recognized as a critical factor in Sick Building Syndrome (SBS) [8], contributing to neuro-physiological symptoms such as fatigue and lethargy that undermine occupational health. Epidemiological evidence and controlled studies link high CO₂ not only to occupant discomfort but also to significant declines in cognitive function and productivity [4,13]. Therefore, objectively characterizing the onset of CO₂-induced drowsiness is essential for addressing these environmental health risks.

In the past few years, researchers have held differing opinions on whether CO₂ causes cognitive decline [14] and induces daytime sleepiness. A review highlighted that while some experiments demonstrate clear deficits, others show inconsistent results, potentially due to differences in exposure methods and the sensitivity of assessment tools [15]. One possible reason for this discrepancy is that in the existing studies on the effects of CO₂ on human performance, sleepiness is traditionally assessed subjectively by self-report [8,16,17]. For example, Snow et al. [18] found that self-reported sleepiness, as assessed by the Stanford Sleepiness Scale (SSS), increased significantly as CO₂ concentrations rose from 830 ppm to 1270 ppm. Similarly, studies using the Karolinska Sleepiness Scale (KSS) observed a significant increase in subjective sleepiness under restricted ventilation [8,17]. However, subjective metrics are prone to individual bias and may not accurately capture the onset of physiological lethargy or the underlying neural mechanisms. To address these methodological limitations, recent research has increasingly turned to electroencephalography (EEG) as an objective measure of neural arousal and vigilance. A systematic review and meta-analysis of EEG-based studies confirmed that elevated indoor CO₂ is consistently associated with alterations in brain wave patterns, specifically increases in low-frequency power bands [19]. Furthermore, a study employing Bayesian statistical analysis demonstrated that EEG signals are more sensitive to CO₂ exposure than subjective sleepiness scales, capable of detecting physiological changes even when participants subjectively report feeling alert [20]. Although EEG has been used to measure neural arousal at very high CO₂ concentrations [21,22] and can be used in neuroscience to objectively characterize sleepiness [23], EEG has not yet been widely used to assess the effects of actual indoor CO₂ concentrations on human performance. Increased power in low frequency bands, such as δ (1–3 Hz), θ (4–7 Hz), and α (8–13 Hz), during the eyes-open state is a well-established marker of reduced vigilance and the transition from alertness to drowsiness [24]. Recent investigations have further validated these EEG markers in office environments, linking CO₂ accumulation to reduced attention and increased “cortical idling,” which suggests a dose–response relationship between CO₂ concentration and neural resource depletion [25].

However, the majority of previous studies have focused on static CO₂ levels. Few studies have examined the dynamic physiological response as CO₂ concentrations gradually rise, which is the typical scenario in occupied buildings when ventilation systems are inactive or insufficient. The rate of change in environmental parameters may induce different physiological adaptation or stress responses compared to steady-state exposure. Therefore, the present study aimed to examine the effects of dynamically increasing CO₂ concentration on daytime sleepiness and EEG signals in a well-controlled environment. Changes in sleepiness were assessed using KSS, along with objective EEG, HR, and HRV data. The findings of this study may serve as a valuable reference for future research investigating the potential of EEG as an objective measure of sleepiness.

2. Materials and Methods

2.1. Subjects

A total of 18 college students (7 males and 11 females) participated in this experiment. Subjects with a history of sleep disorders (screened via the Pittsburgh Sleep Quality Index), neurological, cardiovascular, respiratory, or other medical conditions were excluded from the study. To ensure physiological baselines were not confounded by external stimulants, all subjects were instructed to refrain from consuming caffeinated beverages, medications (including sleeping aids or melatonin), and alcohol for at least 24 h prior to their experimental session. Subjects were also asked to maintain a regular sleep schedule (7–8 h) for three days prior to the experiment. Each subject's clothing thermal resistance was estimated to be approximately 0.6–0.7 Clo, ensuring thermal neutrality at 25 °C. This study was conducted in accordance with the Declaration of Helsinki, and approved by Ethics Committee of Qingdao University of Technology. The demographic characteristics of the participants are summarized in Table 1.

Table 1. The characteristics of the participants in the experiment.

Demographic Variable	Category/Metric	Value	Range
Gender	Male/Female	7/11	-
Age	Years	21.4 ± 1.8	19–24
Anthropometrics	Height (cm)	168.2 ± 7.5	158–180
	Weight (kg)	61.5 ± 9.8	48–76
	BMI (kg/m ²)	21.7 ± 2.2	18.9–24.1

2.2. Experimental Environment Condition

The experiment was conducted in a controlled environmental chamber (3.0 m × 3.0 m × 2.5 m) located on the seventh floor of a teaching building at Qingdao University of Technology (Figure 1a). A controlled laboratory setting was selected over a field study to maximize internal validity by strictly isolating CO₂ concentration as the independent variable. This approach minimizes confounding factors prevalent in real-world buildings, such as fluctuating occupancy noise, uncontrolled thermal stratification, and external electromagnetic interference.

