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Exploring the physiological, neurophysiological and cognitive performance effects of elevated carbon dioxide concentrations indoors

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ABSTRACT

Rationale: An accumulation of CO_2 in occupied indoor spaces is correlated to negative impacts on concentration, sleepiness and aspects of cognitive performance. However, factors such as: (a) the relative effect of CO_2 itself compared to other pollutants; (b) the minimum necessary exposure time for cognitive performance to be affected; and (c) the physiological drivers of cognitive performance reductions due to increased indoor CO_2 concentrations are not yet clear.

Method: A within-subjects counterbalanced study design was used to test cognitive performance, subjective and physiological parameters of 31 volunteers during short (< 60 min) exposures to normal CO_2 (830 ppm) and high CO_2 (2700 ppm, raised by introducing pure CO_2 alongside the occupant generated CO_2). The study was conducted in a small naturally ventilated office and EEG was used as an objective indicator of sleepiness.

Results: The addition of pure CO_2 to the room resulted in the absence of an expected learning effect in two cognitive performance test battery components which could not be explained by any of the physiological, psychological, or reported comfort, sick building syndrome and health variables measured. However, participants who had slept less the previous night appeared more susceptible to becoming sleepier as a result of the increased CO_2.

Contributions: The results suggest (1) the addition of pure CO_2 may influence aspects of cognitive performance after only short exposures (2) these changes occur in the absence of clear physiological drivers, (3) lack of sleep may mediate people's response to higher CO_2 concentration.

1. Introduction

The effects of ventilation and increased carbon dioxide (CO_2) on human performance and physiology is imperfectly understood [1–5]. CO_2 is produced as part of human respiration and is used as a proxy for ventilation rate in a room and thus the likely presence of other indoor pollutants such as volatile organic compounds (VOC's) including human bio-effluents [4,5]. Previous research has sought to determine the effect of reduced ventilation on humans using cognitive performance test batteries [6–8], computerised tests [9–11], and academic performance measured through exam scores [12–14], as well as the effects of increased levels of pure CO_2 on decision making, when ventilation is not restricted [7–9]. Results suggest substantial links exist between reduced or inadequate ventilation and the increased prevalence of sick building syndrome (SBS) symptoms, difficulty concentrating, lower cognitive performance, and lower academic performance [5,6,8,13,15]. While the link between under-ventilation and human performance is clear, three knowledge gaps remain:

(1) It is not conclusively determined whether CO_2 is an innocuous indicator of other harmful indoor pollutants such as volatile organic compounds and human bio-effluents [1,2], or is itself an active contributor to reduced performance in cognitive tasks [7–9].
(2) Temporal dimensions, such as the minimum duration of exposure to poor indoor air quality (IAQ) necessary to cause a measurable reduction in cognitive performance is imperfectly understood. Studies typically utilise exposure durations in excess of two hours [2,5,7,10–12], yet *continuous* exposures of this length are unlikely

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in functional offices, owing to coffee/bathroom breaks, visits to different offices, and actions which reduce the CO_2 concentration such as opening windows.

(3) Despite much literature concerned with measuring the health, comfort, and cognitive performance effects of elevated indoor concentrations of CO_2 (e.g. 1400–5000 ppm), little research reports on the physiological drivers of these changes [1,5]. EEG has enabled insight into changes in brain patterns which occur at very high concentrations of CO_2 (> 50,000 ppm) [13–19], and has been used in sleep science to characterise sleepiness and sleep states [20–22]. However, little is known about the neurophysiological effects of indoor-realistic concentrations of CO_2, and EEG is not typically used as an objective measurement for drowsiness in IAQ research.

To address these three aspects, this study seeks to determine the human performance impact due to additional pure CO_2 over a short period of time that is comparable to uninterrupted work in an office. To understand the drivers of changes to cognitive performance, we couple performance measurements with physiological data using EEG to objectively measure changes to drowsiness as a result of exposure to CO_2.

1.1. Effects of CO_2 concentrations ≥ 50,000 ppm

The physiological and neurophysiological effects of high concentrations of CO_2 have been measured, where a hypercapnic state (elevated arterial CO_2) is induced through direct inhalation of pure CO_2/air mixtures: 5%, (i.e. 50,000 ppm) [16–18,23], 7% [19], 7.5% [14,15], 10% [13] and 20% [24]. Inhalation of 7.5% CO_2 is found to negatively affect mood and induce anxiety [15] and increase subjective feelings of breathlessness [23]. Increased heart rate is also reported during inhalation of 5% [18] and 7.5% CO_2 [15].

Inhalation of ≥ 5% CO_2, causing an increase in the respired partial pressure of CO_2 of ~7–10 mmHg, leads to an overall slowing of the EEG; attenuated power in the higher frequency bands of gamma, beta and alpha [16,23,24] and increased power in the low-frequency power bands of delta and theta [13,17,18]. The increase in low frequency power is indicative of lower neurological arousal, and is used as an indicator of a progression towards sleepiness [17,18]. Despite producing an EEG indicative of sleepiness (i.e. lower neurological arousal), inhalation of 5% [16] and 10% [13] CO_2 concentrations does not affect cognitive performance [13,16].

A key purpose of this present study is to determine the relationship between EEG and cognitive performance at lower levels of CO_2, as neurological arousal is not yet commonly measured in studies involving indoor-realistic CO_2 concentrations.

1.2. Effects of CO_2 realistically achieved indoors (≤ 3000 ppm)

The concentrations of CO_2 measured indoors are an order of magnitude less than of those studies reported above. Yet indoor CO_2 concentrations between 1800–3000 ppm (with and without bio-effluents) have been found to affect decision making, aspects cognitive performance [2,7–9,11,12] and physiological parameters [1,5]. Disagreement in the literature exists as to whether CO_2 itself affects cognitive performance, or whether it is an indicator of other pollutants that do.

