Cerebral oxygenation and cardiac output responses during short repeated sprints exercise and modulatory effect of glucose ingestion

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New findings

1. How does cerebral oxy-, deoxy-, and total haemoglobin change during repeated short cycling sprints.
2. Each repeated short sprints produce a transient increase in O_2Hb, HHb, and tHb, which is positively correlated with cardiac output.
3. After each sprint, O_2Hb, HHb and tHb rapidly return to baseline, despite a still elevated cardiac output.
4. Repeated sprints performance is accompanied with significant decrease in cerebral haemoglobin saturation.
5. Glucose ingestion, although improving performance, does not affect O_2Hb, HHb, and tHb responses to the repeated sprint exercise, but seems to enhance recovery of cerebral tissue oxygenation after repeated sprint exercise.

Abstract

We studied the time profile of changes (Δ) in cerebral oxyhaemoglobin (O_2Hb), deoxyhaemoglobin (HHb) and total haemoglobin (tHb) concentrations during the performance of repeated sprints and its relationship with cardiac output. The modulatory effect of glucose ingestion and acute hyperglycaemia on cerebral haemoglobin responses was also investigated. Ten young male participants performed a set of 10 repeated 6-s sprints on a cycle ergometer after ingesting 70 g of glucose (GLU) or a placebo drink (PLA). Heart rate, stroke volume and cardiac output (impedance cardiography) and ΔO_2Hb, ΔHHb and ΔtHb in the frontal region of the cerebral cortex using near-infrared spectroscopy (NIRS) were continuously measured. Each sprint elicited a significant positive ΔO_2Hb and ΔHHb response that was similar in the two trials. The magnitude of cerebral haemoglobin changes increased with the number of sprint repetitions and was positively correlated with the increase in cardiac output. After each sprint, O_2Hb and HHb rapidly returned to baseline, while cardiac output remained elevated. At the end of the repeated sprints, O_2Hb decreased to below pre-exercise values, whereas HHb and tHb were elevated. After 10-15 min recovery, O_2Hb but not HHb or tHb, returned to pre-exercise values in the PLA, while in the GLU trial O_2Hb increased above pre-exercise values (reactive hyperaemia). We conclude that short sprint exercise induces elevation of O_2Hb, HHb and tHb during the sprint exertion in parallel with the increase in cardiac output. However, together with the transient increase in cerebral haemoglobin, a progressive decline in cerebral oxygen saturation develops during the course of the repeated sprints. Glucose ingestion does not alter cerebral haemoglobin responses to the sprint exercise but seems to be associated with a faster recovery of O_2Hb.

Introduction

Studies measuring cerebral blood flow and blood flow velocity in the internal carotid or middle cerebral artery reveal increased regional and global cerebral blood flow (CBF) during dynamic exercise at moderate-to-severe exercise intensities [i.e., ~ 70% maximal oxygen uptake ($\dot{V}O_{2max}$)] and a return of CBF toward baseline when work rate increases to maximal exhaustive levels (Fisher et al., 2013; Hellstrom et al., 1996; Ide, Boushel, et al., 2000; Jorgensen et al., 1992; Subudhi et al., 2008). Cerebral blood flow at rest and during exercise is tightly regulated in response to changes in arterial blood gases (particularly carbon dioxide), neural activity, blood pressure, and cardiac output (Ainslie et al., 2007; Hoiland et al., 2016; Marsden et al., 2012; Ogawa et al., 2007; Ogoh, Brothers, et al., 2005). In parallel with augmented CBF, cerebral metabolism also rises during dynamic exercise, as shown by the observed decrease in cerebral metabolic ratio, or oxygen-to-carbohydrate index, due to raised cerebral non-oxidative carbohydrate metabolism (Dalsgaard, 2006; Dalsgaard et al., 2004; Ide et al., 1999). Such disparate increase in brain glucose and lactate uptake relative to that of oxygen signals enhanced neural activity (Chuquet et al., 2010; Dienel, 2017; Dienel & Cruz, 2016), and is typically observed during exhaustive exercise (Ide, Schmalbruch, et al., 2000; Rasmussen et al., 2010), when exercise is performed in hyperthermic and hypoxic conditions or when it is performed under increased mental effort (Dalsgaard et al., 2002).

Together with changes in CBF, dynamic exercise also affects cerebral tissue oxygenation (Brassard et al., 2010; González-Alonso et al., 2004). The tissue concentration of oxyhaemoglobin (O_2Hb) and deoxyhaemoglobin (HHb) can be determined non-invasively and with a reasonable spatial resolution (~1-2 cm) using near-infrared spectroscopy (NIRS).

81 According to a meta-analysis of NIRS studies, during submaximal exercise, there is an
82 increase in blood volume and oxygen concentration in the frontal region of the cerebral
83 cortex, which is followed by a decline in cerebral oxygenation when exercise intensity
84 approaches maximal levels, suggestive of inadequate oxygen delivery relative to demand at
85 the end stages of incremental maximal exercise (Rooks et al., 2010). A recent systematic
86 review also reports that prefrontal cortex oxygenation rises with moderate-to-severe
87 intensity dynamic exercise due to increased O_2Hb and unchanged HHb, and declines when
88 exercise intensity increases to near-maximum or maximum intensity (De Wachter et al.,
89 2021). During graded exercise, the breakpoint leading to lowered cerebral oxygenation has
90 been shown to follow the respiratory compensation threshold with a short time lag, as a
91 consequence of cerebral vasoconstriction linked to compensatory hyperventilation-induced
92 hypocapnia (Bhambhani et al., 2007; Globus et al., 1983; Ogoh & Ainslie, 2009; Racinais et
93 al., 2014; Subudhi et al., 2008). The lowered cerebral oxygenation at near-maximal exercise,
94 combined with muscle deoxygenation, may impair muscle recruitment (Racinais et al., 2014)
95 and trigger fatigue, particularly when the exercise is undertaken under hypoxic conditions
96 (Subudhi et al., 2008).

97 Extreme short (<10 s) bouts of all-out sprint interval exercise is an attractive option for
98 achieving cardiorespiratory fitness (Shi et al., 2018), but compared to incremental maximal
99 exercise, the cerebrovascular response during this type of exercise is less documented. A
100 single study investigating cerebral oxygenation, arterial pressure, and cardiac output during
101 30-s all-out cycling exercise performed at normal ambient oxygen levels and in hypoxia
102 showed a linear decrease in middle cerebral artery blood flow velocity (MCA*v*) and a drop in
103 cerebral oxygenation level during the sprint that could only partially be explained by

104 changes in end-tidal carbon dioxide partial pressure ($P_{ET}CO_2$), suggesting that factors other
105 than hypocapnia regulate CBF during high-intensity exercise (Curtelin et al., 2018). Also, the
106 decrease in MCA*v* during the 30-s sprint occurred despite the substantial rise in cardiac
107 output and mean blood pressure (Curtelin et al., 2018). In another study, in which
108 participants performed seven 30-s exercise bouts on a cycle ergometer at a work rate of
109 150% maximal oxygen uptake, the brain's O_2Hb and total haemoglobin concentration (tHb)
110 increased during the first sprint, but with O_2Hb decreasing and HHb increasing from resting
111 levels in the last three sprint bouts, once again without a clear correlation between
112 cerebrovascular response and $P_{ET}CO_2$ (Shibuya et al., 2004). These two studies suggest that
113 contrary to submaximal and maximal exercise during supramaximal exercise CBF and
114 cerebral oxygenation are not strictly dependent on arterial CO_2 levels, blood pressure, and
115 cardiac output. However, 30-s sprints are very exhaustive, with high levels of fatigue and
116 severe drop in power output occurring during a single repetition of this exercise, which
117 could affect cerebrovascular responses. Therefore, the question remains of how cerebral
118 blood volume and oxygenation would change during shorter and repeated sprint efforts and
119 how such changes are related to cardiovascular responses and power output. Repeated
120 sprint-running elicits a high percentage of maximal oxygen uptake, high blood lactate levels,
121 and a cyclic pattern of changes in the difference between O_2Hb and HHb in the contracting
122 muscle whose dynamics follow the sprint/recovery time course (Buchheit et al., 2010), but
123 whether a similar pattern occurs in the brain has not been determined. Also, dynamic
124 cerebral autoregulation responds with a time delay similar to the duration of short sprint
125 bouts (~6 s) (Fantini et al., 2016), which may govern cerebral blood flow response during the
126 performance of a series of short sprints.

127 Blood glucose levels also determine cerebral metabolism and CBF (Xu et al., 2015). Although
128 glucose is the primary energy substrate utilized by the brain, acute hyperglycaemia has been
129 shown to decrease CBF and the cerebral metabolic rate of both oxygen and glucose
130 (Ishibashi et al., 2016; Xu et al., 2015), although opposite findings were reported (Tsuchida
131 et al., 2002). Hence, we also tested the effect of acute glucose intake and subsequent rise in
132 glycaemia in cerebral haemoglobin concentration changes during repeated sprints.