Under baseline conditions, the initial CO₂ concentration was approximately 500 ppm. During the 40-min experimental phase, windows remained closed, and CO₂ levels were allowed to rise dynamically to approximately 2000 ppm (Figure 1b). This concentration is considerably higher than the standard indoor CO₂ levels typically found in offices and classrooms [26–28]. However, such elevated levels are common in occupied buildings with poor ventilation when doors and windows remain closed [8,17,29–31]. The CO₂ elevation process was divided into 4 time intervals for analysis: 0–10 min, 10–20 min, 20–30 min, and 30–40 min. The average CO₂ concentrations during these intervals were

approximately 800 ppm, 1300 ppm, 1700 ppm, and 2000 ppm, respectively. During the experiment, windows remained closed, and ventilation was provided solely through the air-conditioning system. The experimental environment was strictly controlled to maintain thermally neutral conditions. Room temperature was sustained at $25.0\text{ }^{\circ}\text{C} \pm 0.5\text{ }^{\circ}\text{C}$, with relative humidity maintained at $50\% \pm 5\%$. Air velocity in the occupied zone was kept below 0.1 m/s to minimize draft sensations. Illuminance was maintained at approximately 500 lux on the desk surface. Ultra-pure CO_2 gas (99.99%) was introduced into the sealed room from a liquid CO_2 cylinder until the target concentration was achieved. To maintain steady-state concentrations, the room was briefly ventilated manually if CO_2 levels exceeded the specified thresholds. A multi-parameter environmental monitor (Model JTR04; positioned 1.1 m above the floor and a horizontal distance of 0.5 m from the subject's face, corresponding to the breathing zone) was employed to continuously record temperature, humidity, and air velocity, ensuring environmental consistency across all experimental sessions. Furthermore, the room was acoustically treated with sound-absorbing materials, maintaining a background noise level of approximately 35 dB(A) in the absence of indoor activity.

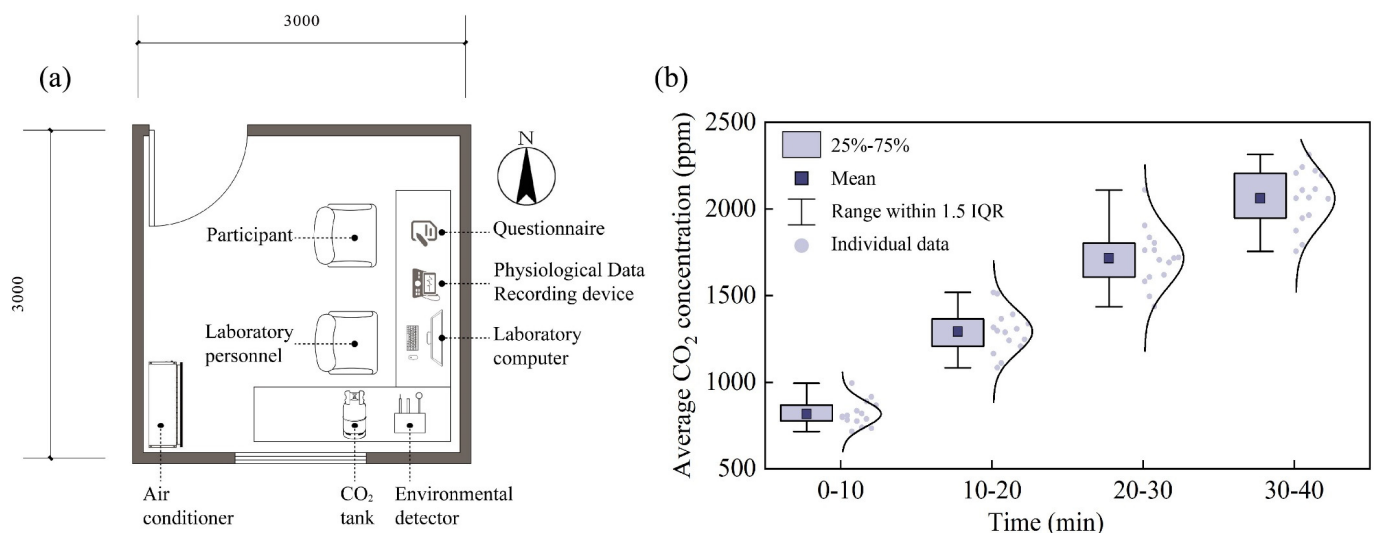


Figure 1. (a) Floor plan of the laboratory; (b) average CO_2 concentration during each time interval.

2.3. Data Collection Equipment and Specifications

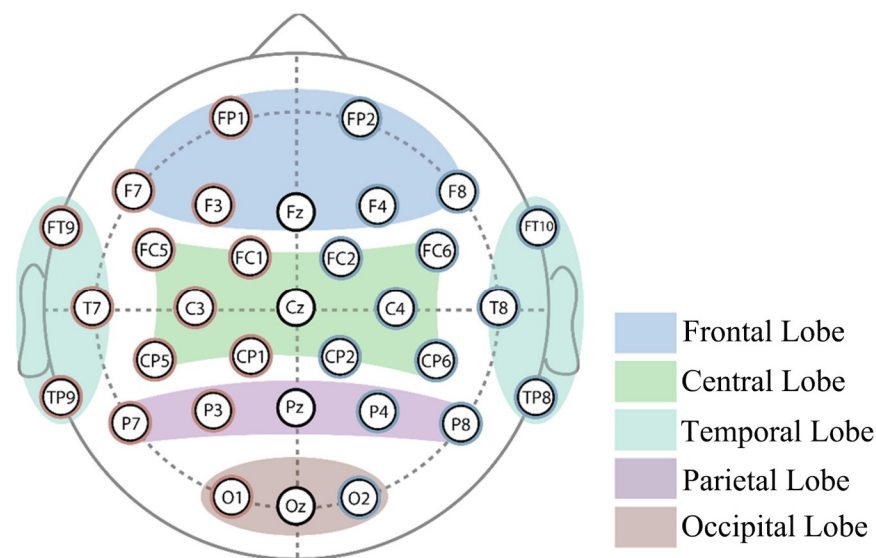
Physiological data collection utilized high-precision, wireless sensing technologies to minimize subject discomfort while maintaining research-grade signal quality. The equipment used in the experiment is listed in Table 2.

