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

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Running head: Cerebral oximetry during short, repeated sprints

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Abstract

In this study we investigated changes in concentrations of cerebral oxyhaemoglobin (O_2Hb), deoxyhaemoglobin (HHb), and total haemoglobin (tHb) during repeated sprints and their relationship with cardiac output. We also examined the effect of glucose ingestion and acute hyperglycaemia on cerebral haemoglobin responses. Ten young male participants ingested either a glucose drink (70 g) or a placebo before performing a set of 10 repeated 6-second sprints on a cycle ergometer. Heart rate, stroke volume, and cardiac output were measured continuously using impedance cardiography, while changes in O_2Hb, HHb, and tHb in the frontal region of the cerebral cortex were measured using near-infrared spectroscopy (NIRS). The results showed that each sprint elicited a transient increase in O_2Hb and to lesser extent HHb concentrations, which was enhanced with the number of sprint repetitions and correlated with cardiac output. After each sprint, O_2Hb and HHb quickly returned to baseline, while cardiac output remained elevated. At the end of the repeated sprints, O_2Hb was decreased to below pre-exercise levels, while HHb and tHb were elevated. After a recovery of 10-15 min, O_2Hb returned to pre-exercise values in the placebo trial, but increased to above pre-exercise values (reactive hyperaemia) in the glucose trial. Our findings suggest that short sprint exercise increases O_2Hb, HHb, and tHb levels during exertion in parallel with cardiac output. However, in addition to the transient increase in cerebral haemoglobin, a progressive decline in cerebral oxygen saturation occurs during repeated sprints. Glucose ingestion does not alter cerebral haemoglobin responses to sprint exercise but appears to be associated with faster recovery of O_2Hb.

Introduction

Studies investigating cerebral blood circulation have reported an increase in regional cerebral blood flow (rCBF) during dynamic exercise of moderate-to-severe exercise intensity [i.e., ~ 70% maximal oxygen uptake ($\dot{V}O_{2max}$)]. However, when the work rate increases to maximal exhaustive levels, rCBF decreases towards baseline [1,2]. In addition to augmented CBF, dynamic exercise also results in an increase in cerebral metabolism, as evidenced by a lowered cerebral metabolic ratio [3,4], indicating enhanced neural activity [5]. This response is typically observed during exhaustive exercise [6], exercise in hyperthermic and hypoxic conditions, or exercise under increased mental effort [4].

In addition to changes in rCBF, dynamic exercise also affects cerebral oxygenation [7]. Changes in concentration of oxyhaemoglobin (O_2Hb) and deoxyhaemoglobin (HHb) in the cerebral tissue can be non-invasively measured with near-infrared spectroscopy (NIRS) with a reasonable spatial resolution (~1-2 cm) and high time resolution. Two independent meta-analyses of NIRS studies have shown that O_2Hb concentration in the frontal region of the cerebral cortex increases during submaximal exercise but

decreases as exercise approaches maximal intensity, suggesting a mismatch between oxygen delivery and demand at the end stages of incremental maximal exercise [8,9]. Such mismatch has been related with a cerebral vasoconstriction in response to exercise-induced hyperventilation and hypocapnia [2,10]. The lowered cerebral oxygenation at near-maximal exercise, combined with muscle deoxygenation, may impair muscle recruitment and trigger fatigue [2].

Extremely short all-out sprint interval exercise (<10 s) is an attractive option for achieving cardiorespiratory fitness [11], but the cerebrovascular response to this type of exercise is unclear. One study showed that a 30-second all-out cycling exercise at normal ambient oxygen levels and in hypoxia resulted in a decrease in middle cerebral artery blood flow mean velocity (MCA*v*) and cerebral oxygenation, which were unrelated to changes in end-tidal carbon dioxide partial pressure ($P_{ET}CO_2$) [12]. In addition, the MCA*v* decrease during the 30-second all-out cycling did not follow the changes in cardiac output and mean blood pressure, which increased during the exercise [12]. In another study, participants performed seven 30-second exercise bouts on a cycle ergometer at a work rate of 150% maximal oxygen uptake, where O_2Hb concentration increased during the first bout but decreased in the last three bouts, and HHb concentration increased from resting levels, without a clear correlation between cerebrovascular response and $P_{ET}CO_2$ [13]. These two studies suggest that, unlike submaximal and maximal exercise, cerebrovascular response during supramaximal exercise is not strictly dependent on arterial CO_2 levels, mean blood pressure, or cardiac output. However, the impact of repeated short sprint efforts on cerebral blood flow and oxygenation, and how it affects performance, remain unknown. It is worth noting that dynamic cerebral autoregulation, which is another major mechanism regulating cerebrovascular function, has a time delay similar to the duration of short sprint bouts (~6 s) [14], which may impair its protective role in preventing a steep rise in cerebral blood flow and the risk of hyperperfusion during all-out exercise.

Blood glucose levels play a role in determining cerebral metabolism and CBF [15]. Although glucose is the primary energy substrate utilized by the brain, acute hyperglycaemia has been shown to decrease CBF and the cerebral metabolic rate of both oxygen and glucose [15,16], although opposite findings have been reported [17]. On the other hand, glucose ingestion has been shown to improve cerebral glucose uptake and reduce perceived exertion during prolonged exercise, suggesting a potential ergogenic effect of glucose through central mechanisms [18]. Hence, we also tested the effect of acute glucose intake and subsequent rise in glycaemia in cerebral haemoglobin concentration changes during repeated sprints and how it relates with performance.

Therefore, this study aimed to investigate changes in cerebral O_2Hb, HHb, and tHb during the performance of repeated cycling sprints and their relationship with both cardiac output and power output. The effect of acute hyperglycaemia on cerebral (prefrontal) oxygenation during the repeated

sprints was also investigated. A NIRS probe scanning the entire prefrontal cortex was used to assess brain oximetry, which allows compensating for interindividual cerebral circulatory regional differences and to investigate differences in cerebrovascular responses to short intermittent cycling sprints across the frontal region of the cerebral cortex.

Methods

Participants and experimental design

Eleven healthy male adults volunteered (Table 1) to participate in this study after providing written informed consent. Participants were free of any metabolic, cardiovascular, or neurological disease and did not regularly participate in exercise training. The study protocol was reviewed and approved by the ethics review board of the University of Macao (Special Administrative Region of Macao, China) and conformed to the ethical principles for research 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 sprint 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). After a warm-up at 25 W for 3 min, participants started cycling at 50 W and 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) [19]. 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 [11].

