

Research Article

## Postprandial Glucose Responses to Standardised Meals Consumed After Moderate- and High-Intensity Exercise Bouts Across Standard School Days in Healthy Adolescents

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### Abstract

Exercise-induced moderation of postprandial glycaemia in adolescents is unclear and has not been examined under free-living conditions. We assessed the effect of moderate-intensity exercise (MIE) and high-intensity intermittent exercise (HIIE) bouts on subsequent postprandial glycaemic responses across three standard school days. Fourteen healthy adolescents ( $13 \pm 1$  years) completed three conditions in the following order across consecutive days: MIE, 30-min continuous brisk walking; CON, no-exercise control; HIIE, 30-min of  $10 \times 30$ -s sprints interspersed with 2.5-min brisk walking bouts. Participants consumed three standardised meals (breakfast, lunch and dinner) at standardised times. Interstitial glucose, energy intake, sedentary time and physical activity were assessed under free-living conditions. Linear mixed models compared glucose outcomes between conditions, and



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Cohen's *d* effect sizes were calculated. Although non-significant, the reduction in post-breakfast glucose iAUC was moderate for MIE ( $-0.24 \text{ mmol}\cdot\text{L}^{-1}$ ;  $P = 0.59$ ;  $d = 0.77$ ) and large for HIIE ( $-0.26 \text{ mmol}\cdot\text{L}^{-1}$ ;  $P = 0.44$ ;  $d = 0.86$ ) compared with CON. Non-significant, moderate ( $0.37 \text{ mmol}\cdot\text{L}^{-1}$ ;  $P = 0.22$ ;  $d = 0.70$ ) and large ( $0.42 \text{ mmol}\cdot\text{L}^{-1}$ ;  $P = 0.20$ ;  $d = 0.81$ ) increases in post-lunch glucose iAUC were observed for MIE and HIIE compared with CON. Nevertheless, the 24-h mean glucose was stable at  $\sim 5.4 \text{ mmol}\cdot\text{L}^{-1}$  across conditions. The glycaemic variability indices calculated over 24-h after the onset of exercise for each condition including standard deviation ( $P = 0.59$ ) and mean amplitude of glycaemic excursion ( $P = 0.82$ ) were not different between conditions. Thirty-minute bouts of MIE and HIIE did not change postprandial glycaemia or 24-h glycaemic variability significantly in the small sample of healthy adolescents. However, the moderate and large effect sizes suggest both MIE and HIIE reduced breakfast glucose iAUC compared with CON, yet led to increases in post-lunch iAUC in the two exercise conditions. The mismatch between the probability values and effect sizes was a consequence of our COVID-reduced sample. The ramifications of these exercise effects are unclear and need to be confirmed in a larger sample of adolescents.

### Keywords

Glycaemic variability; free-living; school-based; glucose iAUC; continuous glucose monitor

## 1. Introduction

Frequent exposure to high glucose concentrations can adversely impact cardiometabolic health [1]. Multiple rises in glucose were found to be more harmful than constant hyperglycaemia in terms of increased oxidative stress production and impaired endothelial function in adults with and without diabetes [2]. Although glucose concentrations above diabetes diagnostic ranges are uncommon in healthy young people, prediabetes hyperglycaemia, defined as having either high fasting plasma glucose ( $5.5$  to  $6.9 \text{ mmol}\cdot\text{L}^{-1}$ ) or high haemoglobin A1c (HbA1c;  $5.7\%$  to  $6.5\%$ ) were found in  $27.4\%$  of apparently healthy adolescents ( $n = 6225$ ) aged 12 to 19 years [3]. The prevalence of prediabetes in the UK population aged 16 to 39 years increased from  $2.8\%$  in 2003 to  $15.6\%$  in 2011 [4]. This is worrying, as evidence suggests progressive increases in cardiovascular disease (CVD) and all-cause mortality with increased glucose even below the limits set for diagnosing diabetes [5, 6]. Additionally, postprandial hyperglycaemia is associated with an increased risk of type 2 diabetes (T2D) [7] and CVD [8] in healthy adults. Thus, interventions to moderate postprandial glycaemic excursions are essential even in healthy adolescents to prevent T2D and CVD developments, especially knowing that people spend most of the time in a postprandial state [9].

Physical activity (PA) is a potent stimulus for improving insulin sensitivity in adolescents, regardless of adiposity status [10]. PA increases the glucose transporter (GLUT4) translocation to the cell membrane independent of insulin (early phase) and improves insulin sensitivity several hours after an exercise bout (late phase) [11]. Thus, it enhances glucose uptake in contracting muscles with lowered blood glucose. A single bout of moderate or high intensity intermittent exercise in healthy adolescent boys acutely improved insulin sensitivity following oral glucose tolerance test (OGTT) with a greater effect after HIIE [12]. Further, the improvement in insulin

sensitivity lasted 17 to 24 h after exercise in healthy adolescents [13, 14], suggesting sequential (immediate and extended for several hours) improvements in glucose regulation. Declining PA is a major concern among young people [15], with the majority (~70%) of the UK adolescents aged 10 to 18 years not meeting the PA guidelines of at least 60 min of moderate to vigorous PA (MVPA) each day, when measured objectively [16]. Thus, promoting PA by targeting this sub-population is vital to prevent serious health complications given the well-established associations between PA, insulin sensitivity and cardiometabolic risk [17, 18].

Studies examining the acute effect of a single bout of PA on postprandial glycaemia in adolescents have been conducted mainly in controlled laboratory settings. Furthermore, these studies provided meals very different from what adolescents typically consume, particularly liquid-based drinks that contained only a single macro-nutrient (e.g., carbohydrates in the OGTT) [12, 13], which have exaggerated glycaemic responses compared to everyday meals [19]. Therefore, studying the acute impact of exercise on the glucose response to ecologically valid meals is needed. Dring and colleagues utilised a more ecologically valid meal and environment; they showed no improvement in postprandial glycaemia, but reduced insulinaemic response to a mixed meal consumed an hour after a 60-min basketball session consisting of warm-up, skill based drills and small sided games [20]. In a separate study, they reported a similar outcome following 30-min ( $P = 0.08$ ) and 60-min ( $P = 0.03$ ) Loughborough Intermittent Shuttle Test [21] completed at the school compared to a no-exercise condition. However, the residual glycaemic and insulinaemic responses to a standardised breakfast did not last 24 h following exercise [21]. Still, few studies have examined the postprandial glycaemic response to subsequent meals consumed in the same exercise day (i.e., several hours after exercise) in adolescents. Some studies suggested the glucose-lowering effect of exercise may appear on subsequent meals consumed several hours after exercise in adults living with [22] or without diabetes [23, 24]. Thus, whether a single exercise bout has a prolonged influence on postprandial glycaemia or glycaemic variability in response to subsequent meals in healthy adolescents consuming ecologically valid meals is not known.