Brain activity was recorded using the Emotiv EPOC Flex Gel Sensor Kit (EMOTIV Inc., San Francisco, CA, USA). This is a wireless, 32-channel EEG system. The system has an effective bandwidth of $0.16\text{--}43\text{ Hz}$, which covers the δ , θ , and α bands relevant to sleepiness analysis. Electrode impedance was monitored and maintained below $5\text{ k}\Omega$ using saline-based gel sensors. The 32 electrodes were arranged according to the international 10–20 system, as shown in Figure 2. EEG data were processed using MATLAB R2024a software.

HR and HRV were monitored using a Healink-R211B sensor (HeaLink Ltd., Bengbu, China) with a sampling rate of 1000 Hz . This high temporal resolution is essential for the accurate detection of R-R intervals required for time-domain HRV analysis.

Table 2. Experiment equipment.

Usage	Model	Test Program	Range	Accuracy/ Sampling Rate
Physiological data monitoring	Emotiv EPOC Flex Gel Sensor Kit	EEG	0.16–43 Hz	128 Hz
		HRV	0.6–40 Hz	1000 Hz
	Healink-R211B	HR	Times/min	1
		CO ₂ concentration	0–5000 ppm	±30 ppm
Environmental Monitoring	Environmental detector-JTR04	Air temperature	−20–70 °C	±0.5 °C
		Relative humidity	0–100%	±3%
		Wind speed	0–50 m/s	±0.1 m/s

**Figure 2.** Electroencephalographic channels and brain regions.

2.4. Experimental Process

Figure 3 illustrates the experimental procedure. Prior to the experiment, each subject was allowed 10 min to acclimate and be fitted with the monitoring equipment. During this phase, a brief introduction to the procedure was provided, lasting no more than 5 min. Upon commencement of the experiment, subjects remained seated and performed a reading task for 40 min under the designated environmental conditions. Throughout this period, the CO₂ concentration was gradually increased, and physiological data were continuously recorded. Subjective questionnaires were administered at three specific time points: at baseline (0 min), 20 min into the session, and at the end of the experiment (40 min). To minimize the potential confounding effects of circadian rhythms and daily fluctuations in alertness, strict temporal control strategies were implemented. First, data collection was distributed over 20 days, making single-day completion logistically impossible. Second, all sessions were scheduled during standard working hours (09:00–11:30 and 14:30–17:00). Crucially, the period immediately following lunch (12:00–14:00) was excluded to mitigate the confounding influence of post-prandial somnolence. Measurement slots were randomized across participants to prevent systematic time-of-day bias.

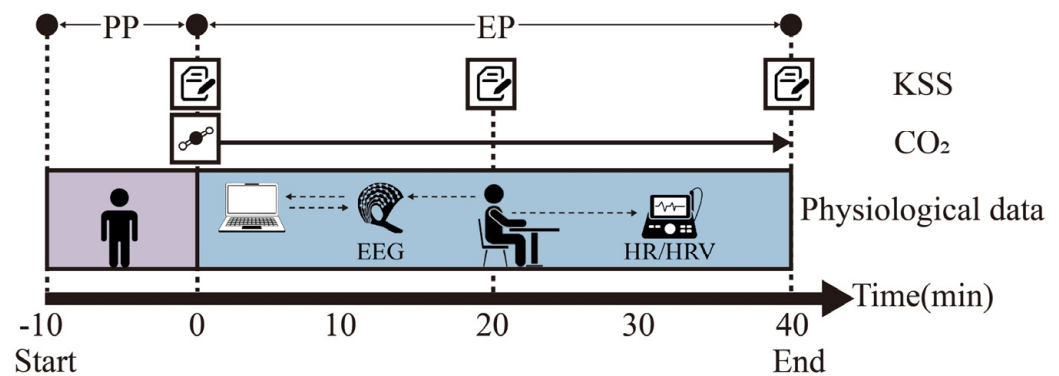


Figure 3. Overview of the experimental procedure. PP: Preparation phase. EP: Experimental phase.

2.5. Psychological and Physiological Indicators

2.5.1. KSS

We used the KSS to assess the degree of sleepiness of the subjects as shown in Table 3. The sleepiness scale was filled out by the subject every 20 min during the elevated CO₂ concentration, in order to minimize interference with the subject's current state and motion artifacts in the EEG signal.

Table 3. KSS.

Name:	Gender:	Age:	
Levels	KSS Definitions		Score
Level 1	Extremely alert		1
Level 2	Very alert		2
Level 3	Alert		3
Level 4	Rather alert		4
Level 5	Neither alter nor sleepy		5
Level 6	Some signs of sleepiness		6
Level 7	Sleepy, but no effort to keep alert		7
Level 8	Sleepy, but some effort to keep alert		8
Level 9	Very sleepy, significant effort to keep alert, fighting sleep		9

2.5.2. Measurement and Analysis of ECG

Electrocardiogram (ECG) is the most commonly used measurement to study the effects of CO₂ on the cardiovascular system. For HR and HRV data, we used the ECG Viewer software (Version 2.8.0) provided by the HeaLink platform for RR interval time series extraction. The experimental data were temporally intercepted and imported into Kubios HRV (Version 3.5.0) software for processing and analysis to obtain quantitative data [32].

