Performance Nutrition

BRIEF REPORT

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Personalised carbohydrate feeding during exercise based on exogenous glucose oxidation: a proof-of-concept study

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Abstract

Background Carbohydrate (CHO) feeding during prolonged exercise is well-established for its ergogenic effects primarily by maintaining glycemia and carbohydrate oxidation. Current CHO intake guidelines propose broad intake recommendations based on exercise duration. Recent evidence demonstrating individual differences in exogenous CHO oxidation suggests a need for personalised approaches. This study aimed to determine whether exogenous glucose oxidation (GLUexo) rates achieved with a high glucose dose (90 g/h) could inform a personalised glucose dose (PC) that maintains comparable GLUexo with reduced intake.

Methods Eleven endurance-trained participants (6 females, 5 males; $\dot{V}O_2$ peak: 59.2 ± 7.3 mL/kg/min) completed two 150-min cycling exercise bouts at 95% of the intensity corresponding to the first lactate threshold with different glucose intakes: a high glucose dose (90 g/h) and a PC dose. Drinks were enriched with U-^{13}C glucose allowing for estimation of GLUexo rates. Peak GLUexo was first determined with the high glucose dose. The PC dose was calculated assuming the peak GLUexo would represent an oxidation efficiency of 80%.

Results The PC dose provided 28 ± 11% less glucose (mean: 65 ± 10 g/h) compared to 90 g/h, yet peak GLUexo was not any lower with PC (0.91 ± 0.19 g/min) vs. 90 g/h (0.90 ± 0.15 g/min; $p = 0.977$). Bland–Altman analysis showed good agreement between trials (mean bias: 0.00 g/min; limits of agreement: ± 0.20 g/min). Differences in plasma glucose, lactate concentrations, or endogenous CHO oxidation rates between conditions were not statistically significant or biologically meaningful. Ratings of perceived exertion and stomach fullness were lower with PC compared to 90 g/h ($p < 0.05$).

Conclusions Personalised glucose feeding strategies based on direct measurement of individual GLUexo rates can optimise glucose provision during exercise by achieving comparable oxidation rates with reduced glucose intake, whilst simultaneously minimising perception of effort and gastrointestinal discomfort.

Keywords Physical activity, Sugars, Sports nutrition, Endurance, Metabolism

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Introduction

The ergogenic effects of carbohydrate feeding during prolonged, strenuous exercise are well established, with physiological mechanisms of action attributed to maintenance of glycaemia and carbohydrate oxidation as well as sparing of endogenous carbohydrate utilisation (i.e., liver and/or muscle glycogen) [1, 2]. Accordingly, athletes' guidelines for carbohydrate feeding during exercise are well developed with recommendations regarding the dose and type of carbohydrate made in relation to the duration of exercise [1, 3]. In respect of carbohydrate dose, the recommendations usually provide a range of carbohydrate amounts within which athletes should target their intake (e.g., 30–60 g/h for exercise lasting 1–2.5 h and up-to 90 g/h for exercise lasting more than 2.5 h) [1, 4]. This is understandable given the breadth of sporting contexts athlete guidelines seek to address, but translating such recommendations into individual, athlete-specific, advice requires fine tuning, often through trial and error.

The optimal amount of carbohydrate to be ingested for improvements in performance during exercise has been suggested to be that which results in maximal exogenous carbohydrate oxidation rates without causing gastrointestinal problems [5]. It was previously suggested that individual differences in exogenous carbohydrate oxidation exist, but they are generally small [3]. However, there is a growing body of evidence that interindividual variations in oxidation rates of ingested carbohydrates are higher than previously thought. A recent study investigated the relationship between body size and power output during cycling exercise as factors affecting the capacity to oxidise exogenous glucose and found appreciable variability between individuals alongside a positive relationship between oxidation rates and body size [6]. Whilst multiple replicates in random order are required to separate inter-individual variance from intra-individual variance in response to carbohydrate ingestion [7], high variability was also discovered in a study in which participants were fed fixed, large amounts (i.e., 120 g/h) of carbohydrate during exercise [8]. During prolonged multiple-hour long endurance events (e.g., cycling and triathlon) high exogenous carbohydrate availability becomes especially important as muscle contraction becomes very reliant on blood glucose [9, 10] and thus suboptimal carbohydrate availability could hinder performance. However, too aggressive carbohydrate feeding has been shown to not only negatively affect gastrointestinal problems [11, 12], but may also accelerate endogenous carbohydrate use [13, 14]. Thus, carbohydrate intakes required to achieve peak exogenous carbohydrate oxidation during exercise, whilst minimising gastrointestinal problems, may differ between individuals, potentially justifying an individualised approach.