Significant reductions in decision making performance using the Strategic Management Software have been recorded after 4 h exposure to CO_2 concentrations of 1400–2500 ppm achieved by continuously injecting pure CO_2 into a well ventilated room (ventilation rate 720 m^3/h) [7,8]. Zhang et al. [2] found no effect of 4.25 h exposure to 3000 ppm CO_2, using a similar protocol involving continuous injection of pure CO_2 in a mechanically ventilated room. This study [2] found significant negative effects on speed at which subjects could carry out addition, their response time in a re-direction task, their cue-utilisation performance, as well as increased self-reported fatigue, increased intensity of reported health symptoms, and reduced perceived air quality, when the same 3000 ppm CO_2 concentration was accompanied by human-bio-effluents. This suggests that bio-effluents rather than pure CO_2 is a key factor affecting human performance [2]. It is proposed that bio-effluents affect cognitive performance through physiological mechanisms, either (a) increased stress/physiological arousal caused by the indoor conditions, or (b) factors such as increased end-tidal $ETCO_2$ (respired CO_2 measured by capnography) and reduced nasal peak flow triggering discomfort, sleepiness, or sick building syndrome (SBS) symptoms [1]. Notably, this second proposition is not completely supported by recent findings that cognitive performance decrements can occur prior to, or in the absence of, occupant discomfort or awareness of reduced air quality [11,12].

1.3. EEG as an objective measurement of drowsiness

EEG is not yet a feature of research assessing the effects of indoor-realistic concentrations of CO_2 upon human performance, despite its use to measure neurological arousal at very high concentrations of CO_2 [13,17,18], and its use in neuroscience to objectively characterise sleepiness [25,26]. In existing studies of CO_2 upon human performance, sleepiness is typically assessed subjectively using self-reporting [2,5,27] and few objective measures of sleepiness are currently utilised in indoor air quality research. One exception is the application of voice analysis [28,29], which is not yet widely applied. This present paper represents a novel use of EEG to objectively measure sleepiness, in conjunction with physiological and cognitive performance parameters. These methods allow insight into the drivers of the cognitive performance declines measured at indoor-realistic CO_2 concentrations.

There are two primary aims to the paper

(1) to measure changes in human performance, physiological, neurophysiological (EEG) and psychological factors as a result of elevated indoor CO_2 concentrations (due to the addition pure CO_2)
(2) to validate EEG as an objective measurement of sleepiness for studies concerned with the effect of the indoor environment on humans.

The study was carried out with human participants who were subject to either normal CO_2 (830 ppm) or high CO_2 (2700 ppm) conditions. Cognitive performance, mood/affect, Sick Building Syndrome symptoms, physiological (respiration, heart rate, skin temperature) and neurophysiological (EEG) parameters were measured, alongside building and IAQ parameters. The interactions between these parameters are compared to findings from the relevant literature and existing understanding of the physiological drivers of human performance effects due to poor IAQ.

2. Methodology

2.1. Participants

We recruited 31 participants to the study between October and December 2017. The protocol and conditions of participation were approved by the University of Southampton Ethical Research Governance Office (Reference no. 30443). Each participant received a £20 voucher for an online retailer. Participants were recruited by advertising the study at multiple locations throughout the University, and convenience sampling involving contacts of the researchers who were not privy to the study aims or protocol. Informed written consent was obtained from each participant prior to commencement of the study.

Participants were allocated to one of four groups (Table 1) and all participants experienced both a normal and a high CO_2 condition. Given the effect of circadian rhythm on alertness (e.g. post-lunch sleepiness [30]) and subjective beliefs around productivity (e.g. being a “morning person”), we controlled for time of day by testing equivalent

Table 1
Overview of study groups.

Group	Time of day	First CO_2 level	Second CO_2 level
1	Morning	High	Normal
2	Morning	Normal	High
3	Afternoon	High	Normal
4	Afternoon	Normal	High

numbers of participants in the morning and afternoon. Morning sessions started between 9:00 and 10.30, and afternoon sessions started between 13:00 and 14.30. The study design also counterbalanced the order of CO_2 treatment given to control for any residual effects of the previous CO_2 condition: normal CO_2 first or high CO_2 first (see Table 1).

Participants were advised that they would be taking part in two experimental conditions, one with a higher level of CO_2, but were not aware of which condition they were entering. Exclusion criteria included current or historic drug/alcohol abuse or panic attacks, pregnancy, current treatment for migraine headaches, current neurological conditions (e.g. Epilepsy), long-term illness, recent or current illness (e.g. flu) on the day of the study. Participants were mostly employees or students of the University who habitually engage in office work, and were aged between 18 and 36 (mean: 22.5, SD: 4.8). Body mass indexes (BMI) were between 18.1 (underweight) and 33.9 (obese) (mean: 23.3, SD: 3.37; healthy). One of the 31 participants was an occasional smoker, no others smoked.

2.2. Study room and preparation

A key motivation for the study was to replicate realistic office conditions, therefore a functional, naturally ventilated office was chosen for the study rather than a laboratory. The office had dimensions 4.0 m by 3.4 m (floor area) by 3.1 m (high), and contained the carpet and furniture of an operational office (Fig. 1). The office was on the fourth floor of a university building in the south of England. Of the two windows on the north and west corner of the room, only the western window (1.7 m high by 0.4 m wide) could be opened and is visible behind the participant in Fig. 1. The CO_2 cylinder is visible in front of the openable window and the numbered arrows indicate the location of the three CO_2 loggers (Fig. 1). The windows were closed while participants were in the room, with ventilation provided through background infiltration.

Room temperature was adjusted to maintain thermally neutral conditions (21–23 °C) and minimise temperature variation. Participants were invited to adjust their clothing before the experiment began, to ensure they were thermally comfortable during the experiment.

