

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

Short title **The effect of CO₂ upon drowsiness**

Author names Stephen Snow¹, Amy Boyson^{1,2}, Marco Felipe-King³, Obaid Malik¹, Louise Coutts¹, Catherine J Noakes³, Hannah Gough⁴, Janet Barlow⁴, m.c.schraefel¹

Affiliations ¹ Electronics and Computer Science, University of Southampton, Southampton, SO17 1BJ, UK,

² Psychology, University of Southampton, Southampton, SO17 1BJ, UK

³ School of Civil Engineering, University of Leeds, Woodhouse lane, LS29JT, Leeds, UK

⁴ Department of Meteorology, University of Reading, Reading, UK, RG6 6AH

Corresponding author, email Stephen Snow, s.snow@soton.ac.uk

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 CO₂ concentrations (average
5 2,700 ± 300 ppm) for approximately 10 minutes and 13 control group participants subject to
6 the same protocol without additional CO₂ (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 CO₂ 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 CO₂ 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 CO₂ on drowsiness compared to other environmental
13 or physiological factors.

14

15 **Keywords:** *EEG; drowsiness; ventilation; CO₂; office; air quality*

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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 CO₂ (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 (CO₂) increases in indoor spaces when
31 ventilation of the space is insufficient to replace stale air [1,2]. CO₂ 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 CO₂ are reported to be typically <
35 3,000 ppm, but whether CO₂ 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 CO₂ is accompanied by human bio-effluents (e.g. the CO₂ concentration is a
39 product of poor ventilation in occupied spaces) [4,13,14] and where pure CO₂ 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- CO₂ (ETCO₂), increased blood pressure, and seemingly increased
43 stress/arousal, as well as reduced cognitive performance [19]. Zhang et al. proposes CO₂ with
44 bio-effluents affects cognitive performance through either (1) stress/arousal or (2)
45 physiological factors such as an increase in ETCO₂ 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 CO₂
48 concentrations above 2,700 ppm, resulted in significantly increased blood-CO₂, 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 CO₂ 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 CO₂ achieved by introducing pure gas into a room correlates to
54 lower decision making capability, cumulatively, there is some evidence that CO₂ 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 CO₂) 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 CO₂ 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 CO₂ than is likely to occur in indoor
78 spaces, e.g. 5% CO₂/air mixture (50,000 ppm) [30–32], or 10% (100,000 ppm) [33] and the
79 resultant hypercapnia (elevated blood CO₂) [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% CO₂/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% CO₂ (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 CO₂ is a pollutant affecting cognitive
88 performance in its own right, with some studies finding evidence that CO₂ 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 CO₂ concentrations. (4) Literature on the effect of CO₂ on resting
95 EEG is presently limited to the human effects of much higher levels of CO₂ [31,33] than could
96 realistically be achieved indoors through human respiration. Comparable studies of office-
97 realistic concentrations of CO₂ 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 CO₂ 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 CO₂ in an office for 10 minutes, as a means of
102 determining the physiological changes of a short-duration exposure to elevated CO₂
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 CO₂ 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 CO₂
107 [4,8,10,12,15,16] and neurophysiology literature on the neurological effects of much higher
108 concentrations of CO₂ [30–33].

109 Materials and methods

110

111 Rationale for study design

112 Our chosen target for CO₂ 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 CO₂ 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 CO₂
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 CO₂ 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 CO₂. 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% CO₂ (75,000 ppm) level
140 of CO₂. 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 CO₂
146 concentrations and 13 participants in the "control group" (CG) for whom CO₂ 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

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-CO₂, 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 CO₂ 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 CO₂ loggers.