Evidence for individualising carbohydrate intake based on the effects of personal factors such as body size/mass [3, 4, 6], sex [15, 16] or diet (e.g., training the gut) [17, 18] on exogenous carbohydrate is either lacking or inconsistent, and even when a positive relationship is found, it only partially explains the observed variance. Whilst these remain important areas of scientific investigation, it is likely that personal, yet unexplained, factors interact to exert an individualised impact of carbohydrate intake on exogenous carbohydrate oxidation. A future advance could, therefore, be tailoring of carbohydrate intakes for athletes based on their individual peak capacity for exogenous carbohydrate oxidation, as previously suggested [2]. Thus, the purpose of the present proof-of-concept study was to establish if peak exogenous glucose oxidation rates obtained when large amounts of glucose (i.e., 90 g/h) were provided during exercise could be used to determine a personalised carbohydrate dose (PC). The hypothesis was that peak exogenous glucose oxidation rates would be comparable between the 90 g/h dose and the PC dose.

Methods

Participants

Eleven participants (6 females, 5 males), all of whom participated regularly in endurance-type activity, completed the study. Participants were classified as healthy by completion of a general health questionnaire, and additional inclusion criteria included completing endurance-type exercise ≥ 3 times per week and a peak oxygen uptake ($\dot{V}O_2$peak) of ≥ 50 mL/kg/min or ≥ 55 mL/kg/min for female and male participants, respectively. Participant characteristics are provided in Table 1. Participants gave their written informed consent to participate in the study, which was approved by the Science, Technology, Engineering and Mathematics Ethics Committee, University

Table 1 Participant characteristics

	ALL	MALE	FEMALE
Age [years]	23 ± 2	24 ± 3	22 ± 0
Height [cm]	171 ± 11	179 ± 10	164 ± 5
Body Mass [kg]	66.4 ± 9.3	73.7 ± 8.4	60.3 ± 4.1
$\dot{V}O_2$ peak [mL/kg/min]	59.2 ± 7.3	65.4 ± 6.0	54.1 ± 2.5
LT1 [W]	194 ± 48	229 ± 51	166 ± 18
LT1 [W/kg]	2.9 ± 0.6	3.2 ± 0.8	2.7 ± 0.2
LT2 [W]	236 ± 55	285 ± 41	195 ± 17
LT2 [W/kg]	3.5 ± 0.6	3.9 ± 0.8	3.2 ± 0.1
Workload [W]	184 ± 46	217 ± 49	157 ± 17

LT1 First Lactate Threshold, *LT2* Second Lactate Threshold

of Birmingham, Birmingham, United Kingdom (Reference: ERN_ 0733).

Experimental design

After baseline/familiarisation testing, each participant performed two main trials in a repeated measures experimental design. Participants were asked to refrain from exhaustive exercise and alcohol as well as to record and replicate dietary intake for 24 h prior to the main trials, which consisted of 2.5 h steady-state exercise on a cycle ergometer at a workload corresponding to 95% of the first lactate threshold (LT1; see *Baseline testing and familiarisation*). During exercise, participants ingested one of two glucose beverages, enriched with U-^{13}C-glucose to permit measurement of exogenous glucose oxidation rates (GLU_{exo}). Indirect calorimetry measurements were taken with expired breath and venous blood samples collected throughout the exercise to characterise substrate oxidation and metabolic responses. The first main trial provided glucose at a rate of 90 g/h during exercise (Trial 90). GLU_{exo} with glucose (or glucose polymer) increase in a dose-dependent manner and plateau when ingested at rates ≥ 60–72 g/h [16, 19], thus a dose of 90 g/h was selected to ensure peak GLU_{exo} would be attained. Peak GLU_{exo} determined for each participant during Trial 90 was used to calculate a personalised glucose dose which was ingested during the second main trial (Trial PC; see *Carbohydrate drinks*). Due to sample processing time for breath ^{13}C:^{12}C ratio analysis, Trial PC took place 2–3 weeks after Trial 90.

Baseline testing and familiarisation

Participants' height (Stadiometer Model 220, Seca, Germany), body mass (Champ II, OHAUSE, Switzerland) and body composition (BODPOD; Cosmed, Italy) were determined. Participants then commenced a submaximal exercise test on a stationary cycle ergometer (WattBike AtomX, WattBike Ltd, UK) to determine LT1. Briefly, exercise began at a workload between 50–100 W and was increased by 25 W every 4 min until the participant attained blood lactate concentrations above the second lactate threshold (LT2). At rest, and during the last minute of each 4-min stage a finger-tip capillary blood sample was obtained and analysed immediately for blood lactate concentration (Biosen C-Line Glucose and Lactate analyser, EKF-diagnostic GmbH, Germany). LT1 and LT2 were determined using the ExPhysLab App (https://www.exphyslab.com). LT1 was defined as the lowest intensity at which there is a sustained increase in blood lactate concentration of more than 0.5 mmol/L above resting values [20] with LT2 defined using the Modified D_{max} method [21, 22]. Heart rate (HR) was measured continuously (Polar 10, Polar Electro Oy, Finland) and Ratings of Perceived Exertion (RPE; [23]) determined at the end of each stage. On completion of this test participants rested for 10–20 min prior to completing a maximal exercise test to determine VO_2peak, using the same cycle ergometer.