Continuous glucose monitoring (CGM) is less invasive than finger-prick blood sampling, and can measure interstitial fluid glucose continuously for up to two weeks [25]. Thus, it provides valuable outcomes on the magnitude and duration of glucose fluctuations (or glycaemic variability) in response to PA interventions under free-living conditions, which may not be reflected accurately using standard laboratory techniques such as OGTT [26]. Numerous studies have utilised CGM technology to examine glycaemic variability and postprandial glucose in response to an acute bout of exercise under free-living conditions in adults living with [22, 27] or without T2D [23, 24, 28]. Studies of a similar approach are lacking in a healthy paediatric population; to our knowledge, only one study using CGM in healthy adolescents found favourable postprandial glucose responses to two meals of different energy content when interrupting sedentary time with short activity breaks involving body-weight resistance exercises [29]. While this previous study focused on interrupting the sedentary time with active breaks, our study investigated the acute effect of pre-breakfast, single bouts of moderate and high intensity exercise on glucose concentrations over standard school days in adolescents under free-living conditions while providing ecologically valid meals at typical meal times, i.e. breakfast, lunch and dinner.

## **2. Materials and Methods**

### **2.1 Participants**

Twenty healthy adolescent girls and boys aged 11 to 14 years participated voluntarily from a local school between November 2019 and March 2020. Participants were recruited through school assemblies, where the aims and procedures of the study were presented. Interested students were asked to collect information packs and discuss the study with their parents/guardians. The participants' general health was screened using a questionnaire, and each of the participant's parents/guardians provided written informed consent with participants giving their assent. The participants were included providing they were healthy, not on medication or suffered from a disease (e.g., diabetes) that may affect glucose metabolism, not presented with any injuries or conditions that prevent them from performing any exercise task (e.g., congenital heart disease, musculoskeletal problems, epilepsy, uncontrolled exercise-induced asthma), no allergy or extreme dislikes to the study meals, and no skin conditions (e.g., allergy) that may affect glucose sensor deployment.

This study was conducted in accordance with the ethical standards of Loughborough University Ethics Committee (HPSC reference number: R19-P147). The ethical approval was obtained in October 2019.

### **2.2 Study Design**

The study was an acute, non-random within measures design involving three experimental conditions performed on three consecutive days in a fixed pre-determined order: 1) moderate-intensity exercise condition (MIE); 2) no-exercise control condition (CON); 3) high-intensity intermittent exercise condition (HIIE). As evidence from healthy adolescents suggest that the acute effect of exercise bout on glycaemia lasted for 24 h [13, 14], the study conditions were designed in a specific order in which HIIE was performed the last as it may have a higher and/or a longer lasting effect. Participants were fitted with a glucose monitor (FreeStyle Libre, Abbott Diabetes Care Inc., UK) and an accelerometer (ActiGraph GT3X+, Pensacola, USA) one week prior to the experimental conditions at the school. The participants were asked to scan the glucose monitor with a handheld reader at least every 7 h to avoid data loss. A glucose monitor was inserted in the participant's non-dominant upper arm and an accelerometer was placed around the waist on the participant's right side during waking hours and removed during water-based activities such as bathing or swimming. Thus, participants wore both a CGM and an accelerometer simultaneously for the study period. Participants attended a preliminary visit in the lab on day 2 or 3 of sensor wear for the glucose monitor validation and baseline measurements. Results from our lab showed mean absolute relative difference of  $13.1 \pm 8.5\%$  (unpublished) when concurrent measures from the FreeStyle Libre and capillary plasma glucose samples were directly compared in healthy adolescents which is similar to the results reported in young people living with diabetes [30].

## **2.3 Preliminary Visit**

### **2.3.1 Anthropometry**

Participants arrived at the laboratory after an overnight fast. Stature was measured using a stadiometer (Leicester height measure, Seca Ltd., Birmingham, UK) to the nearest 0.01 m. Body mass (BM) was measured and percentage body fat (%BF) estimated using bioelectrical impedance (Tanita BC-418MA, Tanita Corporation, Tokyo, Japan), while participants stood barefoot and wearing light clothes, to the nearest 0.1 kg and 0.1%, respectively. Body mass index (BMI) was calculated by dividing the body mass (kg) by the stature squared ( $m^2$ ). Consequently, weight status was determined using age and sex-specific BMI cut off points [31]. Waist circumference was taken from the central point between the 10th rib and the iliac crest using a non-flexible tape measure [32].

### **2.3.2 Oral Glucose Tolerance Test (OGTT)**

The standard OGTT was performed to evaluate the participants' glucose tolerance and insulin sensitivity. After 10 min rest, participants provided a fasting capillary blood sample then consumed a drink containing 1.75 g glucose per kg BM (maximum 75 g standard OGTT procedure) in 300 ml of water within 5 min [33]. Subsequent finger-prick capillary samples were collected at 15-, 30-, 60-, and 120-min intervals after initiating the glucose drink consumption for measuring capillary plasma glucose and insulin. Plasma glucose and insulin were used to calculate the homeostatic model assessment of insulin resistance (HOMA-IR) [34]. Age-, gender-, and BMI-specific percentiles of HOMA-IR were used to identify 'at risk' participants [35].

### **2.3.3 Blood Sampling and Analyses**

To enhance the blood flow to the hand, we asked the participants to immerse their whole hand into a hot water (40°C) container for 5 min, after which the hand was dried immediately, and a finger was cleaned with an alcohol swab then pricked with a lancet (Unistick 3 Extra, Owen Mumford, UK). The first drop of blood was wiped, and 300 to 600  $\mu$ L of blood was drawn into microvette tubes (Sarstedt Ltd., Leicester, UK). The sample tubes were placed immediately into a centrifuge at 12,800 g for 15 min (Eppendorf 5415c, Hamburg, Germany) to allow collection and storage of the resulting plasma at -80°C for subsequent batch analysis. Plasma glucose was analysed using a benchtop analyser (Pentra 400; HORIBA ABX Diagnostics, Montpellier, France) using enzymatic, colourimetric methods (HORIBA ABX Diagnostics). Plasma samples were analysed to determine [insulin] using an enzyme-linked immunosorbent assay (Mercodia AB, Uppsala, Sweden). The intra-assay coefficient of variation (CV) for the duplicate samples was 0.7% for blood glucose and 4.4% for plasma [insulin].