2.5.3. Measurement and Analysis of EEG

The EEG device monitors and records electrical signals from the brain through electrodes placed on the surface of the scalp. When neurons in the brain are activated, a voltage change occurs across the cell membrane, and the electrodes can detect the electrical potential due to the voltage changes occurring simultaneously in tens of thousands of neurons, thus capturing the activity of the central nervous system and reflecting the mental state of the subject. These brain waves are generated by synchronized electrical impulses from a large number of neurons communicating with each other and vary with human behavior and emotions [33]. Specifically, the δ -band has a frequency of 3 Hz and below and tends to be generated during deepest meditation or dreamless sleep; the θ -band has a frequency range of 3–8 Hz and provides a state of

diminished consciousness, light sleep, or extremely relaxed lethargy; and the α -band has a frequency range of 8–13 Hz and is emitted during a state of physical and mental relaxation.

In addition, EEG signals show artifacts (e.g., blinking, muscle movements, etc.). In this study, EEGLAB based on MATLAB open software was used for preprocessing. Raw EEG signals were processed offline using the EEGLAB toolbox in MATLAB to maximize the signal-to-noise ratio. The continuous data underwent band-pass filtering (0.5–30 Hz) to eliminate low-frequency drift and high-frequency noise, supplemented by a notch filter to suppress 50 Hz power line interference. To isolate and remove physiological artifacts such as eye blinks and muscle activity without data loss, Independent Component Analysis (ICA) was performed. Channels exhibiting persistent noise were identified and reconstructed using spherical spline interpolation, after which the clean data were re-referenced to the common average reference (CAR) and segmented into non-overlapping 2-s epochs for subsequent analysis.

The preprocessed EEG signals were first extracted by Fast Fourier Transform (FFT) to extract the power spectral density (PSD) of each frequency band [34], and then, the absolute power was calculated by Wavelet Transform (WT) as shown in Equations (1)–(3).

$$F[k] = \sum_{n=0}^{N-1} x[n]e^{\frac{j2\pi kn}{N}} \quad (1)$$

$$P(k) = \lim_{N \rightarrow \infty} \frac{|F(k)|^2}{N} \quad (2)$$

$$WT(s, \omega) = \frac{1}{\sqrt{s}} \int_{-\infty}^{+\infty} f(t) \varphi\left(\frac{t - \omega}{s}\right) dt \quad (3)$$

F is the Fourier transform, x is the EEG signal, P is the power spectral density, WT is the wavelet transform, s is the scaling factor, w is the translation parameter, and k is 0, 1, ..., $N - 1$.

Studies have shown that sleepiness can be characterized by analyzing preprocessed EEG data [35,36] for increased low-frequency power [37]. For example, it has been shown that the EEG of sleep-deprived subjects during fatigued wakefulness exhibits increased power in the low-frequency range (δ and θ) from 1 to 8 Hz [35]. It has also been shown that EEG power in the α and θ bands increases as subjects become less alert [38]. Therefore, the power of α , δ and θ bands was selected as an index for sleepiness evaluation in this study.

2.6. Statistical Analysis

Statistical analyses were performed using SPSS 26.0 software. The Shapiro–Wilk test was used to verify the normality of the data distribution. Given the within-subject design of the experiment, a One-way Repeated Measures Analysis of Variance (ANOVA) was employed to evaluate the effects of increasing CO₂ concentration (time points) on subjective sleepiness (KSS) and physiological indicators (EEG, HR, HRV). Mauchly’s test was used to assess the assumption of sphericity; if violated, the Greenhouse–Geisser correction was applied to the degrees of freedom. For significant main effects, post hoc pairwise comparisons were performed using the Bonferroni correction to control for Type I errors. To assess practical significance, effect sizes were reported as partial eta squared for the ANOVA main effects and Cohen’s d for pairwise comparisons. A p -value of < 0.05 was considered statistically significant.

3. Results and Discussion

3.1. KSS Score

Figure 4 shows the results of the KSS subjective scores. Elevated CO₂ concentrations caused differences in subjective perceptions. The results show that the degree of sleepiness reported by subjects after exposure to CO₂ was consistently elevated from 0–40 min. Compared to at 0 min, subjects reported increased sleepiness at 20 min and at 40 min. A repeated measures ANOVA determined that subjective sleepiness scores differed significantly between time points. Post hoc tests using the Bonferroni correction revealed that KSS scores increased significantly at 20 min compared to baseline (mean difference = 1.17, $p < 0.01$, $d = 0.83$) and further increased at 40 min (mean difference = 2.50, $p < 0.001$, $d = 1.79$). The difference between 20 min and 40 min was also significant ($p < 0.01$, $d = 0.94$). These large effect sizes ($d > 0.8$) indicate a practically significant impact of dynamic CO₂ elevation on perceived sleepiness.

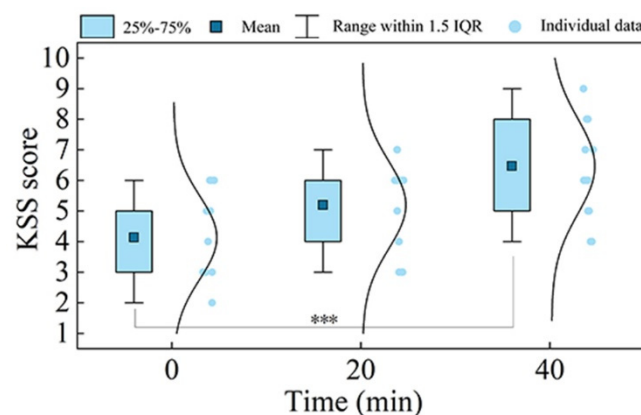


Figure 4. Results of KSS scores (***: $p < 0.001$).

Thus, subjectively, subjects perceived that their daytime sleepiness increased significantly with increasing CO₂ concentration. The results are consistent with Zhang et al.'s findings that when subjects were exposed to high CO₂ concentrations, subjective alertness as measured by the KSS decreased significantly due to limited ventilation [17].