The CO_2 concentration in the room was prepared 15–20 min prior to the beginning of the session (Groups 1 and 3) or in the 15-min break between the normal and high CO_2 conditions (Groups 2 and 4) while the participant was absent. For normal CO_2 conditions, the measured concentration was around 700 ppm at the beginning of the experiment. The chosen target for high CO_2 concentration was 2700 ppm, well above guidelines for CO_2 concentrations in offices (1200 ppm [31]) and classrooms (1500 ppm [6]), but not uncommon in occupied buildings due to poor ventilation when windows and doors are closed [5,32]. It is also comparable with other studies assessing the human performance effects of indoor CO_2 concentration, e.g. 2260 ppm [12] 2500 ppm [7] or 3000 ppm [1,2,9]. Given the disagreement in the literature concerning the effect of pure CO_2 on human performance, the study sought to test the effects of additional pure CO_2 injected into a room. The mean CO_2 concentration recorded from the first two minutes of all high CO_2 sessions was 2680 ppm (*SD*: 130 ppm), made up of 1960 ppm (*SD*: 160 ppm) ultrapure CO_2 (> 99.99% purity), injected from a cylinder, with the remainder either atmospheric, or generated metabolically by the participant and researcher. A pedestal fan was used prior to the experiment to achieve rapid mixing, as per [33]. CO_2 rose gradually by 150–270 ppm during both normal and high CO_2 exposures owing to the respiration of the researcher and participant.

2.3. Experimental protocol

The experimental protocol is described in Table 2. Averaged indoor environmental conditions including CO_2 concentrations for each step of the protocol are listed in Table 3. The participants were met by the researcher in a well ventilated and thermally neutral office away from the study room, where they immediately completed the baseline questionnaires and cognitive performance tasks. When in the study room, participants were instructed to sit at the table in the centre of the room (Fig. 1), remaining as still as possible to minimise movement artefacts in the EEG. Physiological and EEG data were recorded from participants during sessions 1.1–1.3 and 2.1–2.3 (see Table 2).

The two minutes with eyes closed at the start of each Session (Table 2) was necessary to calibrate the artefact reduction algorithm for the EEG analysis. For comparative data analysis, averages were taken for indoor environmental conditions (temperature, humidity, CO_2 concentration) throughout each set, i.e. from the beginning of Session 1.1 until the end of Session 1.3, and likewise for Session 2. This

Fig. 1. Study room showing participant with EEG cap, respiration belt, location of loggers, window and CO_2 cylinder, temperature sensor attached to left hand (out of shot).

Table 2
Experimental protocol. The ~ symbol indicates approximate, i.e. varied among participants. The * symbol indicates fixed timings.

Location	Study segment	Actions/Details	Duration
Study room	**Pre-experiment:** CO_2 *adjusted in study room*	Researcher releases CO_2 into study room using the CO_2 cylinder to achieve target CO_2 concentration of 2700 ppm (Groups 1 and 3) OR researcher checks CO_2 concentration in room is < 800 ppm (Groups 2 and 4)	
Separate testing office	*Informed Consent + Pre-start screening*	Ethical consent gathered, pre-test screening for eligibility, demographics gathered	10 min ~
	***Baseline** Questionnaire 1a (BQ1a)*	Sick Building Syndrome (SBS) symptoms, Stanford Sleepiness Scale, PANAS (state)	5 min ~
	***Baseline** Cognitive Performance test battery (Baseline CPT)*	Stroop Test, Shifting Attention Task, Continuous Performance Test, Four Part Continuous Performance Test	20 min ~
		Researcher and participant enter study room	
Study room	***Baseline** questionnaire 1b (BQ1b)*	Thermal comfort (7 point scale), questions related to perceived air quality, ventilation, air freshness	2 min ~
		Researcher prepares and connects EEG and physiological monitoring equipment to participant; starts recording of physiological parameters	5 min ~
	***Session 1.1** (EEG + physiology recorded)*	Participant sits still with eyes closed	2 min*
	***Session 1.2** (EEG + physiology recorded)*	Participant sits still with eyes open	8 min*
	***Intermediate** Cognitive Performance test battery (Intermediate CPT)*	Identical to Baseline Cognitive Performance test battery	20 min ~
	***Intermediate** Questionnaires (Intermediate Q)*	Thermal comfort (7 point scale), questions related to perceived air quality, SBS symptoms, Stanford Sleepiness Scale, PANAS (state)	5 min ~
	***Session 1.3** (EEG + physiology recorded)*	Participant sits still with eyes open *Remove EEG and Physiology sensors from participant*	8 min*
	Break	Participant leaves room and moves to a different area of the building.	15 min*
	CO_2 adjusted	Researcher ventilates room by opening door and window to reduce CO_2 levels (Groups 1 and 3) OR releases CO_2 into room to target concentration of 2700 ppm (Groups 2 and 4).	
Study room		*Participant returns to study room* Researcher re-connects EEG and physiological monitoring equipment to participant; starts recording of physiological parameters	5 min ~
	***Session 2.1** (EEG + physiology recorded)*	Participant sits still with eyes closed	2 min*
	***Session 2.2** (EEG + physiology recorded)*	Participant sits still with eyes open	8 min*
	***Final** Cognitive Performance test battery (Final CPT)*	Identical to Baseline and Intermediate Cognitive Performance test batteries	20 min ~
	***Final** Questionnaire (Final Q)*	Identical to Baseline and Intermediate Questionnaires	5 min ~
	***Session 2.3** (EEG + physiology recorded)*	Participant sits still with eyes open	8 min*
	End of study debrief	EEG and physiological monitoring disconnected, participant leaves room.	5 min ~
		Approximate duration of engagement for each participant:	**153 min**

Table 3
CO_2 concentration, temperature, and humidity (meañ ± SD) during each Session (Normal CO_2 session .1, .2, and .3, and High CO_2 session .1, .2, and .3) for each condition.

