

**Long title**            **Using EEG to characterise drowsiness during short duration exposure to elevated indoor Carbon Dioxide concentrations**

**Short title**            **The effect of CO<sub>2</sub> upon drowsiness**

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1 **Abstract:** Drowsiness which can affect work performance, is often elicited through self-  
2 reporting. This paper demonstrates the potential to use EEG to objectively quantify changes to  
3 drowsiness due to poor indoor air quality. Continuous EEG data was recorded from 23  
4 treatment group participants subject to artificially raised indoor CO<sub>2</sub> concentrations (average  
5 2,700 ± 300 ppm) for approximately 10 minutes and 13 control group participants subject to  
6 the same protocol without additional CO<sub>2</sub> (average 830 ± 70 ppm). EEG data were analysed  
7 for markers of drowsiness according neurophysiological methods at three stages of the  
8 experiment, Baseline, High CO<sub>2</sub> and Post-Ventilation. Treatment group participants' EEG data  
9 yielded a closer approximation to drowsiness than that of control group participants during the  
10 High CO<sub>2</sub> condition, despite no significant group differences in self-reported sleepiness. Future  
11 work is required to determine the persistence of these changes to EEG over longer exposures  
12 and to better isolate the specific effect of CO<sub>2</sub> on drowsiness compared to other environmental  
13 or physiological factors.

14

15 **Keywords:** *EEG; drowsiness; ventilation; CO<sub>2</sub>; office; air quality*

16

17

18 **Practical implications:**

- 19
- This study introduces EEG as a potential objective indicator of the effect of indoor  
20 environmental conditions upon drowsiness
  - Participants exposed to 2,700 ppm for 10 minutes showed greater evidence of a  
21 progression towards drowsiness (as measured by EEG) than that of participants who  
22 received the same protocol without additional CO<sub>2</sub> (mean 830 ± 70 ppm), despite  
23 similar ratings of subjective sleepiness.
  - Subjective and objectively measured indications of drowsiness were reduced following  
24 ventilation of the room. Future work could explore the potential of regular ventilation  
25 episodes in knowledge work spaces to retain alertness.  
26  
27

28

## 29 Introduction

30 Being a product of human respiration, carbon dioxide (CO<sub>2</sub>) increases in indoor spaces when  
31 ventilation of the space is insufficient to replace stale air [1,2]. CO<sub>2</sub> is thus a useful indicator  
32 of ventilation and, by extension air quality indoors, in occupied spaces [3,4]. A large body of  
33 literature exists relating poor ventilation to mild health symptoms [2,5–7] and lowered  
34 cognitive performance [4,8–10]. Office-realistic levels of CO<sub>2</sub> are reported to be typically <  
35 3,000 ppm, but whether CO<sub>2</sub> itself negatively impacts cognitive performance, or whether other  
36 pollutants such as volatile organic compounds (VOCs), including human bio-effluents, are  
37 responsible, is still unclear [11,12]. Human performance effects have been recorded in studies  
38 both where CO<sub>2</sub> is accompanied by human bio-effluents (e.g. the CO<sub>2</sub> concentration is a  
39 product of poor ventilation in occupied spaces) [4,13,14] and where pure CO<sub>2</sub> gas is added to  
40 a room to achieve steady-state concentrations [12,13,15–18].

41 At a room concentration of 3,000 ppm, human bio-effluents are found to cause an increase in  
42 respired -end-tidal- CO<sub>2</sub> (ETCO<sub>2</sub>), increased blood pressure, and seemingly increased  
43 stress/arousal, as well as reduced cognitive performance [19]. Zhang et al. proposes CO<sub>2</sub> with  
44 bio-effluents affects cognitive performance through either (1) stress/arousal or (2)  
45 physiological factors such as an increase in ETCO<sub>2</sub> and reduced nasal peak flow, triggering  
46 symptoms such as subjective (self-reported) sleepiness, tiredness and headache [19]. One study  
47 found that four hours of exposure to non-ventilated conditions, with average CO<sub>2</sub>  
48 concentrations above 2,700 ppm, resulted in significantly increased blood-CO<sub>2</sub>, heart rate  
49 variability, and increased peripheral blood flow, as well as increased prevalence of health  
50 symptoms and self-reported sleepiness [14]. The study reports that the high CO<sub>2</sub> concentration  
51 itself (separate to bio-effluents) may be a parameter affecting physiology which can lower  
52 functional ability and increase (self-reported) sleepiness [14]. Given findings that 1,400 ppm

53 [15] and 2,500 ppm [16] of CO<sub>2</sub> achieved by introducing pure gas into a room correlates to  
54 lower decision making capability, cumulatively, there is some evidence that CO<sub>2</sub> itself,  
55 independent of other indoor pollutants, may play a role in detrimentally affecting aspects of  
56 work performance [15–17].

57 Drowsiness and fatigue are recognised as important parameters affecting office work and  
58 productivity [14,20]. In this study we focus on drowsiness, (i.e. lethargy or wish to sleep) [21–  
59 23], rather than mental fatigue (i.e. exhaustion or lack of motivation for task(s) due to extended  
60 work effort) [24]. Sub-optimal air quality (i.e. poor ventilation/high CO<sub>2</sub>) is correlated to  
61 increased self-reported sleepiness and fatigue [14]. Yet factors such as sleepiness, drowsiness  
62 and fatigue, when reported in studies assessing the effect of indoor conditions on humans, are  
63 often elicited subjectively through questionnaires only [10,14,18,25]. One study uses voice  
64 analysis to as a means of objectively measuring fatigue [20], but this method has not been  
65 widely adopted. The lack of objective measurement of drowsiness or fatigue may be  
66 problematic, given that self-reporting is identified as a less reliable measurement than objective  
67 measurement [26,27]. On the other hand, fields such as Neurophysiology, have a long history  
68 of objectively measuring sleep, and wakeful sleepiness/drowsiness using  
69 electroencephalogram (EEG). EEG records electrical activity in the brain using electrodes  
70 fitted to a cap, or placed on the scalp directly [28]. EEG data can be analysed to: (a) detect  
71 specific events (event-related potential) or (b) time-averaged power in different frequency  
72 bands [28]. A dominance of low frequency power is typically associated with lower  
73 neurological arousal (delta, theta) [22].

74 The impact of office-realistic concentrations of CO<sub>2</sub> upon objectively measured drowsiness is  
75 a knowledge gap in the literature. Temperature effects on drowsiness using EEG find lower  
76 temperatures are correlated to reduced drowsiness [29]. EEG research to date concentrates on

77 neurological effects of much higher concentrations of CO<sub>2</sub> than is likely to occur in indoor  
78 spaces, e.g. 5% CO<sub>2</sub>/air mixture (50,000 ppm) [30–32], or 10% (100,000 ppm) [33] and the  
79 resultant hypercapnia (elevated blood CO<sub>2</sub>) [30,31,33]. In these studies EEG results are  
80 assessed according to arousal state (i.e. overall changes to low-frequency parameters), but not  
81 drowsiness specifically. Xu et al.[31] found inhalation of a 5% CO<sub>2</sub>/air mixture (50,000 ppm)  
82 caused transition to a lower (brain) arousal state, characterised by a relative increase in delta  
83 power and corresponding decrease in alpha power. Bloch-Salisbury [33] subjected participants  
84 to 10% CO<sub>2</sub> (10,000 ppm) through direct inhalation, finding a significant decrease in both  
85 overall power and a movement of the centroid frequency (i.e. the centrepoint of the mass of  
86 frequencies observed) toward lower frequencies.