### **2.3.4 Peak Oxygen Uptake ( $\dot{V}O_2$ Peak) Determination**

Participants completed a maximal exercise test on the treadmill (Mercury Medical; h/p/cosmos sports & medical GmbH, Germany) to volitional exhaustion. The participants ran at an individual fixed speed (inter-participant range 7 to 10  $km \cdot h^{-1}$ ) and the treadmill belt gradient was raised by 1% every minute until volitional exhaustion was attained. Heart rate was monitored continuously using a chest monitor (RS800CX, Polar Electro, Finland), and the pictorial OMNI (0 to 10) rating of

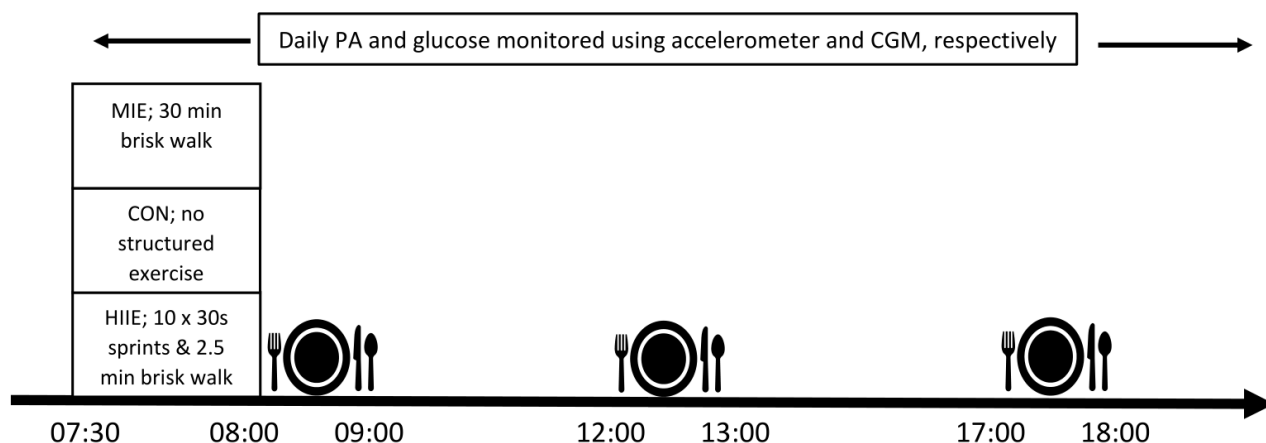
perceived exertion (RPE) scale was determined in the last 10 s of each stage of the maximal test [36]. Expired air samples were collected during at least the last three stages of the maximal exercise test using standard Douglas bag methods for  $\dot{V}O_2$  peak determination.

### 2.3.5 Physical Maturation

Pubertal status was estimated privately using a validated five-point self-assessment of secondary sexual characteristics (i.e., pubic hair growth and breast or genital developments) with the help of a parent/guardian at home [37, 38].

## **2.4 Experimental Conditions**

One week after monitor deployment, participants performed three experimental conditions on three consecutive days in a fixed pre-determined order 1) moderate-intensity exercise condition (MIE); 2) no-exercise control condition (CON); 3) high-intensity intermittent exercise condition (HIIE) and consumed three standardised meals at fixed times. Before participants' arrival, a 10.2 × 13.5 m oval track was marked out with cones spaced at 1.5 m intervals and a safe distance was maintained from the sports' hall walls. Participants arrived in the morning in a group of four, after an overnight fast, at the school sports hall to start the exercise session at 07:30 with 30 min continuous brisk walking (MIE) or 10 × 30 s sprints interspersed with 2.5 min brisk walking bouts (HIIE – total of 5 min sprint and 25 min brisk walking). The two exercise sessions were purposely matched only in duration, not estimated energy expenditure, because time is frequently cited as a barrier to exercise. A breakfast meal was provided after MIE and HIIE at 08:00 (details below section 2.4.1). During CON, participants arrived fasted at the school to consume their breakfast at 08:00 (i.e., there was no structured exercise, nor were they asked to remain seated for 30 min). A schematic representation of the study design is provided in Figure 1. Heart rate (HR) was monitored using a chest monitor (RS800CX, Polar Electro, Finland), and the pictorial OMNI (0 to 10) rating of perceived exertion (RPE) scale was monitored every 10 min during MIE and after each sprint during HIIE. Participants were encouraged to walk briskly (at a pace they could maintain a conversation with someone else) during MIE and repeatedly encouraged to run as fast as possible during HIIE to create a clear difference between the exercise conditions. Lap counts were recorded to calculate the distance covered and individual participant average speed. Participants provided their feelings after MIE and HIIE on an affective feeling scale ranging from -5 (very bad) to +5 (very good) [39] and were asked to avoid structured exercise (e.g., physical education lessons or sports participation) during the remainder of each condition school day. Further, they were asked to refrain from engaging in any moderate-vigorous intensity PA (MVPA) 24 h preceding the first experimental condition (referred to as rest day) and complete weighed food diaries using a food weighing scale (Andrew James UK Ltd., Bowburn, UK). We provided the participants with a cereal bar to consume at 19:20 the evening before each condition, after which they were asked to drink only water to ensure they had fasted for a standardised 12 h when they arrived at the school the following day. The menstrual cycle phase was not controlled in the girls due to logistical reasons related to study design, group-level data collection and the free-living nature of the study, meaning that data collection sessions could not be arranged on an individual basis.



**Figure 1** Schematic of the study design.

#### 2.4.1 Dietary Control During the Experimental Conditions

Participants were provided with three standardised meals (breakfast, lunch, and dinner). They were asked to select from two food/drink options to complement their breakfast and lunch meals (additional material; Table S1). Illustrative pictures of three different meal sizes for breakfast and lunch were presented to participants to help them choose the meal size they perceived would satisfy their hunger (additional material; Table S2). The chosen meals were provided on each of the three conditions. Thus, the energy content and macronutrients distribution were identical within participants across the experimental conditions. The total energy content of the provided meals ranged from 1374 to 2211 kcal (5.75 to 9.25 MJ) per day, and the proportion of macronutrients distributions ranged from 51 to 57%, 24 to 33%, and 14 to 16% of total energy for carbohydrate, fat and protein contents, respectively, depending on the selected portion size. The provided meals complied with the 2015-2020 Dietary Guidelines Recommendations [40]. The food items provided were typical foods consumed by UK adolescents as recent studies showed that packaged pre-prepared meals (e.g., pasta) and packaged bread (e.g., brioche) accounted for 13% and 12% of total energy intake, respectively [41]. Additionally, crisps and chocolate are the most commonly eaten foods among UK adolescents [42].

Participants were asked to consume their meals within 15 to 20 min between fixed times; breakfast (08:00-09:00), lunch (12:00-13:00) and dinner (17:00-18:00). Participants could drink plain water or sugar-free drinks with and between meals. Breakfast was the only meal assessed directly for compliance as researchers monitored the participants eating this meal. For lunch and dinner, participants were asked to record the amount and the time when they started eating their meals in the first condition in a provided food diary and then were asked to replicate and record the amount and meal timing for the subsequent conditions. Breakfast and lunch meals were consumed at the school while the dinner meal (ready meal) was provided in a container and stored in a mini-fridge at the school's reception for the participants to collect by the end of the school day and consume at home.