Session	CO_2 (ppm)	Temperature (°C)	RH (%)
Normal CO_2 .1	720 ± 100	22.0 ± 1.7	39.8 ± 7.9
Normal CO_2 .2	790 ± 100	22.0 ± 1.2	40.1 ± 7.6
Normal CO_2 .3	990 ± 120	22.3 ± 1.2	41.0 ± 7.0
Average (Normal CO_2)	830 ± 110	22.1 ± 1.4	40.3 ± 7.5
High CO_2 .1	2680 ± 130	22.1 ± 1.2	41.6 ± 7.2
High CO_2 .2	2710 ± 130	22.1 ± 1.2	41.8 ± 7.1
High CO_2 .3	2830 ± 180	22.3 ± 1.2	43.1 ± 7.0
Average (High CO_2)	2700 ± 150	22.2 ± 1.2	42.2 ± 7.1

NB RH = Relative humidity.

included the time spent completing the questionnaires and cognitive performance tasks. Participants were exposed to the CO_2 condition for a total of approximately 50 min, cognitive performance testing began after approximately 20 min exposure. The exposure duration of 50 min is short compared to previous studies (e.g. 150 min [7], 235 min [19], 240 min [5]). Our decision to restrict the exposure time to under 1 h reflects the reality that office work is typically punctuated by coffee/bathroom breaks and visits to other rooms with different air quality conditions, and thus continuous exposures of 150–240 min are unlikely in working offices.

2.4. Measurement

2.4.1. Cognitive performance and questionnaires

We used the CNS Vital signs computerised cognitive test battery [34] and included tests of the following domains: executive function, reaction time, working memory, complex attention, simple attention, sustained attention, and cognitive flexibility. These domains were tested through completing the following cognitive tasks on a laptop: (1) Stroop test (2) shifting attention task (3) continuous performance test (4) four-part continuous performance test. Full descriptions of the tasks and domains can be found in the Supplementary Information and in the CNS software documentation [34]. The CNS software provides standardised scores (population mean: 100, σ: 15) for executive function, reaction time, working memory, simple visual attention and cognitive flexibility, calculated through combinations of scores on the respective tests [34]. The choice of performance tasks was based on those used by other authors in this field [5,12,27,35].

The time taken to complete the cognitive tests was approximately 20 min. Questionnaires on subjective parameters were completed on paper and included questions related to Sick Building Syndrome symptoms [36], sleepiness (Stanford Sleepiness Scale [37]), and Positive and Negative Affective State (PANAS) [38].

2.4.2. Environmental measurement

Three factory calibrated Rotronic CL11 (BSRIA, Bracknell, UK) environmental loggers measured temperature, humidity, and CO_2 concentration throughout each experiment. The loggers sampled at 0.1 Hz with a measurement error of ± 30 ppm ± 5% of the measured value for CO_2 concentration, ± 0.3 °C for temperature, and < 2.5% for relative humidity (RH). The CL11's display updates approximately once per second, enabling the researcher to monitor and control the release of CO_2 in the room to a high granularity. The loggers were positioned roughly equidistant to each other around the room (labelled 1–3 in Fig. 1). The logging frequency of the instruments was set to 10 s.

2.4.3. Physiological measurement

Physiological measurements including skin temperature (middle finger, non-dominant hand), pulse rate (finger clip, non-dominant hand) and respiration rate (abdominal belt) were taken from each participant using the Mind Media NeXus-10 MKII[1]. Each parameter was recorded continuously throughout each of the experimental conditions at 32 Hz sampling rate (instrument error ± 2%).

2.4.4. EEG measurements

A Neuroelectrics ENOBIO 20 dry electrode wearable wireless EEG cap[2] was used (19 channel, 10–20 placement, 500 Hz sampling rate, no instrument error given). Reference electrodes were positioned on the participants' bilateral mastoid bone, or mastoid muscle depending on head-shape. EEG was gathered continuously throughout each of the EEG Sessions. Participants were asked to remain as still as possible, to minimise movement artefacts. The EEG and physiological sensors were removed during the break and reconnected shortly after the participant returned to the study room. Due to the difference in logging frequency of the CL11s (0.1 Hz) compared to the EEG measurements (500 Hz), the error on the readings versus that of the condition timings is expected to be ± 20 s. This error was considered acceptable, given the IAQ conditions change gradually throughout the experiments.

2.4.5. EEG pre-processing

EEG data were filtered using a Butterworth filter; low pass at 45 Hz and high pass at 0.15 Hz. Artefact rejection was implemented in two stages. The first used the artefact rejection algorithm WPT-EMD [39,40], which uses a sample of minimum variance EEG taken from the two minute period of closed eyes (Session 1.1 or 2.1). The second stage of artefact rejection involved an amplitude threshold cut-off of ± 100 μV, replacing outlying data with a 10-s moving median around the extreme value. Electrodes showing consistent noise or flat-lined output were removed from the dataset. Due to excessive noise, frontal electrodes were not included in analysis. Eighteen of the 31 participants had sufficiently clean EEG data throughout the experiment and sufficient representation of clean electrodes in each brain region (central, temporal, parietal, occipital) to warrant further analysis.

Bandpower was extracted from the pre-processed continuous EEG for delta (1–3 Hz), theta (4–7 Hz), alpha (8–13 Hz), beta (14–35 Hz), and gamma (> 35 Hz) frequency bands, over one second windows. Average bandpower was computed for central (C3, Cz, C4), parietal (P3, P4, P7, P8, Pz), temporal (T7, T8), and occipital (O1, O2) electrodes for each Session. Slow eye movements can register as low-delta waves between 1 and 1.5 Hz, particularly in the frontal electrodes [41]. Thus to limit any possibility of delta power being contaminated by eye movements, low-delta (< 2 Hz) was not included in the analysis. Gamma was also excluded from further analysis owing to the focus of the study protocol on low frequency behaviour and because gamma represented < 1% of total power at each analysis segment.