87 In summary, (1) findings are mixed as to whether CO<sub>2</sub> is a pollutant affecting cognitive  
88 performance in its own right, with some studies finding evidence that CO<sub>2</sub> affects cognitive  
89 performance [15–17], while others find no evidence of this relationship [10,13,18]. (2) Poor  
90 indoor air quality is correlated to increased subjective drowsiness [14], yet drowsiness is  
91 typically elicited through self-reporting [10,14,18,25], which is less reliable than objective  
92 measurement [26,27]. (3) the field of neurophysiology offer methods of objectively measuring  
93 drowsiness (a precursor to sleepiness) using EEG [23,34], yet these methods have not yet been  
94 applied to office-realistic CO<sub>2</sub> concentrations. (4) Literature on the effect of CO<sub>2</sub> on resting  
95 EEG is presently limited to the human effects of much higher levels of CO<sub>2</sub> [31,33] than could  
96 realistically be achieved indoors through human respiration. Comparable studies of office-  
97 realistic concentrations of CO<sub>2</sub> are not yet available, providing impetus for this present paper.

98 This paper details the novel application of using electroencephalogram (EEG) as a means of  
99 objectively measuring the effect of CO<sub>2</sub> on drowsiness at office-realistic concentrations.  
100 Resting EEG and other physiological and subjective parameters were recorded from

101 participants exposed to  $2,700 \pm 300$  ppm of CO<sub>2</sub> in an office for 10 minutes, as a means of  
102 determining the physiological changes of a short-duration exposure to elevated CO<sub>2</sub>  
103 concentration and testing for EEG data indicative of a progression towards drowsiness. A key  
104 aim of the paper is to explore the effect of CO<sub>2</sub> on drowsiness, given that drowsiness is a  
105 determinant of human work performance [20,35] and compare results to both cognitive science  
106 literature on the cognitive performance effects of office-realistic concentrations of CO<sub>2</sub>  
107 [4,8,10,12,15,16] and neurophysiology literature on the neurological effects of much higher  
108 concentrations of CO<sub>2</sub> [30–33].

## 109 **Materials and methods**

110

### 111 **Rationale for study design**

112 Our chosen target for CO<sub>2</sub> concentration (2,700ppm) reflects a high, but realistic level achieved  
113 in occupied spaces when windows and doors are closed [2,14]. In a meta-review of classroom  
114 ventilation, Fisk [2] found six studies of 20 or more classrooms recorded average or median  
115 CO<sub>2</sub> concentrations between 2,000 and 3,000 ppm. The target concentration is chosen to be  
116 comparable with other studies assessing the human performance effects of indoor CO<sub>2</sub>  
117 concentration, e.g. 2,260 ppm [4] 2,500 ppm [16] or 3,000 ppm [10,17,19]. The duration of  
118 exposure to elevated CO<sub>2</sub> concentration in our study is shorter compared to others [4,14,16],  
119 and relates to our aim to record and analyse EEG continuously throughout the experiment to  
120 provide a novel focus on immediate-term physiological effects of CO<sub>2</sub>. Continuous EEG  
121 recording is less practicable over extended study durations due to the need for participants to  
122 remain still during EEG recordings to ensure clean data [28]. The need to remain still over  
123 extended durations, when combined with a lack of stimulation may produce a tendency to  
124 fidget, which may in turn affect measured EEG parameters, or potentially cause boredom/  
125 drowsiness itself, which could confound determination of drowsiness as caused through  
126 changing indoor environment parameters.

127

## 128 **Participants**

129 A total of 47 subjects were recruited and participated in the study between October 2016 and  
130 February 2017. Usable EEG data was available from 36 of the 47 participants, reflective of the  
131 sensitivity of EEG to movement artefacts and the researchers' wish for data reliability. The  
132 study protocol and conditions of participation were approved by the University of Southampton  
133 Ethical Research Governance Office (ERGO# 30443). Sampling was achieved by advertising  
134 the study on billboards throughout the University, a local supermarket and a departmental  
135 mailing list. Convenience sampling was used for contacts of the research team who were  
136 unaware of the study protocol. The final sample was comprised mostly of students and staff  
137 from the University. Written consent was gathered from each participant prior to their  
138 participation in the study. Exclusion criteria for the study were adapted from those used by  
139 Garner et al. [36], a study where participants were subjected to 7.5% CO<sub>2</sub> (75,000 ppm) level  
140 of CO<sub>2</sub>. Exclusion criteria included current or historic drug/alcohol abuse or panic attacks,  
141 current treatment for migraine headaches, pregnant, current neurological conditions (e.g.  
142 epilepsy), and recent severe illness. Participants were compensated £10 in vouchers for an  
143 online retailer for their participation.

144 Participants were split into two groups. Of the participants with usable EEG data, this involved:  
145 23 participants in the "treatment group" (TG) who received artificially raised CO<sub>2</sub>  
146 concentrations and 13 participants in the "control group" (CG) for whom CO<sub>2</sub> concentrations  
147 were not artificially raised (Table 1). The variance in the size of the groups is due to which of  
148 the participants had sufficiently clean EEG for inclusion and the difficulty in recruiting a larger  
149 sample.

150

151 *Table 1- Participant attributes*

<i>Treatment group</i>	<i>Control group</i>
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<i>Male</i>	12	9
<i>Female</i>	11	4
<i>Median age (years)</i>	23.0	24.5

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152

153 Statistical power analysis was calculated a-priori using G\*Power software [37]. Effect size was  
154 estimated at 0.4 based on similar experiments [12], number of groups = 2 (treatment, control),  
155 number of measurements = 3 (Baseline, High-CO<sub>2</sub>, Post ventilation- defined below),  
156 significance level 0.05. This gave a between factors recommendation for 58 participants, and  
157 recommendations for both within-factors and within-between factors of 18 participants. In this  
158 paper we concentrate on within-factors analysis.

159

## 160 **Study room**

161 A motivation for the study was to replicate office-realistic scenarios. All experiments took  
162 place in a small, carpeted, naturally ventilated office of dimensions 4,000 mm by 3,400 mm  
163 (floor area) by 3,050 mm (high) (Figure 1). The office was on the fourth floor, on the northern  
164 end of a large building in the south of England. The office had two high windows on the north  
165 and west corner of the room. Only the western window could be opened, and is visible behind  
166 the participant in Figure 1. The CO<sub>2</sub> cylinder was positioned directly in front of the openable  
167 window. The door of the room led to a larger reception office which was occupied by one staff  
168 member during some but not all of the experiments. The numbered arrows in Figure 1 point to  
169 the location of the CO<sub>2</sub> loggers.