The VO_2peak test commenced at a workload between 50–100 W and increased by 25 W every minute until task failure or until a cadence of > 50 revolutions per minute could not be maintained. HR and RPE were monitored as describe above. Indirect calorimetry was performed throughout exercise using an online automated gas analyser (Vyntus, Vyaire Medical, USA), to determine oxygen uptake (VO_2) and carbon dioxide production (VCO_2). The volume transducer, oxygen, and carbon dioxide sensors were calibrated before each measurement as per the manufacturer's instructions. VO_2peak was calculated as the highest 30 s average of VO_2. Participants were given a 10-min break before completing a familiarisation session which involved 1-h cycling at an intensity corresponding to 95% LT1 with the provision of glucose beverages during exercise delivering carbohydrate at an ingestion rate of 90 g/h.

Experimental protocol

Participants attended the laboratory in an overnight fasted state between 6:00 and 9:00 AM. Upon arrival at the laboratory, participants were fitted with an intravenous cannula placed in an antecubital vein for subsequent blood sampling. Additionally, a resting expired breath sample was collected into 10-mL evacuated tube (Exetainer Breath Vial, Labco Ltd., UK) which was filled directly from a mixing chamber to subsequently determine the $^{13}C/^{12}C$ ratio in expired CO_2 at rest and every 30 min during subsequent exercise. After 5 min of low intensity warm-up, intensity was increased to 95% LT1 for the subsequent 150 min. Respiratory gas exchanges (VO_2 and VCO_2) were measured, and venous blood sample obtained every 30 min during exercise. At the same time points HR was recorded, RPE was evaluated and gastrointestinal comfort (GC) assessed by a 10-point Likert scale looking at nausea, stomach fullness, and abdominal cramping [24].

Breath and drink samples analysis

^{13}C isotopic enrichment of breath samples was determined using gas chromatography isotope ratio mass spectrometry (Europa Scientific Hydra 20–20, United Kingdom), whereas ^{13}C enrichment of ingested carbohydrates was determined by elemental analyser isotope ratio mass spectrometry (Europa Scientific Hydra 20–20). The isotopic enrichment of expired breath samples and carbohydrate drinks was expressed as δ per mL difference between the $^{13}C/^{12}C$ of the sample and a known

laboratory reference standard using an established equation [25]. $\delta^{13}C$ was then related to an international standard Pee Dee Bellemnitella (PDB). The final enrichment of the drink was 34.74 ± 2.23 δ‰ versus PDB in the Trial 90 and 33.70 ± 2.46 δ‰ versus PDB in Trial PC.

Calculations

Total carbohydrate and fat oxidation rates were calculated using equations by Jeukendrup and Wallis [26] assuming protein oxidation to be negligible:

$$\text{Total Carbohydrate Oxidation}\left[\frac{g}{min}\right] = 4.210 \cdot VCO_2 - 2.962 \cdot VO_2$$

$$\text{Total Fat Oxidation}\left[\frac{g}{min}\right] = 1.695 \cdot VO_2 - 1.701 \cdot VCO_2$$

In which VO_2 and VCO_2 are measured in Litres per minute.

GLU_{exo} were subsequently calculated using the following equation [27]:

$$\text{Exogenous Carbohydrate oxidation}[g/min] = VCO_2 \cdot \frac{\delta Exp - \delta Exp_{bkg}}{\delta Ing - \delta Exp_{bkg}} \cdot \frac{1}{k}$$

where δExp is the ^{13}C enrichment of the expired air at various time points, δIng is the enrichment of the ingested beverage, δExp_{bkg} is the ^{13}C enrichment of the expired air at rest before exercise, and k is the amount of CO_2 (in L) produced by the complete oxidation of 1 g of glucose ($k = 0.7467$ L). GLU_{exo} were analysed for the whole duration of the exercise as 30 min is sufficient for the bicarbonate pool to turn over [28].

Carbohydrate drinks

During the first 2–3 min of exercise, participants were asked to consume an initial bolus (i.e., 300 mL) of a glucose-containing drink. Following this, participants were asked to consume smaller (i.e., 150 mL) dose of the same drink at 15-min intervals during the exercise bout. Total fluid intake during each trial was 1650 mL. The first experimental visit (Trial 90) provided glucose (Dextrose, Bulk, UK) at the rate of 90 g/h (i.e., 225 g in total), whereas during the second experimental visit (Trial PC), personalised dose of glucose was provided as explained below. A small amount (i.e., 0.5 mg per gram of glucose) of stable isotope tracer $U^{13}C_6$-glucose (Cambridge Isotope Laboratories Inc., USA) was added to the drinks to determine GLU_{exo} as explained above.