#### 2.4.2 Processing Glucose Monitor Data

Glucose data were downloaded from the reader using FreeStyle Libre computer software (Abbott Laboratories, Illinois, USA) and exported into Excel files. Before the onset of exercise sessions during

MIE and HIIE, and before breakfast consumption in the CON condition, glucose were defined as fasting. Meal consumption times recorded in the participant's food diary for lunch and dinner were used to identify pre-lunch and pre-dinner glucose. Postprandial glucose were determined as the closest in time glucose readings recorded at 15 min intervals for 2 h following the start of each meal during the experimental conditions (i.e., a total of 9 meals). Fasting (for breakfast) or pre-meal glucose and 2 h postprandial glucose were used to calculate total area (tAUC) and incremental (iAUC) areas under the curve using a time series response analyser (TSRA) [41]. The tAUC and iAUC responses were divided by 120 min to present the values in  $\text{mmol}\cdot\text{L}^{-1}$ . Additionally, the twenty-four-hour glucose data starting at the onset of exercise (07:30) for each experimental condition were examined for completeness (i.e., 96 glucose data points). A day was considered valid for estimating glycaemic variability and was included in the analyses if it contained  $\geq 75\%$  of daily glucose data points (i.e., equivalent to at least 18 h glucose data per day) [43]. Glucose variability indices, including mean glucose, the standard deviation of glucose (StDevG) and mean amplitude of glycaemic excursion (MAGE) were calculated for each experimental condition using EasyGV© Version 9.0 (University of Oxford, Oxford, UK) [44].

#### 2.4.3 Processing Accelerometer Data

ActiGraph raw data were downloaded into 15 s epoch files using ActiLife Software (version 6.10.1, ActiGraph, Pensacola, USA). Then, regenerated data were reduced by removing non-wear time, defined as 60 min of consecutive zeros as recommended [45] using KineSoft Software (version 3.3.80, Loughborough, UK) to generate outcome variables. Daily time spent in sedentary behaviour (SB) and in light (LPA), moderate (MPA), vigorous physical activity (VPA) and combined moderate to vigorous physical activity (MVPA) were determined using Evenson activity cut points [46]. A day was considered valid if a participant wore the accelerometer for at least 8 h [47].

### **2.5 Sample Size Calculation and the Impact of COVID19**

Sample size calculation was based on the power for interaction (3 conditions  $\times$  3 meals) using G\*Power software (version 3.1.9.4). The incremental area under the curve (iAUC) for glucose was the primary outcome. It was estimated that 35 participants were required to detect an effect size of  $d = 0.64$  based on data from a previous study in healthy adolescent boys [12], with 80% power and an alpha level of  $P = 0.05$ . The recruitment target was 42 participants to account for an expected 20% attrition rate. There were up to 45 interested volunteers who were eligible for participation. Yet, we managed to recruit 20 participants before the UK national lockdown in late March 2020 due to the COVID19 pandemic. The possibility of continuing the study was very low due to COVID19 restrictions, and the school could not continue their support of the study for almost two years (2020 and 2021). Therefore, the study was stopped, and the analyses were conducted on the completed data ( $n = 14$ ).

### **2.6 Statistical Analyses**

Differences in postprandial glycaemic outcomes and dietary intakes were examined using linear mixed models, including condition and meal as fixed effects and their interaction. Post-hoc pairwise comparisons between conditions were examined at each meal with Bonferroni correction. Linear



mixed models repeated for condition were used to examine the differences in fasting/pre-meal glucose, 24 h glycaemic variability indices, physical activity and sedentary time (adjusted for wear time), and responses to exercise conditions. Spearman's correlations were performed between post-exercise OMNI RPE and affective scores. The data residuals were checked for normality. Data for dietary intakes and pre-dinner glucose were not normally distributed even after being naturally log-transformed. Therefore, results from raw data are presented for all outcomes. Values are expressed as mean  $\pm$  SD, unless stated otherwise. Statistical analyses were completed using SPSS (version 25.0; SPSS Inc., Chicago, IL). Statistical significance was accepted at  $P < 0.05$ . The importance of reporting effect sizes and providing an interpretation of what these effect sizes mean in terms of practical utility was highlighted recently [48]. In light of this recommendation, effect sizes are also stated to aid with the interpretation of the results of this study. Cohen's effect size ( $d$ ) was calculated to describe the magnitude of difference according to the following thresholds: trivial ( $<0.2$ ), small ( $\geq 0.2$ ), moderate ( $\geq 0.5$ ) and large ( $\geq 0.8$ ) [49].

### 3. Results

Out of twenty participants, ten completed all three experimental conditions, and four completed only one exercise condition (MIE:  $n = 2$  and HIIE:  $n = 2$ ) and CON; all of these participants (6 girls) were included in the final sample. The remaining six participants were excluded from the analyses due to sickness ( $n = 1$ ), completing only one condition ( $n = 1$ ) and discontinuation of data collection due to the COVID-19 lockdown ( $n = 4$ ). Participant characteristics are presented in Table 1. All participants had normal weight status except for two classified as thin (grade 1) according to age and sex-specific BMI cut off points [31]. Two participants were considered overfat according to age and sex-specific body fat reference curves [50] but normal based on age and sex-specific BMI and waist circumference cut off points [31, 32]. One participant was classified as 'at risk' according to age-, gender-, and BMI-specific percentiles of HOMA-IR [35]. Another participant had impaired fasting plasma glucose according to the American Diabetes Association (ADA) criteria (5.6 to 6.9  $\text{mmol}\cdot\text{L}^{-1}$ ). None of the participants had an impaired glucose tolerance, defined as glucose greater than 7.8  $\text{mmol}\cdot\text{L}^{-1}$  at 2 hours after OGTT.

**Table 1** Participant characteristics ( $n = 14$ ).

Variables	Mean $\pm$ SD	Range
Age (y)	12.8 $\pm$ 1.0	11.5-14.4
Stature (m)	1.57 $\pm$ 0.1	1.35-1.75
Body mass (kg)	44.4 $\pm$ 6.9	30.3-56.9
BMI ( $\text{kg}\cdot\text{m}^{-2}$ )	18.0 $\pm$ 1.6	15.4-19.9
Body fat (%)	21.6 $\pm$ 5.8	12.7-31.5
Lean Body mass (kg)	34.7 $\pm$ 5.2	25.1-46.5
Waist circumference (cm)	60.5 $\pm$ 10.9	46.0-87.0
Breast development*	4 (1)	1-4
Genital development*	2 (0)	1-3
Pubic hair development*	3 (2)	1-5
Fasting plasma glucose ( $\text{mmol}\cdot\text{L}^{-1}$ )	4.94 $\pm$ 0.35	4.29-5.76

Fasting plasma insulin (mU·L <sup>-1</sup> )	6.36 ± 3.75	2.07-15.56
HOMA-IR	1.42 ± 0.88	0.44-3.58
$\dot{V}O_2$ peak (mL·kg <sup>-1</sup> ·min <sup>-1</sup> )	39.0 ± 11.0	23.0-58.8

BMI, body mass index;  $\dot{V}O_2$ , oxygen uptake; HOMA-IR, homeostatic model assessment of insulin resistance. CON, control; MIE, moderate-intensity exercise; HIIE, high-intensity intermittent exercise. \*median (interquartile range).