170

171 *Figure 1- Study room showing participant with EEG cap, location of loggers, window and CO<sub>2</sub> cylinder*

172

173 The infiltration rate of the study room with the windows closed was calculated according to  
174 Laussmann et al. [38] using a tracer-gas decay method overnight, with the researcher ensuring



175 the mixing of CO<sub>2</sub> in the room by observing the range of the readings from the three CO<sub>2</sub>  
176 monitors and ensuring all were within instrument error before leaving the room overnight. This  
177 method gave an infiltration rate of  $0.078 \pm 0.002$  ( $R^2 = 0.91$ ) air changes per hour, consistent  
178 with the rubber-sealed windows and minimal air gaps around the door. The value is  
179 approximate, given air exchange rates can differ over time due to differences in temperature,  
180 wind direction and wind speed [39].

181 Carbon dioxide was introduced using a cylinder of ultrapure CO<sub>2</sub> (greater than 99.99% purity)  
182 located in the corner of the room with the outlet attached to pedestal fan to achieve mixing.  
183 The fan was pointed away from the participant and in operation only for the duration of  
184 Condition 3 (see Table 2), when CO<sub>2</sub> was being released, in order to minimise any influence  
185 of air movement on perception or produce possible thermal comfort effects during subsequent  
186 conditions. The target CO<sub>2</sub> concentration once mixed was 2,700 ppm (mean:  $2,700 \pm 300$  ppm  
187 for the duration of Condition 5). Participants were instructed to sit at the table in the middle of  
188 the room while the researcher operated the computer and the gas cylinder behind the  
189 participant. In this way participants were aware the air quality was going to be changed  
190 somehow during the experiment, but were not aware how.

191

## 192 **Experimental Procedure**

193 The experimental protocol took place in the one study room (Figure 1). The study protocol is  
194 summarised below for TG participants (Table 2). CG participants experienced the same  
195 protocol to that of TG participants, except that the CO<sub>2</sub> concentration of the room was not  
196 modified using the cylinder. Instead a pre-recorded and equalized sound was used in place of  
197 the CO<sub>2</sub> gas being released throughout Condition 3 to mimic the sound of the gas release. When  
198 questioned, no CG participant identified the sound as audio playback and thus every participant  
199 assumed their air quality was being modified.

200

201 *Table 2- Experimental protocol*

<b>Condition number</b>	<b>Description</b>	<b>Duration</b>
<i>Pre-start</i>	Ethical consent gathered	
<i>Pre-start</i>	Questionnaire (Baseline)	
1	Eyes closed, window closed, door closed	2 minutes
2	Eyes open, windows closed, door closed	5 minutes
3	Eyes open, windows closed, door closed, CO <sub>2</sub> raised to 2,700ppm, desk fan operational (TG). OR sound played, no CO <sub>2</sub> released (CG)	2-3 minutes (dependent on CO <sub>2</sub> mixing)
4	Eyes closed, window closed, door closed, CO <sub>2</sub> at 2,700ppm (TG) OR CO <sub>2</sub> unchanged (CG)	2 minutes
5	Eyes open, window closed, door closed, CO <sub>2</sub> at 2,700ppm (TG) OR CO <sub>2</sub> unchanged (CG)	8 minutes
6	Eyes open, room ventilated by opening window and door. CO <sub>2</sub> level decreases (TG and CG)	5 minutes
7	As per Condition 6- CO <sub>2</sub> continues to drop	5 minutes

202

203 For comparative data analysis, three two-minute segments were selected for comparison, (1)  
 204 Baseline – the first two minutes of Condition 2; before the environmental conditions were  
 205 changed, (2) High-CO<sub>2</sub> – The last two minutes of Condition 5; beginning when TG participants  
 206 had been exposed to the higher CO<sub>2</sub> concentration for 8 minutes; and (3) Post-Ventilation - last  
 207 two minutes of Condition 7, beginning after 8 minutes of room ventilation. The location of  
 208 these analysis segments within the context of the study protocol are shown in Figure 2.

209

210 *Figure 2- Study protocol with indicative CO<sub>2</sub> level showing location of Baseline, High-CO<sub>2</sub> and Post-Ventilation segments*

211

## 212 Measurement

213 Three factory calibrated Rotronic CL11 (BSRIA, Bracknell, UK) environmental loggers  
 214 measured temperature, humidity and CO<sub>2</sub> concentration throughout each experiment. The

215 loggers were positioned approximately equidistant around the room and are labelled 1, 2 and 3  
216 in Figure 1. The loggers were positioned so as to avoid influence from direct respiration. The  
217 heights of the loggers from the floor were 720 mm (logger 1), 1,545 mm (logger 2) and 1,995  
218 mm (logger 3). The distance from logger 2 to logger 3 was 2,100 mm and logger 1 was  
219 approximately 1,300 mm perpendicular to the participant's heads (Figure 1). Instrument  
220 accuracies for the CL11 are  $\pm 0.3$  ° C (temperature),  $< 2.5\%$  RH (humidity) and  $\pm 30$  ppm  $\pm$   
221 5% of the measured value. The logging frequency of the CL11 monitors was set to 10 seconds  
222 throughout the experiments. The CL11's display updates approximately once per second,  
223 enabling the researcher to monitor and control the release of CO<sub>2</sub> in the room to a reasonable  
224 granularity. The length of Condition 3 (adding CO<sub>2</sub>) was varied according to the time taken to  
225 achieve mixing (Table 2), to enable confidence in the mixing of the room by the start of  
226 Condition 4.

227 EEG data was gathered from each participant using a Neuroelectronics ENOBIO 20 dry electrode  
228 wearable wireless EEG cap (19 channel, 10-20 placement, 500 Hz sampling rate). Two  
229 reference electrodes (DLR, CRL) were positioned on the participants' mastoid muscle. EEG  
230 was gathered continuously throughout each of the experimental conditions (Table 2, Figure 2).  
231 In order to minimise movement artefacts in the EEG, participants were asked to sit quietly and  
232 remain still throughout the experiments except during the short break for the questionnaire  
233 following Condition 5 (refer Table 2, Figure 2).

234 Subjective responses were gathered in relation to experience of sick building symptoms (e.g.  
235 irritated eyes, sore throat, congested nose) [40], positive/negative affect (PANAS) [41],  
236 Stanford Sleepiness Scale [42] and thermal comfort (ASHRAE 7 point scale) [43] were  
237 gathered from participants at Baseline, High- CO<sub>2</sub> and Post-Ventilation segments.

238 As a proof-of-concept, this paper focuses specifically on EEG results and the Stanford  
239 Sleepiness Scale.

240

## 241 *Analysis*

242

### 243 *Environmental measurements*

244 Data from the Rotronic CL11 environmental monitors was downloaded and condition timings  
245 entered retrospectively for analysis. Due to the difference in logging frequency of the CL11s  
246 (10 sec) compared to the EEG measurements (500 Hz), the error on the readings versus that of  
247 the condition timings is expected to be approximately  $\pm 20$  seconds. This error was considered  
248 acceptable given the gradual changes in temperature/humidity and the mixing behaviour of the  
249 CO<sub>2</sub> in the room.