133 Therefore, this study aimed to investigate changes in cerebral O_2Hb, HHb, and tHb during
134 the performance of repeated cycling sprints and their relationship with both cardiac output
135 and power output. The effect of acute hyperglycaemia on cerebral (prefrontal) oxygenation
136 during the repeated sprint was also investigated. A NIRS probe scanning the entire
137 prefrontal cortex was used to assess brain oximetry, which allows compensating for
138 interindividual cerebral circulatory regional differences and to investigate differences in
139 cerebrovascular responses to short intermittent cycling sprints across the frontal region of
140 the cerebral cortex.

141 Methods

142 *Participants and experimental design*

143 Eleven healthy male adults volunteered (Table 1) to participate in this study after providing
144 written informed consent. Participants were free of any metabolic, cardiovascular, or
145 neurological disease and did not regularly participate in exercise training. The study protocol
146 was reviewed and approved by the ethics review board of the University of Macao (Special
147 Administrative Region of Macao, China) and conformed to the ethical principles for research
148 involving human participants (approved number: BSERE22-APP011-FED).

Each participant visited the laboratory on three separate occasions at least one week apart. In the first visit, each participant performed a maximal incremental cycling exercise to determine the individual $\dot{V}O_{2max}$. In the following visits, each participant performed a repeated sprints exercise test on a cycle ergometer after ingesting 70 g of glucose (GLU) or a taste-matched non-caloric placebo (PLA), in a randomised counterbalanced and single blind design.

Instrumentation and measures

Maximal incremental test

The maximal incremental test was carried out on a cycle ergometer (Monark 839E, Varberg, Sweden) to evaluate the individual $\dot{V}O_{2max.}$ After a warm-up at a workload of 25 W for 3 min, participants started cycling at 50 W at a cadence of 60 ± 5 revolutions per minute (rpm), with the workload increasing by 25 W every 3 min. The test was completed when the participant reached volitional exhaustion, with a respiratory exchange ratio (RER) ≥ 1.15, ratings of perceived exertion (RPE) ≥18, and peak HR ≥ 90% of age-predicted maximum (220-age) (Fox 3rd et al., 1971). Respiratory gases were collected by the Vmax Encore System (CareFusion Corp., San Diego, CA, USA) throughout the test, and $\dot{V}O_{2max}$ was calculated as the highest oxygen consumption value averaged over 15 s before the end of the test (Shi et al., 2018).

Repeated sprints cycling exercise

During the two experimental sessions, participants performed a repeated sprints set consisting of 10 repetitions of 6 s maximal cycling sprints interspersed by 24 s resting. The sprints were performed on a cycle ergometer (Monark Ergomedic 894E, Monark Exercise AB,

171 Vansbro, Sweden) connected to a laptop and controlled by software (Monark Anaerobic
172 Test Software, Monark Exercise AB, Vansbro, Sweden). After adjusting the seat height, each
173 participant sat upright on the cycle ergometer, and was instructed to keep the head as
174 steady as possible with the help of a neck cushion to minimise motion artefacts during NIRS
175 recordings. Before the repeated sprints, participants performed a 5-min warm-up, cycling at
176 a power output of 60 W (resistance, 1 kg; cadence, 60 rpm). During the sprints, participants
177 were instructed to use the same ready position, sitting upright and with one leg ready to
178 push powerfully against the pedal and maximally accelerate the cycling crank at the "go"
179 signal. Each participant was free to choose his leading leg in the first session and was asked
180 to use the same leg to initiate all following sprint repetitions. Five seconds into the next
181 sprint, participants moved into the ready position and awaited the verbal go command at
182 the end of a 3-s countdown. A weight equal to 6.5% body mass was applied to the flywheel
183 controlled by the Monark Anaerobic Test Software at the instant the pedalling cadence
184 reached 90 rpm. The controlling software recorded the mean power output for each sprint.

185 *NIRS*

186 A portable 2 x 4-channel continuous-wave NIRS system (Octamon, Artinis Medical Systems,
187 Einsteinweg, The Netherlands) was used to measure changes (Δ) in O_2Hb, HHb, and tHb
188 concentrations using two wavelengths at 750 and 850 nm. Data were acquired with the
189 OxySoft software (OxySoft, Artinis Medical Systems, Einsteinweg, The Netherlands) at a
190 frequency of 50 Hz with a differential path length factor of six. The optodes were attached
191 to a dark ambient light-protecting probe holder, tightly secured to the head by adjustable
192 headbands. The probe was placed over the forehead to scan the underlying prefrontal
193 cortex (four channels on each side). Care was taken to remove the hair under the optodes to

194 ensure optimal optical coupling between optodes and skin. The two light detectors were
195 aligned on Fp1 and Fp2 locations of the 10-20 system for the electroencephalography and
196 surrounded by four light-emitting optodes with an inter-optode distance of 35 mm,
197 providing a total of eight NIRS channels.

198 *Impedance cardiography*

199 Cardiac output was measured continuously during the sessions by signal morphology
200 impedance cardiography (PhysioFlow PF-05, Manatec biomedical, Paris, France). This device
201 measures cardiac output based on changes in thoracic impedance associated with the
202 ejection of blood by the left ventricle in each cardiac cycle, allowing the non-invasive
203 determination of stroke volume and cardiac output (Charloux et al., 2000). After skin
204 preparation to minimise electrical resistance, six Ag/AgCl electrodes (Hewlett Packard 40493
205 E) were placed on the chest wall along the xiphoid process and at the V1 and V6 precordial
206 locations, and at the base of the neck to record the thoracic impedance and
207 electrocardiographic signals. Resting blood pressure was measured by automatic
208 oscillometry to calibrate the cardiac impedance data acquisition. Stroke volume and cardiac
209 output were recorded for each cardiac cycle and measured continuously throughout the
210 trial.

211 *Blood glucose and lactate*

212 Blood glucose measurements were collected with a glucometer (ACCU−CHEK Performa, USA)
213 and the proper test strips (F. Hoffmann-La Roche AG, Basel, Switzerland). Blood lactate was
214 also measured using a handheld device and the appropriate test strips (Lactate Pro 2, AKRAY
215 Europe B.V., Amstelveen, the Netherlands). Both blood analytes were measured in duplicate

216 from capillary blood drawn from a lanceted fingertip at three time points: 1) at baseline,
217 soon after arrival to the laboratory; 2) immediately before starting the exercise protocol;
218 and 3) immediately after completion of the repeated sprints exercise. The two blood sample
219 measurements collected at each time point were averaged for analysis.

220 *Procedures*

221 Participants arrived at the laboratory between 8:00 and 9:00 am after a 12-hours fast. Soon
222 after arrival, participants ingested 300 ml of a drink containing 70 g of glucose (Cambridge
223 Isotope Laboratories, Cambridge, MA, USA) or a taste-matched placebo containing artificial
224 sweeteners (Truvia Calorie-Free Sweetener; Cargill, IL, USA). Afterwards, they rested sitting
225 for the next hour. Fifteen minutes to the end of the one-hour resting, participants were
226 fitted with the cardiac output, and NIRS instrumentations and the two devices were set up
227 for acquisition. The trial started with 10 minutes of seated resting, while wearing a cushion
228 around the neck to reduce head movement.

229 After the 10-min rest, each participant performed a battery of cognitive tests that took
230 approximately 15 minutes to complete (data not shown). Next, they mounted the cycle
231 ergometer and completed the 5-min warm-up followed by the 10 x 6-s repeated sprints.
232 After exercise, there were another 10 minutes resting followed by a second round of
233 cognitive testing (data not shown). Ratings of perceived exertion were obtained at the end
234 of the fifth and tenth sprint repetitions using the Borg's scale.

235 *Data analysis*

236 The NIRS data pre-processing was performed using the Homer2 toolbox (Huppert et al.,
237 2009) for Matlab (Matlab version 9.5.0, release R2018b, The Mathworks Inc., Natick,

238 Massachusetts, USA). First, light absorption data were converted into optical density and
239 haemoglobin concentrations using the modified Beer-Lambert law with a differential path
240 length factor of six. Next, all NIRS signals were low pass filtered at a cut-off frequency of 0.5
241 Hz to remove heartbeat-associated changes in O_2Hb, HHb and tHb. Filtering used a finite
242 impulse response fourth-order Butterworth filter, implemented in the forward and inverse
243 directions to avoid phase filtering artefacts (Huppert et al., 2009). After filtering, the whole
244 NIRS signal was epoched into the individual sprints, starting at minus 5 s and ending at 25 s
245 post the sprint start. Changes in O_2Hb, HHb and tHb were quantified using the median
246 change in the time intervals 0-15 s and 15-25 s, to measure the response during the sprints
247 and during the short time interval separating the sprints, respectively. The median was
248 chosen to quantify ΔO_2Hb, ΔHHb and ΔtHb because of its higher robustness against
249 potential biasing effects caused by motion artefacts and outliers. The time interval 0-15 s
250 was selected following a visual inspection of the transient ΔO_2Hb response accompanying
251 the performance of the sprints (Fig. 3). Because ΔO_2Hb, ΔHHb, and ΔtHb values were not
252 always normally distributed (Supporting information, Figs. S1-S3 and Table S1), they were
253 natural log-transformed for statistical analysis purpose.