Once GLU_{exo} were determined for Trial 90, dose used in Trial PC was established. Current literature suggests that oxidation efficiency of ingested glucose (i.e., amount of glucose oxidised in relation to the amount ingested) is around ~ 70–90% [29]. Based on this, the obtained peak GLU_{exo} was assumed to represent 80% of the dose provided during Trial PC. Mean intake in Trial PC was 65 ± 10 g/h (range: 49–80 g/h).

Blood analyses

Venous blood samples (~ 6 mL) were collected into EDTA tubes, stored on ice and then centrifuged at 4 °C and 1,006 × g for 15 min. Aliquots of plasma were then stored at − 70 °C and later analysed for glucose (Glucose Assay GL3881; Randox, UK) and lactate (Lactate Assay LC3980; Randox, UK) using an automated photometric based clinical chemistry analyser RX Daytona + (Randox, UK).

Statistics

Data were initially tested for sphericity using Mauchly's test. A two-way repeated-measures ANOVA was then conducted to compare differences between conditions. When the assumption of sphericity was violated, the Greenhouse–Geisser correction was applied. For significant time × condition interactions identified by ANOVA, post-hoc comparisons were performed using paired t-tests, with the Tukey correction applied to account for multiple comparisons. In cases where missing values were present (i.e., gas exchange data and blood samples), a mixed-models analysis was used to account for incomplete data. Gas exchange data had 2 missing data points (1 per participant) due to an error with metabolic cart, whereas blood samples had 3 trials without blood being collected (1 full participant and 1 full participant in Trial PC) due to difficulties with cannulations.

Peak GLU_{exo} were compared between conditions using a paired t-test. Normality of the data was assessed with the Shapiro–Wilk test, and the assumption of normality was met. To assess agreement between the two conditions, Pearson's product-moment correlation was assessed and Bland–Altman plots were constructed by plotting the differences between conditions against their average values. Limits of agreement (mean difference ± 1.96 SD) were calculated to quantify the level of agreement.

The associations between peak glucose oxidation rates and participant characteristics (body mass, height and power output) were quantified using Pearson's product-moment correlation coefficients.

A multiple linear regression was conducted to examine the association between predictor variables (Body Height, Body Mass, and Power Output) and the log10-transformed peak GLU_{exo} from the Trial 90. The log10 transformation was applied to normalize the distribution of the dependent variable, reduce skewness, and stabilize variance, ensuring that the assumptions of linear regression were met.

All data are presented as mean ± SD, and statistical significance was set at $p < 0.05$. Data analysis was performed using Prism (Version 10; GraphPad Software, USA).

Results

Stable isotope measurements

Expired breath ^{13}C is shown in Fig. 1. Breath ^{13}C enrichment increased over time ($p < 0.001$), but differences between conditions were not statistically significant ($p = 0.264$) and there was no statistically significant time x condition interaction ($p = 0.491$).

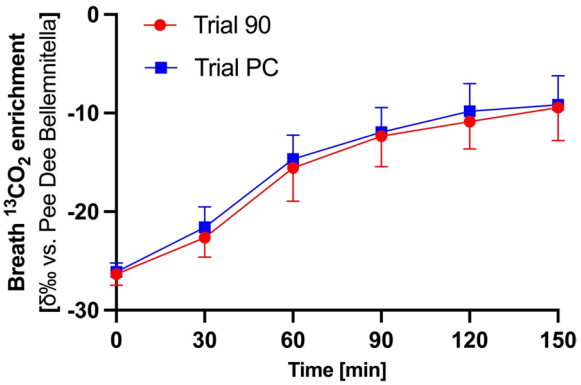

Fig. 1 Breath enrichment over time. Data are Mean ± Standard Deviation. $n = 11$

Substrate oxidation rates

Total CHO oxidation rates (Fig. 2a) displayed little change over time ($p = 0.965$) or between the conditions ($p = 0.199$). Similarly, fat oxidation rates (Fig. 2b) displayed little change over time ($p = 0.780$) or between conditions ($p = 0.592$). Exogenous glucose oxidation rates (Fig. 2c) increased over time ($p < 0.001$) with differences between conditions not statistically significant ($p = 0.473$). The contribution of endogenous carbohydrate oxidation (Fig. 2d) towards total energy turnover decreased over time ($p < 0.001$) but with little differences between the conditions ($p = 0.157$). Peak GLU_{exo} (Fig. 3) displayed little difference between conditions (Trial 90: 0.90 ± 0.15 g/min and Trial PC: 0.91 ± 0.19 g/min; $p = 0.977$) with high degree of correlation (Fig. 3A). Bland–Altman plots with limits of agreement are displayed in Fig. 3B, with limits of agreement (LOA) falling between −0.20 and 0.20 g/min. Oxidation efficiency in Trial PC was 83 ± 9% and in Trial 90 it was 58 ± 9%.

Individual Pearson's product-moment coefficient correlations between height, body mass and power output are presented in Fig. 4. There were no statistically significant correlations.