250

### 251 *EEG pre-processing*

252 EEG data were filtered using a Butterworth filter; low pass at 45 Hz and high pass at 0.15 Hz.  
253 Artefact rejection was implemented in two stages. The first used the artefact rejection algorithm  
254 WPT-EMD [44,45], which uses a sample of minimum variance EEG taken from Condition 2.  
255 The second stage of artefact rejection involved an amplitude threshold cut-off of  $\pm 100$   $\mu$ V, and  
256 replacing outlying data with a 10-second moving median around the extreme value. Electrodes  
257 showing consistent noise or flat-lined output were deleted from the dataset. As mentioned, of  
258 the total 47 participants, 36 participants had sufficiently clean data throughout the experiment  
259 and sufficient representation of clean electrodes in each brain region (frontal, central, temporal,  
260 parietal, occipital) to warrant further analysis.

261 Bandpower was extracted from the pre-processed continuous EEG for delta (0.15-3 Hz), theta  
262 (4-7 Hz), alpha (8-13 Hz), beta (14-35 Hz), and gamma (> 35 Hz) frequency bands, over one  
263 second windows. Average bandpower was computed for frontal (F3, Fz, F4, FP1, FP2), central  
264 (C3, Cz, C4), parietal (P7, P3, Pz, P4, P8), temporal (T7, T8), and occipital (O1, O2) electrodes

265 for each analysis segment (Baseline, High-CO<sub>2</sub>, Post-Ventilation). Gamma was excluded from  
266 further analysis owing to the focus of the study protocol on low frequency behaviour and  
267 because gamma represented < 1% of total power at each analysis segment. Post-hoc analysis  
268 found the lowest delta component (0.15-1.5 Hz) to be contaminated with eye movement  
269 artefacts and was subsequently rejected from analysis. Rather than excluding delta from  
270 analysis completely, and given eye movement artefacts typically occur at approximately 1 Hz  
271 [46], we instead report on high-delta (2-3 Hz) and exclude only low-delta (i.e. all frequencies  
272 < 2 Hz).

273 Mixed model ANOVAs were conducted with factors including electrode region, analysis  
274 segment, group, and frequency to investigate electrophysiological markers of drowsiness  
275 consistent with the literature (detailed below).

276

#### 277 *EEG- drowsiness characterisation*

278 Our characterisation of drowsiness applied to the EEG results is grounded in relevant literature:

279 A meta-review of the psychophysiology of automobile driver fatigue finds changes in delta and  
280 theta strongly linked to the transition towards fatigue [21]. Tired wakefulness among sleep  
281 deprived participants produces an EEG with enhanced power in the low frequency range 1-8  
282 Hz (delta and theta) [22,47]. Providing a greater topographical specificity than previous studies,  
283 Gorgoni et al. finds sleep deprived participants exhibit an EEG involving global increases in  
284 delta and theta (i.e. registered in multiple areas of the brain) [23]. Thus in this study, drowsiness  
285 is characterised by post-hoc analysis of the cleaned EEG data according to an increase in delta  
286 and theta, particularly if these increases are found at multiple brain electrode regions.

287

## 288 Results

289 All statistical analyses conducted and reported in this section relate to data from the three  
290 analysis segments of Baseline, High-CO<sub>2</sub> and Post-Ventilation. Additionally, all analyses and  
291 data reported below relate to the 36 participants with usable EEG data.

292

### 293 Indoor conditions by analysis segment

294 Table 3 below summarises the measured indoor environment parameters at each of the two-  
295 minute analysis segments: Baseline, High- CO<sub>2</sub> and Post-Ventilation (Figure 2), for TG and  
296 CG participants:

297

298 *Table 3: Indoor parameters by analysis segment, TG and CG participants*

<b><i>Treatment group participants</i></b>	<b><i>CO<sub>2</sub> (ppm)</i></b>	<b><i>Temp (°C)</i></b>	<b><i>RH<sup>1</sup> (%)</i></b>
<i>Baseline</i>	670 ± 80	21.8 ± 2.3	44.1 ± 8.2
<i>High-CO<sub>2</sub></i>	2750 ± 160	22.2 ± 2.5	44.6 ± 7.9
<i>Post-Ventilation</i>	850 ± 210	21.5 ± 2.4	43.9 ± 7.5
<b><i>Control group participants</i></b>	<b><i>CO<sub>2</sub> (ppm)</i></b>	<b><i>Temp (°C)</i></b>	<b><i>RH<sup>1</sup> (%)</i></b>
<i>Baseline</i>	660 ± 40	23.6 ± 1.8	37.7 ± 7.6
<i>High-CO<sub>2</sub></i>	860 ± 50	24.3 ± 2.0	37.8 ± 7.3
<i>Post-Ventilation</i>	680 ± 80	23.4 ± 1.8	37.8 ± 7.8

299

300 The mean CO<sub>2</sub> values for the two minute segments of Baseline and High-CO<sub>2</sub> correspond  
301 closely to the mean CO<sub>2</sub> values for TG and CG participants for entire five minute duration of  
302 Condition 2 (650 ± 80 ppm TG, 640 ± 50 ppm CG) and eight minute duration of Condition 5  
303 (2,700 ± 300 ppm in TG, 830 ± 70 in CG). With reference to Table 3, TG participants were  
304 exposed on average to an additional 1,898 ppm of pure CO<sub>2</sub> to that generated by human  
305 respiration alone.

306 To control for possible temperature effects, all participants were able to adjust clothing as they  
307 wished prior to the experiment to ensure comfort. A 3 (analysis segment) by 2 (group) mixed  
308 model ANOVA was run to assess temperature fluctuations. Results show that CG participants  
309 were tested at a significantly higher temperature than TG participants (see Table 3 and Section  
310 0;  $F(1, 34) = 6.30, p = .02, \eta_p^2 = .16$ ). This was due to the majority of CG participants being  
311 tested following the activation of the building's heating systems. Results also showed that  
312 temperature varied significantly between each of the analysis segments irrespective of group  
313 ( $F(1.46, 49.55) = 50.75, p < .001, \eta_p^2 = .60$ ; Sidak post-hoc  $p$ 's  $< .02$ ). Temperature was higher  
314 on average for both groups at High CO<sub>2</sub> relative to the other conditions, due to the doors and  
315 windows remaining closed; additionally, Post-Ventilation was colder than both High CO<sub>2</sub> and  
316 Baseline for both groups due to the windows being open throughout the condition and the  
317 cooler outside air due to the season. However, the difference in temperature between analysis  
318 segments (i.e. Baseline vs High CO<sub>2</sub> vs Post-Vent) did not greatly exceed instrument accuracy  
319 (0.3 °C).

320 The period of ventilation (including the Post-Ventilation analysis segment) was uncontrolled.  
321 During this period, CO<sub>2</sub> concentration (Table 3), as well as air change rate, indoor air velocity  
322 and external noise was variable between participants, depending on external factors such as  
323 wind direction, wind speed and traffic. We did not attempt to isolate, measure or control for  
324 these variables, and include the Post-Ventilation segment in our analysis simply as a reference  
325 period of increased fresh air and sensory disturbance.