254 *Statistical analysis*

255 The Shapiro-Wilk test was used for assessing the normal distribution of data. Repeated
256 measures ANOVA with complete factorial design (cerebral oximetry data: 2 trials x 8
257 channels x 10 sprint repetitions; power output and cardiovascular data: 2 trials x 10 sprint
258 repetitions) was used for testing responses to repeated sprints exercise. The Greenhouse-
259 Geiser correction was applied to within-subject factors violating the sphericity assumption
260 (Mauchly's test p-value < 0.05). In case of significant interaction effects or main effects, post

261 hoc tests were performed employing one-way ANOVA with Bonferroni's correction for
262 family-wise error rate. Significant main effects for repeated sprints were followed by
263 planned contrasts using polynomial fitting, assuming the repeated sprints as an evenly
264 separated quantitative variable. Although polynomial contrast analysis do not specifically
265 compare between two sprint repetitions, it offers relevant insight into the trend effect of
266 the repeated sprints on the response variables. In case of a significant linear contrast,
267 backward differences contrasts were computed (the contrast between two consecutive
268 sprint repetitions, including resting). Further contrasts between the first repeated sprint and
269 the following sprints were also calculated. One sample *t*-test was used for testing the
270 significance of post-exercise mean ΔO_2Hb, ΔHHb and ΔtHb against pre-exercise values
271 (since continuous NIRS measurements are relative, O_2Hb and HHb baseline concentrations
272 are set to zero). Correlation values were computed using the Pearson's coefficient. All
273 statistical tests were performed in RStudio (v. 1.3.1093, 2009-2020 RStudio, PBC) using
274 functions from Rstatix (v. 0.7.0) and R base (v. 4.0.3) packages. Unless otherwise stated, all
275 data are reported as mean ± standard deviation (SD). Significance was accepted at $p < 0.05$.

276 Results

277 The data from one participant were removed from the final analysis due to failure in
278 collecting the NIRS signals, so the final sample size was 10 participants. In one participant,
279 the NIRS signals recorded during three repeated sprints in the two trials were excluded from
280 analysis because of significant motion artefacts. The missing data (48 out of 1600 data
281 points) were replaced with the average from the same individual, channel, and remaining
282 sprints. In one participant (PLA trial), the mean power output in the two final repeated

283 sprints was not measured due to equipment failure. These missing values were replaced
284 with the last mean power output recorded.

285 *Power output and RPE*

286 Figure 1 illustrates the mean power output for each sprint and trial. Overall, the mean
287 power output was higher during the GLU trial than the PLA trial [GLU: 648 ± 183 W; PLA:
288 558 ± 161 W; $F_{(1, 9)} = 6.312$, $p = 0.033$, $\eta^2 = 0.098$]. The higher power output in GLU was the
289 result of enhanced performance during later repeated sprints, in contrast with the PLA trial
290 where power output peaked in the first sprint and decreased thereafter [trial x sprint
291 repetition interaction effect: $F_{(2.79, 25.12)} = 12.314$, $p < 0.000$, $\eta^2 = 0.203$]. One-way ANOVA,
292 testing the effect of trial at each sprint repetition, showed significant differences in mean
293 power output between GLU and PLA for sprint repetition number 1 (PLA higher than GLU), 7
294 and 10 (Bonferroni's corrected $p < 0.05$, Fig. 1). At the end of the fifth sprint, perceived
295 exertion was only moderate (GLU: 11.9 ± 2.1; PLA: 13.2 ± 1.9 units) but increased to
296 maximal or near-maximal values at the end of the repeated sprints set (GLU: 19.3 ± 2.1; PLA:
297 19.3 ± 0.5 units; main effect of sprint repetition, $F_{(1, 9)} = 232.66$, $p < 0.000$, $\eta^2 = 0.856$). No
298 differences between trials or an interaction effect between trial and sprint repetition could
299 be found for RPE scores.

300 *Blood glucose and lactate*

301 Two-way ANOVA revealed a significant effect of time point [$F_{(2, 18)} = 13.143$, $p < 0.000$, $\eta^2 =$
302 0.206] and a significant interaction between time point and trial for blood glucose
303 concentration [$F_{(2, 18)} = 9.666$, $p < 0.001$, $\eta^2 = 0.151$]. At baseline, blood glucose
304 concentration was similar in the two trials (GLU: 5.7 ± 0.5 $mmol \cdot l^{-1}$; PLA: 5.9 ± 0.6 $mmol \cdot l^{-1}$; p

= 0.310), but at the beginning of exercise was significantly elevated with glucose ingestion (GLU: 8.1 ± 1.7 mmol·l^{-1}; PLA: 6.1 ± 0.8 mmol·L^{-1}; p = 0.016). At the end of the repeated sprints, blood glucose concentration was again similar in the two trials (GLU: 6.5 ± 1.6 mmol·l^{-1}; PLA: 5.9 ± 0.9 mmol·l^{-1}; p = 0.254).

Two-way ANOVA revealed a significant effect of time point [$F_{(1.14, 10.3)}$ = 605.698, p <0.000, η^2 = 0.964] and a significant interaction between time point and trial for blood lactate concentration [$F_{(2, 18)}$ = 11.193, p < 0.000, η^2 = 0.156]. At baseline, blood lactate concentration was similar in the two trials (GLU: 1.7 ± 0.9 mmol·l^{-1}; PLA: 1.4 ± 0.4 mmol·l^{-1}; p = 0.335), but increased after glucose ingestion and at the beginning of exercise it was higher in GLU compared to PLA (GLU: 2.4 ± 0.8 mmol·l^{-1}; PLA: 1.6 ± 0.5 mmol·l^{-1}; p = 0.023). Immediately after the repeated sprints, blood lactate was markedly elevated and significantly higher in the PLA trial compared to the GLU trial (GLU: 10.8 ± 1.1 mmol·l^{-1}; PLA: 11.7 ± 1.4 mmol·l^{-1}; p = 0.047).

Heart rate, stroke volume, and cardiac output

Figure 2 depicts the heart rate, stroke volume and cardiac output responses to the repeated sprints. Heart rate increased progressively across the repeated sprints set, rising from a pooled average of 85 ± 4 beats·min^{-1} at rest to 110 ± 5 beats·min^{-1} during the first sprint, further increasing to a peak value of 163 ± 5 beats·min^{-1} in the final sprint [main effect for sprint repetition, excluding resting values: $F_{(3.45, 31.04)}$ = 347.947, p < 0.000, η^2 = 0.884]. No differences in heart rate existed between GLU and PLA trials [$F_{(1, 9)}$ = 0.018, p = 0.897, η^2 < 0.000]. Also, no interaction effect between trial and sprint repetition could be found for heart rate [$F_{(2.67, 24.03)}$ = 0.885, p = 0.453, η^2 = 0.023].

Stroke volume increased from 84.2 ± 5.8 ml at rest to 97.3 ± 8.4 ml during the first sprint, but reached a plateau after the fourth sprint repetition [main effect for sprint repetition, excluding resting values: $F_{(2.47, 22.25)} = 39.546$, $p < 0.000$, $\eta^2 = 0.380$]. No differences between GLU and PLA [$F_{(1, 9)} = 0.195$, $p = 0.669$, $\eta^2 = 0.005$] or an interaction effect between trial and sprint repetition [$F_{(2.14, 19.28)} = 1.199$, $p = 0.326$, $\eta^2 = 0.020$] could be found for stroke volume during the repeated sprints.

Cardiac output increased during the repeated sprints with a curvilinear pattern, rising from a resting cardiac output of 7.2 ± 0.7 $l \cdot min^{-1}$ to a cardiac output of 10.7 ± 1.0 $l \cdot min^{-1}$ in the first sprint and 19.1 ± 1.1 $l \cdot min^{-1}$ at the end of the repeated sprints [main effect for sprint repetition, excluding resting values: $F_{(2.59, 23.31)} = 214.561$, $p < 0.000$, $\eta^2 = 0.792$]. Cardiac output was similar in the GLU and PLA trials [$F_{(1, 9)} = 0.280$, $p = 0.610$, $\eta^2 = 0.005$], and no interaction effect between trial and sprint repetition was found for cardiac output [$F_{(1.93, 17.38)} = 1.122$, $p < 0.346$, $\eta^2 = 0.025$].

In the time intervals between sprints, cardiac output remained elevated, with only a small albeit significant decrease from its level during the sprints [overall mean ± SD during sprinting, 16.2 ± 2.8 $l \cdot min^{-1}$; in between sprints, 16.1 ± 2.8 $l \cdot min^{-1}$; $F_{(1, 9)} = 6.084$, $p = 0.036$, $\eta^2 = 0.003$]. Polynomial contrasts revealed a significant linear and quadratic effect of sprint repetition on heart rate, stroke volume and cardiac output responses. Several backward differences contrasts were also significant (see Fig. 2 for details).

Brain oximetry

Table 2 presents the results for ΔO_2Hb, ΔHHb, ΔtHb during the repeated sprints and the time interval between the sprint repetitions.