2.5. Analyses

EEG was analysed to determine overall changes due to the environmental conditions, and for evidence of sleepiness specifically. Repeated measures ANOVAs were conducted with factors including EEG electrode region, frequency, and condition (normal/high CO_2). Sleepiness was characterised by post-hoc analysis of the cleaned EEG data according to literature [21,42–44]. Sleepiness is characterised by increases in low frequency power [43]; sleep deprived participants' EEG during tired wakefulness exhibits greater power in the low frequency range 1–8 Hz (delta and theta) are global in nature (i.e. registered in multiple areas of the brain) [21]. Accordingly, in this study, sleepiness is characterised as increases in delta and theta, particularly if these increases are global in nature.

Physiological data (heart rate, respiration rate, and skin temperature) was gathered from all 31 participants and down-sampled from 32 Hz to 1 Hz for compatibility of analysis with the environmental data. Averages for each physiological parameter, cognitive performance scores, and answers to individual questions were computed per group and per testing condition (baseline, normal CO_2, high CO_2) and included in the ANOVAs described below.

3. Results

3.1. Indoor air quality parameters

The CO_2 concentration averaged 830 ppm (SD 110) under normal conditions and 2700 ppm (SD 150) in high CO_2 sessions. There was a small variation between groups as shown in Table 3, but this was not significant compared to the difference between the normal and high cases. Table 3 highlights the consistency of the average temperature and relative humidity (RH) readings recorded during the experimental session. All temperature/humidity values recorded fall within ASHRAE 55–2013 [45] using the PMV method, assuming: radiant temperature = air temperature, air speed = < 0.1 m/s, metabolic rate = 1.1 (office work) and clothing level = 1.0 (typical winter indoor).

3.2. Cognitive performance results

All cognitive performance and self-reported measures were subject to 3-way repeated measures ANOVAs to assess the impact of condition: (1) baseline, (2) normal CO_2, (3) high CO_2 (see Table 4). Results show no effect of CO_2 on reaction times, complex attention, simple attention, sustained attention, or working memory. Both cognitive flexibility (F (2, 58) = 4.33, p = .02, η_p^2 = 0.13) and executive function (F (2, 60) = 4.81, p = .01, η_p^2 = 0.14) showed significant effects of condition with scores in the normal CO_2 condition being significantly better than baseline. These findings suggest a possible effect of learning/practice, which is not unexpected, given the cognitive tasks undertaken in the normal CO_2 condition represent the second or third (depending on order of treatment) attempt at the same tests within the relatively short study protocol. Critically, this learning effect was lacking when the participants were exposed to the high CO_2 condition, irrespective of order of treatment (group). This finding suggests that although participants did not exhibit worse performance during the high CO_2 condition, performance still appears negatively impacted, given lack of learning effect relative to the normal CO_2 condition.

3.3. Sick building syndrome symptoms, sleep, and mood/affect

Analysis of participants' experiences of sick building syndrome symptoms in each condition found no significant effects (p's > .05) for any of the symptoms (irritated eyes, sore throat, congested nose, dizziness, nausea, skin irritation/prickly skin, and excessive mental fatigue), suggesting the level of CO_2 has no effect on any of these symptoms for the duration of exposure in the study.

To allow exclusion of any participants who were likely to be sleepy for reasons unrelated to the study (e.g. lack of sleep the previous night, or not having eaten during the day), the self-reported number of hours sleep received the previous night and the amount of time since participants last ate food were recorded. Hours of slept the previous night varied between 4.3 and 9.5 h (mean 7:36 ± 1:27 h) and time since last ate varied between 5 and 192 min. No participants needed to be excluded.

Analysis showed a significant effect of CO_2 condition on self-reported sleepiness (F (2, 58) = 6.84, p = .002, η_p^2 = 0.19). Sidak post-hoc analyses showed increased sleepiness at both normal (p = .006; meañ: 3.03, SD: 1.13) and high CO_2 (p = .006; mean: 3.10, SD: 1.09)

[1] https://www.mindmedia.com/en/products/nexus-10-mkii/.
[2] https://www.neuroelectrics.com/products/enobio/enobio-20/.

Table 4
Standardised scores for Cognitive Performance Measures (mean ± SD) The CNS software computes scores relative to the population mean: (100, σ: 15) for each measure (see Supplementary Information and CNS Vital Signs Documentation [34]).

Cognitive Performance, within-measures (N = 30)	Baseline	Normal CO_2	Additional CO_2
Reaction Time	92.93 ± 14.08	91.27 ± 16.52	94.13 ± 14.42
Complex Attention	92.13 ± 29.20	92.67 ± 32.27	96.80 ± 18.69
Cognitive Flexibility	93.33 ± 6.23	99.43 ± 14.67	98.80 ± 17.05
Executive Function	94.94 ± 16.24	101.32 ± 14.36	100.00 ± 16.56
Working Memory	104.78 ± 8.75	106.59 ± 11.13	108.26 ± 8.65
Sustained Attention	106.26 ± 5.42	104.93 ± 9.88	106.78 ± 5.87
Simple Attention	99.37 ± 17.22	99.07 ± 10.76	95.15 ± 20.60

relative to baseline (mean: 2.50, *SD*: 0.94).