326

### 327 EEG results

328 To test for the effect of elevated CO<sub>2</sub> concentration upon participants' EEG, a 4 (frequency) by  
329 5 (electrode region) by 3 (analysis segment) by 2 (group) mixed model ANOVA was run.

330 Results found a main effect of frequency ( $F(1.08, 36.58) = 89.62, p < .001, \eta_p^2 = .73$ ), electrode  
 331 region ( $F(1.50, 51.13) = 50.52, p < .001, \eta_p^2 = .60$ ), and analysis segment ( $F(2, 68) = 7.98, p$   
 332  $= .001, \eta_p^2 = .19$ ). In addition significant interactions were also found for frequency by region  
 333 ( $F(1.72, 58.56) = 34.57, p < .001, \eta_p^2 = .50$ ), frequency by analysis segment ( $F(2.09, 70.95)$   
 334  $= 9.16, p < .001, \eta_p^2 = .21$ ), region by analysis segment ( $F(2.98, 101.29) = 7.61, p < .001, \eta_p^2$   
 335  $= .18$ ), and frequency by region by analysis segment ( $F(3.73, 126.84) = 4.91, p = .001, \eta_p^2 =$   
 336  $.13$ ). There was no main effect of group, and no significant group interactions.

337 Post-hoc analysis of the main effects (Sidak) showed that each frequency significantly differed  
 338 from the others ( $p$ 's  $< .004$ ) such that high-delta had the highest power, followed by theta, then  
 339 alpha, then beta. Frontal electrodes had greater power than all other regions ( $p$ 's  $< .001$ ).  
 340 Central and temporal electrodes did not differ from each other and neither did parietal and  
 341 occipital electrodes. Frequency power during Baseline was significantly lower than during the  
 342 High-CO<sub>2</sub> ( $p = .001$ ) analysis segment, but did not differ from Post-Ventilation. There was a  
 343 trend toward the Post-Ventilation analysis segment having a lower overall power than the High-  
 344 CO<sub>2</sub> segment ( $p = .09$ ).

345 To investigate the significant interactions, paired-sample  $t$ -tests were computed between the  
 346 Baseline and High-CO<sub>2</sub> analysis segments and the High-CO<sub>2</sub> and Post-Ventilation analysis  
 347 segments for each brain region and frequency, overall and for the TG and CG participants  
 348 separately (Table 4).

349

350 *Table 4- Overall power, within measures, comparison of changes in power by analysis segment for each group. p-values*  
 351 *derived from paired sample post-hoc t-tests*

Overall power, within-measures		High-CO <sub>2</sub> vs Baseline			Post-Vent vs High-CO <sub>2</sub>		
		Overall	Treatment group	Control group	Overall	Treatment group	Control group
Frontal	h-delta	↑ $p < .001$	↑ $p = .01$	↑ $p = .003$	↓ $p = .004$	↓ $p = .07^a$	↓ $p = .02$
	theta	↑ $p < .001$	↑ $p = .004$	↑ $p < .001$	↓ $p = .07^a$	↓ $p = .77$	↓ $p = .53$



	<b>alpha</b>	↑ <i>p</i> = .07 <sup>a</sup>	↑ <i>p</i> = .31	↑ <i>p</i> = .11	↑ <i>p</i> = .73	↓ <i>p</i> = .83	↑ <i>p</i> = .53
	<b>beta</b>	↑ <i>p</i> = .003	↑ <i>p</i> = .09 <sup>a</sup>	↑ <i>p</i> = .007	↓ <i>p</i> = .47	↓ <i>p</i> = .79	↓ <i>p</i> = .48
<b>Central</b>	<b>h-delta</b>	↑ <i>p</i> = .002	↑ <i>p</i> = .02	↑ <i>p</i> = .05 <sup>a</sup>	↓ <i>p</i> = .04	↓ <i>p</i> = .38	↓ <i>p</i> = .04
	<b>theta</b>	↑ <i>p</i> = .14	↑ <i>p</i> = .02	↑ <i>p</i> = .89	↓ <i>p</i> = .36	↓ <i>p</i> = .52	↓ <i>p</i> = .53
	<b>alpha</b>	↑ <i>p</i> = .40	↑ <i>p</i> = .31	↓ <i>p</i> = .98	↓ <i>p</i> = .32	↓ <i>p</i> = .38	↓ <i>p</i> = .65
	<b>beta</b>	↑ <i>p</i> = .36	↑ <i>p</i> = .43	↑ <i>p</i> = .64	↓ <i>p</i> = .35	↓ <i>p</i> = .57	↓ <i>p</i> = .45
<b>Parietal</b>	<b>h-delta</b>	↑ <i>p</i> = .02	↑ <i>p</i> = .04	↑ <i>p</i> = .27	↓ <i>p</i> = .35	↓ <i>p</i> = .55	↓ <i>p</i> = .48
	<b>theta</b>	↑ <i>p</i> = .01	↑ <i>p</i> = .006	↑ <i>p</i> = .55	↓ <i>p</i> = .16	↓ <i>p</i> = .37	↓ <i>p</i> = .26
	<b>alpha</b>	↑ <i>p</i> = .03	↑ <i>p</i> = .001	↑ <i>p</i> = .92	↓ <i>p</i> = .43	↓ <i>p</i> = .32	↓ <i>p</i> = .88
	<b>beta</b>	↑ <i>p</i> = .03	↑ <i>p</i> = .02	↑ <i>p</i> = .60	↓ <i>p</i> = .46	↓ <i>p</i> = .29	↑ <i>p</i> = .93
<b>Temporal</b>	<b>h-delta</b>	↑ <i>p</i> = .13	↑ <i>p</i> = .33	↑ <i>p</i> = .22	↓ <i>p</i> = .34	↓ <i>p</i> = .50	↓ <i>p</i> = .52
	<b>theta</b>	↑ <i>p</i> = .38	↑ <i>p</i> = .62	↑ <i>p</i> = .38	↓ <i>p</i> = .67	↓ <i>p</i> = .31	↑ <i>p</i> = .70
	<b>alpha</b>	↑ <i>p</i> = .77	↑ <i>p</i> = .87	↑ <i>p</i> = .80	↑ <i>p</i> = .81	↓ <i>p</i> = .58	↑ <i>p</i> = .36
	<b>beta</b>	↑ <i>p</i> = .67	↑ <i>p</i> = .86	↑ <i>p</i> = .64	↓ <i>p</i> = .68	↓ <i>p</i> = .79	↓ <i>p</i> = .75
<b>Occipital</b>	<b>h-delta</b>	↑ <i>p</i> = .009	↑ <i>p</i> = .03	↑ <i>p</i> = .14	↓ <i>p</i> = .07 <sup>a</sup>	↓ <i>p</i> = .15	↓ <i>p</i> = .31
	<b>theta</b>	↑ <i>p</i> = .008	↑ <i>p</i> = .03	↑ <i>p</i> = .16	↓ <i>p</i> = .16	↓ <i>p</i> = .61	↓ <i>p</i> = .08 <sup>a</sup>
	<b>alpha</b>	↑ <i>p</i> = .20	↑ <i>p</i> = .18	↑ <i>p</i> = .53	↓ <i>p</i> = .03	↓ <i>p</i> = .26	↓ <i>p</i> = .02
	<b>beta</b>	↑ <i>p</i> = .04	↑ <i>p</i> = .14	↑ <i>p</i> = .16	↓ <i>p</i> = .21	↓ <i>p</i> = .73	↓ <i>p</i> = .09 <sup>a</sup>
<b>Overall</b>	<b>h-delta</b>	↑ <i>p</i> < .001	↑ <i>p</i> = .007	↑ <i>p</i> = .009	↓ <i>p</i> = .01	↓ <i>p</i> = .11	↓ <i>p</i> = .04
	<b>theta</b>	↑ <i>p</i> < .001	↑ <i>p</i> = .003	↑ <i>p</i> = .006	↓ <i>p</i> = .08 <sup>a</sup>	↓ <i>p</i> = .53	↓ <i>p</i> = .03
	<b>alpha</b>	↑ <i>p</i> = .008	↑ <i>p</i> = .11	↑ <i>p</i> = .03	↓ <i>p</i> = .20	↓ <i>p</i> = .17	↓ <i>p</i> = .65
	<b>beta</b>	↑ <i>p</i> = .01	↑ <i>p</i> = .12	↑ <i>p</i> = .03	↓ <i>p</i> = .35	↓ <i>p</i> = .55	↓ <i>p</i> = .49