Oxyhaemoglobin

During the sprints, there was a rapid increase in O_2Hb that peaked near the end of the sprints (Fig. 3; Supporting information, Fig. S4). As for cardiovascular parameters, ΔO_2Hb across the sprints followed a curvilinear pattern. Therefore, three-way repeated measures ANOVA found a significant main effect of sprint repetition on log-transformed O_2Hb concentration response [$F_{(9, 81)} = 3.380$, $p = 0.001$, $\eta^2 = 0.074$]. However, no significant effect could be found for glucose ingestion [$F_{(1, 9)} = 0.930$, $p = 0.360$, $\eta^2 = 0.005$] or NIRS channel [$F_{(7, 63)} = 10.222$, $p = 0.979$, $\eta^2 = 0.002$]. Polynomial fitting of ΔO_2Hb concentration values showed a significant linear effect of sprint repetition on ΔO_2Hb without additional improvement of fitting higher-order components. Further contrast analysis revealed a significantly lower ΔO_2Hb in the first sprint compared to the fourth sprint and the next ones ($p < 0.05$). No backward difference contrasts were found for ΔO_2Hb between two consecutive sprints. After sprint cessation, O_2Hb concentration dropped to pre-sprint levels or below (Fig. 3, Table 2). No significant differences between trials, channel, or sprint repetitions could be found for ΔO_2Hb values in the time interval between sprints.

Deoxyhaemoglobin

Compared to ΔO_2Hb, ΔHHb responses to the repeated sprints were of a smaller magnitude (Table 2, Fig. 3; Supporting information Fig. S4). No significant differences in ΔHHb were found across the repeated sprints [$F_{(9,81)}= 1.446$, $p = 0.182$, $\eta^2 = 0.01$] or NIRS channels [$F_{(7,63)}= 1.531$, $p = 0.173$, $\eta^2 = 0.02$]. Also, ΔHHb was similar in the two trials [$F_{(1, 9)}= 0.476$, $p = 0.508$, $\eta^2 = 0.000$] (Table 2). During the time interval separating the repeated sprints, HHb returned towards pre-exercise levels, with no differences between trials [$F_{(1, 9)}= 0.287$, p =

371 0.605, η^2 = 0.000] or NIRS channel [$F_{(7,63)}$= 1.162, p = 0.337, η^2 = 0.01], but with a significant
372 main effect of sprint repetition [$F_{(9, 81)}$= 2.152, p = 0.034, η^2 = 0.012]. However, polynomial
373 contrasts found only marginal linear (p = 0.052) and quadratic (p = 0.057) effects of
374 repeated sprints on ΔHHb in the time interval between sprints.

375 *Total haemoglobin*

376 ΔtHb resembled ΔO_2Hb, with a marked increase during each sprint bout, followed by a fast
377 decline towards baseline immediately after the end of the sprint, without any effect of
378 glucose ingestion [$F_{(1, 9)}$= 0.970, p = 0.350, η^2 = 0.007] or NIRS channel [$F_{(7, 63)}$= 0.403, p =
379 0.897, η^2 = 0.006]. A significant interaction effect between trial and NIRS channel was found
380 for ΔtHb [$F_{(7, 63)}$ = 2.221, p = 0.044, η^2 = 0.009]. However, when testing the difference
381 between GLU and PLA trials at each channel, no significant differences were found after
382 correction for multiple comparisons. Also, there was a significant difference in ΔtHb across
383 the repeated sprints [$F_{(9, 81)}$= 2.796, p = 0.007, η^2 = 0.054], with a significant linear effect (p =
384 0.008) and with smaller ΔtHb in the first sprint as compared to all the other sprint
385 repetitions (0.003 < p < 0.046) excepting to the third sprint (p = 0.061). Regarding backward
386 difference contrasts, only the difference between the second and first sprints was found
387 significant (p = 0.046). No differences between trials [$F_{(9, 81)}$= 2.017, p = 0.189, η^2 = 0.002],
388 NIRS channels [$F_{(7, 63)}$= 1.016, p = 0.429, η^2 = 0.017] or sprint repetitions [$F_{(9, 81)}$ = 0.866, p =
389 0.560, η^2 = 0.010] were found for ΔtHb during the time interval between sprint repetitions.

390 We also compared the O_2Hb, HHb, and tHb concentrations at the onset of the warm-up, at
391 the end of exercise, and at 10-15 min post-exercise (see Fig. 3, top and middle panels).
392 Because of the small negative ΔO_2Hb after each sprint repetition, at the end of the repeated

sprints set O_2Hb concentration had decreased significantly relative to the time before the warm-up (one sample *t*-test, GLU and PLA pooled together: $t(8) = -2.644$; $p = 0.029$, $n = 9$). This decrease in O_2Hb concentration at the end of the whole repeated sprints set was not affected by glucose ingestion [GLU: -1.16 ± 4.13 μM; PLA: -3.11 ± 2.87 μM; $F_{(1, 7)} = 2.453$, $p = 0.161$, $\eta^2 = 0.096$, $n = 9$]. Contrary to O_2Hb, cerebral HHb concentration increased steadily throughout the repeated sprints set and at the end of the exercise, it was significantly elevated compared to pre-exercise (one sample *t*-test, GLU and PLA pooled together: $t(8) = 3.248$, $p = 0.011$, $n = 9$), without any effect of glucose ingestion [GLU: 3.80 ± 5.95 μM; PLA: 6.44 ± 3.82 μM; $F_{(1, 7)} = 1.541$, $p = 0.254$, $\eta^2 = 0.042$, $n = 9$]. Due to the increase in HHb, tHb at the end of the repeated sprints set was significantly elevated compared to pre-exercise (one sample *t*-test, GLU and PLA pooled together: $t(8) = 3.119$, $p = 0.014$, $n = 9$) and irrespectively of glucose ingestion [GLU: 3.98 ± 5.60 μM; PLA: 5.75 ± 3.86 μM; $F_{(1, 7)} = 0.670$, $p = 0.440$, $\eta^2 = 0.014$, $n = 9$]. After exercise, O_2Hb started to rise towards pre-exercise level. The increase in O_2Hb during recovery was enhanced in the GLU trial [GLU: 2.54 ± 1.80 μM; PLA: 0.95 ± 2.32 μM; $F_{(1, 7)} = 11.263$, $p = 0.012$, $\eta^2 = 0.140$, $n = 9$]. As a result, post-exercise O_2Hb was above pre-exercise values in the GLU trial (one sample *t*-test, $t(8) = 8.099$, $p < 0.000$, $n = 9$) but not in the PLA trial (one sample *t*-test, $t(8) = 2.018$, $p = 0.08$, $n = 8$). At 10-15 min post-exercise, HHb remained similarly elevated in both trials [GLU: 3.42 ± 4.02 μM; PLA: 3.55 ± 3.15 μM; $F_{(1, 7)} = 0.023$, $p = 0.884$, $\eta^2 < 0.000$, $n = 9$] compared to pre-exercise values (one sample *t*-test, GLU and PLA pooled together: $t(8) = 4.557$, $p = 0.001$, $n = 9$). Following the changes in HHb and O_2Hb, tHb concentration also rose after the repeated sprint exercise in both trials [Glu: 3.29 ± 4.07 μM; Pla: 3.12 ± 3.31 μM; $F_{(1, 7)} = 0.050$, $p = 0.829$, $\eta^2 < 0.000$, $n = 9$] and at 10-15 min post-exercise, it was above pre-exercise level (one sample *t*-test, GLU and PLA pooled together: $t(8) = 3.882$, $p = 0.005$, $n = 9$).

Cardiac output and ΔO_2Hb, ΔHHb and ΔtHb relationship

Figure 4 presents the scatter plots with the relationship between cardiac output, power output, and cerebral haemoglobin concentrations (see also Supporting information, Fig. S5). During the sprint exercise, ΔO_2Hb and ΔtHb were positively correlated with cardiac output in both GLU (CO: ΔO_2Hb, R = 0.85, p = 0.0019; CO: ΔtHb, R = 0.86, p = 0.0013) and PLA trials (CO: ΔO_2Hb, R = 0.87, p = 0.0012; CO: ΔtHb, R = 0.85, p = 0.0016). No significant correlation was found between cardiac output and ΔHHb during the repeated sprints. Also, no correlation was found between cardiac output and ΔO_2Hb, ΔHHb and ΔtHb during the time interval between the sprints. During sprints, ΔO_2Hb was negatively correlated with power output in the PLA trial. In the GLU trial, power output was negatively correlated with ΔO_2Hb values during the time interval between the sprints.

Discussion

In this study, we sought to describe the time course of cerebral O_2Hb, HHb and tHb response to the performance of a series of short, repeated sprints and assess the relationship between this response and cardiac output. Additionally, we explored the modulating effect of acute hyperglycaemia on O_2Hb, HHb and tHb changes in response to the repeated sprint exercise. We showed that each of the short, repeated sprints produced a rapid and substantial increase in O_2Hb and, to a lesser extent HHb, resulting in a transient elevation of cerebral blood volume (i.e., increased tHb) and cerebral oxygenation. In the time interval between the repeated sprints, O_2Hb and HHb returned toward or below pre-exercise levels also in a short time. The magnitude of ΔO_2Hb and ΔtHb followed a curvilinear

438 pattern across the repeated sprints set that resembled the heart rate and cardiac output
439 responses to the repeated sprint exercise.