### 3.1 Dietary Intake During the Experimental Conditions

Energy intake for each meal was similar between the conditions (main effect of condition:  $P = 0.683$ ; condition  $\times$  meal interaction:  $P = 0.731$ ). Similar results were found for macronutrient intakes between conditions (main effect of condition:  $P \geq 0.655$ ; condition  $\times$  meal interaction:  $P \geq 0.663$ ). Energy and macronutrient intakes of each meal during the experimental conditions are presented in (additional material; Table S3).

### 3.2 Physiological and Affective Responses to MIE and HIIE

Mean HR was higher during HIIE compared with MIE ( $182 \pm 8$  vs  $132 \pm 15$  b·min<sup>-1</sup>, 95% CI 34 to 65,  $P < 0.001$ ); confirming the achievement of different exercise intensities between the two conditions. Over the 30 min exercise period, participants moved further during HIIE ( $3.0 \pm 0.3$  vs  $2.3 \pm 0.3$  km, 95% CI 0.5 to 1.0,  $P < 0.001$ ) and at a higher average speed ( $8.9 \pm 0.9$  vs  $4.5 \pm 0.7$  km·h<sup>-1</sup>, 95% CI 3.8 to 5.0,  $P < 0.001$ ) compared with MIE (this speed includes the brisk walking recovery periods; mean sprint speed was  $12.9 \pm 1.3$  km·h<sup>-1</sup>). The OMNI RPE scale mean value was higher during HIIE compared with MIE ( $5 \pm 1$  vs  $2 \pm 1$ , 95% CI 1 to 4,  $P = 0.001$ ). Affective perceptions of the exercise conditions were positive but tended to be higher for MIE (HIIE  $2 \pm 3$  vs MIE  $3 \pm 2$ , 95% CI 0 to 2,  $P = 0.057$ ). Large, negative correlations were found between OMNI RPE post exercise and perceived affective score during HIIE ( $\rho = -0.836$ ,  $P = 0.001$ ) and MIE ( $\rho = -0.807$ ,  $P = 0.002$ ).

### 3.3 Accelerometer Data

Table 2 presents the daily time spent sedentary and in free-living PA during the three experimental conditions and the rest day. The two 30 min exercise interventions resulted in 29 min of accelerometry MVPA during MIE and HIIE compared with 6 min of MVPA during free-living CON. Despite the physiological differences induced by MIE and HIIE, the activity intensities according to the Evenson cut points resulted in non-significant but large differences in time spent in moderate (10 vs 15 min,  $P = 0.117$ ,  $d = 2.35$ ) and vigorous (19 vs 13 min,  $P = 0.203$ ,  $d = 1.21$ ) intensity activities between MIE and HIIE, respectively. Yet, the combined MVPA were not significantly different with a trivial effect size ( $d = 0.06$ ). The amount of time spent sedentary, in light PA, and MVPA after the 30 min intervention periods (i.e., from 08:00 onwards) were similar across conditions ( $P \geq 0.169$ ). Thus, this confirms that most of the daily mean difference in MVPA between MIE and CON (i.e., 33 min), and HIIE and CON (i.e., 29 min) derived from the 29 min of captured activity during MIE and HIIE.

**Table 2** Daily time spent sedentary and in free-living physical activity (PA) during the rest day and the three experimental condition days.

	Rest day	MIE	CON	HIIE
<b>Participant with valid wear day (n)<sup>+</sup></b>	12	12	13	12
<b>Wear time (min·day<sup>-1</sup>)</b>	671 ± 106	786 ± 126	806 ± 126	849 ± 166
<b>Sedentary (min·day<sup>-1</sup>)</b>	486 ± 105	547 ± 118 <sup>§</sup>	604 ± 125	562 ± 165 <sup>*</sup>
<b>Light PA (min·day<sup>-1</sup>)</b>	149 ± 63	191 ± 42	166 ± 40	179 ± 50
<b>Moderate PA (min·day<sup>-1</sup>)</b>	25 ± 21	46 ± 16 <sup>§</sup>	31 ± 12	48 ± 16 <sup>§</sup>
<b>Vigorous PA (min·day<sup>-1</sup>)</b>	11 ± 16	32 ± 15 <sup>§</sup>	14 ± 8	26 ± 14 <sup>§</sup>
<b>MVPA (min·day<sup>-1</sup>)</b>	35 ± 35	78 ± 18 <sup>§</sup>	45 ± 16	74 ± 19 <sup>§</sup>

Data were adjusted for wear time in statistical analyses.

<sup>+</sup> PA data were excluded for two participants during the rest day and one participant during CON because they were not valid (i.e., <8 h per day). In addition, PA data were excluded for four participants (MIE: n = 2 and HIIE: n = 2) because they did not complete the exercise conditions.

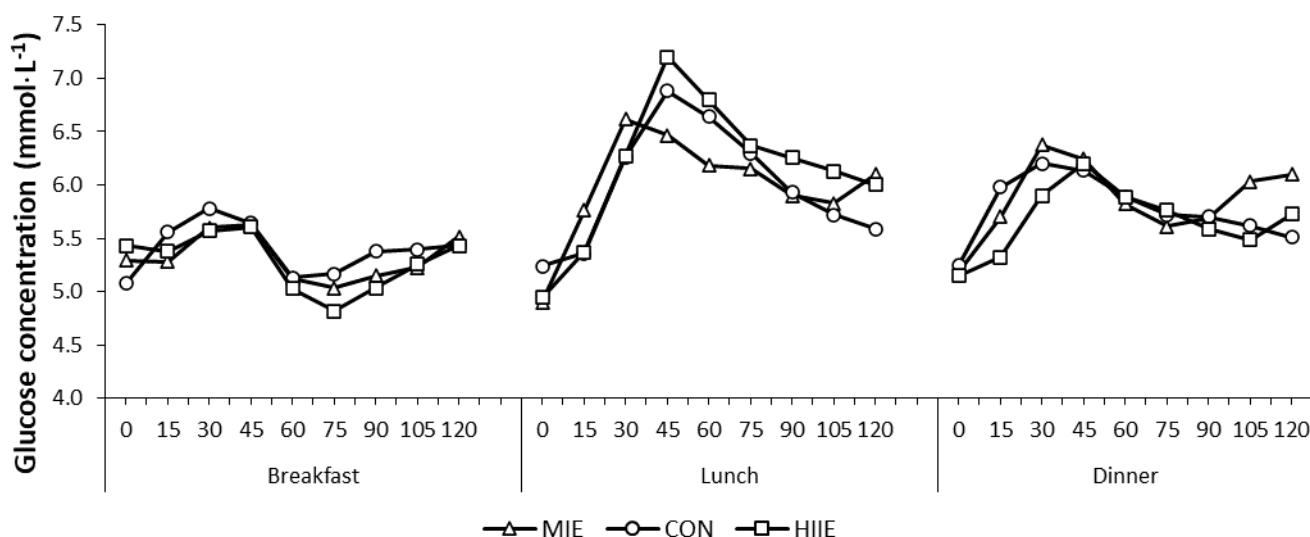
<sup>§</sup> Significant compared with CON (P ≤ 0.040).