Repeated sprints cycling exercise

During the two experimental sessions, participants performed a repeated sprint set of 10 repetitions of 6 s maximal sprints interspersed by 24 s resting, performed on a cycle ergometer (Monark Ergomedic 894E,

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

NIRS

A portable 2 x 4-channel continuous-wave NIRS system (Octamon, Artinis Medical Systems, Einsteinweg, The Netherlands) was used to measure changes (Δ) in O_2Hb, HHb, and tHb concentrations using two wavelengths at 750 and 850 nm. Data were acquired with the OxySoft software (OxySoft, Artinis Medical Systems, Einsteinweg, The Netherlands) at a frequency of 50 Hz with a differential path length factor of six. The optodes were attached to a dark ambient light-protecting probe holder, tightly secured to the head by adjustable headbands and placed over the forehead and the underlying prefrontal cortex (four channels on each side). The two light detectors were aligned on Fp1 and Fp2 locations of the 10-20 system for the electroencephalography and surrounded by four light-emitting optodes with an inter-optode distance of 35 mm, providing a total of eight NIRS channels.

Impedance cardiography

Cardiac output was measured continuously during the sessions by signal morphology impedance cardiography (PhysioFlow PF-05, Manatec biomedical, Paris, France). After skin preparation to minimise electrical resistance, six Ag/AgCl electrodes (Hewlett Packard 40493 E) were placed on the chest wall along the xiphoid process and at the V1 and V6 precordial locations, and at the base of the neck to record the thoracic impedance and electrocardiographic signals. Resting blood pressure was measured by automatic oscillometry to calibrate the cardiac impedance data acquisition following the manufacturer instructions. Stroke volume and cardiac output were recorded for each cardiac cycle and measured continuously throughout the trial.

Blood glucose and lactate

157 Blood glucose measurements were collected with a glucometer (ACCU−CHEK Performa, USA) and the
158 proper test strips (F. Hoffmann-La Roche AG, Basel, Switzerland). Blood lactate was also measured with
159 the Lactate Pro 2 handheld device and respective test strips (AKRAY Europe B.V., Amstelveen, the
160 Netherlands). Both blood analytes were measured in duplicate from capillary blood drawn from a
161 lanceted fingertip at three time points: 1) at baseline, soon after arrival to the laboratory; 2) immediately
162 before starting the exercise protocol; and 3) immediately after completion of the repeated sprints
163 exercise. The duplicate blood sample measurements were averaged for analysis.

164 *Procedures*

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

172 After the 10-min rest, participants mounted the cycle ergometer and completed the 5-min warm-up
173 followed by the 10 x 6-s repeated sprints. After exercise, there were another 10 minutes resting. Ratings
174 of perceived exertion were obtained at the end of the fifth and tenth sprint repetitions using the Borg's
175 scale.

176 *Data analysis*

177 The NIRS data pre-processing was performed using the Homer2 toolbox [20] for Matlab (Matlab version
178 9.5.0, release R2018b, The Mathworks Inc., Natick, Massachusetts, USA). First, light absorption data were
179 converted into optical density and haemoglobin concentrations using the modified Beer-Lambert law
180 with a differential path length factor of six. Next, all NIRS signals were low pass filtered at a cut-off
181 frequency of 0.5 Hz to remove heartbeat-associated changes in O_2Hb, HHb and tHb, using a finite impulse
182 response fourth-order Butterworth filter, implemented in the forward and inverse directions to avoid
183 phase filtering artefacts [20]. After filtering, the whole NIRS signal was epoched into the individual sprints,
184 starting at minus 5 s and ending at 25 s after the sprint start. The median was used to quantify the
185 changes in haemoglobin concentrations in the time intervals 0-15 s and 15-25 s, to measure the response
186 during the sprints and during the rest interval between sprints, respectively. The median was chosen
187 because of its higher robustness against potential biasing effects caused by motion artefacts and outliers.
188 The time interval 0-15 s was selected following a visual inspection of the transient ΔO_2Hb response
189 accompanying the performance of the sprints (Fig. 3). Due to the non-normal distribution of some

190 ΔO_2Hb, ΔHHb, and ΔtHb values (Supporting information, Figs. S1-S3 and Table S1), a natural log-
191 transformation was applied to these variables for the purpose of statistical analysis.

192 *Statistical analysis*

193 The Shapiro-Wilk test was used to verify the normal distribution of the data. To assess the responses to
194 repeated sprint exercise, a repeated measures ANOVA with a complete factorial design (cerebral
195 oximetry data: 2 trials x 8 channels x 10 sprint repetitions; power output and cardiovascular data: 2 trials
196 x 10 sprint repetitions) was conducted. The Greenhouse-Geiser correction was applied to within-subject
197 factors violating the sphericity assumption (Mauchly's test p-value < 0.05). Post hoc tests were performed
198 employing one-way ANOVA with Bonferroni's correction for family-wise error rate. Planned contrasts
199 were performed using polynomial fitting. This approach treated the repeated sprints as a quantitative
200 variable with even spacing. While the polynomial contrast analysis does not directly compare two specific
201 sprint repetitions, it provides valuable information about the overall trend effect of the repeated sprints
202 on the response variables. In case of a significant linear contrast, backward differences contrasts were
203 computed (the contrast between two consecutive sprint repetitions, including resting). Further contrasts
204 between the first repeated sprint and the following sprints were also calculated. One sample *t*-test was
205 used for testing the significance of post-exercise mean ΔO_2Hb, ΔHHb and ΔtHb against pre-exercise
206 values (since continuous NIRS measurements are relative, O_2Hb and HHb baseline concentrations are set
207 to zero). Correlation values were computed using the Pearson's coefficient. All statistical tests were
208 performed in RStudio (v. 1.3.1093, 2009-2020 RStudio, PBC) using functions from Rstatix (v. 0.7.0) and R
209 base (v. 4.0.3) packages. Unless otherwise stated, all data are reported as mean $\pm$ standard deviation
210 (SD). Significance was accepted at $p < 0.05$.

211 Results

212 Due to large noise affecting the NIRS signals, one participant was excluded from the final analysis,
213 resulting in a final sample size of 10 participants. In another participant, the NIRS signals pertaining to
214 three of the sprints in the two trials were removed from analysis because of significant motion artefacts.
215 The missing data (48 out of 1600 data points) were imputed using the average values from the same
216 individual, channel, and the other sprints. One participant in the PLA trial had missing mean power
217 output data for the two final repeated sprints due to equipment failure. The last recorded mean power
218 output was used to replace the missing values.