440 The short sprints in this study elicited an approximately ten-fold increase in O_2Hb, compared
441 to the pulsatile peak increase due to the heartbeat at rest (~0.5 µM). Increased cardiac
442 output and cerebral perfusion pressure, the latter due to elevation of arterial blood
443 pressure, are two credible reasons for this transient increase in O_2Hb and HHb during each
444 short sprint (Cifford et al., 1994; Curtelin et al., 2018; Pott et al., 1997). Although blood
445 pressure was not recorded in this study, an increase in mean blood pressure is expected to
446 occur during all-out sprint exercise, based on previous studies revealing that both heavy-
447 intensity dynamic (e.g., rowing exercise) and resistance (e.g., leg-press exercise) types of
448 exercise cause a rapid increase in diastolic and systolic blood pressures (Cifford et al., 1994;
449 Curtelin et al., 2018; Edwards et al., 2002; Perry & Lucas, 2021). While enhanced central
450 drive and activation of the metaboreflex cannot be discarded as mechanisms contributing to
451 the sprint-induced elevation of blood pressure (Gandevia & Hobbs, 1990), the immediate
452 and transient increase in O_2Hb and HHb during the short sprint exercise suggests a role for
453 mechanical and haemodynamic factors associated with the powerful contraction of the leg
454 and trunk muscles, resulting in brief Valsalva-like manoeuvres and in synchronous elevation
455 of arterial blood pressure (Perry & Lucas, 2021; Pott et al., 1997; Sejersen et al., 2021; Zhang
456 et al., 2004). Although more often associated with heavy static and dynamic resistance
457 exercise (Perry & Lucas, 2021; Pott et al., 2003), Valsalva-like manoeuvres are also
458 characteristically observed during dynamic exercise, such as during the catch phase of
459 rowing (Pott et al., 1997; Sejersen et al., 2021), where there is a cyclical increase in MCAv
460 synchronous with the elevation of intra-abdominal pressure, central venous pressure, and

catch force (Pott et al., 1997). Similarly, the onset of bilateral static leg extension concomitantly rises mean arterial blood pressure, MCAv and cerebral O_2Hb concentration with or without a superimposed Valsalva strain (Pott et al., 2003). Whether a similar Valsalva-like manoeuver occurs during cycling sprints is unclear. However, participants in this experiment cycled against a considerable load (i.e., 6.5% body weight; mean load, 5.8 kg), maintaining high levels of power output (pooled average ~ 600 W) during the entire sprint duration. This power output level is close to that reported during 10 maximal leg press resistance exercise in a fitter population (Gorostiaga et al., 2012), an exercise type that has been shown to increase mean arterial pressure, MCA*v*, and CBF (Perry & Lucas, 2021). Also, the simultaneous contraction of the leg and abdominal muscles during cycling sprint might have produced a transient increase in cardiac output, due to the combined action of the muscle and respiratory pumps (Aliverti et al., 2009).

Cardiac output directly influences CBF and cerebral oxygenation independently from changes in arterial blood pressure (Castle-Kirszbaum et al., 2022; Lie et al., 2021; Ogoh, Brothers, et al., 2005). In our study, cardiac output values during the sprints were moderately and positively correlated with ΔO_2Hb and ΔtHb, which suggests that cardiac output might contribute to changes in CBF and cerebral oxygenation during the repeated sprints. Cardiac output progressively increased over the repeated sprints set, which was akin to the transient ΔO_2Hb response to each sprint exercise, thus suggesting that cardiac output was at least partly driving the rise in O_2Hb during the sprints. The ability of cardiac output in raising CBF independently from cerebral perfusion pressure is controversial. During static leg exercise, changes in MCAv seems to develop in divergence with cardiac output (Pott et al., 2003), but a direct relationship between cardiac output and MCAv has been reported

484 during rest and moderate intensity cycling exercise (Ogoh, Brothers, et al., 2005). However,
485 an enhanced blood pressor response with increasing number of sprint repetitions, as a
486 result of enhanced central drive and exercise pressor reflex (Gandevia & Hobbs, 1990;
487 Stebbins et al., 2002) or stronger Valsalva manoeuver as a response to the developing
488 fatigue and increased feelings of exertion with increasing number of sprint repetitions
489 (Perry et al., 2014), could have also contributed to the increased ΔO_2Hb and ΔtHb across the
490 repeated sprints set, irrespective of cardiac output. Moreover, and in spite of its relationship
491 with O_2Hb and tHb during the sprints, cardiac output seems to have exerted little influence
492 on cerebral haemoglobin concentration levels in the intervals between the repeated sprints.
493 After each sprint, there was a clear deviation between cardiac output and cerebral
494 haemoglobin concentration, with O_2Hb and tHb rapidly returning to baseline despite cardiac
495 output remaining elevated. Therefore, a high cardiac output seems unsufficient to raise
496 O_2Hb and tHb without additional mechanisms that are acting during the sprint exertion. The
497 simplest explanation for the fast decrease in cerebral O_2Hb and tHb concentrations with the
498 end of the sprint is an also sudden decrease in mean arterial blood pressure upon cessation
499 of the heavy cycling exercise (Curtelin et al., 2018; Edwards et al., 2002). Curtelin et al.
500 (Curtelin et al., 2018) reported a drop in mean blood pressure immediately after the end of
501 a Wingate test, which was approximately three times larger than its increase during the
502 effort. Moreover, this decrease in mean blood pressure was accompanied by a significant
503 decline in MCAv, which lasted for approximately 40 s before returning to pre-exercise levels
504 (Curtelin et al., 2018). However, a decrease in CBF at the end of exercise might occur
505 independently from changes in arterial blood pressure. Pott et al. (Pott et al., 1997)
506 reported a continuous decrease in MCAv after each rowing's catch movement in spite of
507 stable mean blood pressure, which these authors suggest could have been caused by the

508 delayed cerebral autoregulation response. A similar event could have taken place during
509 the repeated sprints, considering the match between the repeated sprints duration and the
510 phase delay of cerebral autoregulation (Fantini et al., 2016).

511 The performance of the repeated sprints exercise was accompanied with decreased cerebral
512 O_2Hb and increased in HHb, although with sizeable interindividual variation (Fig 3). The rise
513 in HHb overcame the decrease in O_2Hb, hence cerebral tHb was also increased at the end of
514 the repeated sprints exercise. These alterations in cerebral oxygenation partially agree with
515 MCAv data recorded during the Wingate test, revealing decreased CBF and cerebral
516 oxygenation levels caused by augmented cerebral vascular resistance at later sprint stages
517 (Curtelin et al., 2018). A similar response is observed during heavy resistance exercise, with
518 cerebrovascular resistance increasing during exercise but lowering rapidly once the
519 muscular activity stops (Edwards et al., 2002). Our data extends these observations,
520 showing similarly restrained cerebral blood flow and cerebral oxygen delivery during short
521 cycling sprints, even when perceived exertion is only at moderate levels. Dynamic cerebral
522 autoregulation is a primary mechanism for regulating cerebrovascular resistance. The effect
523 of exercise on this mechanism is controversial, with studies indicating decreased efficacy of
524 dynamic autoregulation during exhaustive aerobic exercise (Ogoh, Dalsgaard, et al., 2005).
525 In contrast, other studies report unchanged or even enhanced cerebral autoregulation
526 during aerobic exercise (Brys et al., 2003). The protracted CBF recovery from hypotension
527 after heavy aerobic exercise (Ogoh et al., 2007) and sprinting (Curtelin et al., 2018) suggests
528 a diminished cerebral autoregulation efficacy, possibly combined with a reduced gain of the
529 arterial baroreflex (Ogoh et al., 2007). Our study cannot address the question of whether
530 performing short sprints affects cerebral autoregulation, but the rapid decrease in O_2Hb at

531 the end of the sprints when cardiac output was elevated suggests normal cerebral
532 autoregulation during this type of exercise. Future studies may test the effect of different
533 sprint/recovery cycle durations on cerebral haemoglobin responses. Also, lowered arterial
534 and brain CO_2 levels and sympathetic activity related to enhanced central drive or activation
535 of muscle metaboreceptors could have also contributed to the lowered oxygen delivery
536 during the repeated sprints (Curtelin et al., 2018). Importantly, a decline in O_2Hb during
537 repeated sprints contrasts with the increase in cerebral oxygenation during lower body
538 resistance exercise (Cherouveim et al., 2022; Zafeiridis et al., 2022). Since decreased brain
539 oxygen saturation is observed in other sprint studies (Curtelin et al., 2018), we may
540 conclude that the extreme intensity of sprint exercise and the recruitment of a large muscle
541 mass reduce cerebral blood flow and lowers cerebral oxygenation. The cessation of the
542 repeated sprints exercise was accompanied by a cerebral reactive hyperaemic response,
543 with a steep increase in O_2Hb within minutes following exercise (Fig. 3). This strengthens the
544 claim of inadequate cerebral oxygen delivery during the repeated sprints exercise.