<sup>\*</sup> Trend compared with CON (P = 0.064).

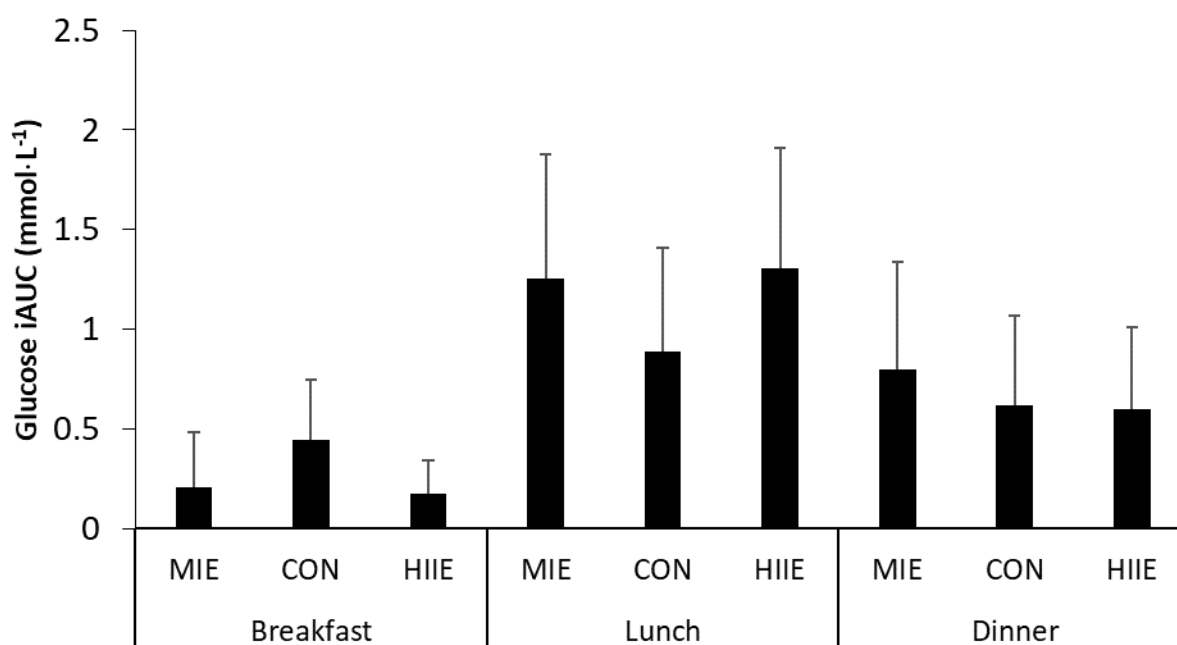
### 3.4 Postprandial Glucose Response to Standardised Meals

Fasting glucose was higher before starting HIIE compared with CON (0.40 mmol·L<sup>-1</sup>, 95% CI 0.04 to 0.76, P = 0.024, d = 0.77), but there were no significant differences between MIE and HIIE (P = 0.679) or between MIE and CON (P = 0.375). Pre-meal glucose were not different between conditions for lunch (P = 0.170) or dinner (P = 0.854).

Changes in glucose during postprandial periods are presented in Figure 2 and postprandial glucose iAUC after each meal across conditions are presented in Figure 3. Postprandial glucose iAUC were not significantly different between conditions (main effect of condition: P = 0.664) and the condition by meal interaction did not reach significance (P = 0.111). In terms of the magnitude of the differences, the reduction in post-breakfast glucose iAUC was moderate for MIE (d = 0.77; -0.24 mmol·L<sup>-1</sup>; 95% CI -0.68 to 0.21, P = 0.589) and large for HIIE (d = 0.86; -0.26 mmol·L<sup>-1</sup>; 95%CI -0.71 to 0.18, P = 0.444) compared with CON, whilst the effect size for the difference between HIIE and MIE was trivial (d = 0.09; -0.03 mmol·L<sup>-1</sup>; 95%CI -0.49 to 0.43, P = 1.000). Post-lunch glucose iAUC, however, increased moderately in MIE (d = 0.70; 0.37 mmol·L<sup>-1</sup>; 95%CI -0.13 to 0.86, P = 0.219) and the change was large in HIIE compared with CON (d = 0.81; 0.42 mmol·L<sup>-1</sup>; 95%CI -0.13 to 0.98, P = 0.203). There was a trivial post-lunch effect size for the difference between HIIE and MIE (d = 0.11; 0.06 mmol·L<sup>-1</sup>; 95%CI -0.49 to 0.60, P = 1.000). The differences in post-dinner glucose iAUC were trivial between HIIE and CON (d = 0.03) and small between MIE and CON (d = 0.41) and HIIE and MIE (d = 0.44).



**Figure 2** Mean changes in postprandial glucose concentration for the control (CON, open circle), moderate- (MIE, open triangle) and high- (HIIE, open square) intensity intermittent exercise conditions. Times for meal consumption were determined as pre-exercise glucose value for breakfast and pre-meal glucose value for lunch and dinner (indicated at 0 min in the figure). Error bars are omitted for increased clarity.



**Figure 3** Postprandial glucose iAUC after each meal across conditions. CON, control; MIE, moderate-intensity exercise; HIIE, high-intensity intermittent exercise; iAUC, incremental area under the curve.

The analyses of postprandial glucose tAUC revealed a non-significant main effect for condition ( $P = 0.967$ ) and non-significant condition by meal interaction ( $P = 0.279$ ). It is worth noting that the effect sizes for the difference in tAUC between conditions for each meal were smaller than that reported for iAUC.