A multiple linear regression was performed to examine the relationship between predictor variables (Body Height, Body Mass, and Power Output) and log10-transformed peak GLU_{exo}. The overall model was statistically significant ($p = 0.037$) and explained 68.1% of

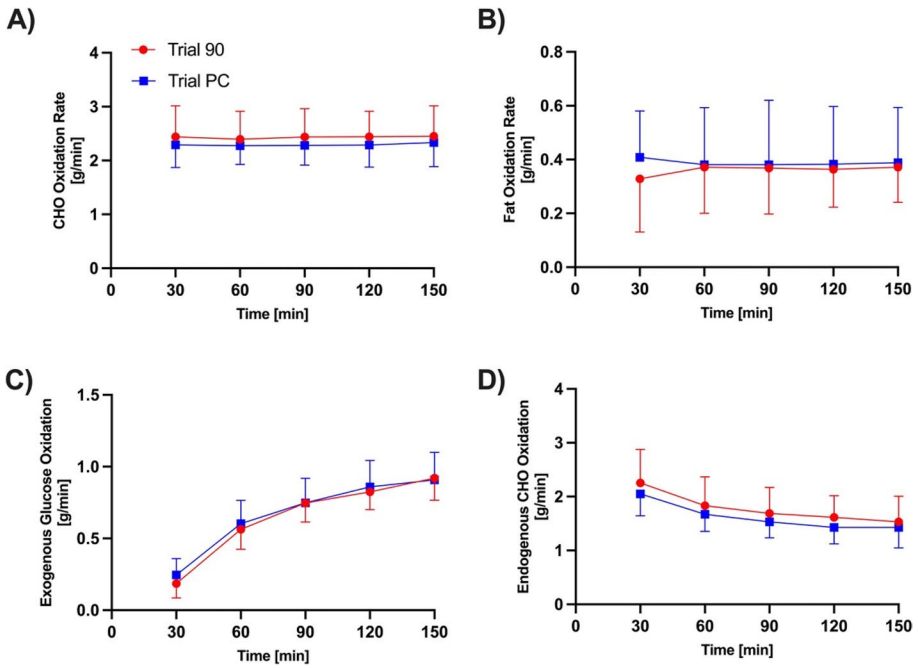

Fig. 2 Total carbohydrate oxidation rates **A**; Fat oxidation rates **B**; Exogenous Carbohydrate oxidation rates **C** and Endogenous Carbohydrate oxidation rates **D**. Data are Mean ± Standard Deviation. $n = 10–11$

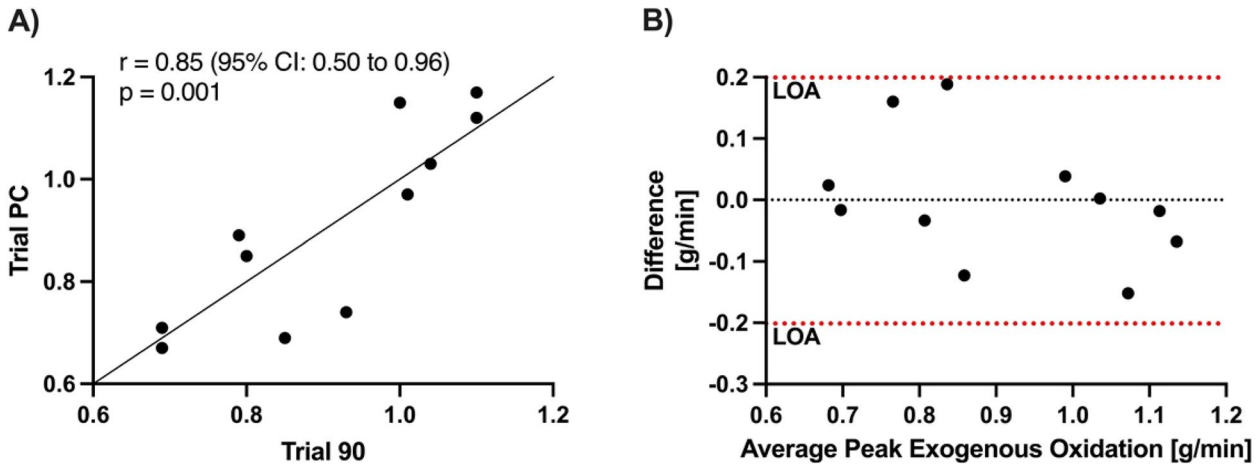

Fig. 3 **A** Pearson's product-moment coefficient correlation including line of identity for peak exogenous carbohydrate oxidation between trials; **B** Bland–Altman plot for peak exogenous carbohydrate oxidation rates. LOA—Limits of agreement

the variability ($R^2 = 0.681$). Power Output ($\beta = 0.001$, $p = 0.045$) and Body Height ($\beta = 0.011$, $p = 0.034$) were significant independent predictors of log10 GLU_{exo}. Body Mass ($\beta = -0.011$, $p = 0.075$) was not a statistically significant predictor.