170

171 *Figure 1- Study room showing participant with EEG cap, location of loggers, window and CO₂ 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 CO₂ in the room by observing the range of the readings from the three CO₂
176 monitors and ensuring all were within instrument error before leaving the room overnight. This
177 method gave an infiltration rate of 0.078 ± 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 CO₂ (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 CO₂ 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 CO₂ 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 CO₂ 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 CO₂ 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*

Condition number	Description	Duration
<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 ₂ raised to 2,700ppm, desk fan operational (TG). OR sound played, no CO ₂ released (CG)	2-3 minutes (dependent on CO ₂ mixing)
4	Eyes closed, window closed, door closed, CO ₂ at 2,700ppm (TG) OR CO ₂ unchanged (CG)	2 minutes
5	Eyes open, window closed, door closed, CO ₂ at 2,700ppm (TG) OR CO ₂ unchanged (CG)	8 minutes
6	Eyes open, room ventilated by opening window and door. CO ₂ level decreases (TG and CG)	5 minutes
7	As per Condition 6- CO ₂ 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-CO₂ – The last two minutes of Condition 5; beginning when TG participants

206 had been exposed to the higher CO₂ 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 CO₂ level showing location of Baseline, High-CO₂ and Post-Ventilation segments*

211

212 Measurement

213 Three factory calibrated Rotronic CL11 (BSRIA, Bracknell, UK) environmental loggers

214 measured temperature, humidity and CO₂ 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 ± 0.3 ° C (temperature), $< 2.5\%$ RH (humidity) and ± 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 CO₂ in the room to a reasonable
224 granularity. The length of Condition 3 (adding CO₂) 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- CO₂ 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 ± 20 seconds. This error was considered
248 acceptable given the gradual changes in temperature/humidity and the mixing behaviour of the
249 CO₂ 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 ± 100 μ 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-CO₂, 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-CO₂ 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- CO₂ and Post-Ventilation (Figure 2), for TG and
296 CG participants:

297

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

<i>Treatment group participants</i>	<i>CO₂ (ppm)</i>	<i>Temp (°C)</i>	<i>RH¹ (%)</i>
<i>Baseline</i>	670 ± 80	21.8 ± 2.3	44.1 ± 8.2
<i>High-CO₂</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
<i>Control group participants</i>	<i>CO₂ (ppm)</i>	<i>Temp (°C)</i>	<i>RH¹ (%)</i>
<i>Baseline</i>	660 ± 40	23.6 ± 1.8	37.7 ± 7.6
<i>High-CO₂</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 CO₂ values for the two minute segments of Baseline and High-CO₂ correspond
301 closely to the mean CO₂ 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 CO₂ 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 CO₂ relative to the other conditions, due to the doors and
315 windows remaining closed; additionally, Post-Ventilation was colder than both High CO₂ 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 CO₂ 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, CO₂ 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 CO₂ 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-CO₂ ($p = .001$) 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 CO₂ segment ($p = .09$).

345 To investigate the significant interactions, paired-sample t -tests were computed between the
 346 Baseline and High-CO₂ analysis segments and the High-CO₂ 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 ₂ vs Baseline			Post-Vent vs High-CO ₂		
		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	↑ $p < .001$	↑ $p = .004$	↑ $p < .001$	↓ $p = .07^a$	↓ $p = .77$	↓ $p = .53$

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

^a 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-CO₂ 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-CO₂ 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*

<i>Self-reported Sleepiness, Average Rating \pm SD, within-measures</i>		
	<i>Treatment group</i>	<i>Control group</i>
Baseline	2.2 \pm .7	2.2 \pm .8
High-CO₂	3.2 \pm 1.1	3.7 \pm 1.0
Post-Ventilation	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 CO₂ concentration. None of the group comparisons for
390 sleepiness approach significance.