3.2. Physiological Data Analysis

3.2.1. EEG

To quantitatively assess the impact of dynamic CO₂ concentration changes on brain activity and daytime sleepiness, EEG data were analyzed across three frequency bands: α , θ , and δ . Figures 5–7 illustrate the absolute power and corresponding brain topographic maps derived from this analysis. In these maps, warmer colors (red) indicate higher energy (greater EEG activity), while cooler colors (blue) represent lower energy.

Figure 5 details the α -band absolute power fluctuations alongside representative topographic maps for a single subject. As shown in Figure 5a, the 0–20 min interval (lower CO₂ concentrations) is characterized by reduced α -band activity, indicated by a predominance of cooler colors and lower PSD. In contrast, the 20–30 min interval (average CO₂ ~1700 ppm) shows heightened activity, particularly in the central, parietal, and temporal regions. Figure 5b quantifies this trend: average α -band power initially increased, peaking during the 20–30 min interval with a rise of 0.15 μV^2 relative to the 0–10 min baseline (average CO₂ ~800 ppm), before subsequently decreasing. This peak suggests an elevation in sleepiness levels. Furthermore, regional analysis (Figure 5c–g) reveals that CO₂ elevation differentially affected brain areas. Activity was most intense in the parietal and temporal lobes (power increases of 0.37 μV^2 and 0.33 μV^2 , respectively), followed by the central and occipital

lobes (increases of 0.12 uV^2 and 0.17 uV^2 , respectively). Consistent with the global trend, absolute α -band power across all regions peaked during the 20–30 min interval.

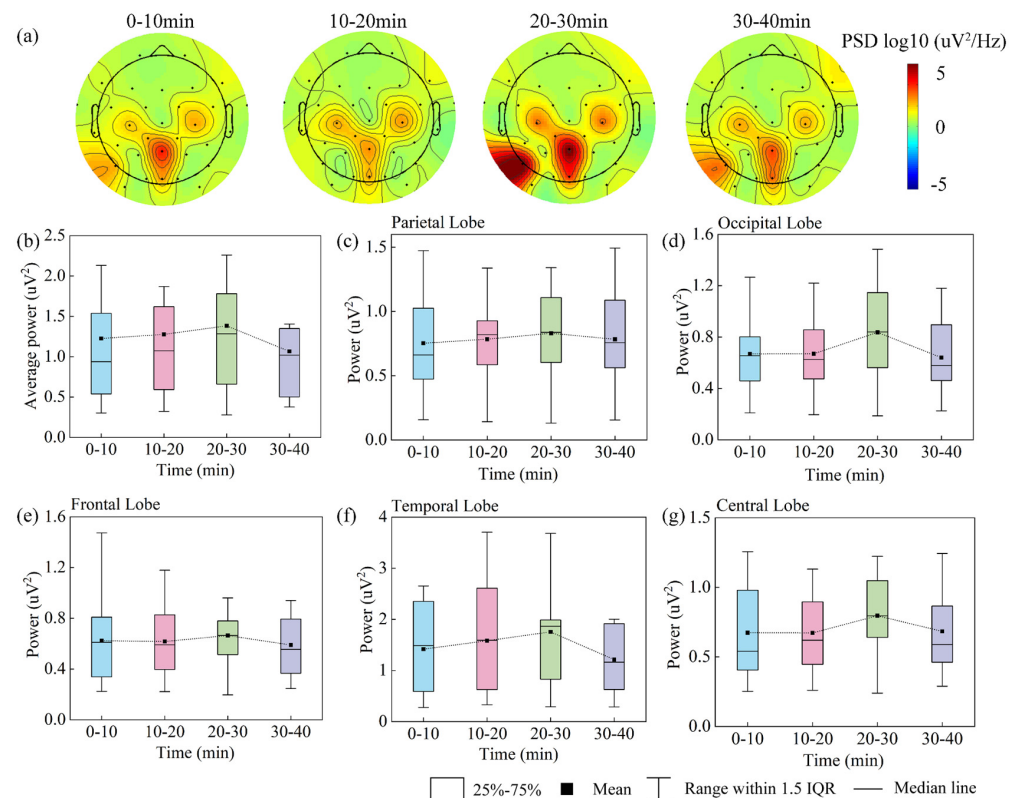


Figure 5. (a) EEG brain maps in the α band; (b–g) absolute power changes during CO_2 concentration dynamics.