Blood plasma metabolites

Plasma glucose concentrations (Fig. 5A) increased slightly from baseline during exercise, showing a significant main effect of time ($p < 0.001$). However, no significant differences were observed between conditions ($p = 0.996$), and there was no significant time × condition interaction ($p = 0.491$). Similarly, plasma lactate concentrations (Fig. 5B) demonstrated a significant main effect of time ($p < 0.001$). In contrast, no significant main effect of condition was detected ($p = 0.181$), nor was there a significant time × condition interaction ($p = 0.492$).

Oxygen uptake, carbon dioxide production and heart rate

Data are presented in Table 2. Oxygen uptake increased significantly over time ($p = 0.026$) and tended to be lower in the Trial PC compared to Trial 90 ($p = 0.060$). However, there was no significant time × condition interaction ($p = 0.713$). Carbon dioxide production also increased significantly over time ($p = 0.018$) and was significantly higher in the Trial 90 compared to Trial PC ($p = 0.043$), with no significant time × condition interaction ($p = 0.870$). HR increased significantly over time ($p < 0.001$) and was higher in the Trial 90 compared to Trial PC ($p < 0.001$). Similarly, no significant time × condition interaction was observed for HR ($p = 0.262$).

Perceptual responses

Data are presented in Table 2. Ratings of RPE increased significantly over time ($p < 0.001$) and were higher in the Trial 90 compared to Trial PC ($p = 0.005$), although there was no significant time × condition interaction ($p = 0.912$). Perception of nausea also increased significantly over time ($p < 0.001$) but did not differ significantly between conditions ($p = 0.080$), nor was there a significant time × condition interaction ($i = 0.156$). Perception

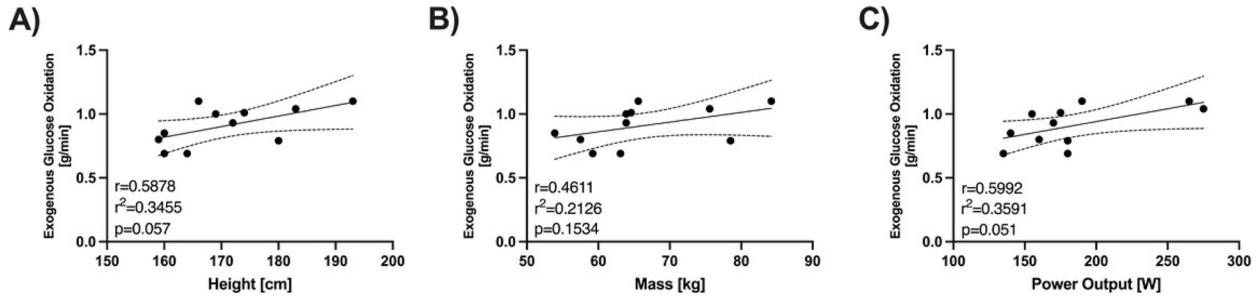

Fig. 4 Pearson's product-moment coefficient correlations between **A** height, **B** body mass or **C** Power output and peak exogenous glucose oxidation rates

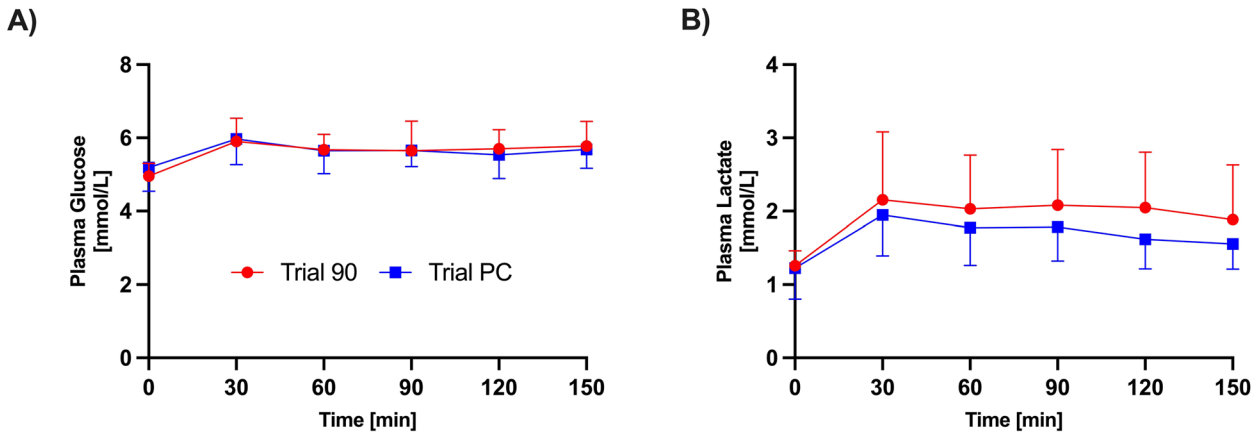

Fig. 5 Blood plasma metabolites. Plasma Glucose (**A**); Plasma Lactate (**B**). Data are Mean ± Standard Deviation. $n = 9–10$