219 *Power output and RPE*

220 Figure 1 displays the mean power output for each sprint and trial. The mean power output was found to
221 be higher during the GLU trial than the PLA trial [GLU: 648 $\pm$ 183 W; PLA: 558 $\pm$ 161 W; $F_{(1, 9)} = 6.312$, p =

0.033, $\eta^2 = 0.098$], particularly in the later sprint repetitions [trial x sprint repetition interaction effect: $F_{(2.79, 25.12)} = 12.314$, $p < 0.000$, $\eta^2 = 0.203$]. According to one-way ANOVA, mean power output was significantly different between GLU and PLA in the first (PLA higher than GLU), seventh and tenth sprint repetitions (Bonferroni's corrected $p < 0.05$, Fig. 1). At the end of the fifth repetition, perceived exertion was moderate (GLU: 11.9 ± 2.1; PLA: 13.2 ± 1.9 units), but RPE reached near-maximal scores at the end of the sprint set (GLU: 19.3 ± 2.1; PLA: 19.3 ± 0.5 units; main effect of sprint repetition, $F_{(1, 9)} = 232.66$, $p < 0.000$, $\eta^2 = 0.856$), with no differences between trials.

Blood glucose and lactate

At baseline, blood glucose concentration was similar in the two trials (GLU: 5.7 ± 0.5 mmol·l^{-1}; PLA: 5.9 ± 0.6 mmol·l^{-1}; $p = 0.310$). However, following glucose ingestion, the blood glucose concentration rose and was significantly higher in the GLU trial compared to the PLA trial prior to exercise (GLU: 8.1 ± 1.7 mmol·l^{-1}; PLA: 6.1 ± 0.8 mmol·L^{-1}; $p = 0.016$). By the end of the repeated sprints, the blood glucose concentration returned to similar levels in both trials (GLU: 6.5 ± 1.6 mmol·l^{-1}; PLA: 5.9 ± 0.9 mmol·l^{-1}; $p = 0.254$).

At baseline, the blood lactate concentration was at the same level in the GLU and PLA trials (GLU: 1.7 ± 0.9 mmol·l^{-1}; PLA: 1.4 ± 0.4 mmol·l^{-1}; $p = 0.335$). However, after glucose ingestion, the blood lactate concentration increased significantly (GLU: 2.4 ± 0.8 mmol·l^{-1}; PLA: 1.6 ± 0.5 mmol·l^{-1}; $p = 0.023$). Immediately after the repeated sprints, the blood lactate concentration was markedly elevated and significantly higher in the PLA trial than in 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 shows the changes in heart rate, stroke volume and cardiac output during the repeated sprints. The heart rate increased progressively throughout the sprints set, reaching its highest value of 163 ± 5 beats·min^{-1} in the final sprint [main effect for sprint repetition, excluding resting values: $F_{(3.45, 31.04)} = 347.947$, $p < 0.000$, $\eta^2 = 0.884$], with no significant difference between GLU and PLA [$F_{(1, 9)} = 0.018$, $p = 0.897$, $\eta^2 < 0.000$].

The stroke volume increased from 84.2 ± 5.8 ml at rest to 97.3 ± 8.4 ml during the first sprint, but remained at a plateau after the fourth repetition [main effect for sprint repetition, excluding resting values: $F_{(2.47, 22.25)} = 39.546$, $p < 0.000$, $\eta^2 = 0.380$]. There was no significant difference in stroke volume between GLU and PLA trials [$F_{(1, 9)} = 0.195$, $p = 0.669$, $\eta^2 = 0.005$].

Cardiac output increased in a curvilinear pattern during the repeated sprints, rising from a resting value of 7.2 ± 0.7 l·min^{-1} to a cardiac output of 10.7 ± 1.0 l·min^{-1} in the first sprint and 19.1 ± 1.1 l·min^{-1} at the

end of the repeated sprints [main effect for sprint repetition, excluding resting values: $F_{(2.59, 23.31)}$ = 214.561, $p < 0.000$, $\eta^2 = 0.792$]. There was no significant difference in cardiac output between GLU and PLA [$F_{(1, 9)} = 0.280$, $p = 0.610$, $\eta^2 = 0.005$], as well as no interaction effect between trial and sprint repetition [$F_{(1.93, 17.38)} = 1.122$, $p < 0.346$, $\eta^2 = 0.025$].

During the time interval between sprints, cardiac output remained high, but decreased slightly, although significantly, from the level attained 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 showed 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 of ΔO_2Hb, ΔHHb, ΔtHb during the repeated sprints and the rest periods between the sprint repetitions.

Oxyhaemoglobin

During the sprints, O_2Hb levels rapidly increased, reaching its peak at the end of each sprint (Fig. 3; Supporting information, Fig. S4). The O_2Hb response to the sprints increased with the increasing number of sprint repetitions [main effect of sprint repetition: $F_{(9, 81)} = 3.380$, $p = 0.001$, $\eta^2 = 0.074$]. However, there was no effect of glucose ingestion [$F_{(1, 9)} = 0.930$, $p = 0.360$, $\eta^2 = 0.005$] or NIRS channel [$F_{(7, 63)}$ = 10.222, $p = 0.979$, $\eta^2 = 0.002$] on this response. Polynomial fitting demonstrated a significant linear effect of sprint repetition on ΔO_2Hb, which did not improve with higher-order components. Additionally, contrast analysis showed significantly lower ΔO_2Hb in the first sprint compared to the fourth and subsequent sprints ($p < 0.05$). After each sprint, O_2Hb concentrations returned to pre-sprint levels or lower (Fig. 3, Table 2). There were no significant differences in ΔO_2Hb values between trials, channel, or sprint repetitions for the rest periods between the sprints.

Deoxyhaemoglobin

In comparison to the changes in O_2Hb, the changes in HHb in response to sprinting were less pronounced and not affected by the sprint repetitions [$F_{(9,81)}$= 1.446, $p = 0.182$, $\eta^2 = 0.01$] (Table 2, Fig. 3; Supporting information Fig. S4). Also, there were no significant differences in the changes of HHb between the two trials [$F_{(1, 9)}$= 0.476, $p = 0.508$, $\eta^2 = 0.000$] (Table 2) or between the NIRS channels [$F_{(7,63)}$= 1.531, $p = 0.173$, $\eta^2 = 0.02$]. During the rest interval between the sprints, HHb concentration returned to pre-exercise levels with no differences observed between the trials [$F_{(1, 9)}$= 0.287, $p = 0.605$, $\eta^2 = 0.000$] or NIRS

channels [$F_{(7,63)}$= 1.162, p = 0.337, η^2 = 0.01], but with a significant main effect of sprint repetition [$F_{(9, 81)}$= 2.152, p = 0.034, η^2 = 0.012]. Polynomial contrasts revealed only marginal linear (p = 0.052) and quadratic (p = 0.057) effects of the repeated sprints in ΔHHb in the rest interval between the sprints.