The Positive And Negative Affect Schedule (PANAS) data were analysed using a 2 (affect: positive, negative) by 3 (condition: baseline, normal CO_2, high CO_2) repeated measures ANOVA. Results showed a main effect of condition (F (2, 58) = 15.45, $p < .001$, $\eta_p^2 = 0.35$), of affect (F (1, 29) = 54.11, $p < .001$, $\eta_p^2 = 0.65$), and a significant condition by affect interaction (F (2, 58) = 7.48, $p = .001$, $\eta_p^2 = 0.21$). Sidak post-hoc analyses show that irrespective of condition, participants had greater positive than negative affect. To analyse the interaction, 3-way repeated measures ANOVAs were conducted for the positive and negative affect separately to investigate the effect of condition. These results show a significant main effect for positive affect only, with higher ratings given at baseline compared to both the normal and high CO_2 conditions (Table 5). These findings suggest that participants became significantly less positive over the course of the study, but not more negative.

3.4. EEG results

EEG data were subject to a 4 (frequency: alpha, beta, high-delta, theta) by 4 (electrode region: central, parietal, temporal, occipital) by 2 (EEG recording session: 1st, 2nd) by 2 (condition: high CO_2, normal CO_2) repeated measures ANOVA. The purpose of the EEG analysis was to determine whether the additional CO_2 caused changes to participants' EEG patterns, in particular, whether these changes were indicative of a progression towards sleepiness.

An increase in sleepiness during the high CO_2 condition using the classification applied (refer Section 2.5) was not found and thus the EEG results do not show any evidence that CO_2 causes increased sleepiness among our participants. Significant main effects were found for frequency (F (1.51, 25.71) = 17.96, $p < .001$, $\eta_p^2 = 0.51$; Greenhouse-Geisser correction applied); regardless of electrode region, EEG recording session, and condition (high/normal CO_2 level), high-delta ($p < .001$) and theta ($p = .002$) had significantly greater power compared to beta, and there was a trend towards high-delta having a greater power than alpha ($p = .07$). Results also found both a significant frequency by EEG session (F (1.90, 32.22) = 3.93, $p = .03$, $\eta_p^2 = 0.19$; Greenhouse-Geisser correction applied) and region by EEG session interaction (F (2.96, 50.33) = 9.97, $p < .001$, $\eta_p^2 = 0.37$; Greenhouse-Geisser correction applied). However, post-hoc analyses using paired-sample t-tests with Bonferroni correction ($\alpha = 0.001$), found that out of 40 comparisons, only 2 were significant: beta at central and temporal electrodes in the high CO_2 condition only (p's < 0.001), with power significantly lower in the 2nd recording session.

Table 5
Average scores (meañ ± SD) for positive and negative affect (PANAS) in each condition.

Condition	Positive Affect	Negative Affect
Baseline	26.07 ± 7.57	12.30 ± 3.16
Normal CO_2	21.73 ± 8.59	11.40 ± 3.30
High CO_2	22.33 ± 7.80	11.60 ± 3.51

Table 6
Skin temperature, respiration rate, and heart rate for each condition and session, mean and SD.

Session	Skin Temperature (°C)	Respiration Rate (breaths/min)	Heart Rate (beats/min)
Normal CO_2 .2	28.7 ± 5.2	16.8 ± 2.4	72.1 ± 7.9
Normal CO_2 .3	27.6 ± 4.6	16.4 ± 2.4	72.1 ± 8.8
Average (Normal CO_2)	28.1 ± 4.8	16.6 ± 2.1	72.1 ± 8.0
High CO_2 .2	28.9 ± 6.1	16.6 ± 2.3	74.5 ± 8.5
High CO_2 .3	28.1 ± 5.3	16.6 ± 2.5	73.9 ± 9.7
Average (High CO_2)	28.5 ± 5.6	16.5 ± 2.3	74.1 ± 8.8

3.5. Physiology results

A 2 (condition; high CO_2, normal CO_2) by 2 (session .2 (1st 8 min eyes open), .3 (2nd 8 min eyes open) repeated measures ANOVA was conducted for skin temperature, respiration rate and heart rate. Means and standard deviations are shown in Table 6.

For skin temperature, a main effect of session was found (F (1, 30) = 17.44, $p < .001$, $\eta_p^2 = 0.37$) with skin temperature in session .2 being significantly higher than session .3, regardless of condition. No significant effects of condition were evident. Respiration rate remained stable over time and was not affected by CO_2 level. For heart rate, a main effect of condition was found (F (1, 30) = 4.85, $p = .04$, $\eta_p^2 = 0.14$), with heart rate in the high CO_2 condition being significantly higher than in the normal CO_2 condition, regardless of session.

3.6. Predicting cognitive performance: regression analyses

Regression models were run for both high and normal CO_2 conditions to assess whether the environmental and/or physiological state immediately prior to completion of the cognitive tests could predict performance on those tests. This was done for only those cognitive tests that showed significant effects of CO_2: cognitive flexibility and executive function.

Variables included were: humidity, temperature, CO_2 level, alpha, beta, high-delta, and theta global power, skin temperature, breathing rate, and heart rate during the first EEG recording session (immediately prior to their taking the cognitive test). Table 7 shows the contribution of each variable to each model.

None of the four overall models were significant. In the normal CO_2 condition, breathing rate and high-delta power are the strongest predictors of cognitive performance, and for the high CO_2 condition, theta power is the strongest predictor, with increases in each of these associated with poorer performance. However, the predictive power of each of these is not sufficient to warrant further investigation.

3.7. Correlations

Correlations were used to assess the relationship between multiple parameters. Table 8 lists the correlations performed and Table 9 presents the results of all *significant* relationships found.

Table 7
Contribution of each of the predictor variables for Cognitive Performance in the High and Normal CO_2 conditions.