391 Discussion

392
393 The effect of office-realistic changes to CO₂ 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 CO₂ 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-CO₂ 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 CO₂ on drowsiness- Relationship between TG and CG 401 participants' EEG

402
403 Results from this study provide an indication that the indoor CO₂ 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-CO₂ 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 CO₂
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-CO₂ analysis segment. This may imply that the increased CO₂

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 CO₂ compared to the CG.
423 Yet the difference between average self-reported sleepiness ratings at High CO₂ 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 CO₂ 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 CO₂ 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 CO₂ 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-CO₂ before dropping to below baseline levels as a result of
450 the ventilation of the room. Related literature finds lower temperatures (without increased CO₂)
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 CO₂;
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 CO₂ 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 CO₂ 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 CO₂
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 CO₂ 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 CO₂ 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 CO₂ 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” CO₂ 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 CO₂), 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 CO₂. Results
508 indicate that even short exposure to elevated levels of CO₂ 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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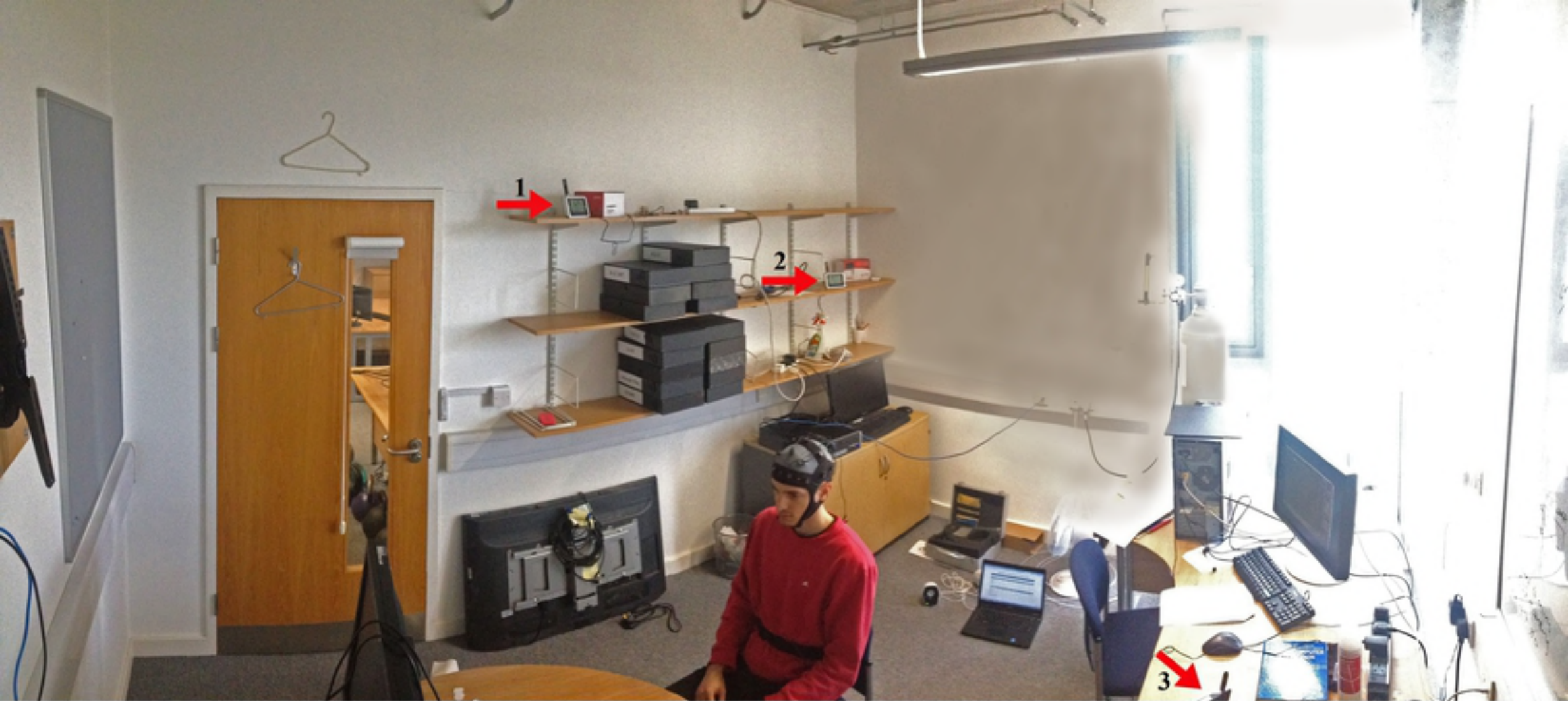