Total haemoglobin

The changes in tHb followed a similar pattern to that observed for O_2Hb, with a marked increase during each sprint, followed by a rapid decline towards the baseline immediately after the sprint. Glucose ingestion [$F_{(1, 9)}$= 0.970, p = 0.350, η^2 = 0.007] and NIRS channels [$F_{(7, 63)}$= 0.403, p = 0.897, η^2 = 0.006] had no effect on the changes in tHb. A significant difference was observed in ΔtHb across the repeated sprints [$F_{(9, 81)}$= 2.796, p = 0.007, η^2 = 0.054], with a significant linear effect (p = 0.008) and lower ΔtHb observed during the first sprint compared to most of the subsequent sprints (0.003 < p < 0.046). No differences in ΔtHb during the time interval between sprints were observed between trials [$F_{(9, 81)}$= 2.017, p = 0.189, η^2 = 0.002], NIRS channels [$F_{(7, 63)}$= 1.016, p = 0.429, η^2 = 0.017], or sprint repetitions [$F_{(9, 81)}$ = 0.866, p = 0.560, η^2 = 0.010].

The concentrations of O_2Hb, HHb, and tHb were compared before, at the end of exercise, and 10-15 minutes post-exercise (see Fig. 3, top and middle panels). The repeated sprint performance caused a significant reduction in cerebral O_2Hb concentration (one sample *t*-test, combining GLU and PLA: t(8) = -2.644; p = 0.029, n = 9) that was comparable between the two trials [GLU: -1.16 ± 4.13 μM; PLA: -3.11 ± 2.87 μM; $F_{(1, 7)}$ = 2.453, p = 0.161, η^2 = 0.096, n = 9]. On the other hand, there was a steady increase in cerebral HHb concentration during the repeated sprints (one sample *t*-test, combining GLU and PLA: t(8) = 3.248, p = 0.011, n = 9), with no effect of glucose ingestion on this response [GLU: 3.80 ± 5.95 μM; PLA: 6.44 ± 3.82 μM; $F_{(1, 7)}$ = 1.541, p = 0.254, η^2 = 0.042, n = 9]. Due to the increase in HHb concentration, tHb also increased after the sprint set (one sample *t*-test, combining GLU and PLA: t(8) = 3.119, p = 0.014, n = 9) regardless of glucose ingestion [GLU: 3.98 ± 5.60 μM; PLA: 5.75 ± 3.86 μM; $F_{(1, 7)}$ = 0.670, p = 0.440, η^2 = 0.014, n = 9].

Following the exercise, there was an increase in cerebral O_2Hb back to baseline levels, particularly in the GLU trial [GLU: 2.54 ± 1.80 μM; PLA: 0.95 ± 2.32 μM; $F_{(1, 7)}$ = 11.263, p = 0.012, η^2 = 0.140, n = 9]. As a result, post-exercise O_2Hb was higher than pre-exercise levels 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 elevated in both trials [GLU: 3.42 ± 4.02 μM; PLA: 3.55 ± 3.15 μM; $F_{(1, 7)}$ = 0.023, p = 0.884, η^2 < 0.000, n = 9] compared to pre-exercise values (one sample *t*-test, combining GLU and PLA: t(8) = 4.557, p = 0.001, n = 9). Following changes in HHb and O_2Hb, tHb concentration also increased after the repeated sprint exercise in both trials [Glu: 3.29 ± 4.07 μM; Pla: 3.12 ± 3.31 μM; $F_{(1, 7)}$

316 = 0.050, p = 0.829, η^2 < 0.000, n = 9], and at 10-15 min post-exercise, it was higher than at pre-exercise
317 (one sample *t*-test, combining GLU and PLA: t(8) = 3.882, p = 0.005, n = 9).

318 Cardiac output and ΔO_2Hb, ΔHHb and ΔtHb relationship

319 Figure 4 illustrates the relationship between cardiac output, power output, and cerebral haemoglobin
320 concentrations (see also Supporting information, Fig. S5). During the sprints, positive correlations were
321 found between cardiac output and the changes in O_2Hb and ΔtHb in both GLU (cardiac output: ΔO_2Hb, R
322 = 0.85, p = 0.0019; cardiac output: ΔtHb, R = 0.86, p = 0.0013) and PLA trials (cardiac output: ΔO_2Hb, R =
323 0.87, p = 0.0012; cardiac output: ΔtHb, R = 0.85, p = 0.0016). No significant correlation was observed
324 between cardiac output and ΔHHb during the repeated sprints, nor between cardiac output and ΔO_2Hb,
325 ΔHHb and ΔtHb during the time interval between the sprints. Conversely, in the GLU trial, power output
326 was negatively correlated with ΔO_2Hb values during the time interval between the sprints.

327 Discussion

328 In this study, our aim was to outline the time profile of cerebral O_2Hb, HHb and tHb responses to a series
329 of brief, repeated sprints and investigate the correlation between this response and cardiac output.
330 Furthermore, we sought to investigate the impact of acute hyperglycaemia on the changes in O_2Hb, HHb,
331 and tHb following the repeated sprint exercise. Our findings indicated that each sprint produced a rapid
332 and significant surge in cerebral O_2Hb concentration, and to a lesser extent HHb, leading to a transient
333 increase in cerebral blood volume (i.e., increased tHb) and oxygenation. During the interval between the
334 repeated sprints, O_2Hb and HHb decreased and returned to, or below, pre-exercise levels rapidly. The
335 variations in ΔO_2Hb and ΔtHb followed a curvilinear trend similar to that observed for heart rate and
336 cardiac output in response to the repeated sprint exercise.

337 During each of the short sprints, there was an increase in O_2Hb, which was approximately ten times
338 higher than the pulsatile peak increase due to the heartbeat at rest (~0.5 μM). Regulation of cerebral
339 blood flow during rest and exercise is closely tied to changes in arterial blood gases (particularly carbon
340 dioxide), neural activity, blood pressure, and cardiac output [21–23]. An increased cardiac output and
341 cerebral perfusion pressure, caused by an elevation of arterial blood pressure, are two possible
342 explanations for the transient increase in O_2Hb and HHb that was observed during each short sprint
343 [12,24,25]. Although we did not record blood pressure, previous studies have shown that heavy-intensity
344 dynamic (e.g., rowing exercise) and resistance (e.g., leg-press exercise) exercise result in a rapid increase
345 in diastolic and systolic blood pressures, therefore it is reasonable to expect that mean blood pressure
346 would increase during all-out sprint exercise [12,25–27]. Although enhanced central drive and activation of the
347 metaboreflex may contribute to the sprint-induced elevation of blood pressure [28], the immediate and
348 transient increase in O_2Hb and HHb during the short sprint exercise suggests a role for mechanical and

349 haemodynamic factors associated with the powerful contraction of the leg and trunk muscles, resulting in
350 brief Valsalva-like manoeuvres and synchronous elevation of arterial blood pressure [24,27,29]. Indeed, the
351 production of Valsalva-like manoeuvres is commonly observed during heavy static and dynamic
352 resistance exercise dynamic exercise [27,30]. However, this manoeuvre can also occur during dynamic
353 exercise, such as during the catch phase of rowing [24,29], and is associated with the cyclical increase in
354 MCAv in synchrony with the elevations of intra-abdominal pressure, central venous pressure, and catch
355 force [24]. Similarly, the onset of bilateral static leg extension simultaneously raises mean arterial blood
356 pressure, MCAv, and cerebral O_2Hb concentration with or without a superimposed Valsalva strain [30].