### 3.5 24 h Glycaemic Variability Indices

Table 3 summarises the 24 h glycaemic variability indices across the three experimental conditions. There were no significant main effects of condition in any of the glycaemic variability indices, including mean glucose ( $P = 0.281$ ), StDevG ( $P = 0.585$ ) and MAGE ( $P = 0.822$ ) calculated over 24 h after the onset of exercise for each experimental condition. The effect sizes ranged from trivial to small between exercise conditions and CON ( $d = 0.12$  to  $0.42$ ).

**Table 3** 24 h glycaemic variability indices during experimental conditions.

	MIE	CON	HIIE
Participant with valid glucose day (n)	11	13	11
Glucose datapoints (n)	88 ± 7	89 ± 7	87 ± 7
Glucose monitoring period (h)	22.0 ± 1.6	22.2 ± 1.7	21.6 ± 1.9
Mean glucose (mmol·L <sup>-1</sup> )	5.31 ± 0.36	5.37 ± 0.33	5.41 ± 0.39
StDevG (mmol·L <sup>-1</sup> )	0.68 ± 0.19	0.63 ± 0.12	0.68 ± 0.18
MAGE (mmol·L <sup>-1</sup> )	1.98 ± 0.78	1.84 ± 0.52	1.95 ± 0.57

StDevG, the standard deviation of glucose; MAGE, mean amplitude of glycaemic excursion. One participant was removed due to insufficient glucose data per day (less than 18 h) for all conditions. No significant differences between conditions ( $P \geq 0.281$ ).

## 4. Discussion

This study is the first, to our knowledge, to examine whether prior (fasted) exercise has favourable effects on postprandial glycaemic responses to three standardised meals, 24 h mean post-exercise glucose and glycaemic variability using CGM under free-living conditions in healthy adolescents. Although there were no significant changes in postprandial glycaemia across the three meals, some potentially interesting pairwise effects were found. The glucose iAUC was lower in both MIE and HIIE conditions than CON at breakfast, but higher at lunch with moderate to large effect sizes. Whether the magnitude of acute changes could have longer-term health consequences if repeated regularly at school is not known. Nevertheless, the 24 h mean glucose was stable around 5.4 mmol·L<sup>-1</sup> across conditions with no meaningful changes in 24 h glycaemic variability measured by StDevG and MAGE detected following MIE and HIIE compared with CON. The findings show that the moderate to large between condition effects in the postprandial periods were not evident when responses were examined over an extended 24 h period in healthy adolescents with well-controlled glycaemic regulation.

Moderating postprandial glycaemic excursions has been found to attenuate oxidative stress, inflammation, and endothelial dysfunction and, therefore has the potential to minimise CVD risk [2, 51]. Post-breakfast tAUC was not significantly different across conditions with small effect sizes. Yet, tAUC does not account for baseline (fasting) concentration, which is particularly important in the current study because fasting glucose was higher in HIIE than CON; the underlining cause of which is unknown as participants reported a fasting state upon school arrival. However, we could

speculate that day-to-day glycaemic variability and/or stress induced increase in glucose in anticipation of high intensity PA might have resulted in high fasting glucose. To account for baseline differences in fasting glucose, post-breakfast iAUC (rather than tAUC) was calculated [52] and showed non-significant but moderate to large reductions after MIE ( $-0.24 \text{ mmol}\cdot\text{L}^{-1}$ ,  $d = 0.77$ ) and HIIE ( $-0.26 \text{ mmol}\cdot\text{L}^{-1}$ ,  $d = 0.86$ ) compared with CON, respectively. Apart from the p-value, which is most likely a result of small sample size, our study showed similar results in terms of the direction and size of the effect to that of Cockcroft and colleagues' study [12]. Cockcroft and colleagues found reductions of  $\sim 0.50$  to  $0.58 \text{ mmol}\cdot\text{L}^{-1}$  (estimated from the figures and converted to  $\text{mmol}\cdot\text{L}^{-1}$ ) in glucose iAUC in response to OGTT (1.75 g CHO per kg of body mass) consumed 10 min after HIIE ( $8 \times 1$  min cycling at 90% of peak power) and work-matched MIE (continuous cycling at 90% of the gas exchange threshold) compared with a rested condition in healthy adolescent boys using capillary blood samples in a laboratory setting (reporting similar effect sizes to ours  $d = 0.64$  to  $0.84$ ,  $P \leq 0.013$ ) [12]. The present study and that of Cockcroft et al. [12] are similar in that 1) acute exercise bouts were performed in a fasted state, 2) the test drink/meal was consumed in close proximity to exercise (immediately vs 10 min after exercise), and 3) mean heart rate (MIE:  $132$  vs  $136 \text{ beats min}^{-1}$  and HIIE:  $182$  vs  $183 \text{ beats min}^{-1}$ ) and exercise duration (MIE:  $29$  vs  $29$  min and HIIE:  $29$  vs  $23$  min including active recovery) were almost the same despite different exercise mode (walking/running vs cycling). Differences in the reduction in absolute glucose iAUC ( $\text{mmol}\cdot\text{L}^{-1}$ ) between the two studies could be due to differences in test meal composition, study setting (free-living vs lab) and methods of glucose measure. Meier and colleagues (2009) found that glycaemic excursions during an OGTT (360 kcal) exceeded those after a large (820 kcal) mixed-nutrient meal test and regular meals consumed under free-living conditions by 20 and 30% in adults with various glycaemic tolerance, respectively [19]. Therefore, the scope for improvement may have been greater in the study by Cockcroft et al. [12] than the present study due to the test drink they used in the OGTT protocol. It is worth noting that the meals provided in the present study were more representative of typical meals than OGTT and were consumed under free-living conditions as opposed to a controlled laboratory setting, thus enhancing ecological validity of the findings from the present study in comparison to previous research. The results from the present study support the potential acute effect of 30 min exercise bouts of different intensities before breakfast on postprandial glycaemia. Whether pre-school PA such as activity programmes or active travel would be an appealing approach to increase adolescents' PA and promote metabolic health warrants investigation as most research is focused on after-school period. Nevertheless, inconclusive promising associations of pre-school activity programmes with health including daily PA, cardiorespiratory and muscular fitness were found in a recent systematic review [53].