<sup>a</sup> Trend (*p* < .10). *Italics* denotes significant *p*-values

352  
353  
354

355 Overall results, irrespective of group, show no changes in the temporal electrode region for any  
356 frequency. The strongest effects from Baseline to High-CO<sub>2</sub> are an increase of frontal high-  
357 delta, theta and beta, central high-delta, and occipital high-delta and theta, as well as global  
358 increases in high-delta, theta, and alpha. Despite a lack of significant group effects in the overall  
359 model, the data presented in Table 4 show a clear difference in the pattern of frequency power  
360 changes across the brain in the two groups. According to the definition of drowsiness employed  
361 (Section 0), the results show the EEG of the TG shows a closer approximation to drowsiness  
362 compared to that of the CG, considering: (a) the increase in delta and theta is more global than  
363 the CG and (b) CG also has a significant overall increase in alpha and beta, while TG increase  
364 is theta and high-delta only.

365

366 **Relationship between EEG and temperature**

367 In order to assess whether any relationship existed between the temperature in the room and  
368 the EEG, Pearson correlations were run for each analysis segment. The results show no  
369 significant correlation between the average temperature during the segment and the global EEG  
370 power of each frequency recorded during that time period. Correlations were also run for each  
371 electrode region. This analysis found a significant negative relationship for alpha power in the  
372 temporal region and temperature during Baseline only ( $r = -.34, p = .04$ ).

373

374 **Self-reported sleepiness (Effect of analysis segment, treatment group, within  
375 measures)**

376 Analysis of questionnaire data on subjective sleepiness found a significant main effect of  
377 analysis segment on self-reported sleepiness,  $\chi^2(2) = 22.84, p < .001$  (Friedman's ANOVA).  
378 Wilcoxon matched pairs post-hoc comparisons show that participants at High-CO<sub>2</sub> had  
379 significantly higher ratings of sleepiness than both Baseline ( $p < .001$ ) and Post-Ventilation ( $p$   
380  $= .01$ ). The Post-Ventilation segment also showed significantly higher ratings of sleepiness  
381 than Baseline ( $p = .01$ ) (Table 5). These p-values remained significant when analysed using  
382 parametric statistics (3-way ANOVA).

383

384 *Table 5- Self-reported sleepiness, average rating with SD, within measures, TG and CG participants*

<b><i>Self-reported Sleepiness, Average Rating <math>\pm</math> SD, within-measures</i></b>		
	<b><i>Treatment group</i></b>	<b><i>Control group</i></b>
<b>Baseline</b>	2.2 $\pm$ .7	2.2 $\pm$ .8
<b>High-CO<sub>2</sub></b>	3.2 $\pm$ 1.1	3.7 $\pm$ 1.0
<b>Post-Ventilation</b>	2.7 $\pm$ 1.2	2.6 $\pm$ 1.0

385 *Stanford Sleepiness index: Likert scale from 1 (wide awake) to 7 (sleep onset soon).*

386

387 The average sleepiness ratings are similar for both TG and CG participants;  $p > .05$  for both  
388 parametric and non-parametric comparisons (Table 5), indicating that subjective sleepiness

389 was not affected by the changes in CO<sub>2</sub> concentration. None of the group comparisons for  
390 sleepiness approach significance.

## 391 Discussion

392  
393 The effect of office-realistic changes to CO<sub>2</sub> on resting EEG represent a knowledge gap in the  
394 literature to date. This study tests the effect of a 2,700 ppm concentration of CO<sub>2</sub> in an office  
395 on resting EEG, analysing EEG results for indicators of a progression towards drowsiness. Data  
396 was analysed at three segments of each experiment; Baseline, High-CO<sub>2</sub> and Post-Ventilation.  
397 This study supports the role of EEG as a means of objectively measuring drowsiness in humans  
398 when affected by changes to the indoor climate.

399

### 400 Evidence for the effect of CO<sub>2</sub> on drowsiness- Relationship between TG and CG 401 participants' EEG

402  
403 Results from this study provide an indication that the indoor CO<sub>2</sub> concentration of 2,700 ppm  
404 had an effect on the EEG indicative of a progression towards drowsiness, when drowsiness is  
405 characterised by a global increase in delta and theta [22,23]. Despite the lack of a significant  
406 effect of group in the overall model, and both groups showing some evidence of a progression  
407 towards drowsiness, the evidence of drowsiness is stronger for the TG (Table 4). A distinct  
408 trend observed among TG participants is the global nature of the high-delta and theta increases  
409 from Baseline to High-CO<sub>2</sub> among TG participants relative to the only frontal increase in these  
410 parameters among CG participants. The findings of this paper reinforce calls for sufficient  
411 ventilation in knowledge work spaces [2] and greater occupant awareness of indoor CO<sub>2</sub>  
412 concentration in these spaces [48].

413 The Post-Ventilation findings show further differences between the TG and CG, where the CG  
414 participants appeared better able to overcome the increased (EEG-assessed) drowsiness  
415 experienced in the High-CO<sub>2</sub> analysis segment. This may imply that the increased CO<sub>2</sub>

416 experienced by TG participants affected the return of the EEG signals to Baseline levels.  
417 However given the difference in sample size between the groups, caution must be taken when  
418 looking at any potential group differences until further research is conducted with larger, more  
419 equal group sizes.