Figure 1

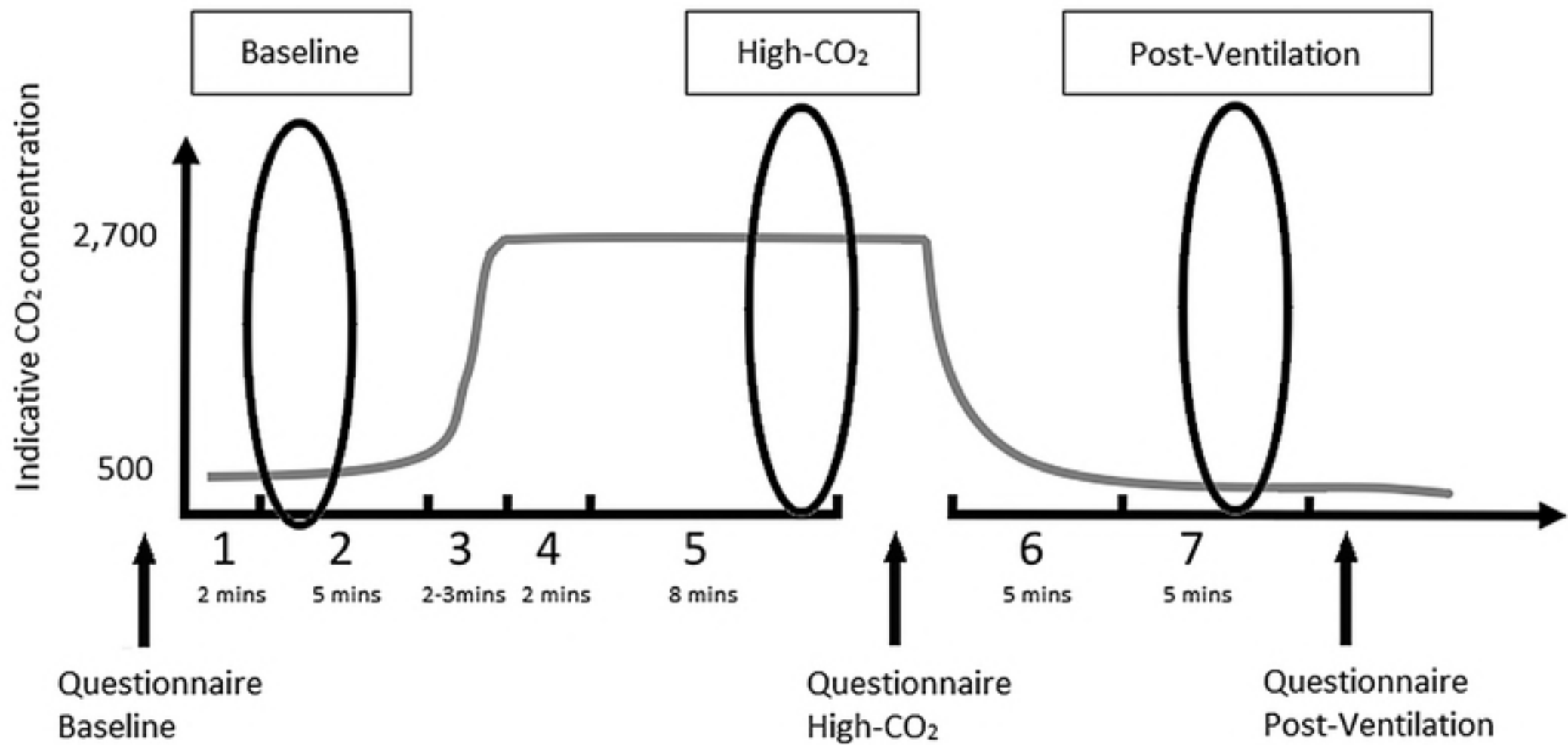


Figure 2