Table 2 Respiratory gas exchange, heart rate and perceptual responses

Time [min]	Trial	VO_2 [L·min^{-1}]	VCO_2 [L·min^{-1}]	HR [bpm]	RPE [6–20]	Nausea [1–10]	S. Fullness [1–10]	A. Cramping [1–10]
30	90	2.6 ± 0.6	2.4 ± 0.5	144 ± 14	12 ± 1	1 ± 1	1 ± 1	1 ± 0
	PC	2.7 ± 0.6	2.4 ± 0.5	139 ± 13	11 ± 1	1 ± 1	1 ± 1	1 ± 0
60	90	2.7 ± 0.6	2.5 ± 0.5	146 ± 13	12 ± 1	2 ± 1	2 ± 1	1 ± 1
	PC	2.6 ± 0.6	2.4 ± 0.5	141 ± 14	11 ± 1	2 ± 1	1 ± 1	1 ± 1
90	90	2.7 ± 0.6	2.5 ± 0.6	148 ± 14	13 ± 1	3 ± 2	3 ± 1	2 ± 2
	PC	2.6 ± 0.6	2.4 ± 0.5	142 ± 14	12 ± 2	2 ± 2	2 ± 2	2 ± 2
120	90	2.7 ± 0.6	2.5 ± 0.5	151 ± 14	13 ± 2	3 ± 2	3 ± 2*	2 ± 2
	PC	2.6 ± 0.6	2.4 ± 0.5	143 ± 11	12 ± 2	3 ± 2	2 ± 2*	2 ± 2
150	90	2.7 ± 0.7	2.5 ± 0.6	152 ± 13	14 ± 2	3 ± 3	3 ± 2*	2 ± 2
	PC	2.7 ± 0.7	2.4 ± 0.6	145 ± 12	13 ± 2	3 ± 2	2 ± 2*	2 ± 2

HR Heart Rate, *S. Fullness* Stomach Fullness, *A. Cramping* Abdominal Cramping, $n = 10–11$

* Denotes a significant ($p < 0.05$) difference between trials at given time point

of stomach fullness increased significantly over time ($p < 0.001$) and was higher in the Trial 90 compared to Trial PC ($p = 0.003$), with a significant time × condition interaction ($p = 0.012$). In contrast, feelings of abdominal cramping did not change significantly over time ($p = 0.130$) and were not different between conditions ($p = 0.557$), with no significant time × condition interaction observed ($p = 0.753$).

Discussion

The purpose of this proof-of-concept study was to determine whether peak exogenous glucose oxidation rates observed with a high glucose intake (90 g/h) could be used to establish a personalised glucose dose (PC). In line with the hypothesis, the PC elicited comparable exogenous glucose oxidation rates during exercise while providing 28 ± 11% less glucose compared to the 90 g/h intake. This reduced carbohydrate load also resulted in lower ratings of stomach fullness and perception of effort. The interpretation and implications of these findings will be further discussed in the context of personalised nutrition strategies.

Current evidence suggests that peak exogenous glucose oxidation rates average approximately 1–1.1 g/min (60–66 g/h) [19] and this evidence is the backbone of the current recommendations for carbohydrate intake during exercise [1, 3–5]. In the sample of the present study, we observed slightly lower peak exogenous glucose oxidation rates (~ 0.9 g/min or 54 g/h). However, the variability in peak glucose oxidation rate was considerable, with rates ranging from 0.7 to 1.1 g/min (42–66 g/h). A recent study reported even larger variability in peak exogenous glucose oxidation rates (~ 0.5–1.5 g/min or 30–90 g/h) [6]. High variability is present also when glucose-fructose based carbohydrates are ingested during exercise [8].

While some degree of variability has been acknowledged in the previous literature [3, 30], the prevailing view has been that this variability is minimal and not worthy of further examination in relation to recommendations for carbohydrate intake during exercise. However, with the growing focus on optimising and individualising nutritional prescriptions, this topic has been revisited. Ijaz et al. [6] sought to clarify the factors contributing to variability by examining the influence body size and exercise intensity has on peak exogenous glucose oxidation rates [6]. Their findings indicated that body size was the best predictor of peak exogenous glucose oxidation rates, and that absolute exercise intensity had little effect. Nevertheless, body size alone could not account for all the observed variability, and carbohydrate intake recommendations based solely on body size could still not be optimal. In the present study, multiple regression analysis revealed that approximately 68% of the variability in exogenous glucose oxidation rates could be explained by a combination of body height and power output. Interestingly, the relationship with body mass did not reach statistical significance. Collectively, individual characteristics likely play a role although unexplained variability remains, preventing accurate predictions of peak exogenous glucose oxidation rates during exercise at the current time.