420

#### 421 [Relationship between self-reported and EEG-measured drowsiness](#)

422 The EEG of the TG more closely approximates drowsiness at High CO<sub>2</sub> compared to the CG.  
423 Yet the difference between average self-reported sleepiness ratings at High CO<sub>2</sub> between CG  
424 and TG is minimal (half the standard deviation), and is not significantly different between  
425 groups ( $p > 0.5$ ), (Table 5). Longer exposures to comparable concentrations of CO<sub>2</sub> with bio-  
426 effluents are found to affect (subjectively assessed) drowsiness: 255 minutes exposure to 3,000  
427 ppm with bio effluents increased subjective sleepiness and difficulty in thinking clearly [10];  
428 235 minutes exposure to 2,260 ppm affected perceived fatigue and perceived lack of energy  
429 [4] and four hours' exposure to CO<sub>2</sub> above 2,700 ppm resulted in increased subjective  
430 sleepiness [14]. The duration of this present study is much shorter than other studies and  
431 subjective sleepiness between groups was unaffected. Given the short duration of the study and  
432 the similarity of subjective sleepiness between groups, a possible explanation here is that both  
433 groups self-report higher feelings of sleepiness simply as a function of time (being sat still in  
434 the same room with no stimulation).

435 Further work is required to determine whether the objectively measured drowsiness indicated  
436 in the EEG results persist over longer timescales, whether self-reported drowsiness is better  
437 correlated to EEG over time, and whether EEG may be used as something of an early warning  
438 system for drowsiness. Small changes in CO<sub>2</sub> can quickly affect blood pH [31], and owing to  
439 the short duration of the experiment, it is possible that EEG results may provide a more timely  
440 indication of physiological changes than subjective sleepiness, though this suggestion needs to

441 be corroborated. Additionally, because both subjectively and objectively measured indications  
442 of drowsiness were reduced following ventilation of the room future work could additionally  
443 explore the potential of regular ventilation episodes in knowledge work spaces to retain  
444 alertness.

445

#### 446 **Relationship between EEG and temperature**

447 Results also show a significant effect of temperature with CG participants, completing the  
448 experiment at a slightly higher temperature than TG participants. Temperature in both groups  
449 increased from Baseline to High-CO<sub>2</sub> before dropping to below baseline levels as a result of  
450 the ventilation of the room. Related literature finds lower temperatures (without increased CO<sub>2</sub>)  
451 are correlated to decreased drowsiness as measured by EEG [29], and increasing indoor  
452 temperatures (i.e. warm discomfort) is correlated to difficulty concentrating [49]. These  
453 findings might explain the higher subjective sleepiness experienced by the CG at High CO<sub>2</sub>;  
454 however, as mentioned, the subjective sleepiness ratings were small and not statistically  
455 significant and all participants were invited to modify their clothing if required in order to  
456 remain thermally comfortable throughout the experiment. Conversely, the TG had a higher  
457 objective indication of drowsiness but were subject to cooler temperatures than the CG,  
458 potentially suggestive that (1) the effects on the EEG of the TG in this study may be attributable  
459 to CO<sub>2</sub> rather than temperature and (2) that subjective and objective determinations of  
460 drowsiness may not be correlated over short timescales. Future research could better control  
461 the temperature of the environment to remove this variable as a potential confound.  
462 Additionally, the correlation between objectively and subjectively measured drowsiness due to  
463 changed CO<sub>2</sub> conditions needs to be further explored, e.g. the potential for EEG to act as an  
464 early warning system for drowsiness.

465

## 466 **Limitations and confounding factors**

467 The results of this study should be viewed in light of its limitations: (1) The duration of  
468 exposure in this study is much shorter than comparable studies of office-realistic CO<sub>2</sub>  
469 concentrations on humans [8,10,14,16,50], and future work is required to determine whether  
470 the changes in EEG with respect to drowsiness are momentary or sustained. (2) Accordingly,  
471 changes in the EEG of the TG should be considered as indicative of a neurological progression  
472 towards drowsiness, rather than definitive drowsiness. (2) While the CO<sub>2</sub> outlet was attached  
473 to a fan, mixing may not have been as effective as is possible in a climate chamber. (3) All  
474 participants assumed that gas was released into the room during the experiment, as the CG  
475 participants were exposed to a pre-recorded and equalized sound to mimic the CO<sub>2</sub> gas being  
476 released throughout Condition 3. Thus the participants were blind to the conditions, but were  
477 not blinded to the fact that the air in the room was (supposedly) being modified. Thus it cannot  
478 be ruled out that some CG may have experienced a placebo reaction. (4) The treatment and  
479 control groups differ in sample size and the study is underpowered with respect to between-  
480 groups analysis (a-priory power analysis N = 58, i.e. 29 per group), potentially explaining the  
481 lack of group effects found in the overall ANOVA. However, even after discarding participants  
482 with poor EEG data, the study is still well powered to make conclusions based on the within  
483 subjects analysis (a-priory power analysis n = 18) of the whole sample, and for the TG. As such,  
484 we are confident in our conclusion that the pattern of results found for this group more closely  
485 approximates drowsiness. The study is only slightly under powered with regards to within  
486 subjects analysis for the CG group only.

## 487 **Future work**

489 To corroborate our findings, future work using EEG as an objective indicator of the effects of  
490 changes to indoor air quality would be helpful. To better isolate CO<sub>2</sub> as a variable in future  
491 studies, we suggest a within subjects study design for future work in order to ensure equal  
492

493 representation in the high and “sham” CO<sub>2</sub> groups. Such a design would control for any  
494 individual differences between the groups. Fully blinding participants to experimental  
495 conditions might also be beneficial. In addition, there are personal factors not controlled for in  
496 this study which could feasibly influence drowsiness, such as number of hours sleep, amount  
497 of time since their last meal, their previous activity before experiment. Future studies should  
498 account for such factors. Given our finding that a 10 minute ventilation period appeared to  
499 reverse the trend towards drowsiness (Post-Vent versus High CO<sub>2</sub>), we suggest further work  
500 investigates the acceptability of periodic drafts in naturally ventilated workplaces as a means  
501 of maintaining vigilance and concentration.

## 502 Conclusion

503 Drowsiness represents an important factor affecting office work and productivity [14,20], yet  
504 many studies assessing the effects of poor indoor environment quality on humans gather only  
505 subjective data for factors potentially affecting work performance such as drowsiness or mood.  
506 In this study we have demonstrated the potential for EEG to be used as an objective  
507 measurement of drowsiness to determine the effect of elevated levels of indoor CO<sub>2</sub>. Results  
508 indicate that even short exposure to elevated levels of CO<sub>2</sub> indoors (TG) can produce EEG  
509 indicative of a progression towards drowsiness. Further work is necessary to corroborate these  
510 findings.

511 Priorities for further work have been outlined including: longer-duration studies using EEG,  
512 full blinding to test conditions, accounting for other potential physiological factors which may  
513 affect drowsiness (e.g. including time since last meal, hours of sleep), and the acceptability of  
514 periodic drafts in naturally ventilated workplaces as a means of maintaining vigilance and  
515 concentration.