545 In this experiment, we exploratory investigated the role of glucose ingestion and acute
546 hyperglycaemia on the response of cerebral haemoglobin to sprint exercise. Although the
547 brain almost exclusively relies on glucose for its metabolism and brain's glucose uptake
548 increases during exercise, hyperglycaemia has been shown to decrease the cerebral
549 metabolic rate of oxygen (Ishibashi et al., 2016; Shemesh et al., 2019) and brain connectivity
550 (Ishibashi et al., 2018) in healthy young participants. Acute and chronic hyperglycaemia also
551 decreases CBF (Duckrow et al., 1985; Shemesh et al., 2019). In our experiment, glucose
552 ingestion did not alter O_2Hb and HHb responses to the repeated sprints exercise, although
553 improving performance. However, reactive hyperaemia was significantly enhanced with

554 glucose ingestion, so that O_2Hb concentration increased faster and reached higher levels in
555 the post-exercise period with glucose ingestion. Whether post-exercise hyperaemia was
556 related to any baseline changes in CBF or cerebral blood volume caused by glucose ingestion
557 cannot be ascertained by our data and warrants further investigation.

558 Study limitations

559 The major limitation in this study is the possible confounding effect of extracerebral (scalp
560 and skull) haemodynamics. Since the emitted NIRS light traverses extracerebral tissues and
561 the cerebral cortex, changes in blood volume and flow in superficial tissues will inevitably
562 contaminate the NIRS measurements. The design of our NIRS probe, with rigid circular
563 optodes holders firmly secured to exert pressure onto the forehead skin underneath the
564 optodes, helps to minimise changes in skin blood flow and their impact on NIRS recordings
565 (Baker et al., 2015). Other procedures employ short separation channels to record the
566 contaminating signal from superficial tissues and use these signals to regress out the
567 influence of superficial tissue haemodynamics on the NIRS signals. However, our NIRS probe
568 did not allow recording from short separation channels and therefore the influence of
569 extracerebral tissues on the measured haemoglobin concentrations could not be
570 ascertained nor removed. The exact impact of exercise on extracerebral haemodynamics is
571 unknown. Facial skin blood flow and vascular conductance have been shown to increase
572 with dynamic exercise intensity without a clear plateauing (Hayashi et al., 2020), whilst
573 ocular circulation is similar to cerebral circulation, decreasing from elevated values to
574 baseline in response to exhaustive dynamic exercise (Ikemura & Hayashi, 2012). These
575 findings suggest that our oximetry data during the repeated sprints exercise mainly
576 originate from cerebral circulation. Future studies may assess the contribution of forehead

skin to changes in haemoglobin concentration in the frontal region of the cerebral cortex during repeated sprints.

Conclusions

Short repeated cycling sprint exercise elicits a transient increase in O_2Hb, HHb and tHb. A positive relationship exists between changes in O_2Hb and cardiac output during the sprint exercise but not during the time interval separating the sprints. Despite its transient increases, O_2Hb declined throughout the repeated sprints set. In contrast, HHb increased, resulting in a progressive decrease in cerebral oxygen saturation as the number of sprint repetitions increase, indicating insufficient brain oxygen delivery during short repeated sprints exercise. Glucose ingestion did not alter O_2Hb, HHb or tHb responses to the repeated sprint exercise but improved sprint performance and was associated with faster O_2Hb increase during recovery.

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Conflicts of interests

The authors report no conflict of interests.

Author contributions

P.A.S. and H.M. conceived the study and were responsible for collecting, analysing and interpreting the data and for writing the original draft. W.Z., W.L. and F.R. helped conducting the experiments and collecting and analysing the data. X.Z. assisted in acquiring and analysing NIRS data. Z.Y. and Z.K. co-supervised and conceived the study and assisted with writing and revising the manuscript.

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838 Table 1: Participants characteristics. Data are presented as mean ± standard deviation; n = 11.

Age (years)	Height (cm)	Weight (kg)	BMI ($kg \cdot m^{-2}$)	% Body fat (%)	$\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR_{peak} ($beats \cdot min^{-1}$)	$\dot{W}_{peak}$ (W)
25 ± 3	174 ± 7	89 ± 11	29 ± 3	30 ± 5	32 ± 2	172 ± 4	132 ± 12

839 BMI, body mass index; $\dot{V}O_{2peak}$, peak value for the rate of oxygen consumption during the incremental maximal exercise test; HR_{peak}, peak value
840 for heart rate during the incremental maximal exercise test; $\dot{W}_{peak}$, peak value for power output during the incremental maximal exercise test.

841

842 Table 2: Mean ± SD values for oxyhaemoglobin, deoxyhaemoglobin, and total haemoglobin concentration changes during the repeated sprints;

843 n = 10.

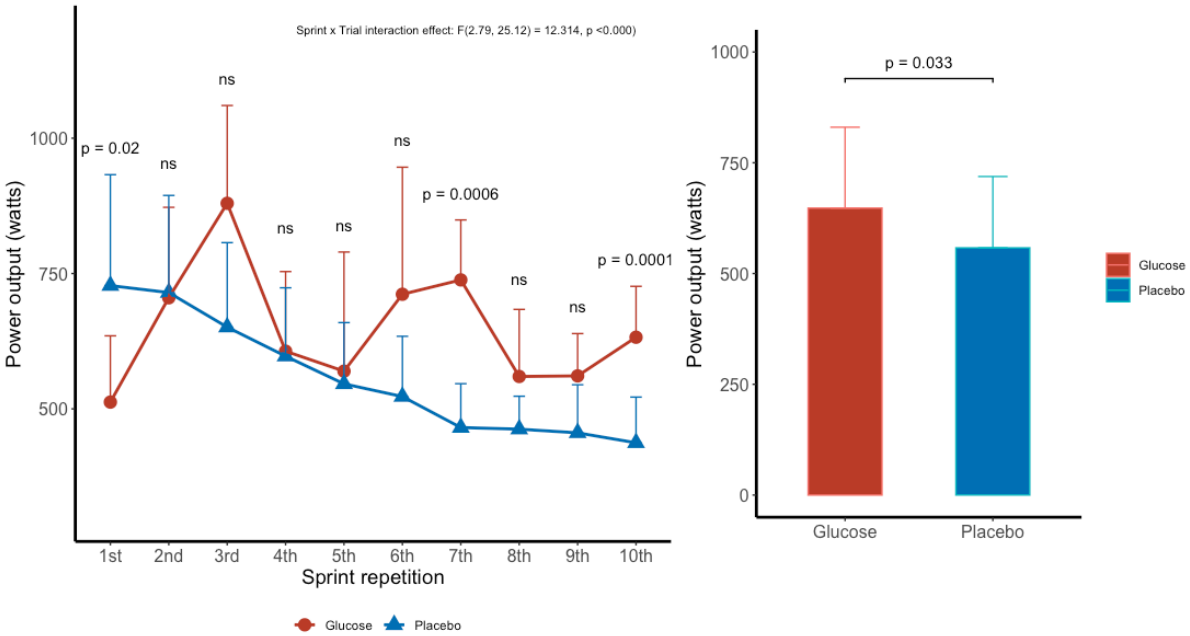
Sprint repetition	**Trial**	**$\Delta O_2Hb_{0\text{-}15\ sec}$ µM**	**$\Delta O_2Hb_{15\text{-}25\ sec}$ µM**	**$\Delta HHb_{0\text{-}15\ sec}$ µM**	**$\Delta HHb_{15\text{-}25\ sec}$ µM**	**$\Delta tHb_{0\text{-}15\ sec}$ µM**	**$\Delta tHb_{15\text{-}25\ sec}$ µM**
1st	Placebo	1.81 ± 1.69	-0.18 ± 1.82	0.02 ± 0.39	-0.25 ± 0.66	1.83 ± 1.84	-0.44 ± 2.06
	Glucose	1.68 ± 1.00	0.30 ± 2.49	-0.11 ± 0.39	-0.35 ± 0.52	1.51 ± 1.96	-0.03 ± 2.42
	Mean ± SD	1.75 ± 1.84	0.06 ± 2.19	-0.04 ± 0.40	-0.30 ± 0.59	1.67 ± 1.90	-0.24 ± 2.25
2nd	Placebo	2.75 ± 1.33	-0.61 ± 1.56	0.25 ± 0.54	0.01 ± 0.49	3.01 ± 1.51	-0.62 ± 1.36
	Glucose	1.99 ± 1.82	-1.04 ± 1.42	0.16 ± 0.33	0.14 ± 0.43	2.16 ± 1.20	-0.88 ± 1.47
	Mean ± SD	2.37 ± 1.64	-0.82 ± 1.31	0.21 ± 0.45	0.08 ± 0.46	**2.59 ± 1.81**[a, b]	-0.75 ± 1.42
3rd	Placebo	2.61 ± 1.52	-0.38 ± 1.40	-0.01 ± 1.18	-0.22 ± 1.25	2.67 ± 1.61	-0.59 ± 2.01
	Glucose	2.24 ± 1.71	-0.55 ± 0.99	0.02 ± 0.29	-0.07 ± 0.43	2.30 ± 1.76	-0.60 ± 1.08
	Mean ± SD	2.42 ± 1.62	-0.47 ± 1.21	0.00 ± 0.85	-0.15 ± 0.94	2.48 ± 1.69	-0.60 ± 1.61
4th	Placebo	3.05 ± 1.71	-0.62 ± 1.43	-0.04 ± 0.82	-0.41 ± 0.93	3.11 ± 2.03	-1.04 ± 2.14