Some studies in healthy adolescents have found attenuated glucose and insulin concentrations in response to OGTT and high-fat test drinks up to 17 to 24 h post exercise [13, 54]. Based on the findings of these studies, it was expected that a single exercise bout would lower blood glucose concentrations after meals consumed later in the same day. Postprandial glycaemic responses did not differ significantly between the conditions for lunch and dinner. The effect sizes for the differences were trivial to small except for post-lunch iAUC glucose (the second meal after exercise), where a non-statistically significant increase ( $0.37$  to  $0.42 \text{ mmol}\cdot\text{L}^{-1}$ ;  $P \geq 0.200$ ) with moderate to large effects ( $d = 0.70$  to  $0.81$ ) was found after MIE and HIIE compared with CON, respectively. The underlying cause for the higher post-lunch glucose concentrations in MIE and HIIE in the present study is not clear. The studies are sparse and inconsistent regarding the residual effect of pre-

breakfast (fasted) exercise on the glycaemic response to subsequent meals. From the literature with adult participants, one laboratory-based study reported no change in postprandial tAUC glucose to lunch consumed ~4 h after 60 min of pre-breakfast brisk walking compared with a no-exercise control in overweight men [55]. Yet, in another free-living study using CGM, the same duration of aerobic exercises (on a treadmill and stationary cycle) lowered postprandial glycaemic excursions for all the meals consumed over two days with a significant reduction of ~14% following lunch (consumed ~5 h after exercise) in adults living with T2D [22]. Perhaps, a longer exercise duration may be required to impose an extended effect on postprandial glycaemia. In addition, the effect of exercise may depend on the participants' glycaemic control. In the present study, the moderate to large between condition effects on post-breakfast glucose did not persist to lower postprandial glucose of subsequent meals following the morning exercise in healthy adolescents.

Glycaemic variability has been identified as an independent risk factor for CVD [5, 56]. We found no meaningful changes with trivial to small effect sizes in glycaemic variability indices calculated by StDevG and MAGE after a single exercise bout. These findings are consistent with previous studies in adults without diabetes [23, 24, 28]. However, in people with diabetes, the results are mixed concerning glycaemic variability reduction after an acute bout of exercise; individuals who have poor glycaemic control (high HbA1c) and high glycaemic variability at the baseline demonstrate greater glycaemic variability reductions after an acute bout of exercise than their counterparts who have better glycaemic control [27]. Thus, an acute bout of at least 30 min of exercise may not be sufficient to reduce glycaemic variability in young, healthy adolescents with no apparent glycaemic dysregulation (i.e., glucose fluctuating within a normal narrow range). It is also worth noting that although it is expected that frequent exercise sessions would improve glycaemic variability, the response seems variable in a highly active population (i.e., athletes). For example, 4 out of 10 trained athletes spent more than 70% of a six-day monitoring period above 6.0 mmol·L<sup>-1</sup> even when the 2-hour postprandial periods were removed [57]. Clearly, there is a need for longer duration studies looking at the effects of exercise on glucose concentration in healthy adolescents (i.e., regular exercise over 14 days using CGM) as there is not enough research data to characterise “normal” glucose responses yet.

The reported mean glucose (5.4 mmol·L<sup>-1</sup>) in the present study was comparable with other studies reporting mean sensor glucose over at least three days of CGM in healthy non-diabetic adolescents (5.3 to 5.7 mmol·L<sup>-1</sup>) [58, 59]. However, glycaemic variability indices including StDevG (0.66 mmol·L<sup>-1</sup> vs 0.89 to 0.91 mmol·L<sup>-1</sup>) was lower, and MAGE (1.92 mmol·L<sup>-1</sup> vs 1.56 mmol·L<sup>-1</sup>) was higher than that reported in healthy adolescents [58, 59]. These discrepancies in glycaemic variability indices may be related to sample size (13 vs 20 and 30), numbers of monitoring days (3 vs 3 to 7 d) or the use of different CGM system (FreeStyle Libre vs Medtronic MiniMed and FreeStyle Navigator or Dexcom G6). In addition, dietary intake was not reported in previous studies, which can contribute to the differences in MAGE as it is influenced by meal-related glycaemic responses in healthy people [60].

The use of CGM allowed us to study glycaemic responses under free-living conditions that represent real-life better than constrained laboratory settings. Our study was limited by a small sample size, which restricted our ability to detect significant findings. The reported non-significant p-value with moderate to large effect sizes in our study reflects the low power of the study but with potentially meaningful responses to exercise. Future studies with a larger sample size are required to examine the acute exercise effect on postprandial glycaemia under free-living conditions. While

the research team supervised breakfast food consumption, lunch and dinner meals were self-reported in the provided food diary. Thus, the timings of lunch and dinner were subjected to inaccurate reporting. Postprandial glucose data were also missing due to infrequent scanning of the sensor ( $n = 6$ ). Therefore, we used a linear mixed model, which has been suggested to have a superior statistical power than repeated-measure ANOVA to handle incomplete and unbalanced data and accounts for between-subject and within-subject variability [61]. Nevertheless, removing participants with incomplete data set did not change the overall results. The study conditions were designed in a fixed, pre-determined order for physiological and logistic reasons. Evidence from healthy adolescents suggests that the acute effect of exercise bout on glycaemia lasted for 24 h [13, 14]. Therefore, the study conditions were designed in a specific order in which HIIE was performed the last as it may have a higher/or a longer-lasting effect. Randomisation and/or balanced order effects may affect the study feasibility considering the constraints of working in a school or under free-living conditions as it may become much more demanding in terms of the participants' time and using the space in the school. In addition, due to logistical reasons related to study design and the free-living nature of the study, which is a novel component of this research, it was not possible to control for the menstrual cycle phase for each individual participant and other markers of glucose control, such as insulin, were not measured to provide a broader insight into the entire glycaemic response.

The energy intake was based on participant's selection of their portion size. While this method represents real-life behaviour, some participants might have consumed more or less energy than their total energy requirements. However, each participant consumed the same energy and macronutrients intake across the three conditions (i.e., meals were standardised within participants).

## **5. Conclusions**

In conclusion, thirty-minute bouts of MIE and HIIE did not change postprandial glycaemia or 24-h glycaemic variability significantly in the small sample of healthy adolescents. However, the moderate and large effect sizes suggest both MIE and HIIE reduced breakfast glucose iAUC compared with CON, yet led to increases in glucose iAUC in the two exercise conditions following the lunch meal. The mismatch between the probability values and effect sizes was a consequence of our COVID-reduced sample. The ramifications of these possible exercise effects are not clear and need to be confirmed in a larger sample of adolescents.

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## **Author Contributions**

Conceptualisation, S.M.A., L.A.B. and K.T.; methodology, S.M.A., L.A.B. and K.T.; investigation, S.M.A.; data curation, S.M.A.; formal analysis, S.M.A. and J.Z-F.; resources, K.T.; writing—original draft preparation, S.M.A.; writing—review and editing, K.T., L.A.B. and J.Z-F.; supervision, K.T. and



L.A.B.; project administration, K.T.; funding acquisition, K.T. All authors have read and agreed to the published version of the manuscript.

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## **Competing Interests**

The authors have declared that no competing interests exist.

## **Additional Materials**

The following additional materials are uploaded at the page of this paper.

1. Table S1: Provided meals (breakfast, lunch, and dinner) during the experimental conditions.
2. Table S2: The three meal sizes for provided breakfast and lunch during the experimental.
3. Table S3: Energy and macronutrient intakes of each meal during experimental conditions.

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