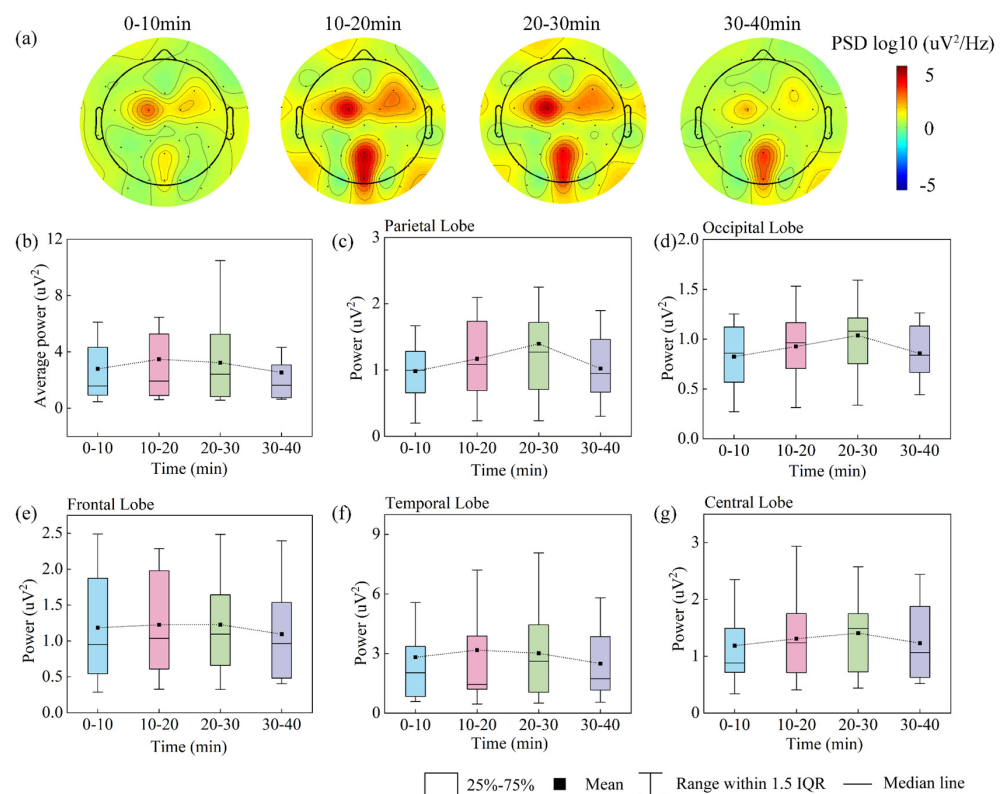


Figure 6. (a) EEG brain maps in the θ band; (b–g) absolute power changes during CO_2 concentration dynamics.

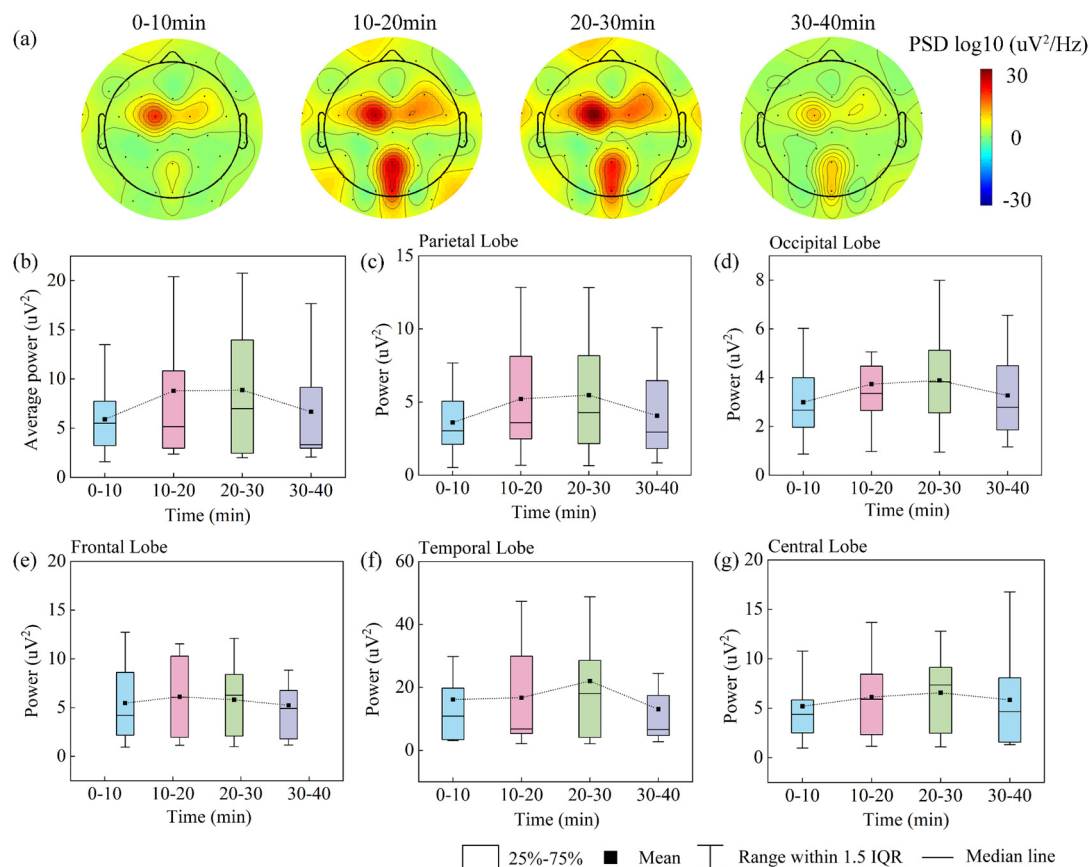


Figure 7. (a) EEG brain maps in the δ band; (b–g) absolute power changes during CO_2 concentration dynamics.

Figure 6 illustrates the absolute power changes and the corresponding topographic distribution of the θ band during the dynamic elevation of CO_2 concentration. As depicted in Figure 6a, the topographic maps for the 0–10 min and 30–40 min intervals exhibit lower PSD and reduced neural activity. In contrast, the 10–30 min interval reveals relatively intense brain activity, predominantly in the central, parietal, and occipital lobes. Figure 6b presents the temporal evolution of the average θ band power. Over the 40-min duration, power levels initially rose, peaking between 10 and 20 min, and plateauing during the 20–30 min interval before subsequently declining. Specifically, compared to the baseline (0–10 min), the average θ power increased by 0.68 uV^2 during the 10–20 min interval and by 0.44 uV^2 during the 20–30 min interval. Regional analysis (Figure 6c–g) revealed spatial heterogeneity in the CO_2 response. While a general trend of increase-then-decrease was observed across the cortex, peak timing differed: anterior regions, including the frontal and temporal lobes, peaked earlier (10–20 min), whereas posterior regions (central, parietal, and occipital lobes) peaked later (20–30 min).