357 Previous studies have shown a direct influence of cardiac output on cerebral blood flow and cerebral
358 oxygenation [23,31]. In this study, we found that cardiac output had a moderate and positive correlation
359 with changes in O_2Hb and tHb during the sprints. This suggests that cardiac output plays a role in the rise
360 of O_2Hb recorded during the sprints. However, the relationship between cardiac output and cerebral
361 blood flow is still unclear and in the present study O_2Hb concentration declined during rest between the
362 sprint bouts despite the increased cardiac output. While previous research has reported a direct
363 relationship between cardiac output and MCAv during rest and moderate intensity cycling exercise (Ogoh
364 et al., 2005), changes in MCAv during static leg exercise seem to develop independently of changes in
365 cardiac output [30]. Regarding our observations, we cannot refute the possibility that with increasing
366 number of repeated cycling sprints there was an enhanced blood pressure response resulting from
367 heightened exercise pressor reflex, central drive, and strongest Valsalva manoeuvre, thus increasing the
368 elevation in O_2Hb concentration across the sprints set, regardless of changes in cardiac output [28,32].

369 Despite its relationship with the changes in cerebral O_2Hb and tHb concentration during sprints, cardiac
370 output seems to have little influence on cerebral haemoglobin concentration levels between sprints.
371 Following each sprint, cerebral oxygenation levels rapidly returned to baseline, while cardiac output
372 remained elevated, indicating that a high cardiac output alone is insufficient to maintain elevated
373 cerebral oxygenation levels. The rapid decrease in cerebral oxygenation levels with the end of each sprint
374 could have been caused by a sudden decrease in mean arterial pressure upon cessation of the exercise [26].
375 Curtelin et al. [12] reported a drop in mean blood pressure immediately after the end of a Wingate test,
376 which was approximately three times larger than its increase during the effort. This decrease in mean
377 blood pressure was accompanied by a significant decline in MCAv, which lasted for approximately 40 s
378 before returning to pre-exercise levels [12]. However, a decrease in CBF at the end of exercise might occur
379 independently from changes in arterial blood pressure. For instance, Pott et al. [24] reported a continuous
380 decrease in MCAv after each rowing catch movement despite stable mean blood pressure, which the
381 authors suggest could have been caused by delayed response of dynamic cerebral autoregulation. A
382 similar event could have taken place during the repeated sprints, given the match between the sprints'
383 duration and the phase delay of cerebral autoregulation [14].

384 The findings of this study showed that the repeated sprints exercise led to a progressive decrease in
385 cerebral O_2Hb and an increase in HHb, with considerable interindividual variation (Fig 3). The rise in HHb
386 outweighed the decrease in O_2Hb, resulting in an increase in cerebral tHb at the end of the repeated
387 sprints exercise. These changes in cerebral oxygenation partially agree with MCAv data recorded during
388 the Wingate test, revealing decreased CBF and cerebral oxygenation levels caused by augmented
389 cerebral vascular resistance at later sprint stages [12]. A similar response is observed during heavy
390 resistance exercise, with cerebrovascular resistance increasing during exercise but lowering rapidly once
391 the muscular activity stops [26]. Thus, the data obtained from this study extends these observations,
392 showing similarly restrained cerebral blood flow and cerebral oxygen delivery during short cycling sprints,
393 even with moderate perceived exertion levels.

394 Dynamic cerebral autoregulation is a primary mechanism for regulating cerebrovascular resistance. While
395 the effect of exercise on this mechanism is controversial, with studies indicating decreased efficacy of
396 dynamic autoregulation during exhaustive aerobic exercise [33], other studies report unchanged or even
397 enhanced cerebral autoregulation during aerobic exercise [34]. The protracted CBF recovery from
398 hypotension after heavy aerobic exercise [35] and sprinting [12] suggests a diminished cerebral
399 autoregulation efficacy, possibly combined with a reduced gain of the arterial baroreflex [35], maybe to
400 compensate previous mismatch between oxygen delivery and demand. While the present study cannot
401 address the impact of short sprint performance on dynamic autoregulation, the rapid decrease of O_2Hb at
402 the end of the sprints when cardiac output was elevated suggests normal cerebral autoregulation during
403 this type of exercise. Future studies may test the effect of different sprint/recovery cycle durations on
404 cerebral haemoglobin responses. Importantly, a decline in O_2Hb during repeated sprints contrasts with
405 the increase in cerebral oxygenation during lower body resistance exercise [36]. Since decreased brain
406 oxygen saturation is observed in other sprint studies [12], it may be concluded that the extreme intensity of
407 sprint exercise and the recruitment of a large muscle mass reduce cerebral blood flow and lower cerebral
408 oxygenation. In addition, the cessation of the repeated sprints exercise was accompanied by a cerebral
409 reactive hyperaemic response, with a steep increase in O_2Hb within minutes following exercise (Fig. 3).
410 This strengthens the claim that cerebral oxygen delivery is reduced during repeated sprint exercise.
411 Finally, the absence of significant increases in both O_2Hb and tHb during the sprint set suggests that
412 repeated sprint performance does not cause a large increase in cerebral blood flow that could
413 endangered the brain.

414 In this experiment, we investigated the effect of glucose ingestion and acute hyperglycaemia on the
415 response of cerebral haemoglobin to sprint exercise. Although glucose is the primary energy source for
416 the brain, hyperglycaemia has been shown to decrease the cerebral metabolic rate of oxygen [16] and brain
417 connectivity [37] in healthy young participants at rest. Furthermore, acute and chronic hyperglycaemia
418 decreases cerebral blood flow [38]. In our study, we found that glucose ingestion did not affect O_2Hb and

419 HHb responses to the repeated sprints exercise, despite improving performance. However, glucose
420 ingestion significantly enhanced reactive hyperaemia, resulting in faster and higher levels of O_2Hb
421 concentration during the post-exercise period. The relationship between post-exercise hyperaemia and
422 any baseline changes in CBF or cerebral blood volume caused by glucose ingestion requires further
423 investigation.