	Glucose	2.67 ± 1.39	-0.51 ± 1.26	0.01 ± 0.48	-0.16 ± 0.46	2.77 ± 1.57	-0.68 ± 1.45
	Mean ± SD	**2.86 ± 1.56**[a]	-0.57 ± 1.34	-0.02 ± 0.67	-0.28 ± 0.74	**2.94 ± 1.82**[a]	-0.86 ± 1.83
5th	Placebo	3.08 ± 1.95	-0.59 ± 0.67	0.10 ± 0.66	-0.16 ± 0.43	3.20 ± 2.22	-0.72 ± 0.82
	Glucose	3.17 ± 1.70	-0.24 ± 1.26	-0.05 ± 0.52	-0.19 ± 0.81	3.16 ± 1.60	-0.41 ± 1.79
	Mean ± SD	**3.12 ± 1.83**[a]	-0.42 ± 1.02	0.02 ± 0.60	-0.18 ± 0.64	**3.18 ± 1.93**[a]	-0.57 ± 1.49
6th	Placebo	3.18 ± 1.66	-0.17 ± 1.08	0.25 ± 0.73	0.00 ± 0.83	3.44 ± 2.00	-0.17 ± 1.70
	Glucose	2.88 ± 1.97	-0.59 ± 1.11	-0.04 ± 0.51	-0.12 ± 0.52	2.93 ± 2.11	-0.73 ± 1.39
	Mean ± SD	**3.03 ± 1.83**[a]	-0.38 ± 1.11	0.10 ± 0.64	-0.06 ± 0.69	**3.19 ± 2.07**[a]	-0.45 ± 1.58
7th	Placebo	2.75 ± 1.52	-0.47 ± 1.25	0.15 ± 0.77	-0.28 ± 0.70	2.94 ± 1.87	-0.74 ± 1.45
	Glucose	2.37 ± 1.25	-0.68 ± 1.02	0.07 ± 0.41	-0.12 ± 0.36	2.49 ± 1.49	-0.80 ± 1.16
	Mean ± SD	**2.56 ± 1.34**[a]	-0.57 ± 1.14	0.11 ± 0.62	-0.20 ± 0.56	**2.71 ± 1.70**[a]	-0.77 ± 1.31
8th	Placebo	3.03 ± 2.78	-0.60 ± 1.49	0.40 ± 1.02	-0.07 ± 0.92	3.47 ± 3.27	-0.66 ± 1.24
	Glucose	3.15 ± 2.34	-0.07 ± 1.10	0.09 ± 0.44	-0.15 ± 0.52	3.26 ± 2.45	-0.21 ± 0.93
	Mean ± SD	**3.09 ± 2.56**[a]	-0.33 ± 1.33	0.25 ± 0.80	-0.11 ± 0.75	**3.36 ± 2.88**[a]	-0.44 ± 1.12

9th	Placebo	3.23 ± 2.70	-0.81 ± 1.33	0.02 ± 1.08	-0.54 ± 1.14	3.28 ± 3.15	-1.37 ± 2.11
	Glucose	2.72 ± 1.42	-0.26 ± 1.01	-0.04 ± 0.45	-0.35 ± 0.57	2.69 ± 1.55	-0.59 ± 1.30
	Mean ± SD	**2.98 ± 2.17**[a]	-0.54 ± 1.21	-0.01 ± 0.82	-0.44 ± 0.90	**2.98 ± 2.49**[a]	-0.98 ± 1.79
10th	Placebo	3.40 ± 3.01	-0.36 ± 1.46	0.10 ± 0.82	-0.29 ± 0.68	3.57 ± 3.56	-0.60 ± 1.70
	Glucose	3.50 ± 1.80	-0.35 ± 1.44	-0.22 ± 0.92	-0.58 ± 1.77	3.35 ± 1.78	-0.92 ± 2.22
	Mean ± SD	**3.45 ± 2.47**[a]	-0.35 ± 1.44	-0.06 ± 0.88	-0.43 ± 1.35	**3.46 ± 2.81**[a]	-0.76 ± 1.98
Overall mean ± SD	Placebo	2.89 ± 2.10	-0.48 ± 1.35	0.12 ± 0.84	-0.22 ± 0.85	3.05 ± 2.44	-0.70 ± 1.73
	Glucose	2.64 ± 1.84	-0.40 ± 1.41	-0.01 ± 0.51	-0.19 ± 0.77	2.66 ± 1.92	-0.59 ± 1.60

$\Delta O_2Hb_{0\text{-}15\ sec}$, median change in oxyhaemoglobin concentration in the time interval 0 – 15 s (sprint); $\Delta O_2Hb_{15\text{-}25\ sec}$, median change in oxyhaemoglobin concentration in the time interval 15 – 25 s (recovery); $\Delta HHb_{0\text{-}15\ sec}$, median change in deoxyhaemoglobin concentration in the time interval 0 – 15 s (sprint); $\Delta HHb_{15\text{-}25\ sec}$, median change in deoxyhaemoglobin concentration in the time interval 15 – 25 s (recovery); $\Delta tHb_{0\text{-}15\ sec}$, median change in total haemoglobin concentration in the time interval 0 – 15 s (sprint); $\Delta tHb_{15\text{-}25\ sec}$, median change in total haemoglobin concentration in the time interval 15 – 25 s (recovery). Positive and negative values indicate increase and decrease in haemoglobin concentrations, respectively, from the time immediately before the onset of the respective sprint repetition (baseline value = 0). Mean ± SD is the average and standard deviation of values from GLU and PLA trials within each sprint. Overall mean ± SD is the average and standard deviation for all values in the respective trial. [a] Significantly different from the first sprint, $p < 0.05$; [b] significantly different from the immediately preceding sprint, $p < 0.05$.

854 Figure 1



855

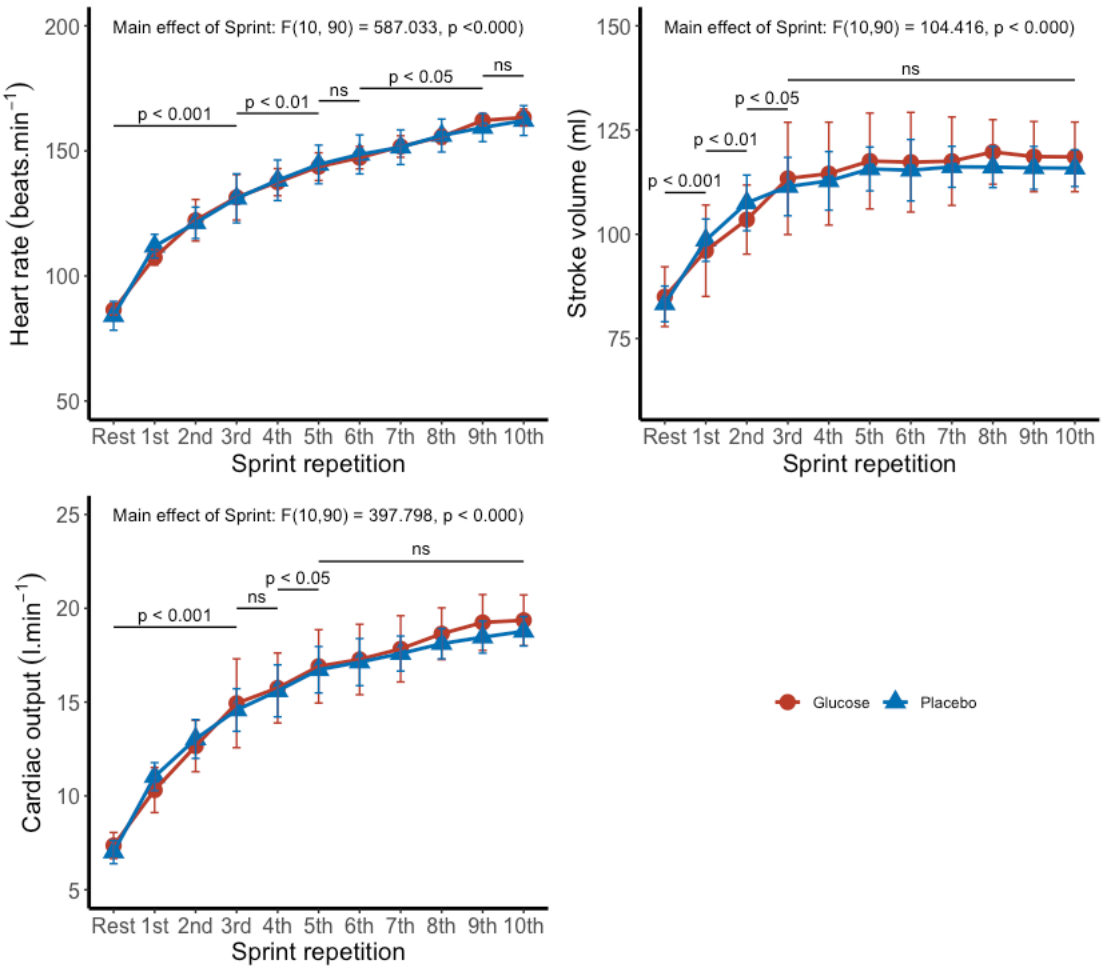
856 Figure 1: Mean power output (mean ± SD) during each repeated sprint for GLU (red line)

857 and PLA (blue line) trials (left panel). Pooled mean power output (mean ± SD) for GLU (red

858 bar) and PLA (blue bar) trials (right panel). Data is mean ± SD; ns, non-significant, n = 10.