Figure 7 displays the absolute power changes and scalp topographies for the δ band under dynamic CO_2 conditions. Similar to the θ band, the topographic maps in Figure 7a indicate lower PSD and smoother brain activity during the 0–10 min and 30–40 min periods, while the 10–30 min interval was characterized by relatively intense activity in the central, parietal, and occipital areas. Figure 7b shows the average power trend, which followed an inverted-U trajectory over the 0–40 min period. The average δ power peaked at 10–20 min, showing an increase of 5.55 uV^2 relative to the 0–10 min baseline. However, regional breakdowns (Figure 7c–g) highlight distinct spatial responses. With the exception of the frontal lobe, the power of the low-frequency band in all other brain regions exhibited a gradual increase from 0 to 30 min, reaching a peak during the 20–30 min interval.

In summary, an increase in power in the low-frequency bands (α , δ , and θ) was observed with increasing CO₂ concentration during 0–30 min. This is consistent with the hallmarks of sleepiness [24] and may reflect a shift in the brain from an alert to a sleepy state. Previous studies have shown that people become drowsy after 35 min of exposure to increased CO₂ levels under high-CO₂ conditions [18]. There is consistency between the results of the present study and previous studies. In addition, it has been shown that the transition to sleep leads to changes in EEG activity in the central and posterior regions of the brain [39]. This finding is consistent with the results of the present study. EEG brain maps showed that the most active regions of the subjects' brains were the central, parietal, and occipital regions as CO₂ concentration increased. And the results of power calculation showed that the absolute power of the low-frequency bands in the central, parietal and occipital regions increased with increasing CO₂ concentration in 0–30 min, and all of them reached the peak value in 20–30 min. However, after reaching the peak, the absolute power of the low-frequency band appeared to decrease with a continued increase in CO₂ concentration. This finding deserves further investigation.

3.2.2. HR and HRV

Figure 8 illustrates the temporal trends of the average HR and HRV-SDNN across four time intervals (0–10, 10–20, 20–30, and 30–40 min). The results indicate a slight increase in HR as the CO₂ concentration rose over the 40-min period. As shown in Figure 8a, the highest average HR was observed during the 30–40 min interval, which was 1.27 bpm higher than the baseline (0–10 min). Similarly, Figure 8b shows that HRV-SDNN values increased with rising CO₂ levels. The average HRV-SDNN reached 53.56 ms during the 30–40 min interval, representing a 16.3% increase compared to the 0–10 min interval (46.05 ms). However, these observed changes in HR and HRV were not statistically significant.

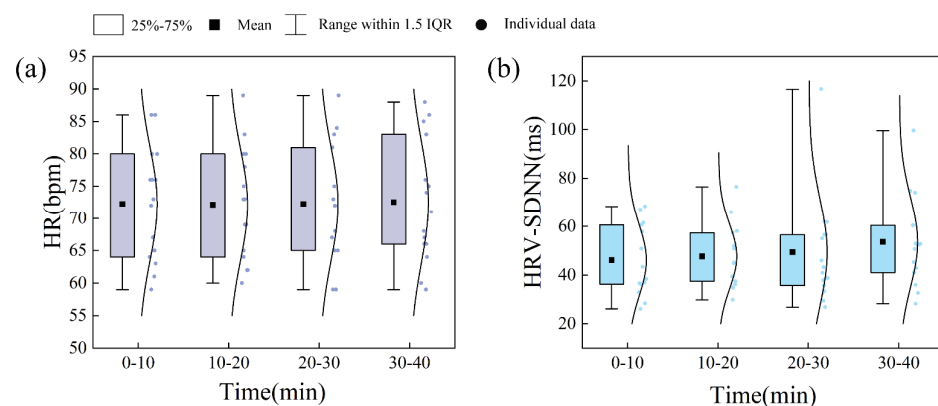


Figure 8. The trend of (a) HR and (b) HRV-SDNN over time.

The lack of statistically significant changes in HR and HRV-SDNN in the present study warrants a nuanced discussion regarding exposure duration and dynamics. In the context of indoor environmental quality research, we define the 40-min duration of our experiment as a ‘short-term’ exposure, distinct from ‘medium-term’ chamber studies (typically 2.5–4 h) [8,17] or occupational exposures (8 h) [4].

Our results stand in contrast to Snow et al., who observed a significant increase in heart rate during a similarly short exposure (<60 min) to 2700 ppm CO₂ [18]. However, our findings align with Zhang et al. and Liu et al., who reported no statistically significant changes in heart rate or blood pressure even after 3–4 h of exposure to pure CO₂ at concentrations up to 3000 ppm [16,17]. We propose that this discrepancy is likely attributable to the dynamic nature of the CO₂ elevation in our protocol. Unlike step-change protocols where subjects are abruptly exposed to high concentrations, triggering an acute stress

response, our subjects experienced a gradual ‘ramp’ from 500 to 2000 ppm over 40 min. Furthermore, Kreska et al. demonstrated that significant alterations in HRV might exhibit a latency effect, appearing only 1 h after the cessation of CO₂ treatment [40]. Thus, the 40-min dynamic window in our study may capture the accumulation phase of physiological load, which was insufficient to overcome the homeostatic resilience of healthy young adults within the observation period.