N = 22	Normal CO_2						High CO_2					
	Cognitive Flexibility			Executive Function			Cognitive Flexibility			Executive Function		
	B	p	sr^2	B	p	sr^2	B	p	sr^2	B	p	sr^2
Humidity	.29	.62	.01	.35	.54	.02	-.15	.82	.002	-.13	.83	.002
Temperature	2.57	.44	.03	3.17	.34	.04	1.66	.70	.006	1.93	.63	.009
CO_2 Level	-.009	.84	.002	-.00003	1.00	.00	-.01	.75	.004	-.006	.87	.001
Skin Temperature	-.74	.40	.03	-.29	.70	.006	.47	.52	.02	.25	.72	.005
Breathing Rate	−4.52	**.04**	.21	−4.60	**.04**	.22	−1.95	.37	.04	−2.12	.30	.04
Heart Rate	.49	.42	.03	.21	.70	.006	.24	.72	.006	.13	.83	.002
Alpha Global Power	4.25	.18	.08	3.40	.27	.03	-.85	.74	.004	-.82	.74	.004
Beta Global Power	.73	.80	.003	1.01	.72	.20	2.93	.64	.009	2.99	.61	.01
High-Delta Global Power	1.63	*.05*	.19	1.51	*.07*	.09	2.31	.20	.07	2.55	.14	.10
Theta Global Power	−5.54	.15	.10	−5.74	.13	.03	−7.39	*.06*	.18	−7.90	**.03**	.22

NB **bold** = significant at 0.05 level; *italics* = trend towards significance at 0.05 level.

Table 8
Outline of the correlations performed in terms of variables included and number.

Dependent Variables	No. of Correlations	Independent Variables
All physiological parameters (Heart rate, respiration rate, skin temperature)	12	All physiological parameters
	12	Temperature
	12	CO_2 level
	12	Humidity
Cognitive performance	6	Hours since last meal
	6	Hours of sleep
	12	PANAS
Self-reported sleepiness (SSI)[a]	3	Hours since last meal
	3	Hours of sleep
All Global EEG parameters[b]	16	Hours since last meal
	16	Hours of sleep
	16	SSI[a,c]
	48	All physiological parameters
	16	Temperature
	16	CO_2 level
	16	Humidity
PANAS[c]	48	All physiological parameters
	64	All Global EEG parameters

[a] Self-reported sleepiness correlations were conducted using Spearman's method.
[b] Where significant correlations were found, additional analyses were undertaken to ascertain whether the effects were specific to a given brain region.
[c] Only Intermediate and Final tests/questionnaires correlated, given no Baseline EEG was recorded.

A key finding is the significant negative relationship between the estimated number of hours sleep the participant had the previous night with high-delta, theta, and beta power in session .3 of the high CO_2 condition (the 2nd EEG recording session of the high CO_2 condition; Table 9). Results show that the less sleep the participant received the previous night, the closer the EEG results approximated drowsiness (greater power in global high-delta and theta) during the high CO_2 exposure. To examine these findings further, additional correlations were carried out for each electrode region to see if these effects were driven by any one region in particular. Results show that only the temporal region (r = - 0.45, p = .04) was implicated in the high-delta relationship, no other specific regions were implicated for any of the frequencies with global effects. These findings suggest (1) participants who may already be affected by lack of sleep could be more susceptible to the effects of CO_2, whilst (2) supporting EEG as an objective measurement of drowsiness, given the correlation between fewer hours of sleep and EEG indicative of sleepiness.

Table 9
List of significant correlations found, with r and p-values.

Dependent Variables		Condition (CO_2)	Session	r	p
CO_2 level	Skin temperature	Normal	.3	.37	.04
Temperature	Skin temperature	Normal	.3	.41	.02
Hours of sleep	Beta power	High	.3	- .49	.02
	High-Delta power	High	.3	- .56	.007
	Theta power	High	.3	- .44	.04
Alpha power	Breathing rate	Normal	.2	.58	.005
	Temperature	Normal	.2	.45	.04
			.3	.47	.03
	Humidity	High	.2	- .49	.02
			.3	- .50	.02
		Normal	.2	- .57	.005
			.3	- .55	.008
Beta power	Humidity	High	.2	- .44	.04
			.3	- .44	.04
High-Delta power	Breathing rate	High	.3	.44	.04
	CO_2 level	Normal	.3	.55	.008
Theta power	Skin temperature	Normal	.2	.45	.04
			.3	.46	.03
PANAS baseline	High-Delta power	High	.2	.49	.02

4. Discussion

Under normal IAQ conditions (the absence of additional pure-CO_2), participants performed better in the subsequent sessions of cognitive performance testing compared to the first. This learning/practice effect was expected, because participants were repeating the same tests within a period of 60 min. Critically, however, short-term exposure to the raised CO_2 concentration (~2700 ppm of which 1960 ppm ± 160 ppm was pure CO_2 released from a cylinder) did not produce this expected learning effect. While the absence of an expected effect should not be considered indicative of the presence of a negative effect, the findings still provide some support to the notion that CO_2, independent of other indoor pollutants such as human bio-effluents, may affect people's ability on decision-making or cognitive tasks [7–9]. These effects were not associated with a lack of sleep, the time since the participant last ate, or the affective state of the participants. Further work is required into the determinants of changes to cognitive performance/inability to learn a task due to elevated CO_2 concentration.

Self-reported sleepiness was significantly higher at normal and high CO_2 relative to baseline, but was not correlated to CO_2. These results echo those of other studies which find exposure to elevated CO_2 affects cognitive performance, without affecting SBS symptoms or other subjectively measured parameters [11,12]. The findings of this study

provide support to (a) the notion that cognitive performance decrements due to poor IAQ can precede awareness of the declining air quality [11,12], and (b) that relying on discomfort as a reminder to ventilate in naturally ventilated offices may not be sufficient to avoid productivity losses, supporting the recommendation for visual CO_2 feedback in naturally ventilated buildings as a reminder to ventilate [46].