424 Study limitations

425 There are several limitations in this study that should be acknowledged. One significant limitation is the
426 unknown effect of extracerebral haemodynamics (scalp and skull) on cerebral oximetry measurements.
427 Since the emitted light from NIRS travels through extracerebral tissues and the cerebral cortex, changes
428 in blood volume and flow in superficial tissues can affect the NIRS measurements. The design of the NIRS
429 probe used in this study, with rigid circular optodes holders firmly secured to apply pressure on the
430 forehead skin underneath the optodes, helped to minimise changes in skin blood flow and their impact
431 on NIRS recordings. Studies have shown that facial skin blood flow and vascular conductance increase
432 with dynamic exercise intensity without plateauing [39], while ocular circulation is similar to cerebral
433 circulation, decreasing from elevated values to baseline in response to exhaustive dynamic exercise [40].
434 Based on these findings, it can be inferred that the oximetry data obtained during the repeated sprints
435 exercise primarily reflect the changes in cerebral circulation. Future studies may assess the contribution
436 of forehead skin to changes in haemoglobin concentration in the frontal region of the cerebral cortex
437 during repeated sprints.

438 Another limitation of this study is the absence of measurements of $P_{ET}CO_2$ and blood pressure, which
439 could have provided a more comprehensive understanding of the physiological changes occurring during
440 the repeated sprints exercise.

441 Perspective

442 We provide evidence that the performance of short cycling sprints produces a significant impact on
443 cerebral oxygenation. We recorded a temporary increase in O_2Hb, HHb and tHb concentrations during
444 the sprints, with the magnitude of these changes growing with the number of sprint repetitions. In
445 addition, we found a positive correlation between changes in O_2Hb and cardiac output, which
446 progressively increased throughout the repeated sprint set and remained elevated during the rest
447 periods between sprint repetitions. However, O_2Hb rapidly returned to baseline or below during these
448 rest periods. Contrary to the sprint response, we observed a decrease in O_2Hb concentration throughout
449 the repeated sprint set. Conversely, HHb increased, suggesting inadequate brain oxygen delivery and
450 insufficient cerebral oxygenation oxygen delivery during the all-out repeated cycling. Although not
451 affecting haemoglobin concentration responses, glucose ingestion did improve performance and was

452 associated with a faster recovery of O_2Hb levels during the post-exercise period. In conclusion, our
453 findings suggest that repeated spring exercise elicits a transient and modest increase in cerebral O_2Hb
454 and tHb during the physical effort itself. However, there is an overall decrease in cerebral oxygenation,
455 leading to a robust post-exercise cerebral reactive hyperaemia in certain individuals. Importantly, our
456 data does not indicate a marked elevation in O_2Hb or cerebral blood flow during short all-out cycling
457 sprints in young, non-trained individuals, thereby reducing the likelihood of cerebral hyperperfusion
458 during this type of exercise.

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Conflicts of interests

The authors report no conflict of interests.

Author contributions

P.A.S. and H.M. conceptualised the study, conducted data collection, analysis, and interpretation, and drafted the original manuscript. W.Z., W.L. and F.R. contributed to conducting the experiments, data collection, and analysis. X.Z. assisted in the acquisition and analysis of NIRS data. Z.Y. and Z.K. contributed to the study conception and provided guidance in manuscript writing and revision.

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584 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

585 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 for heart rate
586 during the incremental maximal exercise test; $\dot{W}_{peak}$, peak value for power output during the incremental maximal exercise test.

587

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

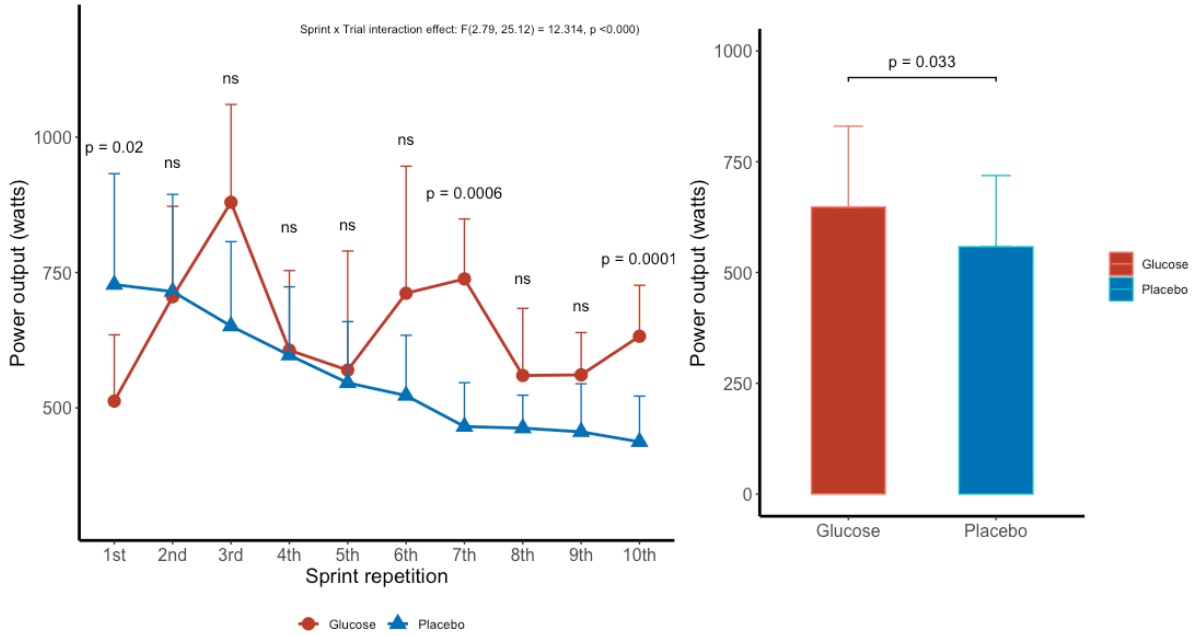
	Mean ± SD	**2.86 ± 1.56**[a]	-0.57 ± 1.34	-0.02 ± 0.67	-0.28 ± 0.74	**2.94 ± 1.82**[a]	-0.86 ± 1.83
5th	Placebo	3.08 ± 1.95	-0.59 ± 0.67	0.10 ± 0.66	-0.16 ± 0.43	3.20 ± 2.22	-0.72 ± 0.82
	Glucose	3.17 ± 1.70	-0.24 ± 1.26	-0.05 ± 0.52	-0.19 ± 0.81	3.16 ± 1.60	-0.41 ± 1.79
	Mean ± SD	**3.12 ± 1.83**[a]	-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, $p < 0.05$.