859

860 Figure 2

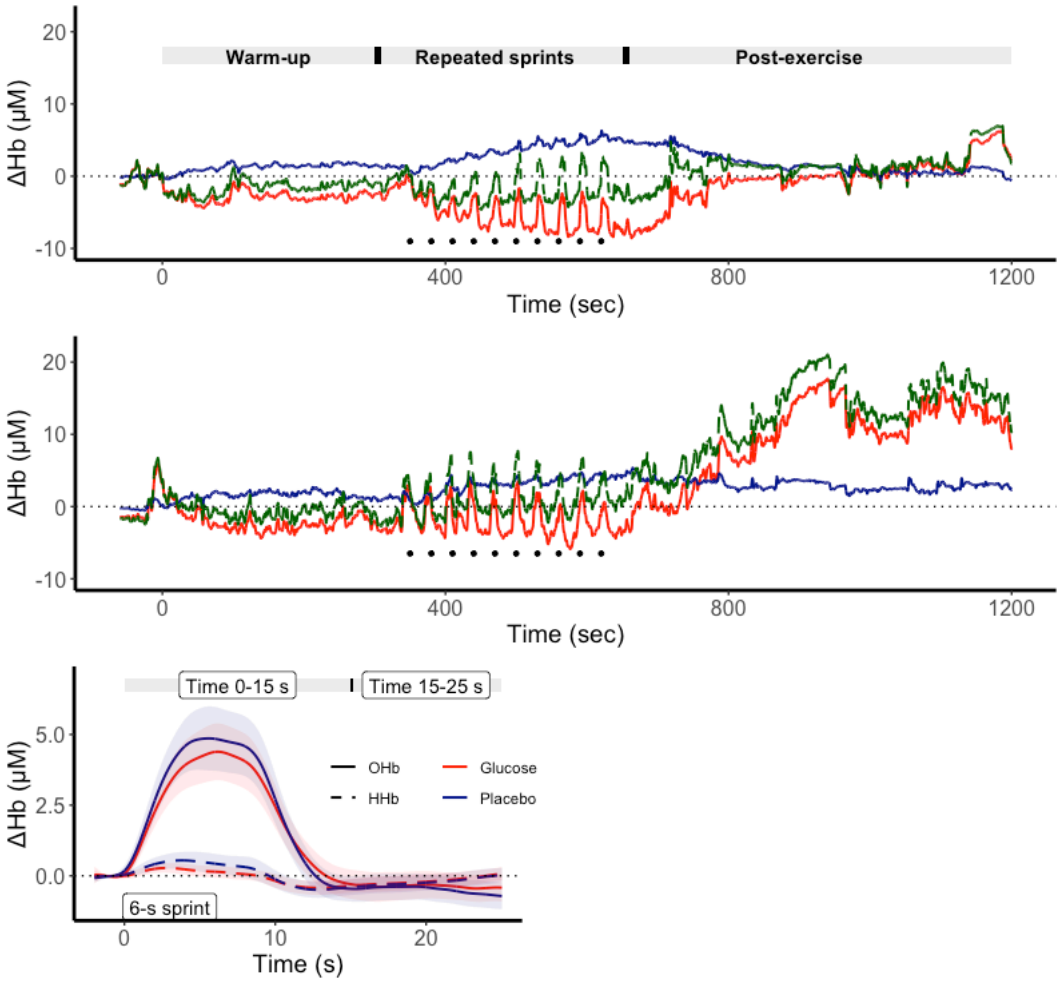


861

862 Figure 2: Heart rate, stroke volume and cardiac output (mean ± SD) at rest and during each
863 repeated sprint for GLU (red lines) and PLA (blue lines) trials. The insert is the output of the
864 two-way repeated measures ANOVA for the significant main effect of sprint repetition,
865 excluding the resting values. P-values are from backward difference contrasts (contrast
866 between one sprint repetition and the one immediately before, including the first sprint and
867 resting values); n = 10.

868

869 Figure 3



870
871

872 Figure 3: Changes in O_2Hb, HHb and tHb during the repeated sprints exercise. Top and

873 middle graphs depict changes in the concentration of O_2Hb (red line), HHb (blue line) and

874 tHb (green line) recorded in two participants. The records start immediately before the

875 warm-up. The transient increase in O_2Hb, HHb, and tHb during each sprint (black dots) is

876 clearly seen in both participants. In the top graph, there was a substantial decrease in O_2Hb

877 concentration and increase in HHb over the repeated sprints exercise and a return of both

878 O_2Hb and HHb toward pre-exercise values during recovery. The participant in the middle

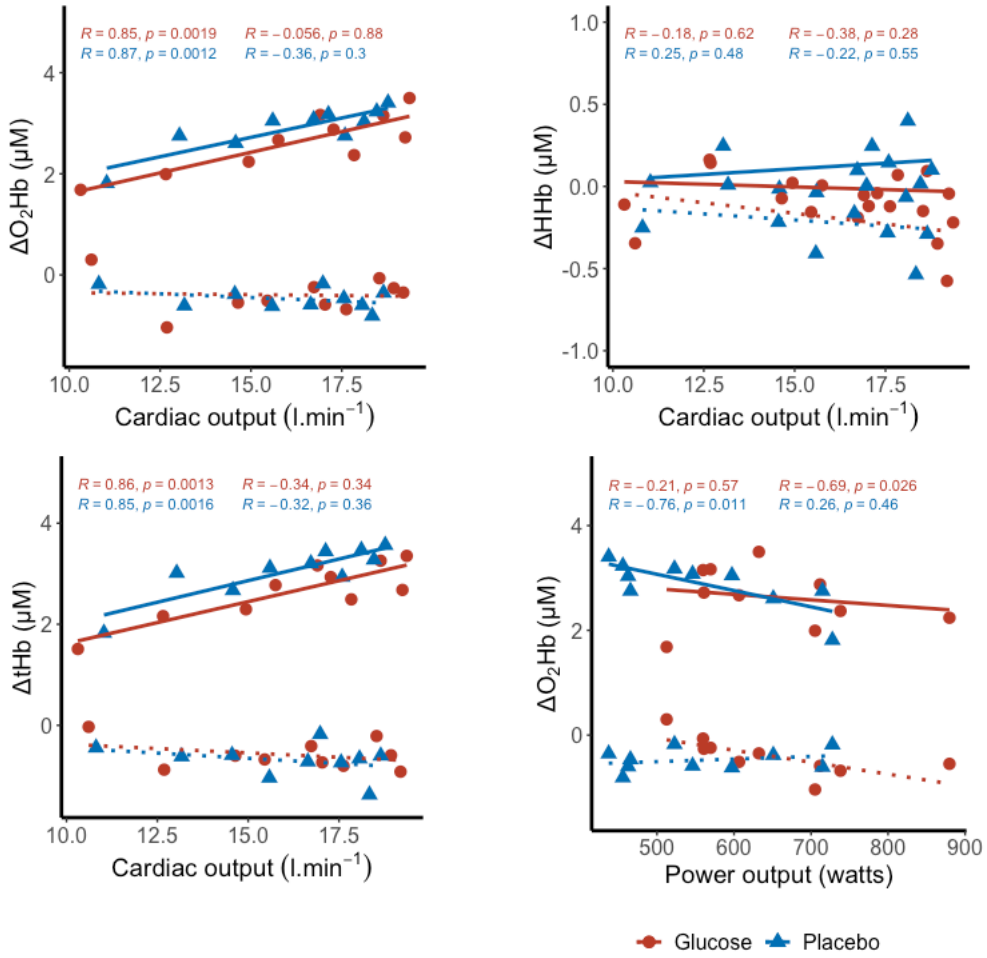
879 graph had lesser changes in O_2Hb and HHb concentration during exercise but substantial

880 post-exercise cerebral hyperaemia. The bottom panel presents the overall average for

881 ΔO_2Hb and ΔHHb during the repeated sprints, showing the rapid increase in cerebral

haemoglobin concentration during the sprint and its fast decrease soon after the end of the sprint. Each tracing is the mean of 10 participants, 10 sprints and 8 channels. The shadowed areas represent the standard error of the mean; n = 10.

886 Figure 4



887

888 Figure 4: Scatter plots depicting the relationship between ΔO_2Hb (upper left graph), ΔHHb
889 (upper right graph), and ΔtHb (lower left graph) with cardiac output and between ΔO_2Hb
890 and power output (lower right graph) during the sprints (solid lines) and during the time
891 interval between sprints (dotted lines) in GLU (red lines and red circles) and PLA (blue lines
892 and blue triangles) trials. The Pearson's R coefficients and respective p-values are presented
893 on the top of the graphs for values during the sprint (left column) and the time interval
894 between the sprints (right column) for GLU (red font) and PLA (blue font) trials. During the
895 sprint, a significant correlation was found between cardiac output and ΔO_2Hb and ΔtHb. No
896 significant correlation was found between cardiac output and ΔHHb and cardiac output and

ΔO_2Hb and ΔtHb during the time interval between sprints. Mean power output was negatively correlated with ΔO_2Hb during the sprints in PLA trial, and negatively correlated with ΔO_2Hb in the time interval between sprints in the GLU trial.