3.2.3. Oxygen Saturation and Pulse Rate Results

Figure 9 shows the trend of average pulse rate and average oxygen saturation of all subjects during the increase in CO₂ concentration. The results showed that the pulse rate of the subjects was maintained in the range of 72.9–76.5 and the oxygen saturation was maintained in the range of 97–99%, and none of them was significantly affected by the increase in CO₂ concentration.

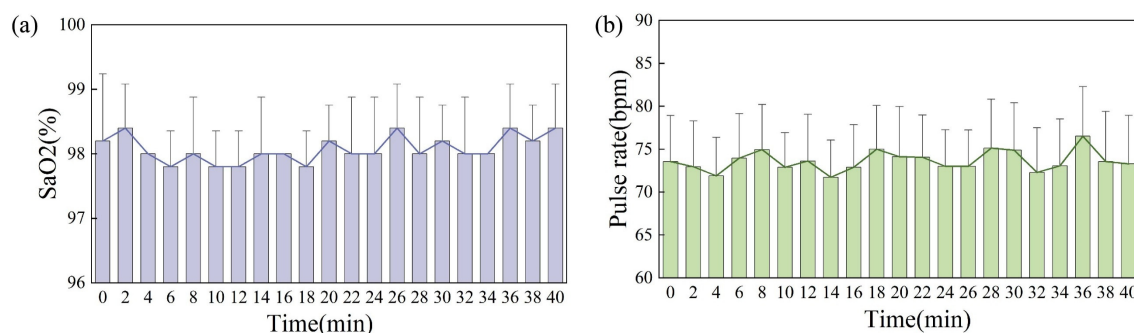


Figure 9. Trends in (a) pulse rate and (b) oxygen saturation over time during a rise in CO₂ concentration.

3.3. Limitations

This study applies EEG technology to measure the effect of dynamically elevated CO₂ concentration on daytime sleepiness, providing useful guidance for researchers to revisit whether EEG can be used as an objective sleepiness determinant. However, this study also has some limitations. First, because the experiment requires wearing EEG equipment, there are some difficulties in the selection of subjects, and the small sample size of the experiment may lead to errors in the results. This study was conducted only on school college students, and the experimental results are not generalizable. In future studies, the sample size should be increased to expand the study group. Secondly, this study did not examine the rate of elevated CO₂ concentration; different rates of elevated CO₂ concentration may have different effects on daytime sleepiness and EEG in humans, and future studies may focus on the effects of different rates of elevated CO₂ concentration. Thirdly, a key limitation is the absence of a control condition with constant CO₂ concentration. Without such a control, it is difficult to fully distinguish the physiological effects of rising CO₂ from time-on-task effects, such as monotony and accumulated fatigue. Future research should employ a crossover design with a stable CO₂ baseline to rigorously isolate the specific impact of CO₂ from exposure duration.

3.4. Implications for Intelligent Building Ventilation Control

While this study primarily focuses on the physiological impacts of dynamic CO₂ concentrations, the quantitative relationships established between CO₂ levels and EEG spectral power offer critical ground-truth data for advanced building control systems. Traditional ventilation strategies, typically based on static CO₂ thresholds, often fail to account for the temporal dynamics of human physiological responses and may react too slowly to prevent the onset of drowsiness. To address this limitation, future research should explore the integration of these physiological feedback loops into building ventilation

control using Reinforcement Learning. Unlike rule-based controllers, RL agents can learn proactive control policies that anticipate physiological decline based on environmental trends. Recently developed open-source frameworks, such as BuildingGym [41], provide a robust platform for this integration. BuildingGym facilitates the training of RL agents by connecting high-fidelity building energy simulations with flexible input interfaces. By utilizing the EEG-based sleepiness markers identified in our study as parameters for the RL reward function, researchers can train intelligent agents to optimize ventilation rates dynamically. This approach aims to achieve a “human-centric” balance, minimizing energy consumption while preemptively mitigating the physiological markers of daytime sleepiness before they significantly impact cognitive performance.

4. Conclusions

CO₂ influences human physiology and contributes to health risks. This study investigated the effects of dynamically elevated CO₂ concentrations on daytime sleepiness and EEG signals in a controlled environment. Subjective ratings, EEG patterns, and physiological indicators were evaluated. Results demonstrate that CO₂ concentration significantly influences daytime sleepiness. Specific conclusions include:

- (1) Subjective sleepiness increased continuously from 0 to 40 min alongside rising CO₂. Compared to baseline (0 min), KSS scores at 40 min significantly increased, indicating a statistically significant change.
- (2) Power spectral analysis revealed increased power in low-frequency EEG bands under elevated CO₂. Specifically, α -band power peaked at 20–30 min, increasing by 0.15 μV^2 compared to 0–10 min. θ - and δ -band power peaked at 10–20 min and remained stable through 20–30 min, increasing by 0.68 μV^2 and 0.55 μV^2 , respectively. However, low-frequency power declined at 30–40 min despite the continued rise in CO₂.
- (3) EEG brain mapping indicated stronger low-frequency activity in the central, parietal, and occipital regions. Power in these regions peaked between 20 and 30 min. However, overall brain activity plateaued as CO₂ continued to rise between 30 and 40 min.
- (4) During the 0–40 min exposure, HR and HRV-SDNN gradually increased with CO₂ concentration. However, these changes were not statistically significant, possibly due to the relatively short exposure duration.
- (5) Throughout the dynamic CO₂ elevation, pulse rates remained between 70 and 80 bpm and oxygen saturation consistently around 98%, indicating no significant impact on these parameters.

Future research should utilize larger, more diverse sample sizes and investigate the impact of varying CO₂ elevation rates on physiological and neural responses.

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