598 Figure 1

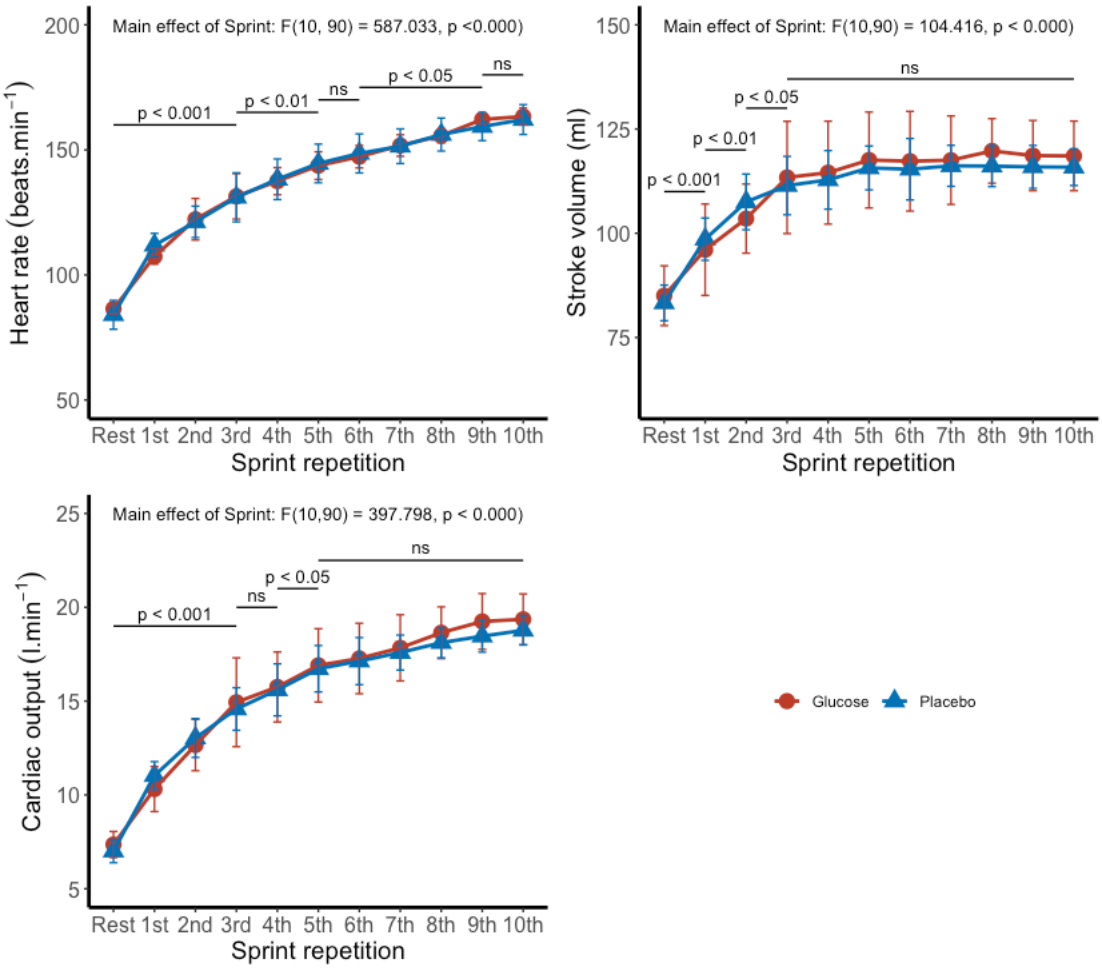


599

600 Figure 1: Mean power output during each repeated sprint for GLU (red line) and PLA (blue line) trials (left
601 panel). Pooled mean power output for GLU (red bar) and PLA (blue bar) trials (right panel). Data
602 presented as mean ± SD, ns indicates non-significant difference, sample size: n = 10.