4.1. Physiological and neurophysiological effects

Aside from the absence of learning effect recorded for executive function and cognitive flexibility, the only other factor significantly affected by the high CO_2 condition was heart rate, where a small but significant increase was found relative to the normal CO_2 condition. These findings may indicate the body regulating the effect of CO_2 on blood pH by increasing heart rate to increase circulation, but the lack of capnography or blood-gas analysis in our study prevents confirmation. The findings do, however, reflect studies at much higher CO_2 concentration where heart rate (HR) is found to increase significantly during inhalation of 5% [18] and 7.5% [15] CO_2/air mixtures.

Further work is required to determine whether the small increase in HR observed in this study is proportional to that measured by studies of higher CO_2 [15,18] and whether HR increases linearly with increased CO_2. Unlike Zhang et al. [2] who found exposure to 3000 ppm CO_2 with bio-effluents increased respiration rate (RR) as well as HR, we found no effect on RR.

4.2. Effects on self-reported and objectively (EEG) measured sleepiness

In this study, the high CO_2 condition (2700 ppm) did not affect participants' EEG, suggesting that at this concentration, there is no correlation between EEG and CO_2, and no correlation between EEG and cognitive performance. Much higher concentrations of CO_2 (50,000 ppm [18], and 100,000 ppm [13]) are found to slow EEG, but not affect cognitive performance [13,16,23].

The results indicate participants recorded relatively slow average EEG throughout with higher high-delta and theta values at both normal and high CO_2. We cannot readily explain why all participants had slow resting EEG, however the significant correlation with the number of hours sleep the previous night (Table 9) could be a factor. The scope of investigation precluded repeated resting EEG readings from all participants over a number of days, which could have more accurately determined whether the CO_2 conditions produced deviations from individual participants' baselines.

Self-reported sleepiness was significantly higher at normal CO_2 and high CO_2 compared to baseline. Because there was no effect of session (i.e. the passage of time during each of the two conditions), the sleepiness results cannot be explained as being a function of time. The increase in ratings instead appears due to the environmental conditions experienced during each of the two conditions. It is possible that the increased sleepiness may be caused by the small increases in CO_2 concentration within each of the sessions due to human respiration, but further work is necessary to determine if such small fluctuations in CO_2 can affect sleepiness.

While the EEG findings do not point to any specific neurophysiological drivers of cognitive performance at indoor-realistic CO_2 concentrations, the correlation analysis provides support for EEG as an objective measure of sleepiness. EEG was not affected by CO_2, but the objectively measured sleepiness results correlated closely to the number of hours sleep participants reported having the prior night. Fewer hours of sleep was correlated to EEG readings indicative of sleepiness in the high CO_2 condition after 35 min exposure to increased CO_2 levels. This finding supports findings from sleep medicine, where sleep deprived patients recorded significantly higher values for high-delta and theta than non-sleep deprived participants [21]. Further, it appears plausible that (relative) sleep deprivation may mediate people's response to higher CO_2 concentration. Once exposed to the high CO_2 condition, those participants who self-reported sleeping less hours the previous night showed significantly higher high-delta and theta values (i.e. significantly higher objectively measured sleepiness). Future research might further explore the possibility that (relative) sleep deprivation may amplify or mediate the effect of elevated CO_2 upon sleepiness.

The correlation between EEG results indicative of sleepiness and the number of hours sleep provides support for EEG as a useful measure of sleepiness, and thus by extension, for predicting work performance, given sleepiness negatively affects work performance [28] and ability to concentrate [43]. Objectively measured sleepiness using EEG may provide a better indication of work performance than cognitive performance test batteries; as participants' desire to succeed on cognitive tests have led to findings that subjects can "overcome" discomfort caused by poor environmental conditions and perform well in cognitive tasks in control trials over limited durations [47]. We recommend future work using EEG as an objective measurement of sleepiness due to changes in indoor conditions.

4.3. Limitations

The benefits of this study are that it controlled for time of day, order of exposure (normal/high CO_2), and possible circadian effects (studies starting in the morning/afternoon). However, this study lacked a baseline recording of EEG at very low levels of CO_2. Such a condition would allow insight into whether the high-delta and theta increases seen in both the normal and high CO_2 conditions are due to the increased CO_2, or whether the participants arrived at the lab with an EEG already indicative of a progression toward sleepiness. Such a study would likely need to be conducted within a climate chamber to remove the bio-effluents produced by the participant and researcher during the session. People are not typically accustomed to sitting still for extended periods of time as is required for the EEG equipment; this consideration influenced the decision for a relative short study protocol. Studies wishing to obtain continuous EEG readings over a longer term should consider the likelihood of increased movement/fidgeting with time, which might influence consideration of the type of EEG equipment used.

5. Conclusion

This paper has sought to measure and explain the effects of additional pure CO_2 upon aspects of cognitive performance, physiological, neurophysiological, mood/affect and subjective factors. Findings suggest: (1) a lack of an expected performance improvement in executive function and cognitive flexibility parameters when CO_2 is artificially raised; (2) this lack of expected improvement can occur without changes to SBS symptoms or perceived air quality, can occur after only short duration exposures to the higher CO_2 conditions, and cannot be explained by physiological, neurophysiological or subjective factors. (3) Individuals already lacking sleep may be more susceptible to the effects of CO_2 in enclosed spaces. These findings provide support to the assertion that CO_2 may be a detrimental pollutant itself. More research is necessary to better isolate the effect of CO_2 on cognitive performance relative to other indoor pollutants such as VOCs, human bio-effluents which also rise with reduced ventilation. Irrespective of the relative impacts of CO_2 compared to these other indoor pollutants, this study supports the case for better IAQ feedback to occupants to help redress the negative human performance impacts of under-ventilation [46]. The paper has additionally contributed to the Building and Environment community the use of EEG as a objective indicator of sleepiness, which might be applied to other studies related to the effect of the indoor environment on human performance.

Conflicts of interest

None of the authors have conflicts of interest with the journal.

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