The present study aimed to explore the individualisation of glucose ingestion rates based on peak exogenous glucose oxidation rates observed when a high dose of glucose (90 g/h) was consumed. It is well established that during exercise, peak oxidation rates do not equate to ingestion rates, with an efficiency of approximately 70–90% [29]. Thus, a slightly larger (i.e., 20%) dose than peak oxidation rates is required to elicit comparable oxidation rates. As a result, glucose dose in Trial PC was 20% higher than measured peak glucose oxidation rates, while still being 28 ± 11% lower than the initial glucose dose. In Trial PC, glucose ingestion rate was 65 ± 10 (range: 49–80) g/h. At this ingestion rate, peak exogenous glucose oxidation rates were not lower than when 90 g/h of glucose was provided. This occurred without a significant change in endogenous carbohydrate oxidation rates, however, it must be noted that present study's design did not allow for partitioning of liver and skeletal muscle glycogen use. Additionally, differences in plasma glucose or lactate concentrations were minimal. Examination of the limits of agreement for peak exogenous carbohydrate oxidation rates between the two conditions reveals that 95% of the differences fall within approximately ± 0.2 g/min, equivalent to ± 12 g/h, which could be a result of daily variability and/or measurement error. Thus, the present data shows that it is possible to elicit peak rates of exogenous glucose oxidation by taking a personalised approach. While on average mean glucose ingestion rate was in line with recommendations for glucose-based carbohydrates (i.e., 60–66 g/h; 4,19), five participants would likely benefit from a dose higher than 66 g and four participants achieved peak exogenous glucose oxidation rates with a dose lower than 60 g/h.

Gastrointestinal problems are common during prolonged endurance events, and undigested or unabsorbed carbohydrates are thought to contribute to these issues [11, 12]. Additionally, evidence suggests that consuming carbohydrates in amounts exceeding an individual's digestive, absorptive and/or oxidative capacity may increase skeletal muscle glycogen utilization, which could counteract the benefits of carbohydrate supplementation [13, 14], something that could not be established from the present study given the lack of muscle glycogen assessment. Personalising carbohydrate intake based on individual peak exogenous carbohydrate (e.g., glucose) oxidation rates may help optimise carbohydrate provision while minimizing the risk of gastrointestinal discomfort and with it associated negative consequences. In the present study, stomach fullness was lower in Trial PC compared to Trial 90, indicating that personalising the glucose dose could help with reducing occurrence of gastrointestinal issues, perhaps through reducing the amount of the unabsorbed carbohydrates in the gut. However, these findings should be interpreted with caution as despite prior familiarisation to the feeding regime to be adopted, Trial 90 always preceded Trial PC. The observed reduction in GI discomfort could therefore be a result of an order effect, which should be investigated in future studies using a randomized or crossover design. The present study only investigated the potential for personalisation of glucose intake during exercise and not a combination of glucose-fructose based carbohydrates that is recommended when ingestion rates are > 60 g/h [1, 4]. Future studies could explore the potential to personalise fructose ingestion rates on the top of individualised glucose ingestion rates to provide athletes with bespoke nutrition recommendations.

In summary, the results of the present proof of concept study demonstrate that with access to an appropriate testing facility and expertise, athletes could personalise their intake of glucose-based carbohydrates to optimise exogenous carbohydrate oxidation during exercise, while minimising the negative effects of under or overfeeding.

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Authors' contributions
TP and GAW contributed to the conception, design, acquisition, analysis, interpretation and manuscript preparation. NCS contributed to the design, acquisition and analysis of data. JGS contributed to the design, interpretation and manuscript preparation.

Funding
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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Participants gave their written informed consent to participate in the study, which was approved by the Science, Technology, Engineering and Mathematics Ethics Committee, University of Birmingham, Birmingham, United Kingdom (Reference: ERN_ 0733).

Consent for publication
Not applicable.

Competing interests
TP has completed paid consultancy for NDuranz. After completion of the research NCS became an employee of Precision Hydration Ltd. For a full list of JTG's disclosures see https://gonzalezjt1.wordpress.com/2024/03/. JTG has received research funding from BBSRC, MRC, British Heart Foundation, Clasado Biosciences, Lucozade Ribena Suntory, ARLA Foods Ingredients and Cosun Nutrition Center; is a scientific advisory board member to ZOE and 6d Sports Nutrition; and has completed paid consultancy for The Dairy Council, PepsiCo, Violicom Medical, Tour Racing Ltd., the European Fruit Juice Association, and SVGC. GAW receives an honorarium for his role as Editor-in-Chief of Performance Nutrition, has received research funding and/or has acted as a consultant for GlaxoSmithKline Ltd (United Kingdom), Sugar Nutrition UK, Lucozade Ribena Suntory Ltd (United Kingdom), Gatorade Sports Science Institute (USA), and Volac International Ltd (United Kingdom).

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