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522

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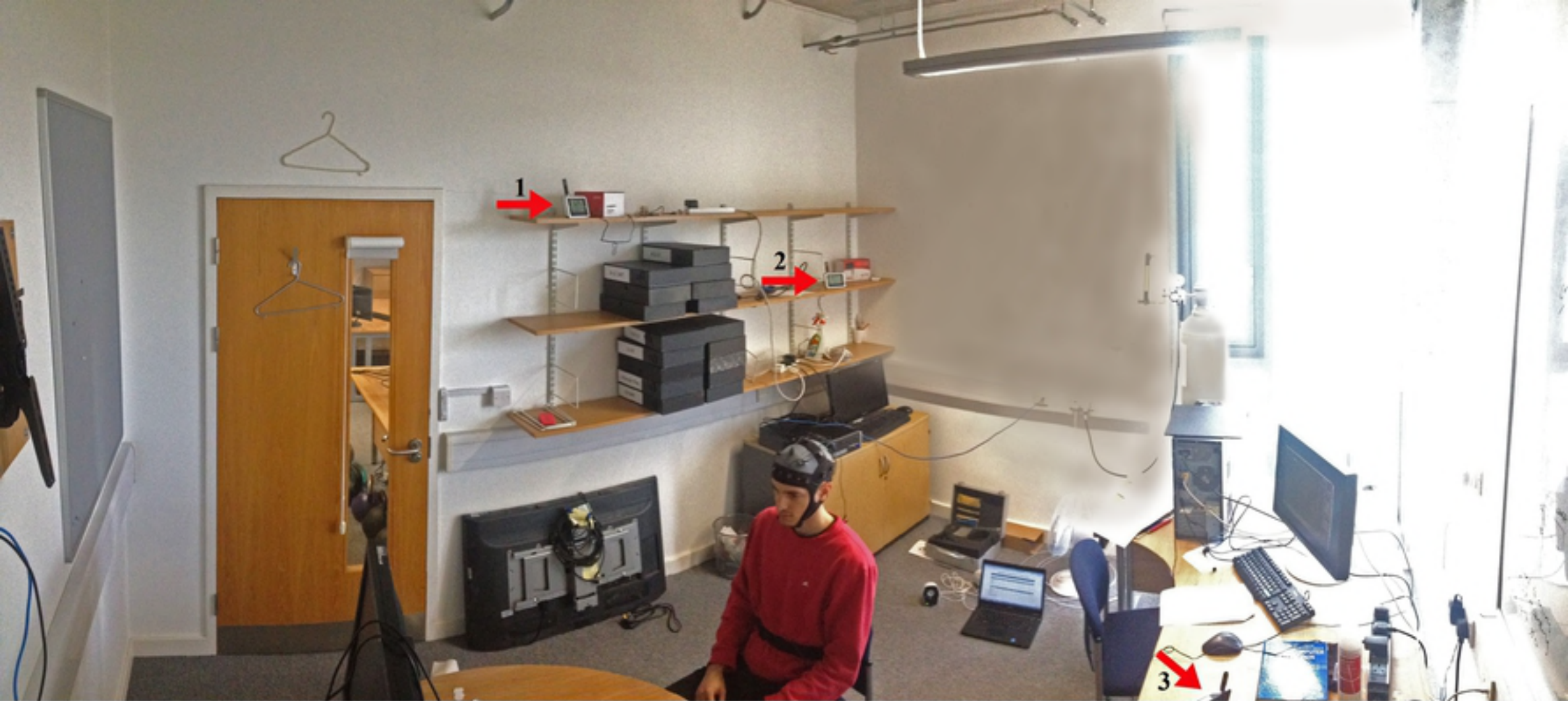


Figure 1

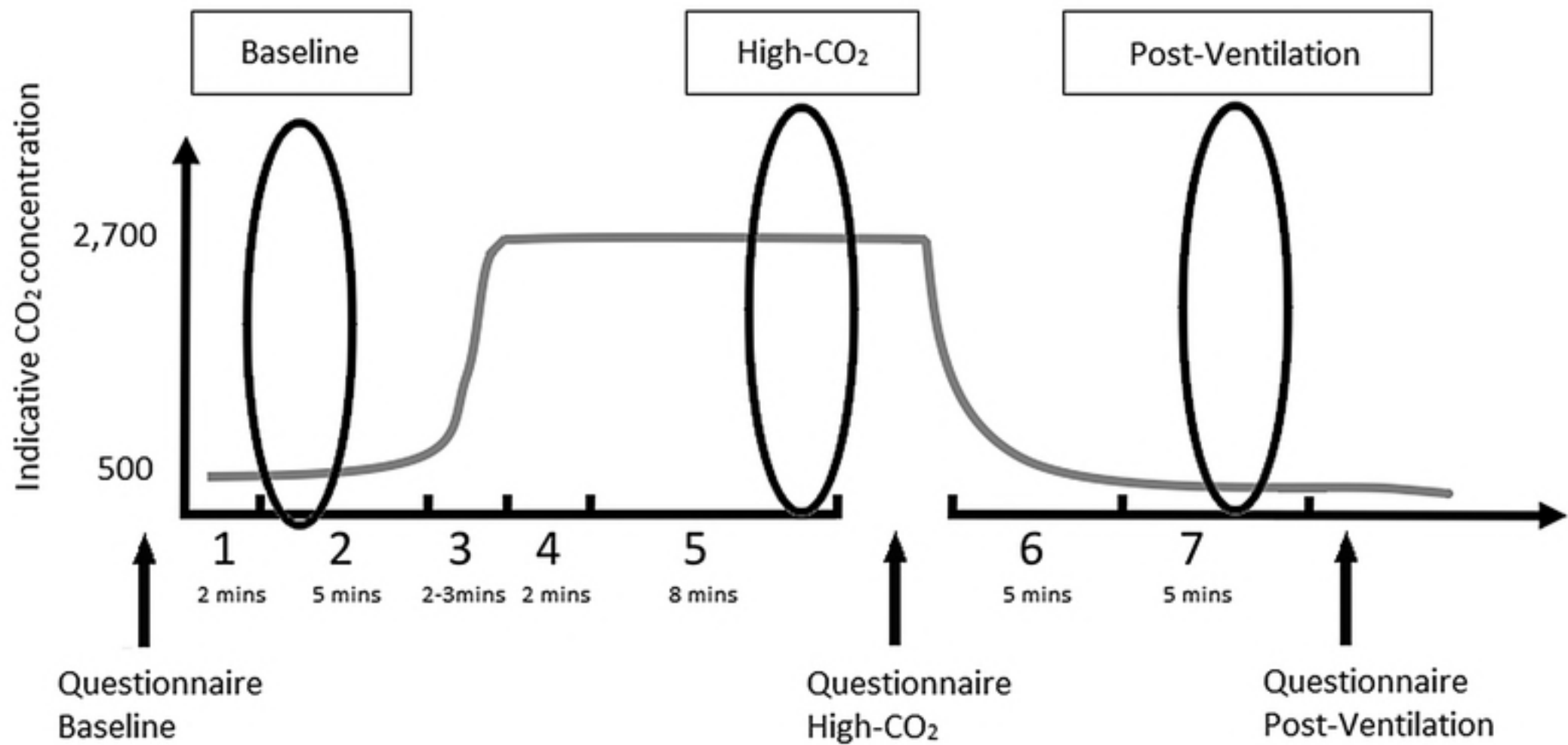


Figure 2