603

604 Figure 2

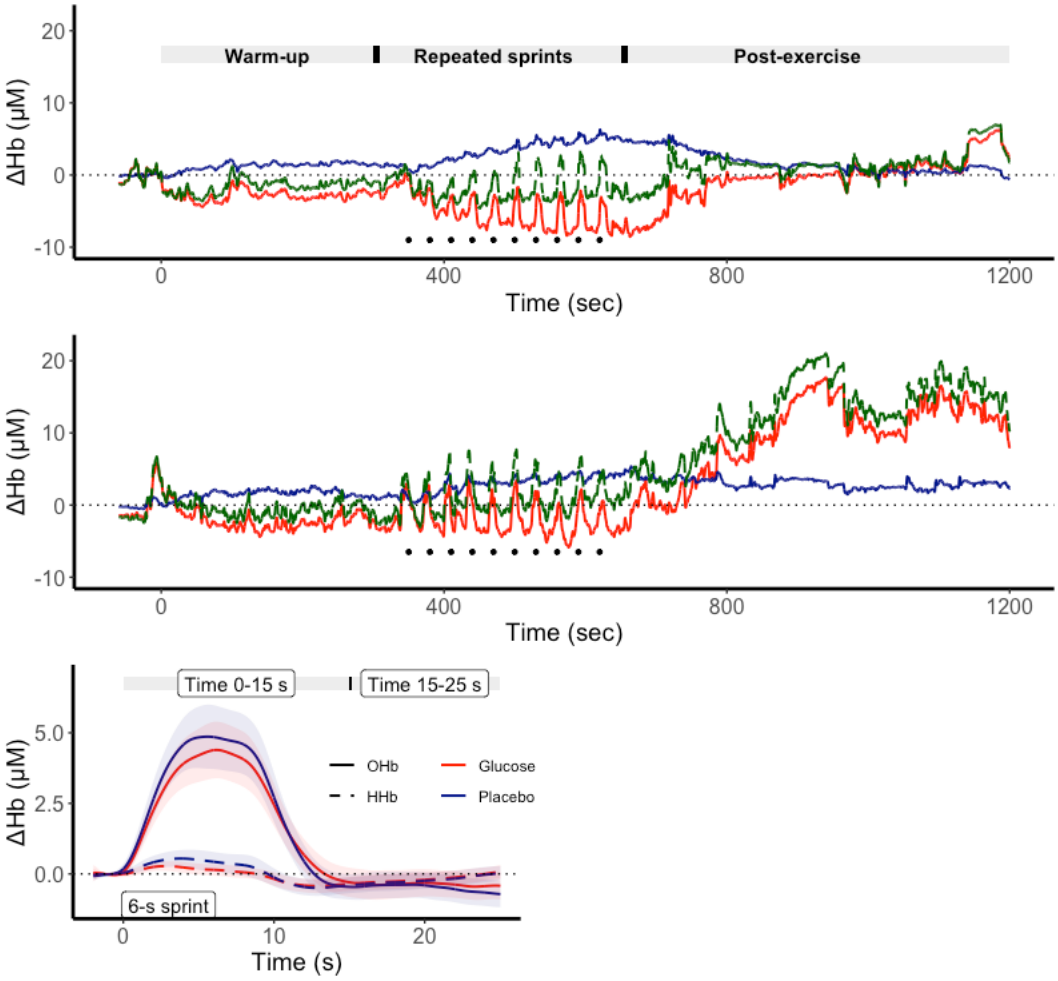


605

606 Figure 2: Heart rate, stroke volume and cardiac output (mean ± SD) at rest and during each repeated
607 sprint for GLU (red lines) and PLA (blue lines) trials. The insert is the output of the two-way repeated
608 measures ANOVA for the significant main effect of sprint repetition (excluding the resting values). P-
609 values are from backward difference contrasts, comparing each sprint repetition with the immediately
610 preceding one, including the first sprint and resting values. Sample size: n = 10.

611

612 Figure 3



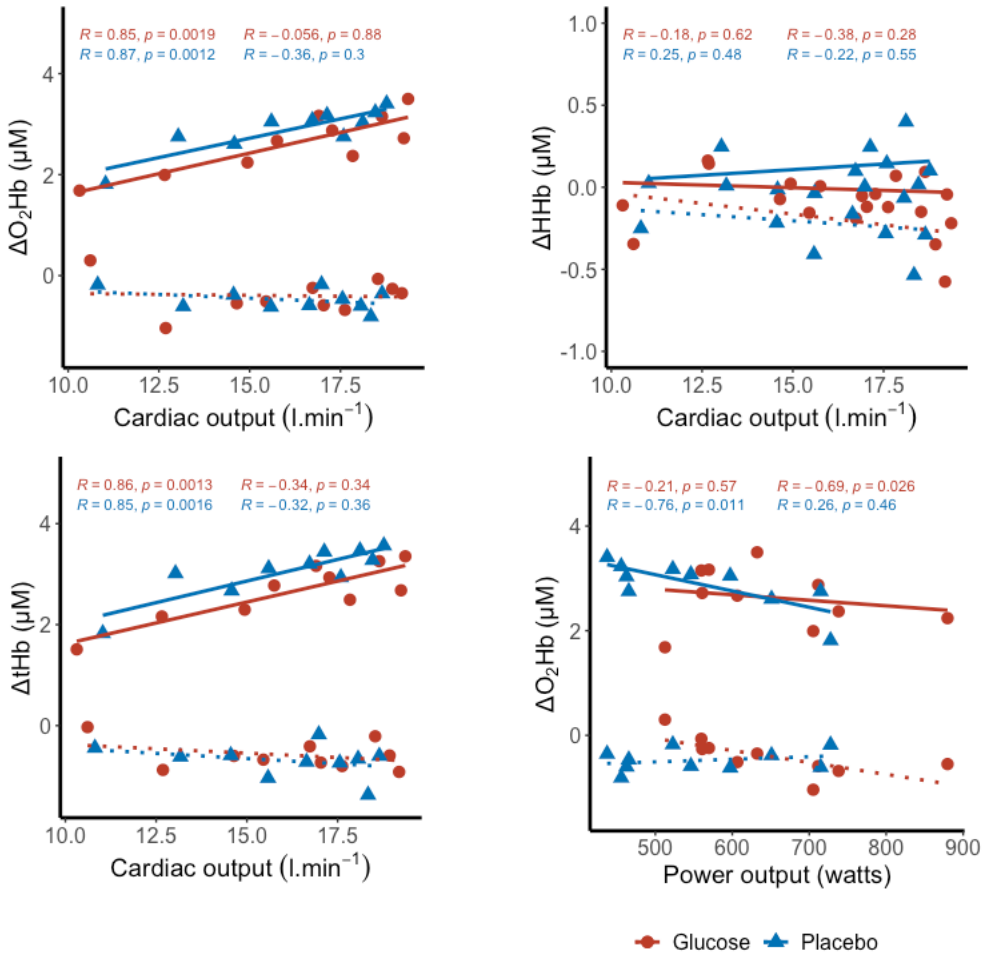
613

614

615 Figure 3: Changes in O_2Hb, HHb and tHb during the repeated sprints exercise. Top and middle graphs
616 display the concentration changes of O_2Hb (red line), HHb (blue line) and tHb (green line) recorded in two
617 participants. The records start immediately before the warm-up. In both participants, a transient increase
618 in O_2Hb, HHb, and tHb during each sprint (black dots) is clearly observed. In the top graph, there was a
619 notable decrease in O_2Hb concentration and increase in HHb throughout the repeated sprints exercise,
620 followed by a return of both O_2Hb and HHb towards pre-exercise values during the recovery period. The
621 participant in the middle graph exhibited lesser changes in O_2Hb and HHb concentration during exercise
622 but displayed substantial post-exercise cerebral hyperaemia. The bottom panel presents the overall
623 average for ΔO_2Hb and ΔHHb during the repeated sprints, revealing the rapid increase in cerebral
624 haemoglobin concentration during the sprint and its quick decrease soon after the end of the sprint. Each
625 tracing is the mean of 10 participants, 10 sprints and 8 channels. The shadowed areas represent the
626 standard error of the mean; sample size: n = 10.

627

628 Figure 4



629

630 Figure 4: Scatter plots illustrating the relationship between ΔO_2Hb (upper left graph), ΔHHb (upper right
631 graph), and ΔtHb (lower left graph) with cardiac output, as well as between ΔO_2Hb and power output
632 (lower right graph), during the sprints (solid lines) and the time interval between sprints (dotted lines) in
633 GLU (red lines and red circles) and PLA (blue lines and blue triangles) trials. The Pearson's R coefficients
634 and respective p-values are presented at the top of the graphs for values during the sprint (left column)
635 and the time interval between the sprints (right column) for GLU (red font) and PLA (blue font) trials.
636 During the sprint, a significant correlation was found between cardiac output and ΔO_2Hb, as well as ΔtHb.
637 No significant correlation was found between cardiac output and ΔHHb, and between cardiac output and
638 ΔO_2Hb or ΔtHb during the time interval between sprints. In the PLA trial, mean power output exhibited a
639 negative correlation with ΔO_2Hb during the sprints, while in the GLU trial, mean power output showed a
640 negative correlation with ΔO_2Hb during the time interval between sprints.

641